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Evolution of cultural communication systems: the coevolution of cultural signals and genes encoding learning preferences

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Abstract

In several communication systems that rely on social learning, such as bird song, and possibly human language, the range of signals that can be learned is limited by perceptual biases – predispositions – that are presumably based on genes. In this paper, we examine the coevolution of such genes with the culturally transmitted communication traits themselves, using deterministic population genetic models. We argue that examining how restrictive genetic predispositions are is a useful way of examining the evolutionary origin and maintenance of learning. Under neutral cultural evolution, where no cultural trait has any inherent advantage over another, there is selection in favour of less restrictive genes (genes that allow a wider range of signals to recognized). In contrast, cultural conformity (where the most common cultural trait is favoured) leads to selection in favour of more restrictive genes.

Introduction

Cultural communication systems arise when signals are acquired by social learning between individuals. The most familiar examples of this are song learning by oscine birds (see Catchpole & Slater, 1995) and human language (see Deacon, 1997). There has been continued interest in the evolution of these types of communication (e.g. Pinker, 1994; Deacon, 1997; Nowicki et al. 1998). However, only a few studies (Cavalli-Sforza & Feldman, 1983; Feldman and Cavalli-Sforza 1984; Aoki & Feldman, 1987, 1989, 1991; Aoki, 1989; Feldman & Aoki, 1992) have taken into account cultural evolution.

The coevolutionary relationship of genetic and vertical cultural transmission has been explored in a number of general models (Feldman & Zhivotovsky, 1992). These models have extended the techniques of theoretical population genetics to cultural transmission, drawing on the analogies between the two forms of transmission. The significance of cultural transmission for the study of human behavioural diversity has been highlighted in a number of anthropological studies (e.g. Cavalli-Sforza et al. 1982; Durham, 1991) as well as in studies of animal behaviour (e.g. Kawai, 1965; Galef, 1988; Laland & Plotkin, 1990; Laland *et al.*, 1995; Lynch, 1996; Payne, 1996). Gene–culture coevolution has been implicated in the evolution of various human genetic traits, including adult lactose tolerance (Feldman and Cavalli-Sforza 1989) and sickle cell anaemia (Durham, 1991).

One key area of interest is the evolution of cultural transmission itself. Here, control of the phenotype is shifted from genes towards culture. Two principal investigations into this transition have focused on the ability of the two forms of transmission to follow environmental change. Boyd & Richerson (1985, 1988 found that culture did prove more effective at tracking environmental change, but the environments had to be sufficiently predictable. Feldman *et al.* (1996) used a different model to demonstrate that the greater the probability of an environmental change, the more difficult it is for social learning to evolve. In a constant environment, Cavalli-Sforza & Feldman (1983) found that genetic transmission out-competed most modes of cultural transmission (except 'one to many' transmission) for an adaptive trait, because of the kin clustering that is entailed by the former.

Song learning by birds is a famous example of cultural transmission in nonhuman animals (Thrope, 1958, see

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Catchpole & Slater, 1995; Kroodsma & Miller, 1996 for recent reviews). The mode of transmission is most often oblique (from a member of the parental generation other than the parent), although some cases of vertical transmission (parent to offspring) have been discovered, and in most temperate species, only males sing (Catchpole & Slater, 1995; Grant & Grant, 1996). However, other empirical findings point to the role of genes in bird song learning. For example, numerous studies have demonstrated how these learned songs tend to be species-specific, and how species respond to playback of their own species' song. Such species specificity could be a result of learning, but if individuals are hand-reared in acoustic isolation from a very young age, and then exposed to tapes of bird song, they still prefer to learn to sing conspecific song (e.g. Marler & Sherman, 1985; Mundinger, 1995). This bias could be because of limitations in production – birds might be physiologically limited in the range of sounds they can produce. However, the evidence does not support this entirely, as demonstrated by experiments with young, hand-reared birds that have not started to sing themselves. Several studies have found that such individuals respond more actively to conspecific song than to heterospecific song (Dooling & Searcy, 1980; Whaling *et al.*, 1997; Nelson, 2000). Putting these empirical data together, it has been suggested (e.g. Marler, 1997) that song learning birds possess a genetically based perceptual bias or predisposition that causes them to recognize conspecific song in a different way from other sounds.

However, within species-typical limits, cultural evolution in bird song has been most successfully fitted to models analogous to genetic drift. Thus Lynch (1996) found that song types are selectively neutral with respect to each other in a range of species, and analogous models have been used to estimate the 'mutation rate' (Ince & Slater, 1980; Lachlan & Slater, in press) or turnover rate (Payne, 1996) of song types.

Together, these data suggest that, on the one hand, for any one species there is a (normally large) number of different song types that have equal efficacy in carrying out their communicative function. Cultural evolution of songs lying within this range is approximately neutral. On the other hand, there also exist genetically transmitted perceptual biases that impose limits, beyond which songs are no longer recognized by conspecifics. Thus songs outside these limits are culturally selected against (they are not learned). Moreover the genes responsible for the perceptual biases may themselves be subject to selection.

Two communicative functions of song, repelling competing males and attracting mates, appear to apply in many species. However, it is not clear why learning is required to achieve these goals. Moreover, while song learning within the oscines appears ubiquitous (Kroodsma & Baylis, 1982), the great variation in song learning strategies would appear to preclude general functional explanations. Many hypotheses focus on putative benefits to singing locally adaptive song types (Kroodsma, 1996). For example, song sharing with neighbours has been correlated with breeding success (Payne et al. 1988; Beecher *et al.*, 2000), perhaps because of improved neighbour recognition.

The existing theoretical frameworks for exploring the evolution of culture are not adequate to explain the maintenance of a communicative trait such as song learning. The hypothesis that song learning might lead to songs that track environmental variation in transmission more efficiently (Hansen, 1979) is only occasionally supported by empirical data (Catchpole & Slater, 1995). And the apparent selective neutrality of alternative song types, within the species-typical limits, suggests that results from models of the evolution of an adaptive trait may not be appropriate to bird song variation.

While differing enormously in many respects, human language has several important features in common with bird song (Doupe & Kuhl, 1999). Of particular relevance here, language is a culturally transmitted trait, with possible genetically based biases. The exact nature of these biases is not known, but some of them may act in a similar way to those involved in bird song. For example, pre-linguistic, neonatal infants exhibit categorical perception of phonemes (Eimas et al. 1987) – in other words, they divide sounds into categories which from an acoustic point of view seem arbitrary. The fact that infants from different cultures do not vary in their perception, while adults do, indicates how genetic and cultural sources of information may be important for the development of language. A second feature in common is that alternative bird song types or human languages both appear to be selectively neutral. One language does not seem to have any great inherent advantage over others.

In this paper, we examine the evolution of genetic biases that influence the learning of cultural signals. The genetic biases are defined as genes that determine the range of signals that individuals recognize and learn. In other words, they impose the limits within which songs are equally well recognized, but outside of which, they are no longer recognized. Importantly, we assume in this paper that the same genes are responsible for the recognition of songs by receivers, and the recognition of songs by individuals that are learning songs for themselves. This seems reasonable to us in some situations, where signallers are also receivers. Here the tasks of recognition are so similar that it seems unlikely that different sets of genes are used. In other situations, where the signallers are males and receivers females, empirical observations support our assumption. In songbird species where females do not sing, females possess similar brain structures to males, use those structures in similar ways, and can be induced to sing learned songs simply by injecting hormones. In other words, the neural organization of song circuits in female songbirds seems homologous to those of males.

This paper describes deterministic evolutionary models of the interaction between genetic biases and culturally transmitted signals. While the signals themselves are selectively neutral as long as they are recognized by others, the limits to recognition are subject to selection. We examine the conditions under which more or less restrictive genetic biases might evolve. For learning to be replaced by genetic transmission of a trait, we follow Lachlan & Slater (1999) in suggesting that selection for more restrictive genetic biases is necessary; and conversely, for learning to evolve from a genetically transmitted trait, selection for less restrictive genetic biases must occur.

In contrast to the models of Aoki and Feldman (Aoki & Feldman, 1987, 1989, 1991; Aoki, 1989; Feldman & Aoki, 1992), we assume that fitness differences between phenotypes only arise through the effect of songs on the outcome of interactions between individuals. There is little evidence that certain song types are intrinsically more successful than others. In our models, cultural transmission is determined by the frequencies of alternative signal types, and by random processes of cultural mutation. Our aim here is to model the neutral cultural evolution of alternative traits within species–typical limits. Our models are similar in some respect to the simulation studies of Lachlan & Slater (1999), but are more general in structure. The use of analytical models requires some simplification of the underlying processes described by the simulation models, but it permits generalizations to be made about the properties of the models over a wider range of parameter values.

Model 1: Inter-sexual interactions

This model investigates effects of mate choice on the gene–culture coevolution of song. It assumes that mate choices are made by females, and that their preferences are based on the same genetic bias that controls song learning by males.

Consider a population with two alleles, *A* and *a*, which affect a bias to learn and recognize culturally transmitted traits. There are two cultural traits: 1 and 2. These alternative cultural traits might correspond to two individual signal types (for example two song types in birds), or they might each correspond to a range of signal types (for example, chaffinch song types with or without terminal 'flourishes'). For reasons of brevity, they shall be referred to as 'songs' in the remainder of this paper. Only males learn songs. Males with allele *A* can learn song 1 only, whereas males with allele *a* can learn either song 1 or song 2, but any individual can only learn one song. The alleles also influence the mating preferences of females, in a similar way to their influence on song learning. Females with allele *A* prefer song 1, whereas females with allele *a* do not have a preference for one song or the other. The strength of this preference is measured using a parameter *s*; females with *A* discriminate against males singing 2 by the factor $(1 - s)$. The transmission of songs is oblique. Males sample the whole population of their parents' generation, and choose which song to learn using the frequency of songs in the parental generation, but are biased according to their allele: males with *A* are unable to learn song 2. After a song is learned, cultural mutation of song 1 into song 2 occurs with frequency μ_1, and cultural mutation of song 2 into song 1 occurs with frequency μ_2. The sex ratio at birth is even, and the alleles *A* and *a* are equally likely to be transmitted from an A × a mating. Given this scenario, six types of phenogenotypes (combinations of cultural traits and alleles) can be defined: *A1* (male), *A2* (male) – which does not exist in practice as allele *A* will not learn song type 2, *a1* (male), *a2* (male), *A* (female), and *a* (female). The frequencies of these phenogenotypes are defined as x_1, x_2 (which is by definition always 0), x_3, x_4, x_5 and x_6 respectively. We have assumed that individuals in the model are haploid. Haploidy simplifies the analysis, but the results should be similar to those for diploids at least in the case of additive directional selection (this has been found for similar gene–culture coevolutionary models – e.g. Cavalli-Sforza & Feldman, 1983; and applies particularly in the absence of dominance).

In general, the aim of our analysis of this model is to find out whether a more restrictive bias (*A*) would be replaced by a less restrictive one (*a*). The first step in this process is to describe how evolution proceeds in the model. This is done by means of a set of recursion equations, which simply show the frequencies of phenogenotypes in one generation (denoted by ′) as functions of the phenogenotype frequencies in the previous one, incorporating the assumptions described above. The recursion equations are:

$$Dx_1' = \frac{x_1x_6 + 2x_1x_5 + x_3x_5 + (1-s)x_4x_5}{4} \tag{1a}$$

$$\begin{aligned} D(x_1 + x_3 + x_4)x_3' &= \{[(1-\mu_1)(x_1 + x_3) + \mu_2x_4][x_1x_6 + 2x_3x_6 \\ &\quad + x_3x_5 + 2x_4x_6 + (1-s)x_4x_5]\}/4 \end{aligned} \tag{1b}$$

$$\begin{aligned} D(x_1 + x_3 + x_4)x_4' &= \{[\mu_1(x_1 + x_3) + (1-\mu_2)x_4][x_1x_6 + 2x_3x_6 \\ &\quad + x_3x_5 + 2x_4x_6 + (1-s)x_4x_5]\}/4 \end{aligned} \tag{1c}$$

$$Dx_5' = \frac{x_1x_6 + 2x_1x_5 + x_3x_5 + (1-s)x_4x_5}{4} \tag{1d}$$

$$Dx_6' = \frac{x_1x_6 + 2x_3x_6 + x_3x_5 + 2x_4x_6 + (1-s)x_4x_5}{4}, \tag{1e}$$

where

$$D = x_1x_5 + x_1x_6 + x_3x_5 + x_3x_6 + x_4x_6 + (1-s)x_4x_5$$

In these recursions, $4D$, the sum of the right-hand sides of eqns 1a–e, is a normalizer, that includes genetic transmission and an even distribution of offspring into males and females. A similar convention is used in eqns 1b and 1c, where the sum of male allele frequencies is required, because which song is learned by a male depends on the frequencies of songs sung in the population (and the first term on the right-hand side of these two equations also refers to cultural transmission). Note that $x_1 = x_5$ and $x_3 + x_4 = x_6$ for all generations after the first.

Under our assumptions, the sex ratio remains even: $x_1 + x_3 + x_4 = x_5 + x_6 = \frac{1}{2}$, and $D = \frac{1}{4} - sx_4x_5$. Comparing eqns 1a with 1d, and 1e with the sum of 1b and 1c, we see that the frequency of A is $2x_1$ and that of a is $2x_6$. Therefore, from eqns 1a and 1e,

$$\left|x_1' - x_6'\right| = \left|\frac{x_1 - x_6}{1 - 4sx_4x_5}\right|. \tag{2}$$

Therefore, if $s > 0$ and $a2$ males and A females are present, the frequencies of a and A diverge. As a result, there can be no stable equilibria where both alleles occur. This means that evolution in this model will always result in either A or a becoming extinct. A situation where both more and less restrictive biases are present simultaneously can never be stable.

The next step in our analysis was to explore the behaviour of the model around the equilibrium points, where either one of the alleles was absent. The aim here is to establish how difficult it would be for one allele to invade a population consisting entirely of the other. First, it is necessary to describe the phenogenotype frequencies that occur at the equilibria. When A is absent, i.e. $x_3 + x_4 + x_6 = 1$, these are:

$$\hat{x}_1 = 0, \quad \hat{x}_3 = \frac{\mu_2}{2(\mu_1 + \mu_2)}, \quad \hat{x} = \frac{\mu_1}{2(\mu_1 + \mu_2)},$$
$$\hat{x}_5 = 0, \quad \hat{x}_6 = \frac{1}{2}. \tag{3}$$

The local stability of this equilibrium to the introduction of allele A was tested by introducing a small amount of allele A near the equilibrium (eqn 3) by setting x_1 and x_5 to the values ε_1 and ε_5, which are small enough to ignore terms of second order and higher. After making these perturbations, we tested whether the recursions described in (eqn 1) brought the phenogenotype frequencies back towards the equilibrium values in (eqn 3). The resulting linear system is:

$$\varepsilon_1 = \frac{1}{2}\left[\varepsilon_1 + \frac{\mu_2 + (1-s)\mu_1}{\mu_2 + \mu_1} \cdot \varepsilon_5\right], \tag{4a}$$

$$\varepsilon_5 = \frac{1}{2}\left[\varepsilon_1 + \frac{\mu_2 + (1-s)\mu_1}{\mu_2 + \mu_1} \cdot \varepsilon_5\right]. \tag{4b}$$

The leading eigenvalue of (eqn 4) is:

$$\lambda = 1 - \frac{s\mu_1}{2(\mu_1 + \mu_2)}. \tag{5}$$

The magnitude of the eigenvalue determines the local stability of the equilibrium; with all eigenvalues <1 in magnitude the equilibrium is stable (that is, if perturbed, it will be returned to), and with any eigenvalue value >1 in magnitude, it will be unstable. Hence, this equilibrium will be locally stable as long as s,μ_1, and μ_2 all have positive values, in which case A cannot invade the population fixed on a.

At the other extreme, when a is absent, the equilibrium phenogenotype frequencies are:

$$\hat{x}_1 = \frac{1}{2}, \quad \hat{x}_3 = 0, \quad \hat{x}_4 = 0, \quad \hat{x}_5 = \frac{1}{2}, \quad \hat{x}_6 = 0. \tag{6}$$

Using the same analysis as before to examine the stability of this equilibrium, the corresponding linear system is:

$$\varepsilon_3 = \frac{(1-\mu_1)[\varepsilon_3 + (1-s)\varepsilon_4 + \varepsilon_6]}{2} \tag{7a}$$

$$\varepsilon_4 = \frac{\mu_1[\varepsilon_3 + (1-s)\varepsilon_4 + \varepsilon_6]}{2} \tag{7b}$$

$$\varepsilon_6 = \frac{\varepsilon_3 + (1-s)\varepsilon_4 + \varepsilon_6}{2}, \tag{7c}$$

and the leading eigenvalue is:

$$\lambda = 1 - \frac{s\mu_1}{2}. \tag{8}$$

Therefore this equilibrium will also always be locally stable, and a cannot invade from fixation on A.

To summarize, both allelic fixation states are stable. However, if the two leading eigenvalues are compared, we see that they are not the same. In particular as long as $\mu_1 + \mu_2 < 1$ (e.g. the mutation rates are both less than 0.5 – a very likely situation), the eigenvalue in eqn 5 will be smaller that that in eqn 8. This means that if the population is fixed on a, and is then perturbed by adding a small amount of A, the model will return to its equilibrium position more rapidly than if the population were fixed on A and then perturbed by adding a small amount of a (for shorthand, we refer in this case to allele a being 'more stable' than allele A). The relevance of this finding is only apparent if we consider the effect of random factors on the model. The domains of attraction for the two allelic fixations are symmetric. However, it might be expected that repeated perturbations from either fixation might eventually lead to the system switching from one equilibrium to the other. The eigenvalues indicate that it should be more difficult for random changes to allow A, the more restrictive allele, to invade a population consisting only of a individuals than *vice versa*.

We tested this prediction by carrying out a series of numerical iterations of the recursions (eqn 1), incorporating random changes in the allele frequencies. Initial starting conditions were the fixation states. Perturbations in the allele frequencies were added at randomly chosen generations (mean probability of a perturbation happening in a given generation: 0.1). A second random factor decided whether the perturbation would lead to an increase in the frequency of A or a. The amount of the perturbation was 0.05. The perturbations were divided between the phenogenotypes according to their frequencies. After 100 generations, the change in allele frequency between two consecutive generations was calculated. If the change in the frequency of the more common allele was less than 0.00001%, the simulation was ended. A total of 10 000 replicates were carried out, starting from both stable equilibria, and the number of times one equilibrium was replaced with the other was recorded. A number of different parameter settings were investigated.

Figure 1 shows the results of the simulation. As predicted, the A equilibrium was more likely to switch to the a equilibrium than vice versa, under all conditions examined, except when either s or μ_1 was very small. This is to be expected from our local stability analysis: as these values become very small, the two eigenvalues both approach one. Cultural mutation rates of around 0.01 appeared to provide the largest difference in the success of the two alleles – and the size of μ_1 (from song 1 to song 2) seemed to have a larger effect on the outcome of the simulation than did μ_2 (from song 2 to song 1).

Our analysis suggests that less restrictive genetic biases are favoured in a neutral model of cultural evolution. Random perturbations in allele frequencies result in the system tending towards fixation of allele a, because of differences in the stability of the fixation states. In the model, fitness costs only arise from the interaction between females with allele A (narrow filters) and males with allele $a2$ (wide filters, and unusual songs). These are the two classes of individuals that mate at lower than expected frequencies, as they are mutually incompatible. Hence the proportion of song type 2 within a males is crucial. When a is rare, type 2 can only arise as a direct result of cultural mutation, but when it is common, a higher frequency arises because of cultural evolution, and this facilitates selection against A with the result that fixation on a is more stable.

Model 2: Intra-sexual interactions

Model 1 entails that only one sex signals, and that the other receives, and that signalling affects the probability of mating. This is a rather limited circumstance, and does not include phenomena such as intra-sexual vocal communication between (typically male) songbirds. Therefore, in our next model, we examine a system in which all individuals possess a cultural signal, and in which there are costs that directly affect the probability of reproductive success.

One type of cost is applied when receivers do not recognize the signal because of their predisposition (as in Model 1). As an example of how this might act, consider two neighbouring male songbirds. Through singing, territorial boundaries are easily maintained, but if one of the males did not recognize the other as a conspecific (because he possessed a narrower perceptual bias), then one might speculate that costly fights would break out more frequently. On average, this type of fight would be expected to be equally costly to both individuals.

A second type of cost may result when two communicating individuals do not possess exactly the same cultural trait. Cultural conformity (Boyd & Richerson, 1985) has been found to be beneficial in several studies of songbird vocal communication (e.g. Payne et al. 1988; Beecher *et al.*, 2000). A similar effect is clearly of importance in human communication, where sharing of vocal cultural traits is necessary for language-based communication, and cultural conformity has been shown to be important in sociolinguistic studies (Chambers, 1995).

Model 2 examines how these different costs affect the evolution of more or less restrictive genetic predispositions. As in Model 1, there are two alleles: a and A, and two songs: 1 and 2. Allele A does not recognize song 2. Combinations of these give rise to three phenogenotypes: $A1$, $a1$, and $a2$, which, in keeping with the notation of Model 1, have frequencies: x_1, x_3, and x_4. Again, cultural transmission is oblique. Songs are copied at random from within the whole population, and cultural mutation between songs 1 and 2 again occurs at rates μ_1 and μ_2. Following transmission is a communication phase during which two fitness costs may be incurred: t is a cost imposed (equally to both signaller and receiver) when two individuals do not possess the same song, while s is a cost imposed (also equally to both signaller and receiver) when the receiver does not recognize the song at all. The latter occurs when an $A1$ individual meets an $a2$ individual, and is analogous to the cost s in Model 1. The recursion equations for this system are given in eqn 9.

In the recursions there are two stages. In the first stage, songs are acquired through the processes of cultural transmission and mutation. In the second stage, individuals communicate, interacting at frequencies proportional to their phenogenotype frequencies, and costs arise from failure to communicate. Here, the denominator W serves the same role as D in Model 1.

Transmission phase

$$x_1' = x_1 \tag{9a}$$

$$x_3' = (x_3 + x_4)(x_3 + x_1)(1 - \mu_1) + (x_3 + x_4)x_4\mu_2 \tag{9b}$$

$$x_4' = (x_3 + x_4)(x_3 + x_1)\mu_1 + (x_3 + x_4)x_4(1 - \mu_2) \tag{9c}$$

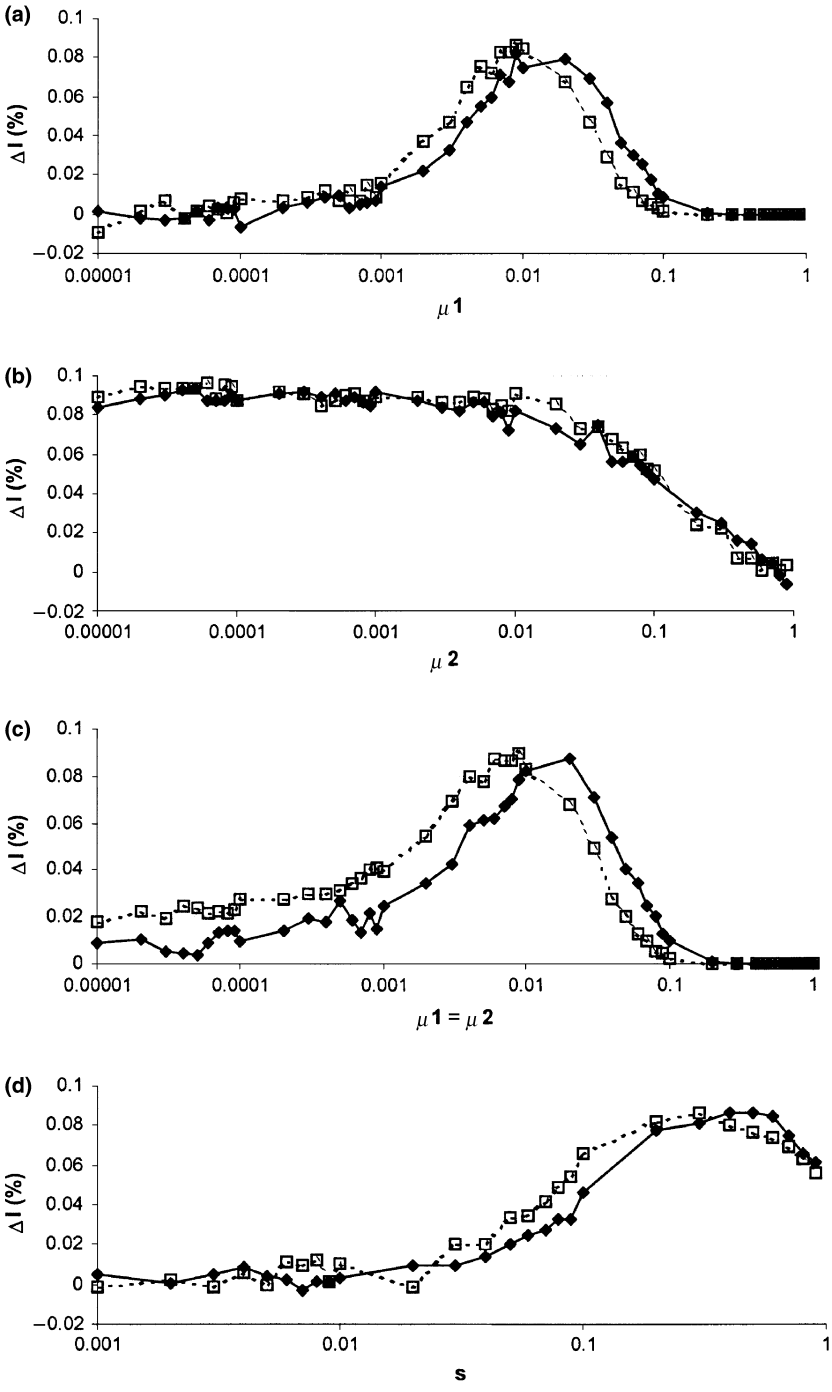

Fig. 1 Results of random walk simulations from the stable equilibria found in Models 1 and 3. ΔI represents the proportion of simulations in which allele *a* invaded, starting from the equilibrium where *A* was fixed minus the percentage of simulations in which allele *A* invaded, starting from the equilibrium where *a* was fixed. Each graph shows the results of varying one parameter value for both Model 1 (oblique transmission, diamonds and solid lines) and Model 3 (vertical transmission, squares and dashed lines). If not otherwise specified, the values of s,μ_1, and μ_2 were set to 0.25, 0.01, and 0.01 respectively.

communication phase

$$Wx_1'' = x_1'(x_3' + x_1') + x_4'x_1'(1 - t - s), \qquad (9d)$$

$$Wx_3'' = x_3'(x_1' + x_3') + x_3'x_4'(1 - t), \qquad (9e)$$

$$Wx_4'' = x_4'^2 + x_3'x_4'(1 - t) + x_4'x_1'(1 - t - s), \qquad (9f)$$

where

$$W = 1 - 2tx_3'x_4' - 2(t + s)x_4'x_1'.$$

Again, the initial step of our analysis is to determine whether any stable internal equilibria exist. From eqns 9a–f:

$$x_3'' + x_4'' - x_1'' = \frac{x_3 + x_4 - x_1 - 2tx_3'x_4'}{1 - 2tx_3'x_4' - 2(t+s)x_1'x_4'}. \qquad (10)$$

Note that if $x_1 > x_3 + x_4$, then $x_1'' > x_1$. Hence, from any starting condition with the A allele frequency higher than that of a, the frequency of A will increase to one. However, if $x_1 = x_3 + x_4$, then in the next generation $x_1'' > x_3'' + x_4''$ if $t > 0$ and $A1$, $a1$, $a2$ are all present. Thus the range of initial A-allele frequencies that result in fixation on A is greater than (0.5, 1.0). From eqn 10, at an internal equilibrium we would have

$$[-2tx_3'x_4' - 2(t+s)x_1'x_4'](x_3 + x_4 - x_1) = -2tx_3'x_4'.$$

As t, $s > 0$, at an internal equilibrium, this would entail $x_3 + x_4 > x_1$. This means that the range of phenogenotype frequencies from which evolution would result in extinction of A is smaller than the range for which a would become extinct.

When $t = 0$ and $s > 0$, $|x_3 + x_4 - x_1|$ increases over time, which is not surprising, as these conditions are directly analogous to those in Model 1. However, this may not be the case when $t > 0$. In this case we were unable to prove analytically that there were no stable internal equilibria. To investigate this, we instead carried out numerical iterations of the recursion system (eqn 9). For each setting of the parameters (t, s, μ_1 and μ_2), 40 starting frequencies of x_1 were used, with $1 - x_1$ divided into a set of 40 frequencies of x_3 and x_4. These starting frequencies were spread uniformly between 0 and 1. The iterations proceeded until frequencies approached within 0.0001 of fixation for one of the alleles, or changed by a factor of less than 1.001 from one generation to the next.

The results of the simulation confirm first that there are no stable internal equilibria for any starting frequency or parameter setting in the model. The second finding confirms that the domain of attraction for fixation on A is always larger than that for a when $t > 0$. This is illustrated in Fig. 2, which shows the minimum starting frequency of x_1 that leads to fixation of allele A.

The next stage of our analysis was to explore the properties of the equilibria that occurred when one or other allele was extinct. Near fixation of A, the model is rather similar to Model 1. The principle eigenvalue of the local linear system is:

$$\lambda = 1 - \mu_1(t+s). \qquad (11)$$

As with Model 1, this suggests that when A is at fixation, the equilibrium is stable to invasion by a. When a is fixed, however, the model becomes more complex. There are three possible equilibria along this edge, given by the solutions to the cubic equation:

$$ax_3^3 + bx_3^2 + cx_3 + d = 0, \qquad (12)$$

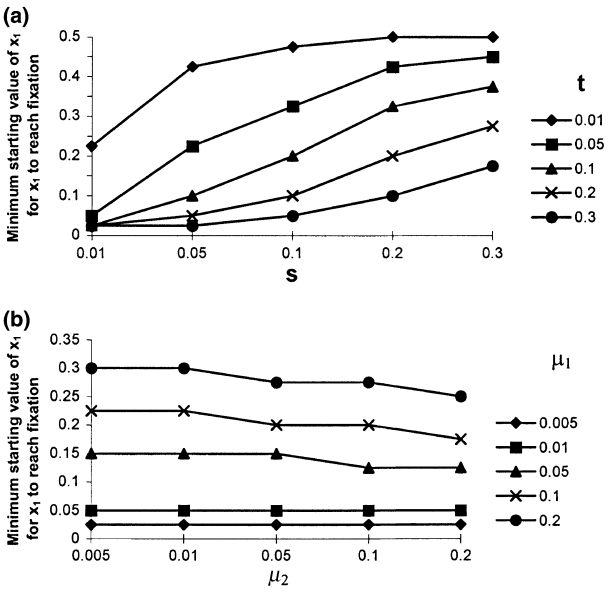

Fig. 2 Global areas of attraction of alleles a and A for certain parameter settings. The graphs show the minimum starting frequency of x_1 that led to fixation on A under the given parameter settings. Here, s and t are the costs referred to in the text; μ_1 and μ_2 are the cultural mutation rates. If not otherwise specified, the values of s and t were 0.2, and μ_1, and μ_2 were 0.05.

where

$$a = -2s(1 - \mu_1 - \mu_2)^2,$$
$$b = t[(1 - \mu_1 - \mu_2)^2 + 2(1 - \mu_1 - \mu_2)(1 - 2\mu_2)],$$
$$c = -\mu_1 - \mu_2 - t[(1 - \mu_1 - \mu_2)(1 - 2\mu_2) - 2\mu_2(1 - \mu_2)],$$
$$d = \mu_2 - t\mu_2(1 - \mu_2).$$

As this system is hard to analyse, we first considered two special instances of this model:

(1) $t = 0$

This is a similar situation to that found in Model 1, with no cost to nonmatching. Equation 12 simplifies, and there is a single equilibrium with a fixed:

$$\hat{x}_1 = 0, \quad \hat{x}_3 = \frac{\mu_2}{\mu_1 + \mu_2}, \quad \hat{x}_4 = \frac{\mu_1}{\mu_1 + \mu_2}. \qquad (13)$$

This is very similar to the first boundary equilibrium obtained for Model 1. The leading eigenvalue for the local linear stability of this equilibrium is:

$$\lambda = 1 - s\frac{\mu_1}{\mu_1 + \mu_2}. \qquad (14)$$

which is very similar to that for Model 1, as might be expected. For $t = 0$, the comparison between the two stable equilibria yields the same result as for Model 1 – the equilibrium where the wide allele (a) is fixed is more stable provided the sum of the mutation rates is less than 1, i.e. $\mu_1 + \mu_2 < 1$. In other words, there seems

to be little effect on the outcome of the models if the only difference is that in one males signal and females receive, while in the other all individuals both signal and receive.

(2) $\mu_1 = \mu_2 = \mu$ *(Equal cultural mutation rates)*

In this case, eqn 12 can be solved explicitly, giving three equilibria:

$$\left(\hat{x}_1 = 0, \quad \hat{x}_3 = \frac{1}{2}, \quad \hat{x}_4 = \frac{1}{2}\right) \quad \text{and}$$

$$\left(\hat{x}_1 = 0, \quad \hat{x}_3 = \frac{1}{2} \pm \frac{\sqrt{t-4\mu}}{2(1-2\mu)\sqrt{t}}, \quad \hat{x}_4 = 1 - \hat{x}_3\right). \quad (15)$$

There are now three equations along the edge because the conformity factor t tends to favour one song-type predominating. The stability of the first of these (the only valid solution when $t < 4\mu$) to invasion by A is governed by the eigenvalue:

$$\lambda = \frac{1 - \frac{1}{2}(t+s)}{1 - \frac{1}{2}t}. \quad (16)$$

As before, we can compare the eigenvalues for when A or a are extinct to examine how susceptible the equilibria are to invasion. If $s > 0$, the equilibrium with a fixed will be more stable provided

$$s > t\frac{\mu(2-t)}{1-\mu(2-t)}. \quad (17)$$

For the other equilibria, the leading eigenvalue is:

$$\lambda = \frac{1 - \frac{1}{2}(t+s)\left(1 \mp \frac{\sqrt{t-4\mu}}{\sqrt{t}}\right)}{1-2\mu}. \quad (18)$$

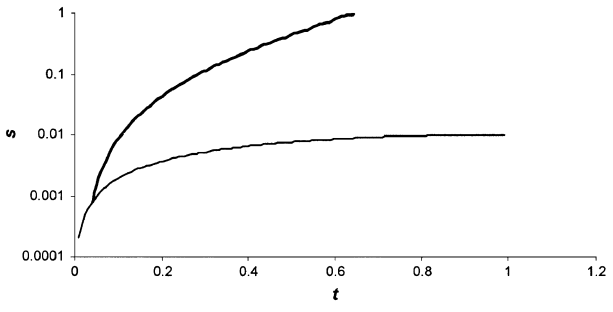

Fig. 3 Comparison of the eigenvalues in Model 2. The axes show values of t and s, and the lines represent the boundaries between conditions where fixation on a is most stable (above the lines) and where fixation on A is most stable (below the lines). The heavier line is the average of the results for equilibria 2 and 3, the lighter line is the result for equilibrium 1. μ = 0.01.

Comparing this to the eigenvalue obtained near fixation of A (eqn 11), gives the following condition under which fixation at a is more stable than fixation at A:

$$s > \frac{4\mu\sqrt{t}}{\sqrt{t}(1-2\mu+4\mu^2) \mp \sqrt{t-4\mu}} - t. \quad (19)$$

These comparisons of local stability (eqns 17 and 19) allow us to examine how the two factors t and s act either in favour of the more restrictive allele A or the less restrictive allele a. This is shown in Fig. 3. In fact, they agree with our numerical analysis of global attraction in that the cost t appears to favour (i.e. creates larger area of attraction, and makes more stable at equilibrium) allele A, whereas the cost s favours allele a. It is difficult from these findings, however, to assess under what precise values of t and s the two alleles are favoured overall. In order to clarify this we iterated the recursion set (eqn 9) numerically in a drift simulation similar to that used in Model 1. For situations in which the cultural mutation rates were not equal, it was necessary first to approach the equilibria along the $a1$–$a2$ edge. When two stable equilibria existed along this edge, the iteration was carried out twice for each parameter setting – once for each equilibrium – and the results averaged.

The results of the numerical work (Fig. 4) confirmed our predictions. Allele a was favoured only in the absence of t (the analogous situation to Model 1), or at low values of t. Overall, s had to be considerably larger than t for allele a to be selected. For example, with μ_1 and μ_2 both equal, and $s = 0.25$, t had only to be >0.06 for allele a to be favoured over all cultural mutation rates. The cultural mutation rate had a much lower effect on the relative success of the alleles than did t and s. As in Model 1, μ_1 had a much larger effect than μ_2.

In summary, Model 2 shows firstly that selection for wider predispositions, in the form of a cost caused by failure to recognize another communicating individual, also occurs when communication is intrasexual, or independent of sex. It also suggests that one of the simplest forms of interaction among cultural signals – a benefit to cultural conformity – creates selection in the opposite direction, namely, for more restrictive predispositions.

Model 3: Inter-sexual interactions with vertical cultural transmission

Model 3 is a modification of Model 1 where instead of oblique cultural transmission of songs, we use vertical transmission (from father to son). In the following description, all symbols are identical to those used in Model 1.

The following is the recursion system for this model:

$$Dx_1' = \frac{x_1x_6 + 2x_1x_5 + x_3x_5 + x_4x_5(1-s)}{4}, \quad (20a)$$

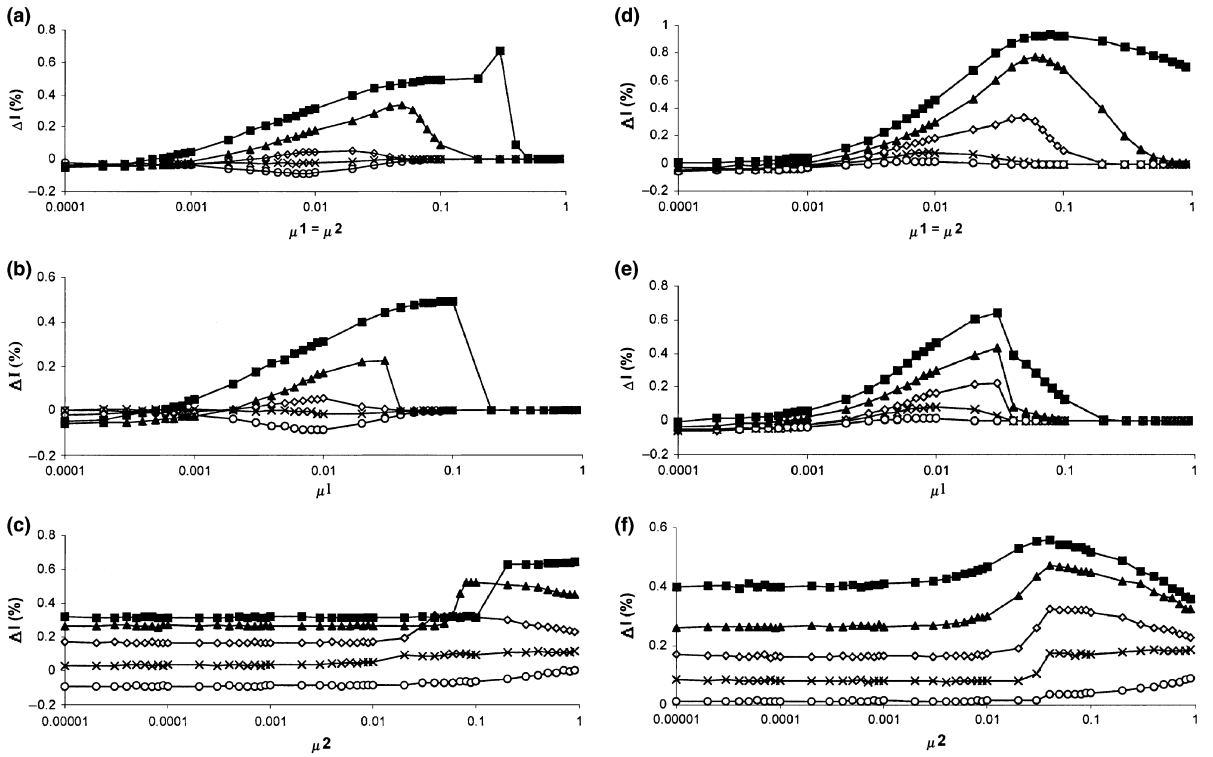

Fig. 4 The results of random walk simulations from the stable equilibria found in Model 2. As in Fig. 1, ΔI represents the proportion of simulations in which allele A invaded, starting from the equilibrium where a was fixed minus the percentage of simulations in which allele a invaded, starting from the equilibrium where A was fixed. If not otherwise specified, $s = 0.25$, $t = 0.25$, $\mu_1 = 0.01$, $\mu_2 = 0.01$. In Figs. 4a–c, $s = 0.25$, while t is varied: squares: $t = 1$; triangles: $t = 0.25$; diamonds: $t = 0.1$; crosses: $t = 0.05$; circles: $t = 0$. In Figs. 4d-f, $t = 0.25$ while s is varied: squares $s = 0$; triangles: $s = 0.1$; diamonds: $s = 0.25$; crosses: $s = 0.5$; circles: $s = 1$.

$$Dx_3' = \{x_1x_6(1-\mu_1) + x_3x_5(1-\mu_1) + 2x_3x_6(1-\mu_1) + x_4x_5(1-s)\mu_2 + 2x_4x_6\mu_2\}/4 \quad (20b)$$

$$Dx_4' = \{x_1x_6\mu_1 + x_3x_5\mu_1 + 2x_3x_6\mu_1 + x_4x_5(1-s)(1-\mu_2) + 2x_4x_6(1-\mu_2)\}/4 \quad (20c)$$

$$Dx_5' = \frac{x_1x_6 + 2x_1x_5 + x_3x_5 + x_4x_5(1-s)}{4} \quad (20d)$$

$$Dx_6' = \frac{x_1x_6 + x_3x_5 + 2x_3x_6 + x_4x_5(1-s) + 2x_4x_6}{4} \quad (20e)$$

where:

$$D = \frac{1}{4} - sx_4x_5. \quad (20f)$$

As in Model 1, the assumptions entail that sex ratio remains even: $x_1 + x_3 + x_4 = x_5 + x_6 = \frac{1}{2}$. Clearly, $x_1 = x_5$ and $x_3 + x_4 = x_6$ after the initial generation. Therefore, from eqns 1a and 1e:

$$|x_1' - x_6'| = \frac{1}{1 - 4sx_4x_5}|x_1 - x_6|. \quad (21)$$

As long as $s > 0$, the frequencies of alleles a and A will diverge from one generation to the next. Therefore the only equilibria are fixations on a and A. We next investigate their local stability.

For fixation on a, the equilibrium is:

$$\hat{x}_1 = 0, \quad \hat{x}_3 = \frac{\mu_2}{2(\mu_1 + \mu_2)}, \quad \hat{x}_4 = \frac{\mu_1}{2(\mu_1 + \mu_2)}, \quad \hat{x}_5 = 0, \quad \hat{x}_6 = \frac{1}{2}. \quad (22)$$

The stability of this equilibrium is governed by:

$$\lambda = 1 - \frac{s\mu_1}{2(\mu_1 + \mu_2)}. \quad (23)$$

At fixation of A we have:

$$\hat{x}_1 = \frac{1}{2}, \quad \hat{x}_3 = 0, \quad \hat{x}_4 = 0, \quad \hat{x}_5 = \frac{1}{2}, \quad \hat{x}_6 = 0. \quad (24)$$

and the stability of this equilibrium to invasion is governed by:

$$\lambda = 1 - \frac{Q}{2} + \sqrt{Q^2 - 2s\mu_1}. \qquad (25)$$

where

$$Q = \frac{1 + \mu_1 + \mu_2 - s\mu_2 + s}{2}. \qquad (26)$$

At a polymorphic equilibrium, from eqn 21 we have $\hat{x}_5 = \hat{x}_6 = \hat{x}_1 = \hat{x}_3 + \hat{x}_4 = \frac{1}{4}$. Hence, from eqn 20c we have at equilibrium

$$\left(\frac{1}{2} - \frac{sx_4}{2}\right)x_4 = \frac{\mu_1}{32} + \frac{\mu_1(\frac{1}{4} - x_4)}{8} + \frac{\mu_1(\frac{1}{4} - x_4)}{4} + (1 - \mu_2)\left[\frac{(1 - s)x_4}{8} + \frac{x_4}{4}\right]. \qquad (27a)$$

This simplifies to

$$4sx_4^2 + x_4[(1 - \mu_2)(3 - s) - 3\mu_1 - 4] + \mu_1 = 0. \qquad (27b)$$

As eqn 27b is positive at $x_4 = 0$ and negative at $x_4 = \frac{1}{4}$, there is a single valid root $\hat{x}_4 = \frac{1}{4} - \hat{x}_3$ which lies on the separatrix of the domains of attraction to the two genetic fixations.

Comparing the stability of the two equilibria, it can be shown that the first equilibrium (eqn 22) is more stable than the second (eqn 24) if:

$$s\mu_1(1 - \mu_1 - \mu_2)[\mu_1 + (1 + s)\mu_2] > 0, \qquad (28)$$

that is, as long as $s > 0$ and $\mu_1 + \mu_2 < 1$. As these conditions would always be true in the model, we can conclude that it is more likely that allele *a* would invade a population of *A* than vice versa. To verify this conclusion, we again used a random walk simulation. The results of this (Fig. 1) were very similar to those of Model 1. Under all parameter settings with $s > 0$, allele *a* was more successful than allele *A*. In summary, rather similar results were obtained from the model, irrespective of whether oblique or vertical cultural transmission occurred.

Discussion

Although genetic predispositions have long been known, or suspected to underlie many kinds of learning (e.g. Bolhuis, 1996; Marler, 1997), the evolutionary implications of this developmental mechanism have not been extensively examined. When the learning involved leads to cultural transmission, an especially interesting case arises, as two separate evolutionary systems contribute to the same phenotype. The models studied here demonstrate that in this situation, cultural evolution is likely to strongly influence the evolution of genetic predispositions that restrict learning. However, we found that different cultural and communicative processes influenced this evolution in different ways. If individuals also relied on their predisposition to recognize suitable individuals to interact with, we found selection for less restrictive predispositions. If, however, it benefited individuals to share exactly the same cultural phenotype as those with whom they were interacting (cultural conformity), selection for more restrictive predispositions arose. Processes of cultural mutation have a considerable effect in amplifying these tendencies.

The significance of the evolution of more or less restrictive genetic predispositions was discussed by Lachlan & Slater (1999), who argued that selection for increased width would be sufficient for the evolutionary maintenance of vocal learning because learning could only disappear through the increasing constriction of the predisposition; eventually there would be no possibility for learning to affect the phenotype, and therefore nothing to favour learning. They also found in their spatial simulation that wider predispositions were more successful than narrower ones, given that the predispositions for learning were also used by females for identifying potential mates, suggesting that once cultural transmission of bird song evolved, it was unlikely for a population to revert to genetic transmission. They called this effect the cultural trap hypothesis. The results of this paper explain in more detail why less restrictive predispositions might be more successful. As our models show, when a new allele invades, it is subject to frequency-dependent selection against it, whether it is more or less restrictive. However, individuals with less restrictive alleles tend to avoid this selection because they can learn the mutually acceptable signal type. A second result of our models is that less restrictive predispositions were found to be more successful in a wider variety of more general situations, indicating that this effect may be found in nonterritorial situations (i.e. without local song copying), and that it might occur in the interactions between males, as well as the interactions between males and females. Finally, in an even more general interpretation of these models, one could regard *A* as a 'non-learning' allele and *a* as an allele supporting learning. In this situation the origin, as well as the maintenance, of learning might be explained.

A number of authors have argued that benefits resulting from song sharing between individuals, especially between neighbours, might provide an evolutionary explanation for song learning (Kroodsma, 1996; Brown & Farabaugh, 1997). However, our results suggest that, at least under very general conditions, this is not the case. Selection of cultural conformers leads to the evolution of narrower filters, and this progressive constraint on song learning could eventually lead to disappearance of learning. Again, this perspective can be supported by viewing the cultural traits as either ranges of song-types, or as individual song types. In the latter scenario, learning is effectively removed by selection for conformers.

In our models, cultural conformity appeared to produce a much stronger effect than the cultural trap (Fig. 3). However, two conditions change this bias. The first is that the cost incurred from failing to share a cultural trait with another individual is lower than that incurred from failing to recognize a conspecific (i.e. $s > t$). As the latter is likely to be important in species recognition, this condition may be met. The second condition is that the cultural mutation rate from the universally recognized cultural trait to the trait only recognized by individuals possessing allele a is lower than the cultural mutation rate in the opposite direction. This condition might be met if the two cultural traits represented ranges of 'song types', and allele a represented an incremental increase in the width of the genetic bias. A more specific spatial simulation model supports this finding in the case of territorial songbirds (Lachlan, 1999).

Recently, biologists studying bird song have begun to investigate in greater depth the interactions between ecology, function and developmental mechanism (Kroodsma, 1996). We would argue on the basis of our findings here that any such research programme should consider the evolutionary nature of cultural transmission, and its interactions with genetic evolution. Not only do cultural evolutionary models serve as a useful way of posing testable hypotheses from evolutionary arguments, but as in this case, they also provide results that would seem counterintuitive without this perspective.

Processes such as we have described here leading to changes in width of predisposition for song birds, may also occur in the evolution of human language. However, the increased structural and social complexity of human cultural communication means that we must be much less certain about the applicability of the model. For example, in our models, cultural mutation is a random event. This is likely to be unrealistic for language. The adoption of novel communication traits has been shown to be strongly biased by factors such as social dominance (Chambers, 1995). Nevertheless, these models may serve to illustrate some of the evolutionary interactions that may have occurred between the cultural and genetic transmission of language.

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