Review

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Ecological influences on human behavioural diversity: a review of recent findings

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Human societies are remarkably variable in terms of their size, complexity, social structure, marriage systems and norms. This diversity has sometimes been raised as an obstacle to taking an evolutionary approach to human behaviour. However, a substantial proportion of the variation between human societies might represent local adaptation to ecological conditions and would thus be very much amenable to evolutionary explanation. I review recent studies correlating inter-population differences in humans with ecological factors, specifically pathogen prevalence. Many questions remain unanswered, such as whether we correctly understand the causal pathways and what the mechanisms producing local adaptation are, but the strength of correlations between social and ecological parameters is striking.

Understanding human diversity

A central challenge facing anyone who studies the behaviour of humans is that it is not the same everywhere. Although there are human universals [1], these are instantiated in each society in strikingly different ways. Human societies vary from small and egalitarian to large and stratified; from polygynous to polyandrous; from strict life-long pair bonding to much more fluid mating arrangements; and from son-biased to daughter-biased in their transmission of resources. Moreover, anyone who has experience of travel will agree that norms of expression and behaviour vary in much less tangible ways too.

The diversity of human behaviour has sometimes been viewed as a problem for biological approaches to studying humans. That is, the existence of inter-population behavioural differences that are unlikely to reflect genetic differences has been taken to show that biology is insufficient for understanding human behaviour and that we need to add a whole new type of explanation, usually couched in terms of culture, a force whose laws "are independent of the laws of biology" [2]. Even Richard Dawkins, a strong advocate of adaptationism, concludes his book *The Selfish Gene* with a discussion of the argument that human behaviour is driven by the dynamics of a new system of replicators – memes – that can over-ride standard adaptationist reasoning [3]. Meanwhile, those who wish to argue for adaptationist approaches to modern humans find themselves forced to downplay the extent of human behavioural diversity, as if admitting diversity were to concede ground to non-evolutionary approaches [4].

However, there is nothing surprising, from an evolutionary perspective, about different populations of a generalist species exhibiting different behaviours. Baboons, for example, have different social structures and activity patterns in different parts of their range. This is a consequence of local adaptation to prevailing ecological conditions [5]. Given that humans occupy a much broader range of habitats than baboons, a great degree of inter-population behavioural diversity would be expected even if humans had no special derived traits. As for culture, in the sense of socially transmitted learned information, humans undoubtedly rely on this to a very considerable extent, but it is not the only mechanism that can lead to inter-population differences in behaviour. Thus, when we observe such differences, whether cultural transmission is the source is an empirical question. Moreover, even when culture is important, it is best seen as a proximate mechanism that often, but not always, leads to locally adaptive behaviour rather than as a force independent of biology [6,7]. This means that we often expect to see the same outcomes at equilibrium regardless of whether the mechanism of local adaptation is cultural transmission, individual learning, other forms of plasticity or genetic evolution.

The question as to what extent human inter-population diversity reflects adaptation to the local habitat has a long and rather inconclusive history in anthropology (Box 1). However, in the last few decades evidence has begun to emerge that the distribution of human diversity is more strongly associated with ecological factors than had been realized. All of these findings have been made possible by the use of comparative evidence. That is, instead of studying a single society, researchers have shifted to assembling geographically explicit databases encompassing many different populations and several different social and environmental variables. This has the advantage of making manifest large-scale patterns, such as the latitudinal gradient in the density of ethnolinguistic groups (Figure 1), but the limitation that it is an essentially correlational methodology.

This paper reviews these recent findings, concentrating on a number of studies that have taken pathogen diversity as their main explanatory variable. My purpose is not to

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Box 1. A brief history of cultural ecology

There is a long tradition of using ecology to explain why societies are the way they are [32], going back at least as far the anthropologist Julian Steward [33,34]. However, this cultural ecology perspective did not become central and a fair generalization remains that social scientists usually only consider the endogenous dynamics of societies: ''A culture utilizes and modifies natural resources and landscape...in a particular way....because of a particular heritage. We do something in such and such a way because this is the way our people do it'' [35].

Progress in cultural ecology has been impeded by four main issues. First, many cultural ecology studies were based on data from one society. Although the detail of such research is admirable, adaptive explanations based on a single case are always vulnerable to the charge of a just-so story. Thus, in evolutionary biology the use of comparative evidence has become a key method for testing adaptive hypotheses [36] and the work reviewed in this paper reflects an attempt to do the same for social patterns in humans.

Second, some have objected that cultural ecology reduces human social life to protein and profit, failing to recognize the multiplicity of influences on human behaviour [37]. Objections of this type represent a double confusion. They conflate the claim that there is a non-random relationship between environmental variable X and societal feature Y with the claim that environmental variables are sufficient to explain all variation in societies. In fact, the first of these is often true and the second is always false. They also conflate proximate mechanisms and ultimate causes. Humans are proximately influenced by many things, including ideologies and symbolic meanings, but this sheds no light on why certain ideologies or meanings (or the behaviours they inspire) persist in certain places and not in others [35].

Third, much of the early cultural ecology was naively group-selectionist, finding group-level advantages for behaviours such as warfare and infanticide [38,39]. This is theoretically problematic and so cultural ecology in the 1980 s reoriented itself towards individual-level explanations [32]. All of the ideas reviewed in this paper are based on individual-level advantage. Note, however, that social learning in humans generates within-group similarity and between-group variation, so where there are social processes with multiple stable evolutionary equilibria, between-group selection can act as an equilibrium selection mechanism [40].

Finally, cultural ecological research has generally been vague about the mechanisms that lead to the emergence and maintenance of locally adaptive traits. We need to understand how human beings decide on particular courses of action given particular environmental inputs, including but not limited to information from other human beings. We also need to understand how individual decisions scale up to produce emergent properties of whole societies. In other words, we need to build stronger bridges between macroscopic properties of societies and individual psychology (Box 2).

argue for or against the authors' explanations of the patterns observed. Instead, I have two claims to make. First, regardless of the causal pathways, these studies suggest that human ways of life are strongly shaped by ecology in a broad sense (meaning all non-anthropogenic aspects of the local environment). Second, although it is difficult to tease apart the different causal pathways involved, we already have the tools for many of tasks required and rapid progress in this area can be anticipated if the best methodologies are followed.

Associations between ecology and social parameters

Table 1 lists seven examples of recently documented associations between ecological variables and social parameters. The outcomes involved vary from macroscopic societal variables, such as ethnic group size, to marriage patterns, reproductive behaviour and individual psychological variables, such as personality and attitudes. All of the studies find moderate to strong associations between the ecological predictor(s) and outcome variables, even after adjustment for various controls. For full accounts of the studies, I refer the reader to the original papers. Here, I discuss some of the potential problems of interpretation. I consider three issues in turn: whether the associations are spurious; how we can discriminate between alternative causal accounts of the associations; and what other types of investigation could be carried out to strengthen the claims made about the influence of ecology on human behaviour.

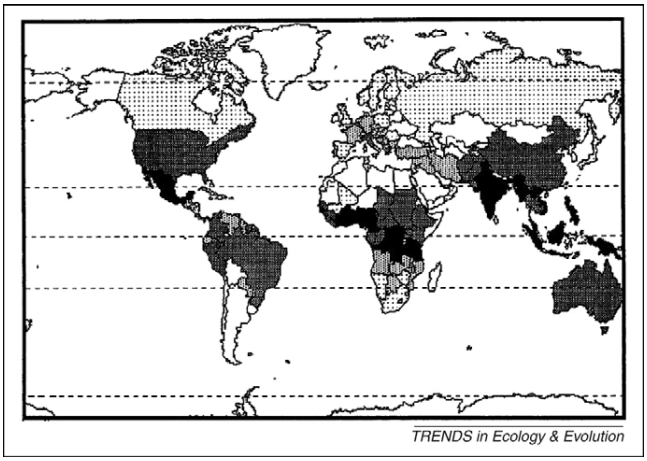

Figure 1. Relative language density of the world's countries (darker shading indicates more languages per unit area). Ethnolinguistic groups generally have smaller ranges closer to the equator, similar to the pattern observed for species in many taxa. Reproduced from Ref. [14].

The danger of spurious associations

Spurious associations can arise in correlational data if the sampling units are not statistically independent. For example, for countries of Eurasia there is a strong statistical association between the use of pharyngeal consonants in speech and the aridity of the environment. All the desert countries of North Africa and the Levant use pharyngeals, whereas none of the temperate countries of Europe do so. However, all the Levantine countries speak the Arabic language, so in fact we have only one data point that is pseudo-replicated by sampling Arabic several times. Pharyngeal consonants did arise in an arid environment, but only once, so we have no ability to tell whether this was just chance.

Non-independence of sampling units is known in anthropology as Galton's problem and a number of methods have been suggested to deal with this issue [8,9]. Non-independence can often be detected by spatial autocorrelation analyses and samples can be designed to minimize the problem, as in the standard cross-cultural sample [10]. However, this sharply decreases the potential sample size and shared historical influences that do not follow geography can go undetected. Another solution is to place societies onto a phylogeny based on classification of the languages that they speak and use phylogenetic methods developed for evolutionary biology [9,11]. This technique is useful in a number of cases but is not

Table 1. Recently documented associations between ecological factors and attributes of human societies

Social variable	Ecological factors	Pattern	Methods	Control for non-independence	Explanation(s) offered
Range size of ethnic groups [13–17,53–55] or religions [16]	Latitude, rainfall, temperature, seasonality, pathogen prevalence	Lower latitude, less seasonality or higher pathogen loads→smaller ethnic groups	*Samples:* Various approaches using ethnolinguistic atlases or by-country databases *Predictors:* Latitude as a proxy for ecology [53], climate data [13,14,17,54,55] or pathogen prevalence scores [15–17] *Controls*: Country size [14–16], economic development [15,16] and democracy [16]	None [13,14,53,55], sample structure [17] or stratification by region [15,16,54]	(a) Spatial and temporal variation in resource availability drives patterns of dispersal and social exchange [13,14] (b) High pathogen prevalence inhibits development of expansionist states and armies [17] (c) High pathogen prevalence favours ingroup assortative sociality, outgroup avoidance and limited dispersal, leading to cultural divergence [15,16]
Degree of polygyny [18]	Pathogen prevalence	Higher pathogen load→society more polygynous	*Sample:* Standard cross-cultural sample (SCCS [10] 186 ethnographic societies) with multiple indices of polygyny *Predictors:* Prevalence scores for seven key pathogen classes	Sample structure, stratification by region	Where pathogen stress is high, it becomes more important for women to seek high genetic quality in a mate, reducing the proportion of acceptable males
Fertility rate [56]	Pathogen prevalence	Higher pathogen load→higher fertility rate	*Sample:* 150 countries with total fertility rate *Predictors:* Prevalence of major pathogens from disease control data *Controls:* Country size, urbanization, economic development and life expectancy	Dummy variables representing geographic position, major ethnic groupings and religions; autocorrelation analysis	People respond to pathogen stress with increased reproductive rate
Parental care [46]	Pathogen prevalence and frequency of famine	Very high pathogen load→shorter or less parental care	*Sample:* SCCS, using age at weaning, scores for maternal and paternal care *Predictors:* Pathogen prevalence scores, famine *Controls:* Numerous, including social complexity, subsistence pattern, marriage system, latitude and modernization	Sample structure; region as a dummy variable; correction of standard errors for clustering by language family	As the rate of extrinsic mortality increases beyond a certain point, parents reduce investment in each offspring to increase the number (see fertility rate above)
Mate preferences [47]	Pathogen prevalence	Higher pathogen load→stronger preferences for health and attractiveness	*Sample:* Preference survey data from 37 countries *Predictors:* Pathogen prevalence scores *Controls:* UN indices of gender equality, level of economic development, latitude	Region as a dummy variable	Where pathogen prevalence is high, people increase their preference for health and physical attractiveness in a mate as good immunity becomes more important
Sexual restrictiveness (among other personality variables) [57]	Pathogen prevalence	Higher pathogen load→more restricted sexual behaviour	*Sample:* Self-report measure from samples in 48 countries *Predictors:* Estimates of the historical prevalence of nine major disease classes *Controls:* Life expectancy, economic development, latitude, temperature	None	Where disease risk is high, caution in sexual encounters is selected for
Individualistic versus collectivistic orientation [58]	Pathogen prevalence	Higher pathogen load→greater collectivism	*Sample:* 98 countries, various mainly self-report measures of individualism and collectivism *Predictors:* Estimates of the historical prevalence of nine major disease classes, contemporary disease control data *Controls:* Population density, economic development, economic inequality, life expectancy	Grouping into larger regions likely to be historically independent	Collectivistic behaviours mimimize exposure to outgroup members and uphold local hygienic norms

universally applicable. The Japanese language has no demonstrable phylogenetic relationship to Chinese but this cannot be taken to demonstrate that China has had no historical influence on Japanese society. Many important historical influences are not co-transmitted with language. Catholic religion, for example, might well influence patterns of fertility but the language tree would not pick up the fact that the Igbo of Nigeria are mainly Catholic whereas their Yoruba neighbours are not.

Researchers respond to this type of problem by including control variables for shared factors they suspect are important, but there might always be historical confounds that have not been identified. Some studies therefore include a dummy variable for each continent or region, which researchers hope will pick up any unidentified region-specific factors. This strategy would be much more powerful if linear mixed models or generalized linear mixed models (GLMMs) were used instead of ordinary regression models [12]. In these models, random effects specific to each country or society (if necessary, nested within larger regions or groups) can be included in the model. Moreover, the random effects of different sampling units can covary. This means that the model can capture shared historical influences without having to know *a priori* what these are. In addition, in GLMMs all the data for individuals can be used for cases such as individualism scores or fertility rather than having to derive an average for each country and use that as the datum in a regression analysis. Most of the studies in Table 1 show some awareness of Galton's problem and take some steps to mitigate it, but the danger of spurious associations is substantial in some cases and none of the studies used GLMMs.

Adjudicating between alternative explanations

Since we are dealing with correlational data in these studies, we need to separate the association documented from the causal hypothesis proposed to explain it. Even if the association is real, the reasons for it could be quite different from those proposed. For example, ethnic groups tend to have smaller ranges at the equator than towards the poles. One interpretation focuses on temporal variability in food production: at the equator, food production is constant through the year and thus local settlements have reduced need for nomadism or broad networks of exchange to buffer shortfalls [13,14]. The consequence is ethnolinguistic units of smaller scale. Alternatively, it could be that the high disease burdens in equatorial environments select for limited dispersal and outgroup avoidance, again leading to ethnolinguistic divergence [15,16]. Another alternative is that a high pathogen load mitigates against the development of large settlements and armies at the equator, constraining the potential size of social formations [17]. Other possibilities, or combinations, could undoubtedly also be envisaged. Such difficulties beset the interpretation of other patterns besides ethnic group size. Societies tend to be more polygynous where pathogen load is high (near the equator). This could be because women are selected to become more discriminating of male genetic quality where pathogens are more dangerous [18]. However, it could also be because the sex ratio is more female-biased in tropical countries [19], leading to a marriage market with an excess of women, which would favour polygyny.

There are a number of points to make about these problems. First, none of them contradicts the general claim that ecology is an important influence on human societies. If it were confirmed that polygyny is driven by variation in the sex ratio, this would still be an ecological effect because ecology must ultimately explain the latitudinal gradient in human sex ratio [19]. It might still be a pathogen effect because the infectious disease burden could drive sex-ratio variations through differential susceptibility of male babies [20]. Even if the reason for greater polygyny in the tropics were lower levels of economic development, this could be viewed as partly a consequence of ecology because economists have argued that eco-climatic variables exert a significant effect on economic productivity and growth [21,22].

Thus, when alternative explanations for the association are raised, they are often still ecological hypotheses at some level. What these amount to is the claim that the expected *mediators* of the relationship between the ecological predictor and the social outcome are different from those proposed. Thus, one way to adjudicate between competing accounts is to use a multivariate technique that tests between alternative mediation pathways, namely structural equation modelling (SEM) [23]. SEM is a multi-equation regression technique for examining the response of a variable to changes in several interconnected predictors. SEM makes it possible to estimate the total effect of an ecological parameter on a social parameter and to examine how much of this effect is direct and how much is mediated by other measured variables [24].

Although none of the studies in Table 1 uses SEM, it would be useful to do so for three reasons. First, pathogen diversity, which is a central explanatory variable in most of the studies, is itself strongly conditioned by more basic ecological predictors such as temperature and rainfall [17,25]. Thus, SEM might help to distinguish between direct effects of temperature and rainfall on human societies and indirect effects caused by the increase in pathogen load when temperature and rainfall are high. Second, SEM would help to identify social variables affected by other social variables and thus to discriminate between, for example, direct effects of pathogen load on fertility and indirect effects via economic development. Third, SEM facilitates the identification of latent variables within multivariate data. For example, age of weaning and fertility rate might respond as a single variable (fast life history [26]) to changes in ecology and such a coupled response could be identified using SEM.

The authors of the studies in Table 1 are well aware of the problems of interpretation and many include several control variables in their models, such as economic development when examining individualism or latitude when examining mate preferences. Such a strategy is conservative because these are not exactly confounds so much as related variables that could be somewhere on the same causal pathway. Thus, controlling for these variables and still finding a result is rather strong support for the hypothesis. However, use of SEM would make it unnecessary to compare latitude and pathogen load as predictors

Box 2. What are the proximate mechanisms driving local adaptation?

A number of different proximate mechanisms might be involved in local adaptation in humans. The first possibility is genetic differences. Although there is evidence of ongoing selection in the human genome [41,42], genetic diversity is humans is overwhelmingly distributed at the within-population level, with relatively little between-population variance [43], so genetics does not seem to be the most promising source of explanation for between-society patterns, even if within-population heritabilities are high. Moreover, the social change observed in response to many changes in environmental conditions is too rapid to reflect genetic evolution in many cases [29].

A second possible mechanism is developmental induction. This refers to evolved mechanisms that alter the adult phenotype, usually permanently, in response to early life inputs or conditions. For example, within Western societies, women who were of low birth weight for gestational age are more likely to reproduce early when they are adults [44]. This could reflect an evolved mechanism to adjust life-history strategy in response to phenotypic state. The striking cross-society differences in age at first reproduction [45] and age at weaning [46] might be driven by cross-society differences in birth weight for gestational age, which could in turn be influenced by ecology.

A third possible mechanism is context sensitivity (also called evoked culture [47], although this term is slightly misleading [48]). In these cases, behavioural flexibility is subserved by evolved psychological mechanisms that take particular classes of cue as input and output different behaviours or motivations. For example, societies subject to food shortage have norms of beauty favouring larger female body size than societies in which food is abundant, for fairly obvious adaptive reasons [49]. Hunger in Western males is sufficient to make them prefer larger female bodies than those they would favour in a satiated state [50]. Differences in body preferences might thus be at least partly evoked by the cue of current appetite, providing a mechanism for between-society differences and predicting, correctly, that these can be abolished very quickly when individuals move environment [51]. Like developmental induction, a requirement for context sensitivity is that humans have experienced a range of environmental conditions recurrently over evolutionary history. However, the if–then mappings do not have to be completely prespecified. Associative learning, for example, can be viewed as a mechanism to produce context sensitivity without precise prespecification of what will work in the local environment.

The final possible mechanism is true culture, or social learning. Social learning – copying the behaviour of others in the population – can be selected for when the costs of individual learning are substantial and there is some cross-individual and cross-time consistency in which behaviours are optimal [6,7]. However, social learning is under frequency-dependent selection and thus at equilibrium there will always be some mix of individual experimentation and social learning [52]. Whenever there is some bias in who is copied (e.g. differential copying of those who are most successful) then social learning will lead to locally adaptive behaviour much of the time [6]. However, social learning can lead to considerable adaptive lags, particularly when the cost of individual learning is high.

and would instead make it possible to estimate how much of the difference in mate preferences in temperate latitudes is due to reduced pathogen load and how much is due to other processes.

Beyond multivariate correlations

Even with best multivariate practice, uncertainty regarding the validity of causal claims can remain. There are not enough documented independent replicates of human social evolution to test all possible competing models conclusively using cross-sectional comparisons, and true experiments cannot be carried out for obvious reasons. Thus, we have to look to other types of data, specifically data from within-society individual variation and from changes over time, to extrapolate and confirm claims. However, this approach requires specification of the nature of the proximate mechanisms involved (Box 2) because different types of mechanism that could be driving local adaptation lead to different predictions.

If local adaptation to ecology in a particular case is driven by context-sensitive psychological mechanisms, then psychological responses of individuals in real time can be used to provide support for causal accounts. For example, making disease cues temporarily salient makes people favour ingroups over outgroups more strongly [27], which is a component of the pathogen-load account of the distribution of ethnic groups. Reminding men of mortality increases their desired number of children [28]. The cause of mortality was not specified but these study results could be related to between-society differences in fertility.

If adaptation is driven by mechanisms of developmental induction, then these real-time effects should not be expected. However, within-society individual differences might shed light on between-society patterns. If, for example, it could be shown within Western societies that maternal infection during pregnancy or disease early in life leads to faster female maturity, earlier reproduction and shorter breastfeeding, then the between-society association of life history with pathogen load would be comprehensible as a simple scaling up of individual exposures. If the mechanisms involved in adaptation are mainly social learning, it becomes more difficult to use within-society variation to understand between-society variation. This is because the dynamics of social learning often produces relative within-group uniformity even if individuals are not all experiencing the same ecological inputs [6].

The other potentially relevant sources of information are longitudinal. As disease exposures change, then, *ex hypothesi*, behaviours, attitudes and social structures should follow suit [29]. Particularly valuable will be any type of natural experiment in which an intervention causes lower disease exposures in one sub-population and not in another that is otherwise similar; examples of such scenarios in other domains have been reported [30,31]. However, predicting what will happen in these experiments of nature again depends on an understanding of the proximate mechanisms. Context sensitivity predicts an almost immediate response; developmental induction predicts a lag of a generation or more; social learning predicts a potentially long lag followed by an S-shaped diffusion curve as a new behaviour spreads through the population.

Conclusions

In this review I have discussed intriguing new findings showing associations between ecological conditions and human behavioural diversity. I have stressed some of the uncertainties involved in interpreting these patterns but that these uncertainties can be, and in some cases already have been, addressed. Our success in moving forward from this point will depend on our ability to

integrate different types of data (comparative, experimental, historical), and different facets of the explanation (functional, mechanistic, ontogenetic). I should also stress that not all between- (or indeed within-) society variation in human behaviour is driven by ecology. The endogenous dynamics of social forces, especially once social learning is involved, means that human history is massively path-dependent and involves large amounts of pattern and structure that could not be predicted from ecological context alone. This much is not in dispute. However, the data suggest that ecological factors do at least constrain the directions that social evolution can take.

An understanding of the interplay between social and ecological conditions contributes to the breaking down of boundaries. Such an understanding can transcend the obstructive barriers that exist in academia between the social and biological sciences, between anthropology and psychology, and between the study of humans and of the other species on this planet. Whatever the specific results of future research, this seems a worthwhile endeavour.

References

1 Brown, D.E. (1991) *Human Universals.* McGraw-Hill
2 Murdock, G.P. (1932) The science of culture. *Am. Anthropol.* 34, 200–215
3 Dawkins, R. (1976) *The Selfish Gene.* Oxford University Press
4 Pinker, S. (2002) *The Blank Slate: The Modern Denial of Human Nature.* Penguin
5 Hill, R.A. and Dunbar, R.I.M. (2002) Climatic determinants of diet and foraging behaviour in baboons. *Evol. Ecol.* 16, 579–593
6 Boyd, R. and Richerson, P.J. (1985) *Culture and the Evolutionary Process.* University of Chicago Press
7 Henrich, J. and McElreath, R. (2003) The evolution of cultural evolution. *Evol. Anthropol.* 12, 123–135
8 Eff, E.A. (2004) Does Mr. Galton still have a problem? Autocorrelation in the standard cross-cultural sample. *World Cultures* 15, 153–170
9 Mace, R. and Pagel, M. (1994) The comparative method in anthropology. *Curr. Anthropol.* 35, 549–564
10 Murdock, G.P. and White, D.R. (1969) Standard cross-cultural sample. *Ethnology* 8, 329–369
11 Mace, R. and Holden, C.J. (2005) A phylogenetic approach to cultural evolution. *Trends Ecol. Evol.* 20, 116–121
12 Bolker, B.M. *et al.* (2009) Generalized linear mixed models: a practical guide for ecology and evolution. *Trends Ecol. Evol.* 24, 127–135
13 Nettle, D. (1996) Language diversity in West Africa: an ecological approach. *J. Anthropol. Archaeol.* 15, 403–438
14 Nettle, D. (1998) Explaining global patterns of language diversity. *J. Anthropol. Archaeol.* 17, 354–374
15 Fincher, C.L. and Thornhill, R. (2008) A parasite-driven wedge: infectious diseases may explain language and other biodiversity. *Oikos* 117, 1289–1297
16 Fincher, C.L. and Thornhill, R. (2008) Assortative sociality, limited dispersal, infectious disease and the genesis of the global pattern of religion diversity. *Proc. R. Soc. B Biol. Sci.* 275, 2587–2594
17 Cashdan, E. (2001) Ethnic diversity and its environmental determinants: effects of climate, pathogens, and habitat diversity. *Am. Anthropol.* 103, 968–991
18 Low, B.S. (1990) Marriage systems and pathogen stress in human societies. *Am. Zool.* 30, 325–339
19 Navara, K.J. (2009) Humans at tropical latitudes produce more females. *Biol. Lett.* 5, 524–527
20 Wells, J.C.K. (2000) Natural selection and sex differences in morbidity and mortality in early life. *J. Theor. Biol.* 202, 65–76
21 Mellinger, A.D. *et al.* (2000) Climate, coastal proximity and development. In *Handbook of Economic Geography* (Clark, G.L. *et al.*, eds), pp. 169–194, Oxford, Oxford University Press
22 Gallup, J.L. and Sachs, J.D. (2000) Agriculture, climate, and technology: why are the tropics falling behind? *Am. J. Agric. Econ.* 82, 731–737
23 Grace, J.B. (2006) *Structural Equation Modelling and Natural Systems.* Cambridge University Press
24 Nettle, D. *et al.* (2007) Cultural diversity, economic development and societal instability. *PLoS ONE* 2, e929
25 Guernier, V. *et al.* (2004) Ecology drives the worldwide distribution of human diseases. *PLoS Biol.* 2, 740–746
26 Promislow, D.E.L. and Harvey, P.H. (1990) Living fast and dying young: a comparative analysis of life-history variation amongst mammals. *J. Zool.* 220, 417–437
27 Faulkner, J. *et al.* (2004) Evolved disease-avoidance mechanisms and contemporary xenophobic attitudes. *Group Process. Intergroup Relat* 7, 333–353
28 Mathews, P. and Sear, R. (2008) Life after death: an investigation into how mortality perceptions influence fertility preferences using evidence from an interrnet-based experiment. *J. Evol. Psychol.* 6, 155–172
29 Thornhill, R. *et al.* (2009) Parasites, democratization, and the liberalization of values across contemporary countries. *Biol. Rev.* 84, 113–131
30 Costello, E.J. *et al.* (2003) Relationships between poverty and psychopathology – a natural experiment. *J. Am. Med. Assoc.* 290, 2023–2029
31 Gibson, M.A. and Mace, R. (2006) An Energy-saving development initiative increases birth rate and childhood malnutrition in rural Ethiopia. *PLoS Med.* 3, e87
32 Orlove, B.S. (1980) Ecological anthropology. *Annu. Rev. Anthropol.* 9, 235–273
33 Steward, J.H. (1968) The concept and method of cultural ecology. In *International Encyclopedia of the Social Sciences* (Sills, D.L., ed.), pp. 337–344, MacMillan
34 Steward, J.H. (1955) *Theory of Culture Change. The Method of Multilinear Evolution.* University of Illinois Press
35 Denevan, W.M. (1983) Adaptation, variation and cultural geography. *Prof. Geogr.* 35, 399–406
36 Harvey, P.H. and Pagel, M. (1991) *The Comparative Method in Evolutionary Biology.* Oxford University Press
37 Sahlins, M.D. and Service, E. (1960) *Evolution and Culture.* University of Washington Press
38 Freeman, M.M.R. (1971) A social and ecological analysis of systematic female infanticide amongst the Netsilik Eskimo. *Am. Anthropol.* 73, 1011–1078
39 Vayda, A.P. (1974) Warfare in ecological perspective. *Annu. Rev. Ecol. Syst.* 5, 183–193
40 Soltis, J. *et al.* (1995) Can group-functional behaviors evolve by cultural group selection? An empirical test. *Curr. Anthropol.* 36, 473–494
41 Hawks, J. *et al.* (2007) Recent acceleration of human adaptive evolution. *Proc. Natl. Acad. Sci. U. S. A.* 104, 20753–20758
42 Voight, B.F. *et al.* (2006) A map of recent positive selection in the human genome. *PLoS Biol.* 4, 446–458
43 Rosenberg, N.A. *et al.* (2002) Genetic structure of human populations. *Science* 298, 2381–2385
44 Nettle, D., D.A. Coall & T.E Dickins (2009). Birthweight and paternal involvement predict early reproduction in British women: Evidence from the National Child Development Study. American Journal of Human Biology, doi:10.1002/ajhb.20970
45 Low, B.S. *et al.* (2008) Influences of women's reproductive lives: unexpected ecological underpinnings. *Cross-Cult. Res.* 42, 201–219
46 Quinlan, R.J. (2007) Human parental effort and environmental risk. *Proc. R. Soc. B Biol. Sci.* 274, 121–125
47 Gangestad, S.W. *et al.* (2006) Evolutionary foundations of cultural variation: evoked culture and mate preferences. *Psychol. Inq.* 17, 75–95
48 Sear, R. *et al.* (2007) Synthesis in the human evolutionary behavioural sciences. *J. Evol. Psychol.* 5, 3–28
49 Ember, C.R. *et al.* (2005) Valuing thinness or fatness in women: re-evaluating the effects of resource scarcity. *Evol. Hum. Behav.* 26, 257–270
50 Nelson, L.D. and Morrison, E.L. (2005) The symptoms of resource scarcity. Judgements of food and finances influence preferences for potential partners. *Psychol. Sci* 16, 167–173
51 Tovee, M.J. *et al.* (2006) Changing patterns of attractiveness as observers are exposed to a different culture. *Evol. Hum. Behav.* 27, 443–456

52 Rogers, A.R. (1988) Does biology constrain culture? *Am. Anthropol.* 90, 819–831
53 Mace, R. and Pagel, M. (1995) A latitudinal gradient in the density of languages in North America. *Proc. R. Soc. Lond. B Biol. Sci.* 261, 117–121
54 Collard, I.F. and Foley, R.A. (2002) Latitudinal patterns and environmental determinants of recent human cultural diversity: do humans follow biogeographical rules? *Evol. Ecol. Res.* 4, 371–383
55 Moore, J.L. *et al.* (2002) The distribution of cultural and biological diversity in Africa. *Proc. R. Soc. Lond. Ser. B Biol. Sci.* 269, 1645–1653
56 Guegan, J.F. *et al.* (2001) Disease diversity and human fertility. *Evolution* 55, 1308–1314
57 Schaller, M. and Murray, D.R. (2008) Pathogens, personality and culture: disease prevalence predicts worldwide variability in sociosexuality, extraversion and openness to experience. *J. Personal. Soc. Psychol.* 95, 212–221
58 Fincher, C.L. *et al.* (2008) Pathogen prevalence predicts human cross-cultural variability in individualism/collectivism. *Proc. R. Soc. B Biol. Sci.* 275, 1279–1285