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Do transmission mechanisms or social systems drive cultural dynamics in socially structured populations?

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Cultural traits spread via multiple mechanisms among individuals within social groups, including via transmission biases that occur when subordinates copy from dominants (prestige transmission), or via common cultural trait variants that are favoured over rare ones (consensus transmission). Most animal populations are subdivided into social groups where cultural learning occurs, yet theoretical studies of cultural trait transmission have tended to focus on within-group transmission dynamics. We developed an agent-based model of cultural transmission in socially structured populations in which a trait arises in one individual and either persists until a stable population equilibrium is reached, or goes extinct. With this model, we systematically varied group size, rates of dispersal among groups, mortality rates, transmission characteristics, the benefit of the cultural trait (including possibly negative benefits), and whether individuals disperse locally or randomly. We used generalized linear models to examine how changes in these parameters influence trait extinction, equilibrium prevalence and time to equilibrium. Four traits increased the probability of extinction: smaller group size, higher background mortality, lower transmission rate and more costly traits (although costly traits sometimes reached an equilibrium). Local dispersal and biased transmission mechanisms (prestige and consensus) had no significant effects on extinction probability, and similar patterns were found for equilibrium prevalence. We found that a lower dispersal rate and local dispersal slowed the time required for a trait to reach equilibrium, as did smaller groups, lower transmission rates and lower costs. Collectively, these analyses reveal that prestige and consensus transmission have weaker effects than other factors associated with demographic and social conditions.

A striking feature of human evolution is the incredible diversity of cultures that exist around the world. For example, linguists have counted over 6000 languages (Gordon 2005), and humans are thought to practise more than 4300 religions (faith groups). Many human cultural traits are likely to be adaptive, such as those related to resource allocation and health practises, and are thus subject to natural selection (Mesoudi et al. 2004). Other cultural traits, such as decorations on pottery, are probably driven less by natural selection, but they may provide social or sexual benefits that indirectly translate to higher reproduction. Some persistent cultural traits in humans are even associated with costs. For example, a celibate priesthood dramatically reduces the reproductive success of individuals that become priests, while scarification, excision and circumcision increase the risks of lethal infections, especially in societies living without access to safe medical practises. Potential cultural traditions also have been documented in many nonhuman systems, including nut cracking in chimpanzees, *Pan troglodytes* (Boesch et al. 1994; Boesch & Boesch-Achermann 2000), potato washing in Japanese macaques, *Macaca fuscata* (Kawai 1965), and tool use in New Caledonian crows, *Corvus moneduloides*, to obtain invertebrates from the vegetation (Hunt & Gray 2003). Understanding the spread of cultural traits in nonhuman systems could provide insights to human evolution and the factors leading to the explosive growth of cultural traits in the human lineage.

A critical question in studies of cultural evolution involves features that affect the dynamics of cultural traits, both in terms of the proportion of individuals that express the trait and the rate at which the behaviour spreads through a population. In addition to the cost or benefit of the trait in question, two factors are thought to be important to the spread of cultural traits: the mechanism by which behaviours are learned and the social context in which

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transmission takes place. First, concerning mechanisms, cultural transmission usually occurs through social learning, in which an individual learns a new behaviour by watching other individuals perform the behaviour. In nonhuman primates, for example, social learning has been proposed in the case of potato washing in Japanese macaques and nut cracking in chimpanzees (Kawai 1965; Boesch & Boesch-Achermann 2000). Importantly, different models of cultural transmission may operate, depending on the social system in which the individual is embedded and the mechanisms by which traits are acquired. For example, individuals may be more likely to copy the behaviours of more dominant individuals, which would be adaptive if dominants possess behavioural traits that make them more successful (Boyd & Richerson 1985; Boesch & Tomasello 1998; Henrich & McElreath 2003). Similarly, individuals may be more likely to adopt traits when more individuals in the group express the trait through a 'conformity' or 'majority rule' mechanism (Boesch & Tomasello 1998; Henrich & McElreath 2003; hereafter called consensus transmission). While these transmission biases are not mutually exclusive, neither are they completely congruent.

Second, social context is likely to be important for the spread of cultural traits. At the population level, most primates and humans live in socially structured populations, and the limited evidence available suggests that cultural traits tend to spread more commonly among members of the same social group than between groups (Kawamura 1959; Boesch 2003; Leca et al. 2007). Within social groups, the rate of cultural transmission is expected to be higher when group sizes are larger, with larger numbers of more tolerant individuals providing more opportunities for invention and social learning (van Schaik et al. 1999). Opportunities for learning can be modified by other factors, such as proximity of individuals and their capacity for social learning (van Schaik & Pradhan 2003). Mortality rates and movement between social groups can also be important in a socially structured population. If dispersal occurs only between neighbouring groups and at a low rate, for example, then the trait in question may take longer to establish in the larger population, and will thus be more prone to cultural extinction if the group is lost because of other factors. Similarly, if individuals that possess a costly cultural trait die at a higher rate, fewer other individuals will have an opportunity to learn the skills that are needed to express the behaviour.

In this paper, we use an agent-based model (Grimm & Railsback 2005) to investigate how cultural traits spread through animal social systems, focusing in particular on features involving group size, dispersal and background mortality (i.e. a death rate that is independent of expressing the cultural trait). We also examine how different mechanisms of social learning, specifically involving prestige and consensus models, influence the spread of cultural traits, and how different probabilities of acquiring the trait and the selective benefits (or costs) of the trait affect transmission dynamics. The model is spatially explicit and incorporates three social transmission mechanisms, and individuals can disperse either locally (a spatial model) or randomly to any of the groups (a nonspatial model). In addition, the model allows for variation in group size, dispersal rates, mortality and the selective benefit (or cost) of the cultural trait (expressed by adjusting the baseline mortality rate among individuals with the trait). The model can therefore be applied to study cultural traits in a wide range of systems in which individuals live in socially structured populations, including humans, nonhuman primates and other animals. Our work adds to a growing number of agent-based models of cultural trait transmission, including in the context of foraging (van der Post & Hogeweg 2006, 2008) and the spread of traits through social networks (Franz & Nunn 2009).

Social learning is a key component of the model. We call the two roles in this exchange the 'observer', who learns the behaviour, and the 'performer', who expresses the behaviour and therefore serves as the role model for social learning to take place. We investigated three different transmission mechanisms (Boesch & Tomasello 1998; Henrich & McElreath 2003). The first transmission mechanism, referred to as the random transmission model, is the simplest. In this scenario the probability of cultural transmission between two individuals is independent of sex, social affiliation, the proportion of group-mates with the trait and dominance rank. The other mechanisms represent modifications of the random model. In the prestige transmission model, transmission probabilities are positively correlated with the dominance rank of the individual expressing the trait (Boyd & Richerson 1985; Henrich & Gil-White 2001). Consensus transmission addresses the importance of social conformity, with increasing probability that an individual adopts a trait as the proportion of groupmates expressing the trait increases (Boyd & Richerson 1985; Henrich & Boyd 1998). We designed the simulation model so that the mean rate of transmission would be approximately equal across the three transmission mechanisms.

We investigated four main questions regarding the relative effects of social system and transmission characteristics on the spread of cultural traits. In particular, we were interested in how these features interact to determine the probability of extinction, the equilibrium proportion of individuals with the trait, and the time to equilibrium.

(1) Does local dispersal in a spatially explicit model (i.e. local dispersal) produce different outcomes than a nonspatial model, in which dispersing individuals can move to any social group? Random movement from one group to any other group increases the probability that a dispersing individual with the trait will land in a group that has yet to experience the trait. Once within a group, the trait is expected to spread rapidly. Thus, random movement should increase the rate of trait spread and favour the establishment of traits in the population. In contrast, local dispersal should slow the rate of cultural dispersion at the population level. Less is known about how local dispersal affects the prevalence of a trait or its probability of extinction, but we expect that spatially localized traits are more likely to go extinct through stochastic processes.

(2) Do cultural traits spread more rapidly, and reach higher prevalence, in populations composed of larger social groups, or in populations characterized by higher rates of individual movement among groups? These two social parameters could interact, with larger groups potentially producing more migrants that carry the trait to other groups. Here, we focus on actual movement of individuals between groups (migration), thus assuming that casual observation of individuals in neighbouring groups is insufficient for social learning to occur (cf. Boyd & Richerson 2002).

(3) How does mortality affect the prevalence of a cultural trait in a population? In epidemiological models, higher rates of mortality remove individuals carrying a disease from the population, making it more difficult for the pathogen to become established and reducing overall prevalence (Anderson & May 1979; Thrall et al. 2000). Similar principles should apply to cultural traits. Thus, increased background mortality (i.e. mortality that is independent of the expression of the cultural trait) should negatively affect the equilibrium prevalence of the trait. The selective advantage of cultural traits should modify these patterns. Higher benefits (holding costs constant) should lead to lower mortality among individuals with the trait and result in more opportunities for the trait to spread. Traits with a net cost should lead to the opposite pattern, resulting in lower prevalence and increased probability that the trait will go extinct.

(4) How do social learning mechanisms influence the spread of cultural traits? One aspect of social learning involves the probability that a trait will spread from one individual to another.

A higher rate of transmission (β) could increase prevalence, or reduce the time until equilibrium prevalence is reached. Another aspect of social learning involves the transmission mechanisms discussed above, which effectively modify β based on individual characteristics (prestige transmission) or prevalence of the trait in a group (consensus transmission). Compared to a random model, do cultural traits spread more rapidly or reach higher equilibrium prevalence under a prestige or consensus model?

METHODS

Simulation Model Structure

We developed a simulation model using the computer package MATLAB (version 7, Natwick, MA, U.S.A.) to simulate the spread of an introduced cultural trait in a socially structured population. The basic design of the model was developed as part of a previous investigation of the effect of host social group structure on the spread of an emerging infectious disease (Nunn et al. 2008). In that study, an initial infection was introduced into a population of susceptible hosts. Individuals that died from disease were not replaced (as is typical of wildlife epidemics), and group composition was allowed to depart from initial conditions as animals died or dispersed from groups. Here, we extend the model to study the spread of culturally inherited traits by including three different transmission mechanisms and adaptive value to the trait (i.e. positive or negative net benefits, representing a beneficial or costly trait, respectively).

We were particularly interested in examining the spread of cultural traits in a spatial context, given that previous studies on infectious disease have shown that spatial structure can significantly impact disease dynamics and longer-term evolutionary processes (Thrall & Antonovics 1995; Gandon et al. 1996; Boots & Sasaki 1999; Roy & Kirchner 2000; Carlsson-Graner & Thrall 2002; O'Keefe & Antonovics 2002). For each simulation run, groups of individuals were formed based on user-specified values for group size. Groups were distributed on a 12×12 matrix (i.e. 144 groups on a square lattice) and formed as random draws from a Poisson distribution assuming an equal number of males and females. Deaths, births and dispersal of individuals will tend to cause the initial social conditions to drift over a simulation run, especially when simulations are run for many time steps. To deal with this issue, we retained a matrix of the initial numbers of males and females in each group. This 'initiating matrix' was used to stochastically adjust probabilities associated with demographic parameters (birth and dispersal) to help maintain initial conditions for each group throughout a simulation run.

The cultural trait was initiated in a single individual, and the trait was allowed to spread through the population in discrete time steps. In each time step, an individual remained in its original group or dispersed to other groups in the population, as determined by the probability of dispersal per time step. We assumed that dispersing individuals lacked contact with conspecifics. We further assumed that dominance rank of a migrant equalled the rank of that individual in the previous group and that this rank did not affect the probability of emigrating or immigrating. Individuals that dispersed were not allowed to enter groups from which they had most recently departed. The simulation was allowed to run until the cultural trait either went extinct in the population or the prevalence of the cultural trait stabilized at a nonzero value.

Mechanisms of Cultural Transmission

Cultural traits spread by social learning within groups, and the probability of transmission (β) represents the per-contact probability of an observer acquiring a cultural trait from an individual that expresses the behaviour. Mechanistically, β encapsulates the combined probability that one individual expresses the trait while another naïve individual can view and potentially learn from the performer, including the time needed for the observer to learn techniques associated with performing the trait. Thus, lower values of β could represent behaviours that are more complex (and thus more difficult to learn) or behaviours that are performed more rarely. Individuals that acquire the trait serve as performers in the next time step, and agents retain the trait throughout their lives in a simulation run. In our model, the selective benefits (or costs) of cultural traits are expressed by altering the background probability of death (see below).

In the random model, contact rates and per-contact probabilities of transmission were equal among all individuals in a social group regardless of dominance rank and the proportion of individuals expressing the trait. Contacts within groups were assumed to have no spatial restrictions, in comparison to contacts between groups (where contact can occur only through dispersal). Thus, contact rate increased with group size, analogous to predictions from standard mass-action epidemiological models (May & Anderson 1979; Anderson & May 1981). Analytically, the probability of a susceptible individual not acquiring the trait as a result of contacts with members of its group is equal to $(1-\beta)^I$, where I represents the number of individuals in a social group expressing the cultural trait. Thus, the overall probability that an individual learns the trait from one or more performers in a time step is given by $1-(1-\beta)^I$.

The prestige model calculates the individual probability that a trait spreads between individuals based on the rank of the performer, under the assumption that observers prefer to copy more dominant individuals within the population, including the possibility that animals possess simple heuristics in which subordinates emulate dominants as a way to learn successful foraging, competitive and hunting behaviours (Boyd & Richerson 1985; Boesch & Tomasello 1998; Henrich & McElreath 2003). At the time of group formation, individuals were assigned dominance ranks (d_i) using values from a uniform distribution. Use of a uniform distribution was preferred to the normal or other distributions because it captures the essence of dominance as a linear ranking, while also allowing some fine differences between individuals in rank. In the process of simulating the spread of cultural traits in the prestige model, user-defined values of β were adjusted as follows for spread of a trait from performer i to observer j:

$$\beta' = \beta\,(0.01 + 1.98\,r_i)$$

where r_i' is the standardized rank of the performer, with standardization of ranks within each group ranging from 0 to 1 ($r_i' = (r_i - r_{\min})/(r_{\max} - r_{\min})$). This procedure gives a range of values for β′ of 0.01 to 1.99 times the user-specified β, with the midpoint centred on the user-specified value β. In this way, the individual with the lowest possible dominance rank (=0) had an adjusted β greater than zero ($\beta' = 0.01$), thus preventing deterministic extinction of the trait if the first performer of the trait happened to be the lowest-ranking individual in a group. When $\beta > 0.5$, the probability of transmission could exceed 1 for higher-ranking individuals. As our values of β were always less than 0.04 (Table 1), this should have no effect on model outcomes. Although a stronger version of the prestige model might not allow transmission from the lowest-ranking performer to occur, it is worth noting that in our model, the probability of transmission for the lowest-ranking individual is two orders of magnitude smaller than a middle-ranking individual; thus, rank has substantial effects on the probability of transmission. In one run of the simulation using the prestige model, we found that the normalized dominance rank of the performer was higher than the observer ($t = 37.7$, $N = 427$

Table 1
Parameter values investigated in the simulation

Symbol	Definition	Range of values
g	Average number of individuals in groups	4 to 40
d	Baseline probability of dispersal per day	0.0001 to 0.02
β	Per-contact transmission probability	0.0001 to 0.04
m	Baseline mortality rate per day	0.0001 to 0.04
c	Benefit or cost of cultural trait (multiplier for m)	0.001 to 2
S	Spatial vs nonspatial model (categorical)	0,1
T	Transmission model (categorical, corresponding to random, consensus and prestige transmission)	0,1,2

transmission events, $P < 0.0001$), where the average rank of the source was 0.667 and the average rank of the recipient was 0.495. For the other transmission models, both performer and observer had similar ranks (average of 0.50).

In the consensus model, transmission rates were adjusted based on the percentage of individuals in the group that expressed the trait, p. The adjusted β was calculated using a linear transformation:

$$\beta' = \beta + (p - 0.5)\beta$$

Thus, when less than half of the group members expressed the trait, β was adjusted downwards, and when the majority of individuals expressed the trait, β was adjusted upwards. It is important to stress that only the per-contact probability of transmission parameter (β) was adjusted, and this reflects the probability of transmission between two individuals in the same group; the mass-action effect of increasing numbers of 'culturally infected' individuals represents an independent effect that tends to increase the spread of traits as more individuals in a group acquire the behaviour.

The consensus and prestige models were designed to be as simple as possible in their implementation and to have equal average probabilities of transmission, thus allowing comparison among the different transmission models. We acknowledge, however, that different mechanisms of maintaining a constant 'average' transmission rate are possible and could produce dynamics that differ from those reported here. We consider this in more depth in the Discussion, along with alternative forms of biased transmission that could be investigated in the future.

Maintaining Starting Conditions: Deaths, Births and Dispersal

The causes of death were identified during a simulation run as being due to background mortality (m), such as predation and old age, or the presence of a costly cultural trait (because this increased mortality rates through a linear transformation of background mortality). An individual that died from natural causes was replaced by an individual of the same sex. Newly generated healthy individuals were placed in one of the existing groups with a probability that was adjusted according to how current group composition compared to the initiating matrix. If the number of individuals of the sex of the individual being replaced was less than the initiating values for that group, then the probability of assignment was increased. The new group was then determined based on a random draw from a list of all groups, with each group listed once and groups that were deficient given an additional entry. Thus, individuals could be added to any of the groups, but the addition was more likely if the group showed a deficit in the number of individuals of that sex, relative to the initiating matrix. As in our previous model (Nunn et al. 2008), we assumed that mortality rates were independent of age and that deaths attributed to a costly trait were not replaced by new individuals (as might be expected if populations are unable to respond demographically to these losses in the time horizons simulated here).

To investigate the effect of selective benefits (and costs) of a cultural trait, we assumed that selection on cultural traits acts by increasing or decreasing mortality. The mortality rate of individuals with the cultural trait was multiplied by a selection multiplier, s_m, which was user-defined and ranged from 0.001 to 2 (Table 1). Thus, selective benefits produced a death rate that was as low as 1/1000 of the baseline mortality ($s_m = 0.001$), and selective costs could increase baseline mortality by as much as 2 times ($s_m = 2$). In exploratory simulations, values of $s_m > 2$ tended to result in rapid and consistent extinction of the trait. Because individuals that died from expressing a costly trait were not replaced in the simulations, a costly trait might be expected to go extinct over the longer term in the simulated populations.

We also varied the rate of dispersal, which was measured as the per-day probability that an individual disperses from a group (Table 1). We assumed that dispersal was more likely for groups in which the number of individuals of a particular sex was above the initiating values for the number of individuals for that sex, thus using a procedure similar to that described above for mortality to maintain the initial population structure. Once dispersal was initiated, individuals were capable of entering a new group as soon as the next day. The dispersing individual moved in a random walk on the two-dimensional lattice of cells representing the different social groups. The lattice was bounded spatially and was not reflective; thus, a dispersing individual that hit a boundary did not move in that time step. When floaters entered a new group, they were capable of transmitting cultural traits as early as the next daily time step of the simulation.

In summary, group composition was adjusted to maintain initial, user-specified values by preferentially adding individuals to groups with a deficiency in males or females through births and removing individuals from groups with an excess number of males or females through dispersal events.

Sampling Parameter Space and Simulation Procedures

To explore how different parameters influence cultural dynamics, we undertook multivariate analysis using random sampling. Random sampling was conducted using Latin hypercube sampling (Seaholm et al. 1988; Blower & Dowlatabadi 1994; Rushton et al. 2000), which is a type of stratified Monte Carlo sampling that has been used in epidemiological modelling and is more efficient in this context than random sampling regimes or those that include all possible parameter values (Seaholm et al. 1988; Blower & Dowlatabadi 1994). Seven parameters were varied in the Latin hypercube sample: transmission model, group size, transmission probability, background mortality, net benefit of the cultural trait, rate of dispersal and a spatial versus nonspatial dispersal model. Table 1 gives ranges of parameter values. The discretely coded parameters (transmission model, spatial model) were represented as continuously varying traits in the Latin hypercube sample, and binned into equal numbers of the discrete traits. We assessed the sample size needed for the Latin hypercube sample by computing the theoretical variance and relative bias of parameter estimates for a range of possible sample sizes. To obtain rough approximations of the aforesaid variance and bias, we fit preliminary models from a few pilot simulation runs. From these computations, we determined that a sample size of 1500 would be sufficient to investigate the effects of parameter variation shown in Table 1.

As noted above, each simulation run continued until the prevalence of the cultural trait reached equilibrium, or prevalence fell to 0 (i.e. the cultural trait went extinct). For cases in which the trait

persisted, equilibrium prevalence was determined empirically. Specifically, the simulation was stopped when six interrelated conditions were met. (1) The cultural trait had spread to all groups (even if subsequently going extinct in one or more groups). The correlation between time and prevalence (2) was nonsignificant at $P > 0.05$ and (3) explained less than 1% of the variation over the previous window of 200 time steps. Similarly, we examined the standard error of overall prevalence and required that it (4) was nonsignificant at $P > 0.05$ and (5) explained less than 1% of the variation over the previous 200 time steps. (6) Finally, we required that the median standard error of overall prevalence was less than the median for 200 time steps. We also examined variation in the time to equilibrium, defined as the first time step in which the estimated equilibrium value was reached, and we investigated factors that led to extinction of the trait. Figure 1 provides an example from one simulation run. The trait spread rapidly and reached an equilibrium prevalence of about 0.80 among individuals in the population after approximately 500 time steps. From this, the equilibrium prevalence was calculated as 0.798 and the time to equilibrium following infection of all groups occurred on day 504. To satisfy the criteria for identifying equilibrium, the duration of the actual simulation was an order of magnitude longer than the time to equilibrium, with these criteria finally satisfied on day 5537.

Analyses of Simulation Output

We analysed the output from the simulation using both generalized linear models (GLMs) and regression and classification trees (De'ath & Fabricius 2000; Roff & Roff 2003). We constructed three linear models to explain the simulation outcomes in terms of their parameters. First, all variables were scaled to the unit interval so that the magnitudes of their fitted effects could be compared on an absolute scale. To test for possible interaction effects among the simulation settings, we fitted each model using two sets of explanatory variables: a 'reduced set' incorporating only main effects and a 'full set' including all possible interactions. The reduced set comprised the seven variables in Table 1 and the particular interaction of background mortality (m) and the cost multiplier of the trait (c), both of which were hypothesized to drive the response. The second set included these factors along with all 27 possible pairwise interactions. For each of the three outcomes, the full and reduced models were compared using a likelihood ratio test and Wald test (McCullagh & Nelder 1989) to assess the significance of the full set of pairwise interactions. All linear models were estimated using standard packages from the R statistical software (The R Foundation for Statistical Computing, Vienna, Austria). When plotting modelled output, we used default values of $g = 16$, $d = 0.008$, $\beta = 0.004$, $m = 0.01$, with no costs or benefits for the cultural trait, a nonspatial model, and random transmission within groups.

Regression and classification trees were calculated for the analysis of extinction probability and time to equilibrium using the Statistics Toolbox in MATLAB v. 7.0. We split impure nodes when the number of observations for that node was 100 for regression trees (time to equilibrium analysis) and 10 for classification trees (extinction analysis). After creating an initial tree using the simulation output, we used 10-fold cross-validation to identify the pruning level with the minimal cost (De'ath & Fabricius 2000), identified as the tree with the minimum error rate. Using this pruned tree, we calculated the percentage of variance explained by comparing predicted and observed values for the regression trees.

RESULTS

General Patterns

The simulation model produced a diversity of outcomes, with some traits quickly going extinct and others reaching an equilibrium in which the majority of individuals in the population expressed the trait. These variable outcomes reflected both stochastic effects and the effects of the parameters on the simulation dynamics. Among the 1500 simulations, the cultural trait persisted in 52.5% of the runs, as defined by the equilibrium conditions described in the Methods; in the remaining simulation runs, the cultural trait went extinct. Of the simulations resulting in trait persistence, the model ran for an average of 1513 time steps (range 453–11 198 time steps). In cases of extinction, the model ran for an average of 842 time steps (range 1–7477 time steps). In cases of trait persistence, the average proportion of individuals expressing the trait was 0.931 (range 0.19–1.0) and the time to reach this equilibrium 'prevalence' was 447 time steps (range 18–8893 time steps). In cases of extinction, the trait spread to an average of 81.2 groups prior to going extinct (range 1–144). Thus, even traits that eventually went extinct often spread widely in the population.

The net benefit of the trait varied in the Latin hypercube sample (along with other parameters in Table 1). In general, traits with higher costs tended to go extinct, while higher benefits favoured the establishment of a cultural trait (Fig. 2). Remarkably, in 57.7% of

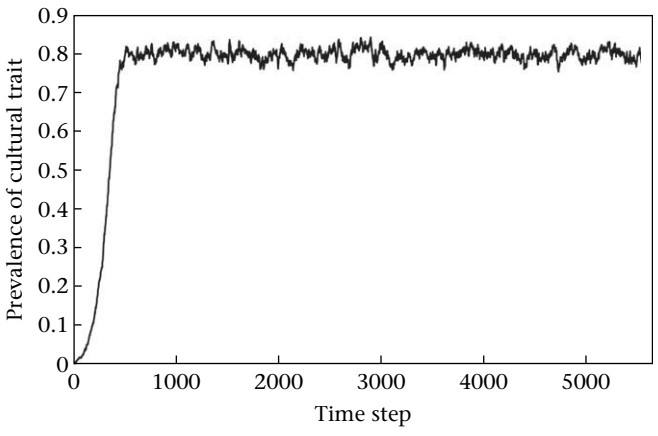

Figure 1. Detecting equilibrium. Plot shows output from one simulation run using default parameters. The text provides details on equilibrium prevalence and the time step at which this was first reached, as calculated by the simulation program.

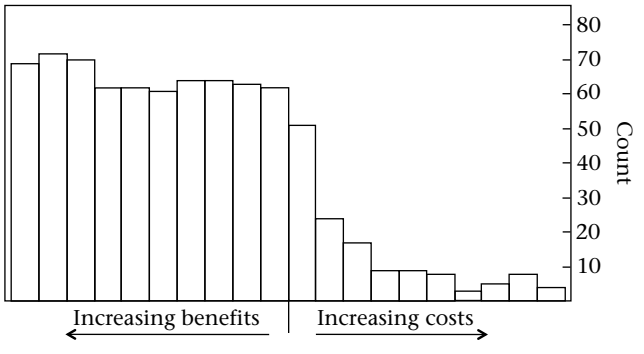

Figure 2. Trait persistence in relation to net benefits of the cultural trait. Bars indicate number of cases in which the trait reached an equilibrium, as compared to the alternative of going extinct. Increasing benefits are shown to the left of the central line, while increasing costs are shown to the right. Results are based on the output from 1500 simulations. The Latin hypercube sample provided a flat distribution for the values, including costs of the trait shown along the X axis, so this plot reveals that higher costs are associated with higher extinction, but that some costly traits nevertheless reached an equilibrium as defined in this study.

simulations of costly traits, the trait managed to spread to all 144 groups in the population (although not all groups necessarily had the trait simultaneously). In 68% of these cases of pervasive spread, however, the costly trait subsequently went extinct. Thus, costly social traits spread widely in the simulated populations, but these traits typically failed to reach a stable equilibrium and eventually went extinct. The analyses below provide more insights into how costs affect trait establishment and spread.

Probability of Extinction

We first investigated the factors that influence the probability of extinction. We fitted a logistic regression model for the full and reduced variable sets, treating extinction as the binary outcome for all 1500 simulations. Using the Wald test for the significance of the pairwise interaction effects in the full model, we found them to be nonsignificant ($\chi^2_{27} = 8.31$, $P = 0.99$). Consequently, we settled on the reduced model (Table 2). The main drivers in this model were trait cost, background mortality, transmission probability and group size. We found that group size had a strongly negative effect on the probability of extinction (Fig. 3a). The effect of trait cost and background mortality on extinction was stronger (based on the parameter estimates) (Fig. 4). The probability of extinction increased with cost, and was further driven by an interaction effect with background mortality. Transmission probability (β) had a negative coefficient, indicating that increases in β reduced the risk of trait extinction. We also found that higher rates of dispersal reduced the probability of extinction, although this effect was not significant ($P = 0.07$). In contrast to these factors, the coefficients associated with the transmission model and spatial models were negligibly small and not significant, indicating that the results were similar across all transmission models and were minimally affected by either local dispersal or transmission biases.

To visualize the effects of the parameters on the probability of extinction, we also ran a classification tree analysis. The resulting tree (Fig. 5) revealed that traits were more likely to go extinct at higher costs and higher mortality. The tree also predicted that for beneficial traits, a higher transmission probability (β) reduced the probability of extinction. The classification tree analysis confirmed the interaction between costliness of the trait and mortality in the generalized linear model (Table 2), but failed to detect an effect of group size. The tree also provided no evidence for effects of local dispersal or transmission model.

Equilibrium Prevalence

The second set of analyses involved the factors that influenced the proportion of individuals that expressed the cultural trait at equilibrium (i.e. equilibrium prevalence). For the 787 simulations in which the trait did not go extinct, we fitted a binomial GLM for prevalence, modelling the mean proportion of individuals that had

Table 2
Parameter estimates in the logistic regression model for extinction probability

Parameter	Estimate	SE	*P*
Intercept	−3.35	0.68	<0.001
g	−1.42	0.38	<0.001
d	−0.67	0.37	0.070
β	−1.81	0.39	<0.001
m	0.04	1.03	0.971
c	6.01	0.92	<0.001
T (consensus vs random)	0.37	0.26	0.153
T (prestige vs random)	0.36	0.27	0.178
S (spatial vs nonspatial)	0.16	0.21	0.453
*m***c*	7.93	1.92	<0.001

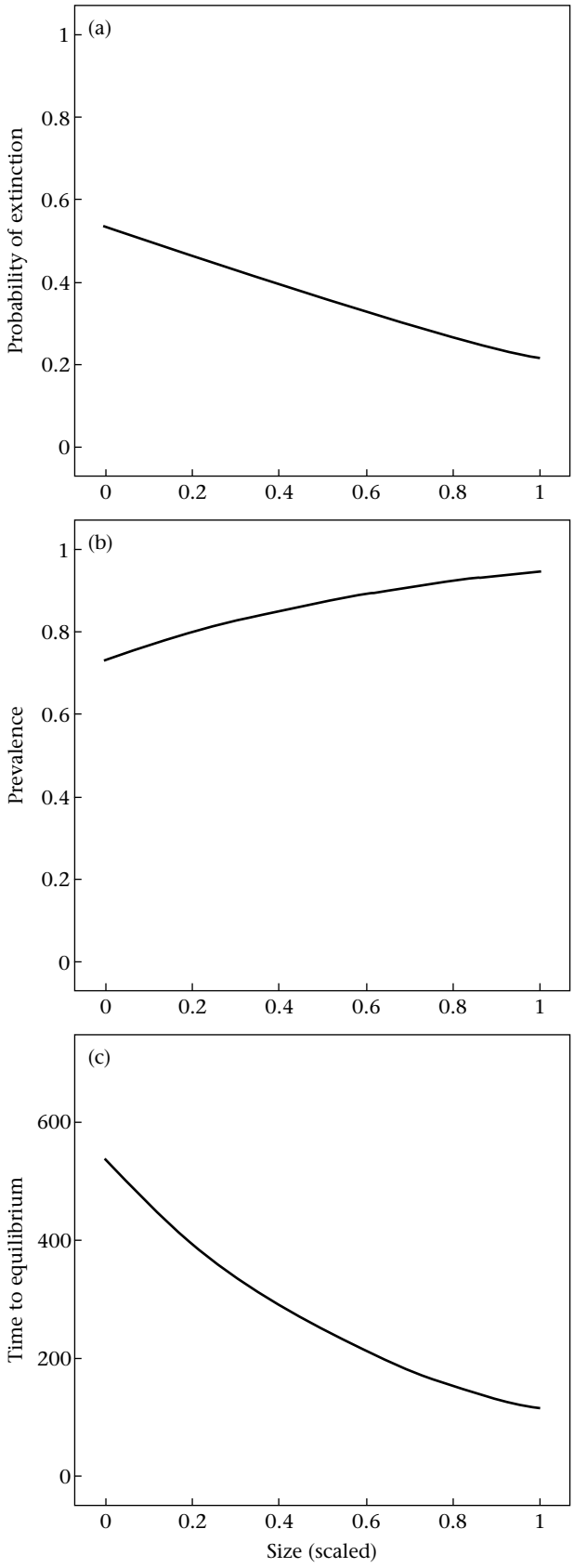

Figure 3. Modelled (a) extinction rate, (b) prevalence and (c) time to equilibrium by scaled group size. The rates correspond to default values of the other settings (see Methods).

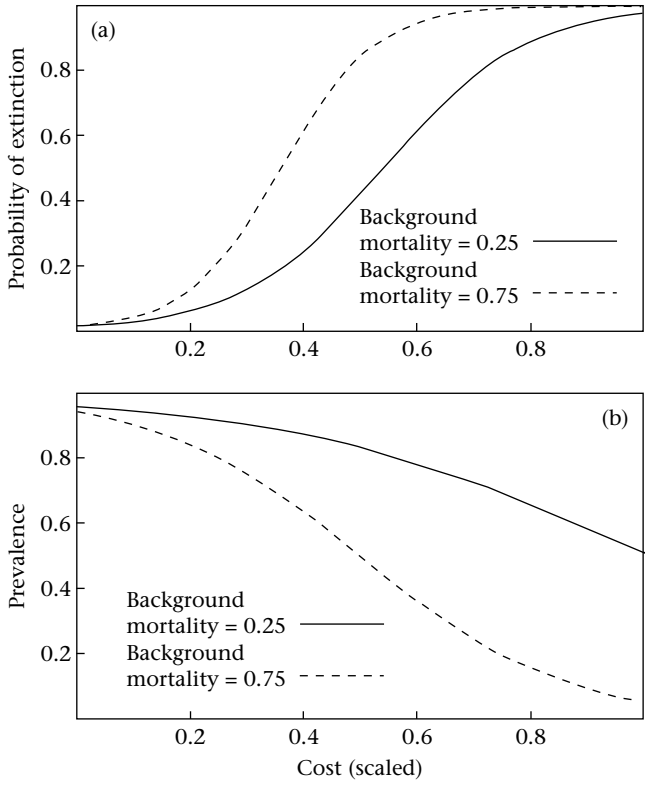

Figure 4. Modelled (a) extinction rate and (b) prevalence across a range of cost levels at two background mortality rates, *m* (scaled on the interval 0 to 1). The other settings are at default values (see Methods).

the trait at the end of the simulation. We again tested for interaction effects additional to mortality × cost using the likelihood ratio test and found none to be significant ($\chi^2_{27} = 26.07, P = 0.49$), leading us to accept the reduced model. The resulting regression estimates are shown in Table 3.

As expected, most of the coefficients for the extinction model were reversed in sign for the model describing equilibrium prevalence (i.e. factors that increase prevalence should decrease the probability of extinction). The relative magnitudes of the parameters varied. In the binomial GLM for prevalence, the transmission probability (β) had a major impact on prevalence of the cultural trait, with greater values of β increasing equilibrium prevalence. Group size (Fig. 3), background mortality and trait cost all affected prevalence,

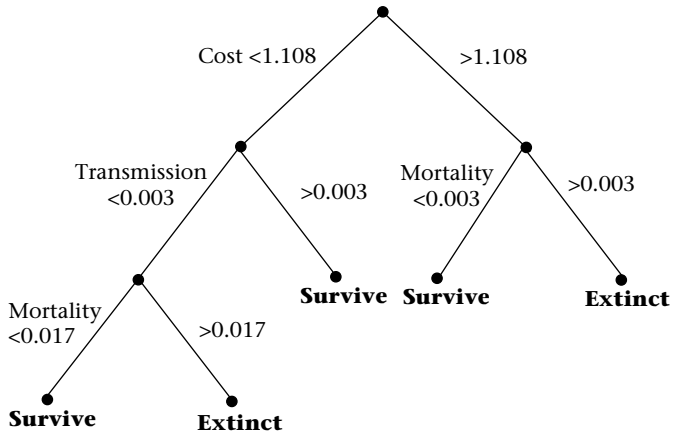

Figure 5. Classification tree for extinction. Extinction is indicated as a dichotomous trait on the tips of the tree, where 'survive' indicates that the trait is predicted to reach an equilibrium rather than go extinct.

Table 3
Parameter estimates in the binomial GLM model for trait prevalence

Parameter	Estimate	SE	*P*
Intercept	2.43	0.18	<0.001
g	1.84	0.12	<0.001
d	0.12	0.11	0.293
β	2.91	0.13	<0.001
m	−0.79	0.24	0.001
c	−1.95	0.31	<0.001
T (consensus vs random)	0.14	0.08	0.065
T (prestige vs random)	−0.09	0.08	0.214
S (spatial vs nonspatial)	0.04	0.06	0.455
*m***c*	−4.89	0.58	<0.001

with a strong interaction between mortality and cost (Fig. 4). The results were again similar across most transmission and spatial models, although consensus transmission tended to result in higher prevalence (as compared to the random model).

Time to Equilibrium

We analysed the factors that influence the speed with which the trait spreads in the population by again focusing on the 787 simulations in which the traits reached an equilibrium. Because the equilibrium times were highly right-skewed, we fitted a log-linear model of time to equilibrium. In this case, the full model, with main effects and all pairwise interactions, yielded a significantly better fit than the reduced model, leading the likelihood ratio test to reject the reduced model ($\chi^2_{27} = 49.31, P = 0.005$). Table 4 shows the most significant effects and interactions from the full model, which explained 75% of the variation in log-transformed time to equilibrium.

The major drivers of time to equilibrium were group size, dispersal rate, cost of the trait and transmission probability. Time to equilibrium decreased with larger group sizes (Fig. 3) and greater dispersal rates (Fig. 6). A strong negative coefficient indicated that greater transmission probabilities (β) also increased the rate at which a cultural trait penetrated a population (Fig. 6). Among this set of simulations that resulted in equilibrium, higher costs were associated with more rapid establishment of equilibrium prevalence. The analysis also revealed several significant interaction effects. The combination of greater transmission probability and greater cost and background mortality increased time to equilibrium substantially. While the results were similar across transmission models, time to equilibrium was generally much greater in the spatial model than in the nonspatial model (Fig. 6).

We also ran a regression tree analysis to illustrate the effects of the parameters in Table 1 on the time to equilibrium, which was log-transformed for this analysis (Fig. 7). The resulting tree explained 64% of the variation in the time required for a cultural trait to reach equilibrium. Dispersal rate was found at the highest

Table 4
Significant parameter estimates in the log-linear model for time to equilibrium

Parameter	Estimate	SE	*P*
Intercept	7.52	0.22	<0.001
g	−1.68	0.26	<0.001
d	−1.21	0.25	<0.001
β	−1.56	0.27	<0.001
c	−1.23	0.33	<0.001
S (spatial vs nonspatial)	0.82	0.15	<0.001
*d**β	−0.47	0.23	0.042
β**m*	0.69	0.23	0.003
β**c*	1.38	0.33	<0.001
(27 others)			

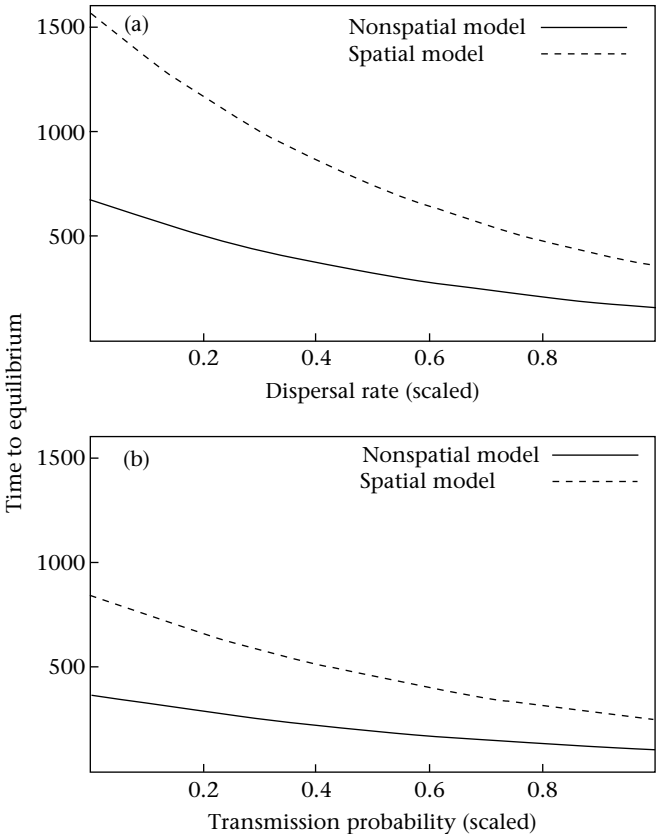

Figure 6. Modelled time to equilibrium by (a) dispersal rate and (b) probability of transmission (β). The plots show the effects of dispersal rate, transmission probability and spatial model on time to equilibrium. The other settings are at default values (see Methods).

node, as well as in lower parts of the regression tree; in all cases, higher rates of dispersal reduced the time required for a trait to reach equilibrium. Subsequent effects were different at low and high rates of dispersal. When the probability of dispersal was less than 0.0047, group size significantly influenced the time required for a trait to reach equilibrium; with group sizes less than 20.3, the time to equilibrium was predicted to be markedly higher (based on the log-transformed durations given on the tips of the tree). By contrast, at higher rates of dispersal, a nonspatial model resulted in a marked increase in the rate of trait spread at the population level (predicted values of 2.50 for local dispersal, versus 2.13 for random dispersal to any group). As expected, a higher transmission probability increased the rate of trait spread. However, prestige and consensus transmission again had no effects on cultural trait dynamics at the population level.

DISCUSSION

In animal societies, most social learning occurs among individuals within groups, and the same was likely to be true of prehistoric human populations. In socially structured populations, establishment of a cultural trait at the population level requires that the trait spread beyond a single social group, yet with few exceptions (e.g. Henrich & Boyd 1998; Boyd & Richerson 2002), most work on cultural transmission has focused on within-group dynamics. We investigated a set of transmission mechanisms, including biases due to prestige or consensus transmission, and social system parameters to determine which factors influence cultural dynamics in socially structured populations. Among the transmission parameters, the transmission probability (β) affected trait persistence and equilibrium levels of trait prevalence, with higher transmission probability resulting in higher prevalence (and also more rapid spread of the trait). Among the social system parameters, increased group size favoured the establishment of the trait and enhanced its spread, while increased mortality and trait costs increased the probability of trait extinction and reduced equilibrium prevalence. Remarkably, we found that transmission biases involving prestige or consensus effects had no significant effects on trait dynamics at the population level (although consensus transmission showed evidence for some weak effects on equilibrium prevalence). Another interesting result was that local dispersal slowed the rate of trait spread in the population, but had no significant effects on the probability of extinction or prevalence.

One conclusion from these analyses is that the effects of biased transmission involving consensus and prestige effects were minor relative to other factors. Biased transmission may have minor effects because cultural traits can spread rapidly within groups, and the mass-action effect of increasing the number of animals acting as performers may outweigh any minor adjustments in transmission probability caused by prestige or consensus mechanisms. We designed the simulation so that the mean rate of transmission would be approximately equal across the three transmission models that we used. We suggest that effects of biased transmission, if they exist, are weaker than other effects, such as the costliness of the cultural trait.

We acknowledge, however, that different implementation of the consensus and prestige models could alter this conclusion. Instead of our simple model of 'linear majority rules' for the consensus model, for example, the probability of transmission could have a different shape, including possibly a more rapid rise at lower prevalence, which could alter the dynamics to speed up trait spread and reduce extinction risk. Similarly, we assumed that the effect of dominance was linear, and that only the rank of the performer was relevant (rather than the difference in the ranks of observer and performer). If we assumed instead that dominants were also more likely to acquire beneficial traits (i.e. that a link exists between dominance rank and the acquisition of beneficial traits), this could impact cultural trait dynamics (e.g. Boyd & Richerson 1985; Boesch & Tomasello 1998; Henrich & Gil-White 2001; Henrich & McElreath 2003). Similarly, we might expect that migration is more likely by lower-ranking individuals. In such a case, the rate of trait spread could slow, as lower-ranking individuals moving into a new group would be less likely to be copied. Thus, our model provides a foundation for exploring the conditions under which prestige and consensus mechanisms have an impact on par with the effect of social system parameters.

An almost limitless set of cultural transmission mechanisms is possible (e.g. Laland 2004), and thus we were forced to select a small subset of key factors that might bias transmission (Boesch & Tomasello 1998; Henrich & McElreath 2003). We further aimed to implement these transmission models as simply as possible, for example, by using linear transformations of the probability of transmission based on dominance rank of culturally skilled individuals (prestige model) or the percentage of animals in the group that expressed the trait (consensus model). Future research could consider variants on these models, and also constraints. For example, there could be greater opportunities for transmitting traits within the sexes than between them (e.g. clothing fashions). Similarly, social groups themselves are often composed of networks of interactions involving kin, alliances and sexual partners, and some traits might be transmitted vertically from mother to offspring. Age effects might also be important, with transmission to an observer more likely during age-specific periods when learning

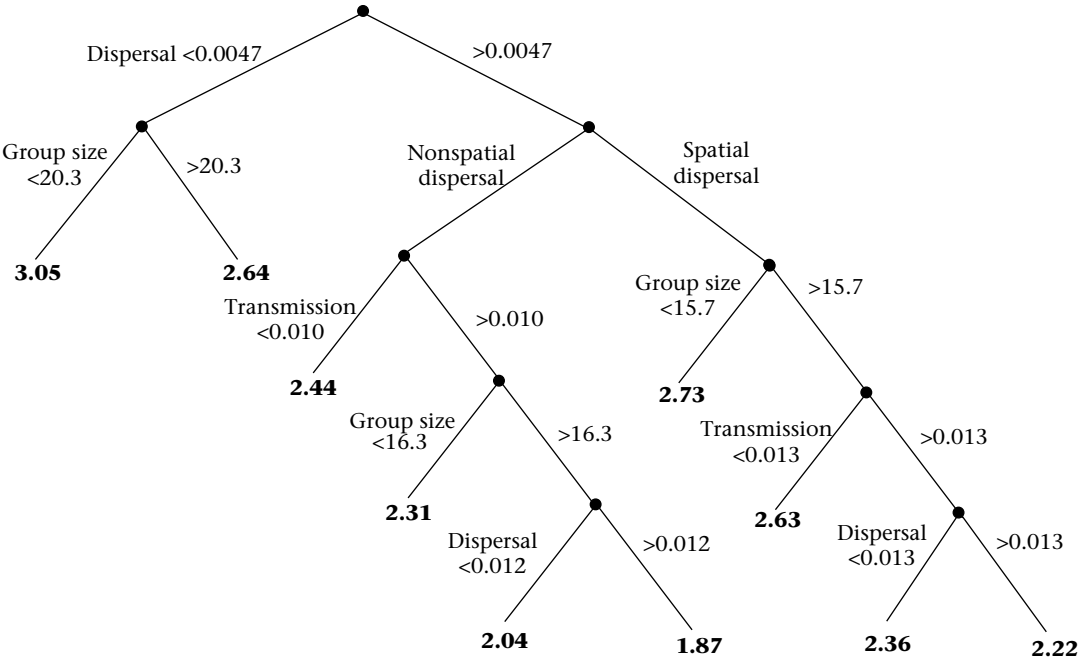

Figure 7. Regression tree for time to equilibrium. Time to equilibrium is log-transformed, with predicted values shown at the tips of the tree.

is more likely, or the behaviours themselves only expressed at a particular life stage; such effects would be expected to slow the spread of the cultural trait. It would also be interesting to investigate competition among traits that have different transmission mechanisms or benefits to individuals with the traits. Finally, it is worth keeping in mind that the prestige and consensus models are not mutually exclusive. Although we treated them separately here, it might be interesting to investigate their combined effects on cultural trait dynamics.

The social factors that we investigated have clear analogies to the spread of infectious disease in socially structured populations, particularly for costly cultural traits that can negatively impact fitness. Returning to the case of individuals copying dominants, for example, similar patterns can be found with sexually transmitted diseases (STDs). In epidemiological models of STDs in animals, more dominant individuals are more likely to be infected, and thus more likely to spread the disease (Thrall et al. 2000; Kokko et al. 2002). Similarly, disease spread can be impacted by group size, patterns of dispersal, and mortality rates (Anderson & May 1991; Wilson et al. 2003; Nunn & Altizer 2006).

However, important differences exist between the spread of cultural traits and infectious disease, particularly with regard to the selective benefits of many cultural traits (in comparison to costs usually associated with disease). As compared to disease transmission, for example, cultural evolution in socially structured populations is likely to set up a group selection scenario, in which advantageous cultural traits could lead to larger groups and higher rates of dispersal (Wilson 1983; Soltis et al. 1995). In addition, cultural traits tend to spread directly between individuals in close proximity, while infectious diseases can be transmitted indirectly (e.g. through vectors or contaminated soil). Lastly, innovation is possible in cultural systems, even if it is often 'primed' by previous innovations or cultural structures, whereas infectious diseases do not typically arise de novo in a population (although they could appear to do so when spillover from a reservoir host occurs, or when hybridization among pathogens opens up new hosts to exploit). In other words, you do not actually have to have direct contact with an 'infected' individual to get a good idea; individual learning can also play a role, and is ultimately responsible for the origin of cultural behaviours.

A beneficial cultural trait is expected to spread rapidly and reach high prevalence, and our simulations confirmed this expectation under a wide range of conditions. Advantageous behaviours are also likely to reduce the likelihood of group extinction, which could create opportunities for group selection in natural situations. On the other hand, one can easily think of cultural traits that are clearly not advantageous for survival, yet spread throughout populations. These are superficially similar to establishment of infectious diseases, which entail a cost to the host but still can reach a stable equilibrium. Our simulations suggest that costly cultural traits can spread widely, but as costs increase, the probability of extinction also increases.

The results of our analysis suggest that the explosion of cultural behaviours and variants in human evolution should have resulted when group size, contact between groups and the benefits of cultural traits increased. Many cultural traits in humans are technological. Hence, these traits would be likely to carry a very strong benefit, favouring their establishment. Second, the higher technological skills seen in human evolution, with the inclusion of many stone tools, could reasonably have led to a reduction in mortality rates. This would have favoured the further development of larger social groups, which, as we saw in our analysis, favour the establishment of cultural traits. Lastly, in comparison to other apes, early humans lived in more dispersed social groups, in much larger home ranges, and they maintained regular contact with more than their direct neighbours; these social groups probably had more contact with other groups as trade took place. Our results suggest that these contacts would have increased the rate at which cultural traits spread, and might have reduced the probability that they went extinct.

To conclude, it is useful to return to the four questions that we posed in the Introduction. The simulations revealed that local dispersal increases the time required for a trait to reach equilibrium (Question 1) and that cultural traits are buffered from extinction in larger groups (Question 2). We also found that higher rates of dispersal increase the rate of trait spread in the population, with weaker effects (approaching significance) on the probability of

extinction. In terms of mortality, we found that mortality rates affect cultural dynamics, including through effects of the cultural trait on mortality itself (Question 3). Thus, higher costs of the trait and higher background mortality increase extinction probability and reduce the prevalence of the trait. Lastly, we found that the rate of transmission affects all of the outcome variables that we examined, but that transmission mechanisms involving prestige or consensus had no statistically discernible effects on trait dynamics (Question 4). As noted above, this conclusion could be sensitive to how prestige and consensus transmission were implemented, and therefore should be explored further in future research. Along similar lines, it would be interesting to explore other transmission mechanisms that might influence the spread of traits among contact networks within groups, including vertical transmission, sex- and age-specific transmission, and patterns of kinship.

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References

Anderson, R. M. & May, R. M. 1979. Population biology of infectious diseases: part 1. *Nature*, **280**, 361–367.

Anderson, R. M. & May, R. M. 1981. The population-dynamics of micro-parasites and their invertebrate hosts. *Philosophical Transactions of the Royal Society of London, Series B*, **291**, 451–524.

Anderson, R. M. & May, R. M. 1991. *Infectious Diseases of Humans: Dynamics and Control*. Oxford: Oxford University Press.

Blower, S. M. & Dowlatabadi, H. 1994. Sensitivity and uncertainty analysis of complex-models of disease transmission: an HIV model, as an example. *International Statistical Review*, **62**, 229–243.

Boesch, C. 2003. Is culture a golden barrier between human and chimpanzee? *Evolutionary Anthropology*, **12**, 82–91.

Boesch, C. & Boesch-Achermann, H. 2000. *The Chimpanzees of the Tai Forest*. Oxford: Oxford University Press.

Boesch, C. & Tomasello, M. 1998. Chimpanzee and human cultures. *Current Anthropology*, **39**, 591–614.

Boesch, C., Marchesi, P., Marchesi, N., Fruth, B. & Joulian, F. 1994. Is nut cracking in wild chimpanzees a cultural behaviour? *Journal of Human Evolution*, **26**, 325–338.

Boots, M. & Sasaki, A. 1999. 'Small worlds' and the evolution of virulence: infection occurs locally and at a distance. *Proceedings of the Royal Society of London, Series B*, **266**, 1933–1938.

Boyd, R. & Richerson, P. J. 1985. *Culture and the Evolutionary Process*. Chicago: University of Chicago Press.

Boyd, R. & Richerson, P. J. 2002. Group beneficial norms can spread rapidly in a structured population. *Journal of Theoretical Biology*, **215**, 287–296.

Carlsson-Graner, U. & Thrall, P. H. 2002. The spatial distribution of plant populations, disease dynamics and evolution of resistance. *Oikos*, **97**, 97–110.

De'ath, G. & Fabricius, K. E. 2000. Classification and regression trees: a powerful yet simple technique for ecological data analysis. *Ecology*, **81**, 3178–3192.

Franz, M. & Nunn, C. L. 2009. Network-based diffusion analysis: a new method for detecting social learning. Proceedings of the Royal Society of London. *Series B*, **276**, 1829–1836.

Gandon, S., Capowiez, Y., Dubois, Y., Michalakis, Y. & Olivieri, I. 1996. Local adaptation and gene-for-gene coevolution in a metapopulation model. *Proceedings of the Royal Society of London Series B-Biological Sciences*, **263**, 1003–1009.

Gordon, R. G. 2005. *Ethnologue: Languages of the World*, 15th edn. Dallas, Texas: SIL International. http://www.ethnologue.com/.

Grimm, V. & Railsback, S. F. 2005. *Individual-based Modeling and Ecology*. Princeton, New Jersey: Princeton University Press.

Henrich, J. & Boyd, R. 1998. The evolution of conformist transmission and the emergence of between-group differences. *Evolution and Human Behavior*, **19**, 215–241.

Henrich, J. & Gil-White, F. J. 2001. The evolution of prestige: freely conferred deference as a mechanism for enhancing the benefits of cultural transmission. *Evolution and Human Behavior*, **22**, 165–196.

Henrich, J. & McElreath, R. 2003. The evolution of cultural evolution. *Evolutionary Anthropology*, **12**, 123–135.

Hunt, G. R. & Gray, R. D. 2003. Diversification and cumulative evolution in New Caledonian crow tool manufacture. *Proceedings of the Royal Society of London, Series B*, **270**, 867–874.

Kawai, M. 1965. Newly acquired pre-cultural behavior of the natural troop of Japanese monkeys on Koshima Islet. *Primates*, **6**, 1–30.

Kawamura, S. 1959. The process of sub-culture propagation among Japanese monkeys. *Primates*, **2**, 43–60.

Kokko, H., Ranta, E., Ruxton, G. & Lundberg, P. 2002. Sexually transmitted disease and the evolution of mating systems. *Evolution*, **56**, 1091–1100.

Laland, K. N. 2004. Social learning strategies. *Learning & Behavior*, **32**, 4–14.

Leca, J. B., Gunst, N. & Huffman, M. A. 2007. Japanese macaque cultures: inter- and intra-troop behavioural variability of stone handling patterns across 10 troops. *Behaviour*, **144**, 251–281.

McCullagh, P. & Nelder, J. 1989. *Generalized Linear Models*. London: Chapman & Hall.

May, R. M. & Anderson, R. M. 1979. Population biology of infectious diseases: part II. *Nature*, **280**, 455–461.

Mesoudi, A., Whiten, A. & Laland, K. N. 2004. Perspective: is human cultural evolution Darwinian? Evidence reviewed from the perspective of *The Origin of Species*. *Evolution*, **58**, 1–11.

Nunn, C. L. & Altizer, S. M. 2006. *Infectious Diseases in Primates: Behavior, Ecology and Evolution*. Oxford: Oxford University Press.

Nunn, C. L., Thrall, P. H., Harcourt, A. H. & Stewart, K. 2008. Emerging infectious diseases and animal social systems. *Evolutionary Ecology*, **22**, 519–543.

O'Keefe, K. J. & Antonovics, J. 2002. Playing by different rules: the evolution of virulence in sterilizing pathogens. *American Naturalist*, **159**, 597–605.

van der Post, D. J. & Hogeweg, P. 2006. Resource distributions and diet development by trial-and-error learning. *Behavioral Ecology and Sociobiology*, **61**, 65–80.

van der Post, D. J. & Hogeweg, P. 2008. Diet traditions and cumulative cultural processes as side-effects of grouping. *Animal Behaviour*, **75**, 133–144.

Roff, D. A. & Roff, R. J. 2003. Of rats and Maoris: a novel method for the analysis of patterns of extinction in the New Zealand avifauna before European contact. *Evolutionary Ecology Research*, **5**, 759–779.

Roy, B. A. & Kirchner, J. W. 2000. Evolutionary dynamics of pathogen resistance and tolerance. *Evolution*, **54**, 51–63.

Rushton, S. P., Lurz, P. W. W., Gurnell, J. & Fuller, R. 2000. Modelling the spatial dynamics of parapoxvirus disease in red and grey squirrels: a possible cause of the decline in the red squirrel in the UK? *Journal of Applied Ecology*, **37**, 997–1012.

van Schaik, C. P. & Pradhan, G. R. 2003. A model for tool-use traditions in primates: implications for the coevolution of culture and cognition. *Journal of Human Evolution*, **44**, 645–664.

van Schaik, C. P., Deaner, R. O. & Merrill, M. Y. 1999. The conditions for tool use in primates: implications for the evolution of material culture. *Journal of Human Evolution*, **36**, 719–741.

Seaholm, S. K., Ackerman, E. & Wu, S. C. 1988. Latin hypercube sampling and the sensitivity analysis of a Monte-Carlo epidemic model. *International Journal of Bio-Medical Computing*, **23**, 97–112.

Soltis, J., Boyd, R. & Richerson, P. J. 1995. Can group-functional behaviors evolve by cultural group selection? An empirical test. *Current Anthropology*, **36**, 473–494.

Thrall, P. H. & Antonovics, J. 1995. Theoretical and empirical studies of metapopulations: population and genetic dynamics of the Silene-Ustilago system. *Canadian Journal of Botany, Supplement*, **73**, S1249–S1258.

Thrall, P. H., Antonovics, J. & Dobson, A. P. 2000. Sexually transmitted diseases in polygynous mating systems: prevalence and impact on reproductive success. *Proceeding of the Royal Society of London, Series B*, **267**, 1555–1563.

Wilson, D. S. 1983. The group selection controversy: history and current status. *Annual Review of Ecology and Systematics*, **14**, 159–187.

Wilson, K., Knell, R., Boots, M. & Koch-Osborne, J. 2003. Group living and investment in immune defence: an interspecific analysis. *Journal of Animal Ecology*, **72**, 133–143.