Prestige as a Driving Force in Cultural Transmission

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26 Abstract

Copying our role-models have always been an efficient method of acquiring knowledge. Copying successful role-model is one of the methods of cultural transmission of traits. In this paper, we study the various biases and their effects of the population and it's evolution. The most common bias when choosing a role-model to copy is success bias, i.e copying whoever appears successful to us. This estimation is based on the performance of the role-model alone, without any other factors. Here, we study another factor we believe aids to better model the cultural inheritance of traits in a large population. Influence bias is a bias evaluated by the number of copiers a role-model already has. In our model we combine these components to what we call the **Prestige bias** and analyze its relationship to the dynamics of the population. We successfully found mathematical approximations to our model, easing the mathematical analysis and the computation power required for simulations. We show the value of these approximations using simulations, and their robustness to variations such as mutation and other relaxations required for the mathematical proofs. In the binary form of our model, we found alternatives to Kimura's equations for approximating fixation probability and time to fixation of an invading advantageous trait, in both a constant and changing environment. We show that Influence solely affects the effective population size. We found that influence acts as an accelerator for a state of the population, matching the rich getting richer it was based on. We believe such model better describes how humans acquire knowledge from one another, mainly in the last years where social networks are very popular. Social networks allow easy access to estimate number of copiers a role-model has, with little to no effort.

46 Introduction

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Traits transmission is when an individual passes on a trait, genetic or behavioral, to another individual. Transmission in nature manifests in two main ways: genetic and cultural. Genetic transmission is when an individual, or several, transmit their genes to their offspring by duplication of their own cells. Cultural transmission is the way individuals transmit cultural traits (i.e behavior) from one another, typically via teaching and demonstrating. Cultural transmission is most common in humans (Cavalli-Sforza and Feldman, 1981, pg. 3) and in primates like chimpanzees (Horner et al., 2010; Kendal et al., 2015). The common cultural traits in humans are behavioral patterns, like personalities and habits, transmitted via observations and verbal discussions. Henrich and McElreath (2007) suggest that cultural learning may be particular to humans, but McComb et al. (2001) suggest that it appears in other mammals as well, elephants for example:

... the possession of enhanced discriminatory abilities by the oldest individual [matriarch] in a group can influence the social knowledge of the group as a whole.

They showed that once a matriarch is removed from the group, the group's survival instincts are inferior. They support their hypothesis by exacting an experiment: playing audio recordings of African elephants, showing that groups with a matriarch recognize and react better to hostile or

friendly calls than the groups without one. Moreover, cultural transmission appears in other species, even simpler than mammals, such as *Drosophila*. Battesti et al. (2012) show that oviposition site
choice in fruit flies is culturally transmitted. They showed that flies without experience in choosing sites, after spending some time with "experienced" flies, chose the same type of site without directly observing this behavior. Battesti et al. (2012) mention that how the information is transferred is still an open question, but suggest that the flies may use olfactory cues, like other animals such as rodents and bees.

Cultural transmission is similar to genetic transmission in many ways, while different in others. Similar to genetic transmission, the effects of culturally transmitted traits can be physiological rather than behavioral, and transmitted from parents to offspring. For example, parents can teach their children to be strong or tall, within some biological limits, by instructing them to maintain a healthy diet and engage in physical activity. Contrary to genetic transmission, the sources of the traits can be many, and not only parents. They can even be unrelated, like teachers, celebrities, coaches, the media, or any stranger that comes in contact with them. Cultural transmission can be vertical, where parents transmit to their children, but also oblique, where other adults transmit traits to children (not their own). Horizontal transmission is also possible, where peers transmit traits to one another. Lastly, vertical transmission in the opposite direction is possible too, where parents copy traits from their children (e.g playing video games) as Cavalli-Sforza and Feldman (1981) and Creanza et al. (2017) suggest. In addition, even when a cultural trait is disfavored by natural selection, it still may spread across a population given transmission biases strong enough to negate the selection bias (Boyd and Richerson, 1988, Ch. 8 pg. 279).

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Transmission bias occurs when a trait has a disproportionate probability from its frequency in the population to be transmitted. For example, Eickbush et al. (2019) show that there are genes of yeast called wtf genes, that bias their transmission to the gametes. They secrete a long life expectancy poison, together with a short life expectancy antidote, so a gamete without the gene will perish (the poison will outlive the antidote). Transmission biases, though exist in genetic transmis-90 sion, are probably more common in cultural transmission. Much like mutation in genetic evolution, one could learn behavioral patterns or traits on his own, usually referred to as innovation, also called individual learning, and just like mutation, without it humans might have been remained at the stone age, or even go extinct. Rendell et al. (2010) suggest that success biased social transmission contribute more to the general success of the population than individual learning. They conducted a tournament for developing learning strategies of a population, where each participant need to devise a strategy. Each strategy must define when individuals should observe and copy from others, and when to engage in individual learning. The best strategies contained a high percentage of social learning relative to individual learning, even when the error when copying was as high as almost 0.5. It is important to add that all of the strategies include some percentage of individual learning, and without it the results would be a lot worse. In addition to Rendell et al. (2010), Fogarty et al. 100

(2017) define different types of transmission biases based on success. They define several types of role-model choosing methods, all assuming that the copier correctly identifies the successful ones. 102 Both studies assume that individuals can successfully evaluate successful individuals. Boyd and Richerson (1988, Ch. 5) suggest that the evaluation of success can be divided into three groups: 104 direct bias, indirect bias and frequency-dependent bias. A direct bias is when a variation of a trait is more attractive than others, and is evaluated by directly testing the variation of the trait. For example, an individual observing a Ping-Pong match between two others can try both of the pad-108 dle grips it observed, and decide what grip is better for it. An indirect bias is when an individual uses the value of one trait to determine the attractiveness of another, so it *indirectly* evaluates the attractiveness of the role-model. Continuing with the example, a bystander could copy the paddle 110 grip of the Ping-Pong player who scored more points in the match. A frequency-dependent bias is when an individual has a probability to copy a variant of the trait that is nonlinear to the trait's 112 frequency in the parent's generation. Continuing with the example, when an individual is 80% likely to copy the common paddle grip even when only 60% of the population is using it, it is said to be frequency-biased.

116 Frequency bias could be negative too. Aljadeff et al. (2020) show that societies under competitive conditions are likely to develop diversity in foraging specialization rather than uniformity.

118 Prestige means having a good reputation or high-esteem, therefore does not directly signify success (although it may imply it), making it an indirect bias. Both Boyd and Richerson (1988, 120 Ch. 8) and Fogarty et al. (2017) claim that prestige biases are probably more common in humans than success biases. Boyd and Richerson (1988, Ch. 8) add that maladaptive traits may spread widely in a population, if the indirect bias is strong enough. They claim the bias could lead to a runaway process, caused by a cultural equivalent of sexual selection (Andersson, 1994). On the 124 other hand, Henrich and Broesch (2011b) claim that prestige biases, over generations, can lead to cultural adaptations. According to them, prestige can make a maladaptive trait spread in the population, but can also accelerate the spread of adaptive traits as well. Prestige bias is often 126 mentioned in the literature, but seldom modeled. Boyd and Richerson (1988) have modeled the prestige bias, but didn't include the effects the copiers of a role-model has on the probability of 128 other individuals to choose the same role model.

This effect is similar to conformity (Denton et al., 2020), which is usually modeled as a different bias. Conformist learning (imitating locally common behaviors) is a known bias in cultural transmission
(Molleman et al., 2013), and we suggest that prestige bias is made up by both indirect bias and a new type of conformity. Our new component, influence, is assigned to a role-model, contrary to
conformity, which refers to the frequency of a trait in the population, regardless which individuals posses it. The goal of this study is to define a more realistic model for prestige bias and
analyze the dynamics of the population it causes.

Today, due to social media, it is easier than ever to estimate the influence individuals have over others, therefore it is probably a major part of humans decision-making process. For example, the number of *followers* a person has in the mobile application *Instagram* may significantly affect

how his beliefs are perceived by the population. We want to create a model that better fits reality and simulate scenarios that better mimic cultural transmission dynamics. With a more accurate
 model of prestige bias, we may understand better how cultural traits are transmitted, and why. Moreover, we could better explain the cause for the spread of maladaptive traits, or the acceleration
 of adaptive traits often seen in humans.

Models and Methods

146 Reminder: A Wright-Fisher model is a mathematical model meant to describe a genetic drift process. This model assumes that generations do not overlap and that each copy of the gene found
148 in the new generation is drawn independently at random from all copies of the gene in the old generation.

A Moran model assumes overlapping generations. At each time step, one individual is chosen to reproduce and one individual is chosen to die. In our models we harness these two models and modify them to describe new mathematical models that we use to expand the basic indirect bias model Boyd and Richerson (1988) suggest.

154 Continuous Model

Consider a population of N individuals, each individual has one trait on a continuous scale. Every generation, N naive individuals (copiers) must choose a trait to copy from one of the individuals of the previous generation (role-models). Similar to a Wright-Fisher model, we assume the generations don't overlap. We base our model on the model of Boyd and Richerson (1988), by assuming only oblique transmission of the traits (Indicator trait - A). Unlike their model, we omit a second trait called Indirectly biased trait to lower complexity. The model's state at time t can be described by:

162
$$\vec{A}_t = (A_{t,1}, \dots, A_{t,N})$$
 (1)

where \vec{A}_t is a vector describing the indicator traits at time t, and \vec{A}_0 is drawn from a standard normal distribution. Each individual from generation t+1, a *copier*, inherits traits like so:

$$A_i' = F_i(\vec{A_t}) \tag{2}$$

where A'_i is the indicator and indirect trait values correspondingly, that copier i acquires. We use A'_i as an alias for $A_{i,(t+1)}$ for simplicity for the transition between generations $t \to t+1$. F is a function over the t generation traits vector, and is defined differently for every implementation of the **Generic model**.

170 **Success bias.** Boyd and Richerson (1988, Ch.8, p.247-249) describe a method of inheritance using a blend, i.e weighted average of the trait of the entire generation. They define F as a weighted

172 average of the role-models' traits in a single generation:

$$F_i(\vec{X}) = \sum_{j=1}^{N} \left(G_{ij} \cdot X_{ij} \right) \tag{3}$$

174 where $G_{i,j}$ is:

$$G_{ij} = \frac{\beta(A_{ij})}{\sum_{l=1}^{N} \beta(A_{il})} \tag{4}$$

We define G_{ij} to be the *Success bias* of role-model j in the eyes of copier i. $A_{i,j}$ is the absolute indicator trait value copier i estimates role-model j has:

$$A_{i,j} = A_j + e_i, (5)$$

where e_i is the copier's error of estimation, $\vec{e} \sim N(0, \frac{1}{\eta^2})$. $\beta(X)$ is the bias function, meant to quantify the success bias of a role-model:

$$\beta(A_{i,j}) = b \cdot \exp\left(-\frac{(A_{i,j} - \hat{A})^2}{2J}\right),\tag{6}$$

where \hat{A} is the optimal indicator value and J, b are model parameters to control the "strength" of the bias. $G_{i,j}$ is therefore the relative success score copier i assigns to role-model j, resembling relative fitness in genetic transmission models.

Random choice transmission. Boyd and Richerson (1988) note that the method of transmission they use in their model has alternatives. We follow their suggestion and create a model similar to theirs, with random choice as a transmission method: The probability of copier i to choose role-model j as his role-model to copy its traits from is $G_{i,j}$. Once a copier chose its role-model, it will copy both its traits only from his role-model, instead of a weighted average of the entire role-model generation:

$$A_i' = A_{i,j} \tag{7}$$

192 **Influence bias.** Copiers choose their role-models one by one. After copier i chose a role-model, we denote K_{ij} as the number of copiers that chose role-model j until that point, such that $\sum_{j=1}^{N} K_{i,j} = i$. The stochastic process of role-model choice,

$$\{\vec{K}_i\}_{i=1}^N, \quad \vec{K}_i = (K_{i1}, \dots, K_{iN}),$$
 (8)

196 is described by the recurrence equation

$$K_{i,j} = K_{i-1,j} + S_{i,j}, \quad i, j = 1, 2, \dots, N$$
 (9)

where $S_{i,j} = 1$ if the *i*-th copier chose role-model *j* and 0 otherwise, and the initial state is $K_{0,j} = 0$.

The probability that the i-th copier chose role-model j

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$$G_{i,j} = P(S_{i,j} = 1 | S_{1,j}, S_{2,j}, ..., S_{i-1,j})$$
 (10)

is the prestige of role-model j in the eyes of copier i. This prestige $G_{i,j}$ is determined as follows. 202 First, role-model j is characterized by its indicator value A_j as before, and the estimated indicator value by copier i, $A_{i,j}$ remains as eq. (5). Finally, the prestige $G_{i,j}$ of role-model j in the eyes of copier i is determined by the estimated biased indicator value $\beta(A_{i,j})$ and the number of copiers that chose role-model j before copier i, $K_{i-1,j}$,

206
$$G_{i,j} = \frac{\alpha_j \cdot \beta(A_{i,j}) + (1 - \alpha_j) \cdot K_{i-1,j}}{W_i}, \tag{11}$$

where the weight α_j is a characteristic of role-model j that determines the relative significance of the indicator and the influence in the prestige, and W_i is a normalizing factor to ensure $\sum_{j=1}^{N} G_{i,j} = 1$,

$$W_{i} = \sum_{j=1}^{N} \left(\alpha_{j} \cdot \beta(A_{i,j}) + (1 - \alpha_{j}) \cdot K_{i-1,j} \right).$$
 (12)

210 Binary model

The indicator trait can now manifest in only two phenotypes, and for simplicity we define they can be either \hat{A} or A. In the binary model, the influence is determined by the number of copiers already chosen **any** role-model with either A or \hat{A} , as all role-models with A will contribute to the probability of the trait to be inherited just the same (can be proved with simple induction). Simply put, assuming there are two role-models with the A trait, the probability a copier will copy from either role-model will be the same, and the probability the A trait will be inherited is the sum of both role-models. In the general case, the probability of the i-th individual to inherit trait A, based on eq. (22) is:

$$P_{i,A} = \frac{(N-X)\alpha'\beta(A) + K_A}{i - 1 + (N-X)\alpha'\beta(A) + X\alpha'\beta(\hat{A})} = \frac{(N-X)\alpha'\beta(A) + K_A}{i - 1 + (N-X)\alpha'\beta(A) + \alpha'X}$$
(13)

- 220 where X is the number of role-models with trait \hat{A} and K_A is the number of copiers that already chose A.
- The model begins with the first generation having a single individual with \hat{A} , and the rest have A. The process itself is the same stochastic process as the continuous model.

224 Methods

The main methods we used to experiment and compare our models is using computer generated 226 simulations. In order to establish our claims and base our mathematical approximations of our models, we used the χ^2 test for the full continuous model, and the Kimura's equations of fixation probability and time to fixation for the binary model.

Results

230 Approximations

Currently $\{\vec{K}_i\}_{i=1}^N$ is a stochastic process where each state depends on the previous state, i.e a Markov chain. We wanted to find an equivalent stochastic process that has the same joint distribution on $\{\vec{K}_i\}_{i=1}^N$, but it is possible to evaluate the joint distribution directly without evaluating all the marginal conditional distributions: eq. (9), eq. (10).

We found two approximations to our process, which are summarized here and explained in 236 detail later on:

- K_{i,j} follows the general binomial distribution defined by Drezner and Farnum (1993). Moreover, E[K_{N,j}] = N · G_{1,j} if e = e_l = e_m for all l, m. That is, the expected number of copiers of role-model j equals its prestige in the eyes of the first copier, multiplied by the total number of copiers. In addition, we find that when α is homogeneous, α_l = α_m for all l, m, then E[K_{N,j}] = β(A'_j) / β(A'), where A'_j is the estimated indicator value A'_j = A_j + e, and β(A') is the population mean estimated indicator value. That is, the expected number of copiers of a role-model equals its relative biased indicator value, similar to the role of relative fitness in population-genetic models.
- 2. The role-model choice process eq. (8) is equivalent to a Pólya urn model if $e_l = e_m$ for all l, m. Therefore, $\vec{K}_i = (K_{i,1}, \dots, K_{i,N})$ follows a Dirichlet-Multinomial distribution,

$$\vec{K}_i \sim DM(N, \vec{G}_1), \tag{14}$$

where $\vec{G}_1 = (G_{1,1}, \dots, G_{1,N})$. Note that here $G_{i,j}$ is only a function of the indicator values A_j and the weights α_j .

250 General Binomial Distribution Approximation

The general binomial distribution (GBD) is achieved by a series of Bernoulli experiments, with possible dependency between experiments.

Proposition: The number of copiers $K_{i,j}$ follows the GBD, $K_{i,j} \sim GBD(i, \alpha_i \cdot \beta(A'_j))$, when 254 $e_l = e_m$ for all $l, m \in N$ and $A'_j = A_j + e$

Proof: We'll denote $Q_j(k,i) = P(K_{i,j} = k | K_{i-1,j})$ as the probability that exactly k out of i copiers choose role-model j, using conditional probability and eq. (9):

$$Q_j(k,i) = P_j(S_{i,j} = 1|k-1,i-1) \cdot Q_j(k-1,i-1) + P_j(S_{i,j} = 0|k,i-1) \cdot Q_j(k,i-1)$$
 (15)

258 where $S_{i,j} = 1$ when the *i*-th copier chooses role-model *j*.

We see that eq. (15) is equivalent to eq. (2.1) that Drezner and Farnum (1993) define. $Q_j(k, N)$ 260 is the probability that k out of N copiers choose role-model j at the end of the process, which by our previous notation is $k = K_{N,j}$. By describing the process of eq. (8) as (Drezner and Farnum, 262 1993) did, we've completed the proof.

Corollary 1: $\mathbb{E}[K_{N,j}] = N \cdot G_{1,j}$.

In (Drezner and Farnum, 1993, equation 2.3), they show that the expected value of k is: $\mathbb{E}[k] = N \cdot Q_j(1,1)$ (using different notations). $Q_j(1,1)$ is the initial probability to choose role-model j, before any choices are made. $Q_j(1,1) = G_{1,j}$ by definition, therefore we can say that $\mathbb{E}[K_{N,j}] = N \cdot G_{1,j}$.

Corollary 2: $\mathbb{E}[K_{Nj}] = \alpha_j \cdot \beta(A'_j) / \overline{\alpha \cdot \beta(A')}$.

270 **Proof:** The initial prestige of role-model j based on eq. (11) is:

$$G_{1,j} = \frac{\alpha_j \cdot \beta(A'_j)}{\sum\limits_{m=1}^{N} \alpha_m \cdot \beta(A'_m)}$$
(16)

272 The denominator of eq. (16) can also be formulated as:

$$\sum_{m=1}^{N} \alpha_m \beta(A'_m) = N \cdot \overline{\alpha \cdot \beta(A')}$$
(17)

274 where $\overline{\alpha\beta(A')}$ is the mean value of $\alpha_m \cdot \beta(A'_m)$ for all m. Using eq. (17) we get:

$$\mathbb{E}[K_{Nj}] = \alpha_j \cdot \beta(A'_j) / \overline{\alpha \cdot \beta(A')}$$
(18)

276 , completing our proof.

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The special case where $\alpha = \alpha_l = \alpha_m$ for all $l, m \in N$ is interesting, because we can evaluate the expected number of copiers using a linear equation:

$$\mathbb{E}[K_{Nj}] = N \cdot \frac{\alpha \cdot \beta(A'_j)}{\sum_{m=1}^{N} \alpha \cdot \beta(A'_m)} = \beta(A'_j) / \overline{\beta(A')}$$
(19)

where the only variable is A'_j , because $\overline{\beta(A')}$ is the mean of the distribution we draw the indicator values from, modified by some constant parameters of β . We can then denote $L = 1/\overline{\beta(A')}$ and write:

$$\mathbb{E}[K_{Nj}] = L \cdot \beta(A_j') \tag{20}$$

284 Dirichlet-Multinomial Distribution Approximation

Reminder: Pólya urn model is a stochastic process that is defined as such: The process consists of N draws from an urn with an initial amount of colored balls of M colors. When a ball is drawn, it is then placed back in the urn together with an additional new ball of the same colour.

288 Let $\vec{U_i} = \{u_{i,1}, u_{i,2}, ..., u_{i,M}\}$ where $u_{i,j}$ is the number of balls of the j-th color in the urn after i draws. Let $S_{i,j} = 1$ when drawing a j colored ball on the i-th draw, and 0 otherwise. The probability that $S_{i,j} = 1$ given $\vec{U_{i-1}}$ is:

$$P(S_{i,j} = 1|\vec{U_{i-1}}) = \frac{u_{i-1,j}}{\sum_{m=1}^{M} u_{i-1,m}} = \frac{o_j + w_{i-1,j}}{\sum_{m=1}^{M} o_m + w_{i-1,m}}$$

$$= \frac{o_j + w_{i-1,j}}{i - 1 + \sum_{m=1}^{M} o_m}$$
(21)

where o_j is the initial number of balls of the colour j in the urn, and $w_{i,j}$ is the number of new balls that were added to the urn after i draws of the color j.

294 **Proposition:** process $\{\vec{K}_i\}_{i=1}^N$ is equivalent to a *Pólya urn model* when $e = e_i = e_j$ and $\alpha = \alpha_j = \alpha_i$ for all $i, j \in N$.

296 **Proof:** We denote α' as the odds ratio between the weights of the indicator and the influence $(\alpha' = \frac{\alpha}{1-\alpha})$. Using eq. (11) we get:

$$G_{i,j} = \frac{\alpha \cdot \beta(A'_j) + (1 - \alpha) \cdot K_{i-1,j}}{W_i} \cdot \frac{1 - \alpha}{1 - \alpha}$$

$$= \frac{\alpha' \beta(A'_j) + K_{i-1,j}}{\sum_{m=1}^{N} \alpha' \beta(A'_m) + K_{i-1,m}}$$

$$= \frac{\alpha' \beta(A'_j) + K_{i-1,j}}{i - 1 + \sum_{m=1}^{N} \alpha' \beta(A'_m)}$$
(22)

298

We see that eq. (21) and eq. (22) are equivalent when setting M = N, $o_j = \alpha' \beta(A'_j)$, $w_{i,j} = K_{i,j}$, 300 completing the proof.

Corollary 1: In their paper, Frigyik et al. (2010, section 2) prove that the proportion of different colored balls in a *Pólya urn model* will converge to the Dirichlet distribution as the number of draws approaches infinity, based on *Martingale Convergence Theorem* (Durrett, 1999). We therefore have

an approximation for the relative "weight" or the proportion each role-model has when evaluated as a role-model. Drawing from a Multinomial distribution where the parameters are the modified weights gained from the Dirichlet distribution is viable for selecting the role-model for the next generation. We can therefore sample from a Dirichlet-Multinomial distribution to approximate how many copiers each of the role-models will have: $\vec{K}_i \sim DM(N, \vec{G}_1)$.

Numeric validation: We showed our process is DM (Dirichlet-Multinomial) distributed when there are no errors when copying or evaluating the traits, and when α is homogeneous in the population. To support our proof, we tested our approximation empirically using computer simulations.

312 To test our hypothesis, we used a *goodness of fit* method known as *Pearson's chi-squared test*. In this test, one can reject or accept the null hypothesis, which in our case is the hypothesis that the 314 simulations results were drawn from a DM distribution.

To use this test, we ran many simulations of our original model, and used the mean distribution 316 of copiers. This mean distribution is our observed distribution, and we tested it with the DM expected distribution, using said chi-squared test. We tested multiple variations of the trait weight 318 parameter (α) .

In all our tests, the p-value was 1. This means that the probability to reject the null hypothesis is essentially nonexistent (the usual threshold for a p-value needed to reject H_0 is 0.05 or lower). In addition, we found out that for high α values (above 0.5), very few simulations are needed to reach p-value 1. (less than five simulations for $\alpha = 0.9$, and less than 20 for $\alpha = 0.7$) For very low α values, which means very high influence weight, the number of simulations needed was 100, which is still a relatively small amount. To verify our codes results, we also ran the test for different distributions, for example the uniform distribution (all role-models have exactly one copier).

All these tests resulted in a p-value 0, which means we can likely reject H_0 for these distributions, as expected.

Once we validated our proof for a single iteration of the model, we went on to more complex validations for the entire model.

330 Numeric comparisons

We're interesting in studying the difference between the real binary model as we defined in eq. (13), 332 and the Dirichlet-Multinomial approximation. Specifically, we're interesting in the fixation probability of the favored trait (\hat{A}) and its time to fixation.

The first step was to find the number of simulations needed to sufficiently approximate the real model with the DM approximation. From fig. 1 we see that 1000 simulations or higher is enough.

The next step was to see how the observed metrics (fixation probability and time) varies when relaxing our assumptions we used to prove the DM approximation. First we relaxed our assumption of no mutation. To include mutation in the binary model, it needs to be redefined, since in the original model it was based on the fact the traits are drawn from a continuous scale. In the binary

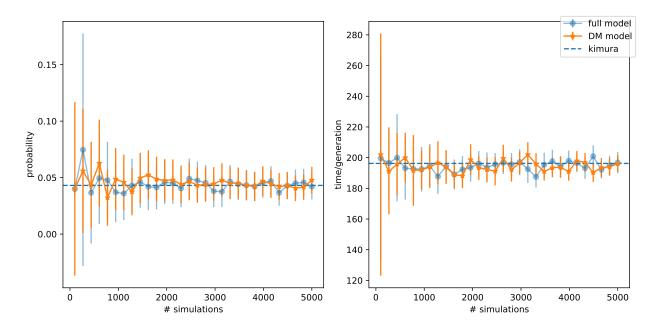


Figure 1: The number of simulations needed to get a good approximation. At 1,000 the approximation is good enough. Error bars represent 95% confidence interval. Population size $N=1000,~\alpha=0.5,~J=1,~\hat{A}=1,A=0.7,~\beta(A)=0.956.$

model mutation will be manifested as an error when evaluating the bias itself. This is easily done by using a heterogeneous J parameter, which controls the strength of the success bias in eq. (6).

In fig. 2 we see the comparison when heterogeneous mutation is applied to both models. When mutation is applied, we sample J_i for each copier i from a normal distribution with varying scale (variance). We can see that even when the standard deviation is 0.1, the metrics of both models are both similar, and close to the Kimura approximation (more details in the next section).

In fig. 3 we relaxed our assumption of a homogeneous α , and used a heterogeneous α instead. Similar to the mutation comparison, we drew α_j for each role-model j from a normal distribution with varying scale. We again see that the metrics of both models are similar in the entire spectrum of our x-axis, and the Kimura approximation is within both confidence intervals.

350 Fixation probability and time - binary model

Kimura's approximation: After establishing a case in the favor of our DM approximation, we wanted to use it to examine the behavior of the population. Specifically, we wanted to analyze the influence of the indicator weight (α) on the fixation probability and time to fixation of the favored phenotype in a binary model. For simplicity, we don't include mutation rate in the binary model approximations. Following Durrett (2008), we used our DM approximation of the model to find the effective population size. From eq. (13) we can derive that the process of inheritance in our binary model is DM distributed with a parameters vector of size two: V = (α'X, (N - X)α'β(A)).

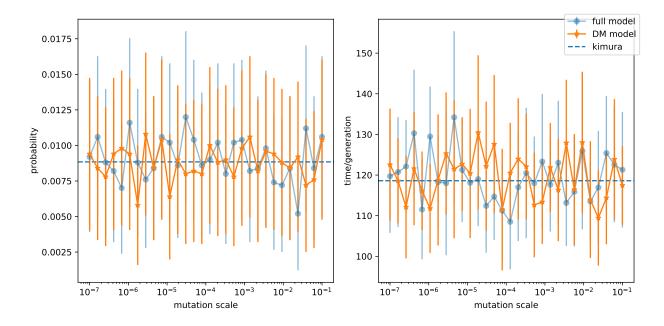


Figure 2: Comparison of the DM approximation and the full model when mutation is included. Even high mutation rate doesn't worsen the approximation, and the data points are close to the mathematical estimation (Kimura's). Error bars are 95% confidence intervals, and are condensed (+- 0.01 probability and +-40 generations) 5000 simulations per data point, $N=1000, \alpha=0.1, \hat{A}=1, A=0.7, J\sim N(1,x^2)$ where x is the mutation scale in the x-axis.

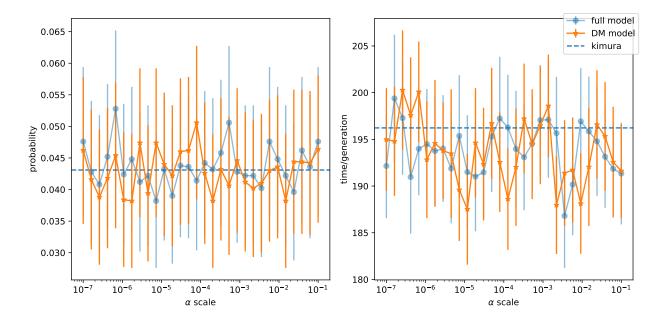


Figure 3: Comparison of the DM approximation and the full model when success weight is heterogeneous. High success weight variance distances the approximation and the full model of generations to fixation from the Kimura's approximation, but not by much (confidence intervals still cover it). Error bars are 95% confidence intervals, and are less condensed (+- 0.03 probability and +-40 generations) 5000 simulations per data point, N = 1000, $\alpha \sim N(0.5, x^2)$, $\hat{A} = 1$, A = 0.7, A = 0.7, A = 0.956.

358 Proposition: 1-β(A) is equivalent to the selection coefficient s in a classic Wright-Fisher model in the diffusion equations meant to approximate the fixation probability and time of the advanta 360 geous trait.

Proof: Let x be the frequency of type \hat{A} in the population with N individuals. Let X be the number of individuals of type \hat{A} so x = X/N. X' is the number of individuals with \hat{A} in the next generation and x' their frequency. By definition $\beta(\hat{A}) = 1$, and for simplicity we'll denote $\beta(A) = \beta$ 364 $(\beta < 1)$.

The expected number of individuals of a DM distribution is:

$$E[X'] = N \frac{\alpha_1}{\alpha_1 + \alpha_2},\tag{23}$$

where $\alpha_1 = \alpha' X$ and $\alpha_2 = \alpha' (N - X) \beta$, from eq. (13). We want to use frequencies instead of quantities to follow Durret's process so:

$$E[x'] = E[\frac{X'}{N}] = \frac{1}{N}E[X']$$
 (24)

370 Putting it together we get:

374

$$E[x'] = \frac{1}{N} N \frac{\alpha' x N}{\alpha' x N + \alpha' N (1 - x) \beta}$$

$$= \frac{x}{x + (1 - x) \beta}$$
(25)

which is identical to the equation in the top of page 253, chap 7.2 in Durrett (2008). We therefore use the same approximation and say that:

$$E[x'] = \frac{x}{x + (1 - x)\beta} = \frac{x}{x + (1 - x)(1 - s)} =$$

$$= x + x(1 - x)s + o(s)$$

$$= x + x(1 - x)(1 - \beta) + o(1 - \beta)$$
(26)

By definition, x is constant, so E[x] = x. We continue to calculate E[x' - x]:

376
$$E[x'-x] = E[x'] - E[x] = x(1-x)(1-\beta) + o(1-\beta)$$
 (27)

where when substituting $1-\beta$ with s, we get the same result as Durrett (2008) which is the desired result.

Proposition: $Ne = \alpha N + (1 - \alpha)$, where Ne is the effective population size of our binary model.

380 **Proof:** The variance of a DM distribution is:

$$V(X') = N \frac{\alpha_1}{\alpha_1 + \alpha_2} (1 - \frac{\alpha_1}{\alpha_1 + \alpha_2}) (\frac{N + \alpha_1 + \alpha_2}{1 + \alpha_1 + \alpha_2})$$
(28)

382 And again, we want to use frequencies so:

$$V(\frac{X'}{N}) = \frac{1}{N^2}V(x') \tag{29}$$

Putting it together with our model's notations:

$$V(x') = \frac{1}{N^2} N \frac{x}{x + (1 - x)\beta} \left(1 - \frac{x}{x + (1 - x)\beta}\right) \left(\frac{N + \alpha' x N + \alpha' N (1 - x)\beta}{1 + \alpha' x N + \alpha' N (1 - x)\beta}\right)$$
(30)

Like Durret, we'll use the zero order of the approximation when $\beta \approx 1$,so:

$$\frac{x}{x + (1 - x)\beta} \approx x \tag{31}$$

388 and we also use $\beta \approx 1$ for the entire variance expression and get:

$$V(x') \approx \frac{1}{N}x(1-x)\left(\frac{N+\alpha'xN+\alpha'N-\alpha'xN}{1+\alpha'xN+\alpha'N-\alpha'xN}\right)$$
$$= x(1-x)\left(\frac{1+\alpha'}{1+\alpha'N}\right)$$
(32)

390 Again following Durret we want to calculate:

$$V(x' - x) = V(x') - V(x) \approx x(1 - x)(\frac{1 + \alpha'}{1 + \alpha' N})$$
(33)

392 because x is a constant so V(x) = 0

In our model, α' is the odds ratio of a parameter we called "indicator weight", denoted in our 394 model as α , so:

$$\alpha' = \frac{\alpha}{1 - \alpha} \tag{34}$$

Combining eq. (33) and eq. (34) we get:

$$V(x'-x) \approx x(1-x)\left(\frac{1+\frac{\alpha}{1-\alpha}}{1+\frac{\alpha}{1-\alpha}N}\right)$$

$$= x(1-x)\left(\frac{\frac{1-\alpha+\alpha}{1-\alpha}}{\frac{1-\alpha+\alpha}{1-\alpha}}\right)$$

$$= x(1-x)\left(\frac{1}{1-\alpha(1-N)}\right)$$

$$= x(1-x)\left(\frac{1}{\alpha N+(1-\alpha)}\right)$$

$$= x(1-x)\frac{1}{N_e}$$
(35)

Using our substitute for a selection coefficient, $1 - \beta$, and the effective population size N_e , we can estimate the fixation probability and time of our binary model.

400 The fixation probability derived from Kimura is therefore:

$$P_{fix} = \frac{1 - e^{-2(1-\beta)N_e x}}{1 - e^{-2(1-\beta)N_e}}$$
(36)

where x is the initial frequency of the advantageous phenotype \hat{A} .

The time to fixation can be estimated by:

$$T_{fix} = \frac{1 - P_{fix}}{1 - \beta} \int_0^x \frac{e^{2(1-\beta)\xi} - 1}{\xi(1-\xi)} d\xi + \frac{P_{fix}}{1-\beta} \int_x^1 \frac{1 - e^{-2(1-\beta)(1-\xi)}}{\xi(1-\xi)} d\xi$$
 (37)

where the integrals cannot be solved in closed form, so we can only estimate them numerically.

To validate our math we ran multiple simulations comparing our binary model with the classic Wright-Fisher model, using different α and β each time, and using the corresponding values of s and N_e for the WF simulations. In fig. 4 we changed α (and N_e accordingly) and used a constant β . In fig. 5 we changed β and used a constant α . In both cases we can see that the two models behave similarly, and both are approximated well by the Kimura's equations: eq. (36) and eq. (37).

Changing environment

412 After finding good estimations for our model in a constant environment, when the favorable trait is always \hat{A} , we want to find an estimation for our model in a changing environment.

For that we will find an expression for the expected and variance of the change in frequency between t generations. Let $s_t = N(1 - \beta_t)$, and $S_n = \sum_{i=1}^n s_i$, where β_t is $\beta(A)$ at time/generation t.

416 **Proposition:** $E\left[\frac{X_t}{N} - x\right] \simeq \frac{1}{N} S_t x(1-x)$, $V\left(\frac{X_t}{N}\right) \simeq \frac{1}{N_e} t x(1-x)$, where x is the initial frequency of the favorable/invading trait and X_t is the number of individuals with the trait at time t.

The proof is based on the proof of Ram et al. (2018), proving a similar scenario.

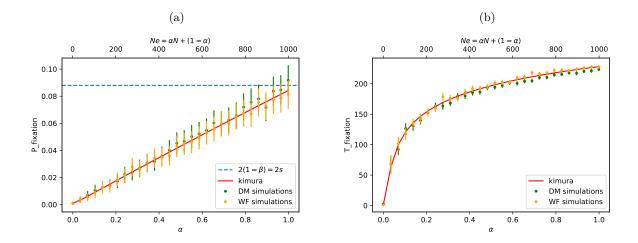


Figure 4: Comparison of the DM approximation and the WF model for different values of the effective population size. The approximation seems very good, and is also condensed around the mathematical equation expectancy. Error bars are 95% confidence intervals. Effective population calculated by $N_e = \alpha N + (1 - \alpha)$. 5,000 simulations per data point, N = 1,000, $\hat{A} = 1$, A = 0.7, $J = 1,1 - \beta = s = 0.044$.

Proof by induction: From eq. (27) we know that

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$$E\left[\frac{X_{t+1}}{N} - \frac{X_t}{N} \middle| X_t\right] = \frac{X_t}{N} \left(1 - \frac{X_t}{N}\right) (1 - \beta_{t+1})$$

$$= \frac{1}{N} \frac{X_t}{N} \left(1 - \frac{X_t}{N}\right) s_{t+1}$$
(38)

Also note that using the definition of $V(y) = E[y^2] - (E[y])^2$

$$E\left[\frac{X_t}{N}\left(1 - \frac{X_t}{N}\right)\right] = E\left[\frac{X_t}{N} - \left(\frac{X_t}{N}\right)^2\right]$$

$$= E\left[\frac{X_t}{N}\right] - E\left[\left(\frac{X_t}{N}\right)^2\right]$$

$$= E\left[\frac{X_t}{N}\right] - V\left(\frac{X_t}{N}\right) - \left(E\left[\frac{X_t}{N}\right]\right)^2$$
(39)

we can now use the induction assumption of $V(\frac{X_t}{N})$ and get

$$E\left[\frac{X_t}{N}\left(1 - \frac{X_t}{N}\right)\right] \simeq E\left[\frac{X_t}{N}\right] \left(1 - E\left[\frac{X_t}{N}\right]\right) - \frac{1}{N_e}tx(1 - x) \tag{40}$$

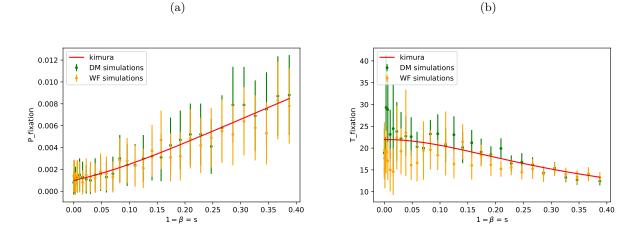


Figure 5: Comparison of the DM approximation and the WF model for different values of the selection coefficient, manifested as success bias in our model. The approximation seems good, and is also condensed around the mathematical equation expectancy. Error bars are 95% confidence intervals. Effective population calculated by $N_e = \alpha N + (1 - \alpha)$. 5,000 simulations per data point, N = 1,000, $\hat{A} = 1$, A = 0.7, J = 1, $\alpha = 0.01$.

From eq. (38) we know that

426

428

$$E\left[\frac{X_{t+1}}{N} - \frac{X_t}{N}\right] = \frac{1}{N} s_{t+1} E\left[\frac{X_t}{N} \left(1 - \frac{X_t}{N}\right)\right]$$

$$\simeq \frac{1}{N} s_{t+1} \left(E\left[\frac{X_t}{N}\right] \left(1 - E\left[\frac{X_t}{N}\right]\right) - \frac{1}{N_e} tx(1-x)\right)$$

$$\simeq \frac{1}{N} s_{t+1} \cdot E\left[\frac{X_t}{N}\right] \left(1 - E\left[\frac{X_t}{N}\right]\right) - \frac{1}{N_e N} s_{t+1} tx(1-x)$$
(41)

Now we'll omit $O(\frac{1}{Ne \cdot N})$ and get

$$E\left[\frac{X_{t+1}}{N} - \frac{X_t}{N}\right] \simeq \frac{1}{N} s_{t+1} \cdot E\left[\frac{X_t}{N}\right] \left(1 - E\left[\frac{X_t}{N}\right]\right) \tag{42}$$

We'll now look at the induction assumption to see that

430
$$E\left[\frac{X_t}{N} - x\right] = E\left[\frac{X_t}{N}\right] - E[x] = E\left[\frac{X_t}{N}\right] - x, \tag{43}$$

so using the assumption we get

$$E\left[\frac{X_t}{N}\right] \simeq \frac{1}{N} S_t x (1-x) + x$$

$$1 - E\left[\frac{X_t}{N}\right] \simeq 1 - \frac{1}{N} S_t x (1-x) + x \tag{44}$$

we'll use both expressions in eq. (42) and get

434
$$E\left[\frac{X_{t+1}}{N} - \frac{X_t}{N}\right] \simeq \frac{1}{N} s_{t+1} \left(\frac{1}{N} S_t x(1-x) + x\right) \left(1 - \frac{1}{N} S_t x(1-x) + x\right)$$
$$\simeq \frac{1}{N} s_{t+1} \cdot x(1-x)$$
(45)

after again omitting $O(\frac{1}{N^2})$ parts of the equation. To conclude our proof, we see that

$$E\left[\frac{X_{t+1}}{N} - x\right] = E\left[\frac{X_{t+1}}{N} - \frac{X_t}{N}\right] + E\left[\frac{X_t}{N} - x\right]$$
(46)

so again using the induction assumption, together with eq. (45) we get

$$E\left[\frac{X_{t+1}}{N} - x\right] \simeq \frac{1}{N} s_{t+1} \cdot x(1-x) + \frac{1}{N} S_t \cdot x(1-x)$$

$$\simeq \frac{1}{N} x(1-x)(S_t + s_{t+1})$$

$$\simeq \frac{1}{N} S_{t+1} x(1-x)$$
(47)

which proves the first part of our preposition.

440 For the second part, we'll use a property of variance:

$$V\left(\frac{X_{t+1}}{N}\right) = E\left[V\left(\frac{X_{t+1}}{N}\middle|X_t\right)\right] + V\left(E\left[\frac{X_{t+1}}{N}\middle|X_t\right]\right) \tag{48}$$

using eq. (38) we see that:

438

$$E\left[\frac{X_{t+1}}{N}\middle|X_{t}\right] - E\left[\frac{X_{t}}{N}\middle|X_{t}\right] = \frac{1}{N}s_{t+1}\frac{X_{t}}{N}\left(1 - \frac{X_{t}}{N}\right)$$

$$E\left[\frac{X_{t+1}}{N}\middle|X_{t}\right] = \frac{X_{t}}{N} + \frac{1}{N}s_{t+1}\frac{X_{t}}{N}\left(1 - \frac{X_{t}}{N}\right)$$

$$(49)$$

Using eq. (35) we get:

$$V\left(\frac{X_{t+1}}{N}\middle|X_t\right) = \frac{1}{N_e}\frac{X_t}{N}\left(1 - \frac{X_t}{N}\right) \tag{50}$$

and using the equation $y'(1-y') \simeq y(1-y)$ on the first part of eq. (48) we get:

$$E\left[V\left(\frac{X_{t+1}}{N}\middle|X_{t}\right)\right] = \frac{1}{N_{e}}E\left[\frac{X_{t}}{N}\left(1 - \frac{X_{t}}{N}\right)\right] \simeq \frac{1}{N_{e}}x(1 - x)$$
(51)

and moving on to simplify the second part of eq. (48) using eq. (49):

$$V\left(E\left\lceil \frac{X_{t+1}}{N} \middle| X_t \right\rceil\right) = V\left(\frac{X_t}{N} + \frac{1}{N}s_{t+1}\frac{X_t}{N}\left(1 - \frac{X_t}{N}\right)\right)$$
(52)

and now, because $\frac{X_t}{N}$ is a frequency, i.e $0 \le \frac{X_t}{N} \le 1$, we know that $V\left(\frac{X_t}{N}\left(1 - \frac{X_t}{N}\right)\right) \le \frac{1}{4}$. We therefore see that:

452
$$V\left(\frac{1}{N}s_{t+1}\frac{X_t}{N}\left(1 - \frac{X_t}{N}\right)\right) \le \frac{1}{4N^2}s_{t+1}^2 \tag{53}$$

and so it can be ignored. Combining our equations we get:

$$V\left(E\left\lceil \frac{X_{t+1}}{N} \middle| X_t \right\rceil\right) = V\left(\frac{X_t}{N}\right) + O\left(\frac{1}{N^2}\right) \simeq V\left(\frac{X_t}{N}\right)$$
 (54)

Using the induction assumption and eq. (51):

456
$$V\left(\frac{X_{t+1}}{N}\right) \simeq \frac{1}{N_e}x(1-x) + \frac{1}{N_e}tx(1-x) \simeq \frac{1}{N_e}x(1-x)(t+1)$$
 (55)

proving the second part of our preposition.

458 Following our proof, we can say that after many cycles, we can use a modified version of our fixation probability:

$$P_{fix} = \frac{1 - e^{-2\frac{S_n}{n}N_e x}}{1 - e^{-2\frac{S_n}{n}N_e}}$$
 (56)

where $\frac{S_n}{n} = \frac{k-l}{k+l}(1-beta)$, n=k+l. Put into words, we use the average selection coefficient of a cycle (k+l) as the selection coefficient in our original equation. In our proof we showed that the expected change in frequency and variance is only manifested in the selection coefficient S_n , and that we can use those modified equation as a base for Kimura's equation.

We wanted again to validate our results, using simulations. This time, the number of parameters increased: in addition to α, β , there are also k, l as model parameters.

We again tried different variations of the parameters, changing only one of them at a time. In 468 fig. 6 we can see that α on it's own doesn't cause any deviation for the the estimation. β however affects the results greatly.

We plotted along the modified estimation the original Kimura's estimation, as a limiter. We suspect that when β is too large, there won't be many cycles in the simulations. This might

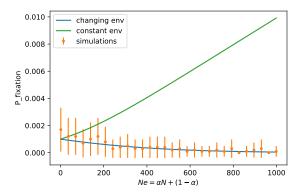
2 happen if either the population reaches a high frequency of the ideal trait after only a few cycles, or it get extinct very quickly, because the advantage it had in the k generations wasn't sufficient,

474 and the same s becomes a greater disadvantage when the environment changes, resulting in a fast extinction.

In the larger values of beta we even see a deviation from the original estimation environment, but it's to be expected, because Kimura's equations are only viable for small s values.

We then also tried changing the composition of the cycle, by keeping a constant n = 40, but changing k, l accordingly.

In fig. 7 we see that the larger k relative to l, the closer the modified equation is to the original estimation of the constant environment. When using higher values of n, the simulation results doesn't fit the equation result as with lower values. This is due to the fact that our proof, and



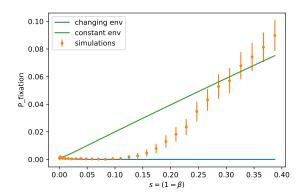


Figure 6: Model simulations compared with both the constant environment and the changing environment equations with different effective populations sizes and selection coefficients. Changing the effective population size doesn't affect the approximation, and it is condensed the mathematical expected values across all values. High values of success bias (s > 0.1) will distance the simulations from the changing environment expected values. Very high values (s > 0.35) will even deviate from the constant environment expected values. This is expected because Kimura's approximation are only viable for low selection coefficient values. 10,000 simulations per data point, N = 1,000, $\hat{A} = 1$, A = 0.9, J = 1.

therefore our equation is more accurate when more cycles occur. When n is high, there will be less cycles, and the simulations will get closer to the constant environment equation.

Discussion

486 Cultural transmission is the phenomenon of which cultural elements, in the form of attitudes, values, beliefs, and behavioral patterns, are transmitted between individuals, typically via copying. 488 Some cultural traits can be more likely to be copied by others, regardless of their frequency in the population. Such transmission biases are common in cultural transmission processes. Many models 490 are based on the assumption that success can be correctly identified, and easily copied. Here we assume that success isn't correctly identified, therefore individuals may use other indicators to try 492 and estimate the success of potential role-models. We believe, as Fogarty et al. (2017) suggest, that prestige biases are more common in nature than success biases, since estimating success is 494 harder. We believe prestige is composed of two main components: a trait that indicates success (but doesn't guarantee it), and the influence the individual already has on others, i.e number of individuals already chose him as a role-model. We suggest a model for prestige bias, inspired by 496 the model Boyd and Richerson (1988) have suggested, and added the influence bias to it. We approximated our models using various distributions, and compared them to the original model 498 using simulations. We showed that a Rich getting richer type of model can be approximated well by the general binomial distribution and the dirichlet multinomial distribution. We experimented 500 with constant and changing environment in our model, and created a variation of a binary model

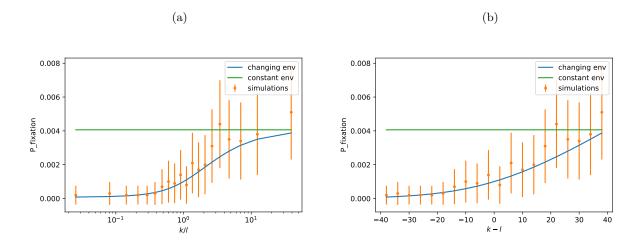


Figure 7: Model simulations compared with both the constant environment and the changing environment equations for different compositions of the environment cycle. When k < l the approximation is good. When k > l, the approximation and the simulations are both very close to the constant environment approximation. 10,000 simulations per data point, N = 1,000, $\hat{A} = 1$, A = 0.8, A =

for easier mathematical and computational analysis. We believe that in this era of social media it is easy to estimate one's influence over others. It is therefore crucial to model the cultural biases
 more realistically than success bias based model, and we believe including influence is crucial for that purpose.

With a more realistic model of a common cultural transmission bias, we may be able to better understand decision-making processes in humans, including life-changing choices such as occupation or a life partner. Our model can be expanded in multiple ways: observing the effects of different bias functions, including errors in estimating the influence, combining factors of cost when copying from an influential role model (not all could afford to copy from the most popular role-model), and analyzing the differences when including several optimal values for the indicator trait (multiple preference traits in the population).

Summaries

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514 Animal leadership (King and Cowlishaw, 2009)

There are two main approaches to decision makings of groups in nature: leadership and consensus. 516 Prey leaders would lead the pact when traveling, while other animal group leaders will decide on a nesting site or foraging patches. They found out that leadership is observed mostly when there 518 is a profound social network in the group, and when there are individuals that present leadership behavior. Leaders would usually be high ranking members in the group, such as elders, individuals 520 with many kin relations, or posses other dominant traits. When no individual posses such traits, or when the social network is lacking, a consensus is more likely to occur. When a leader is present, 522 they will have greater selection costs, such as higher risk for predation, being poisoned by unknown experimental patch, but also greater benefits. For example, given the route to the foraging site was successful, the leader and his closest followers would gain most of the food, unlike in a consensus, 524 where the food would be shared more equally. It appears leaders appear in simple organisms as well, like fish. In these organisms however, the leader would usually be the hungriest or the weakest, while 526 the rest would prefer to follow, minimizing their costs. In baboons however, King and Cowlishaw 528 (2009) describe many benefits for the closest associates of the dominant male, such as protection from predators. This is an instance of sexual-selection, where the leader, whose survival chances are lower, gains more sexual partners due to the benefits. ("The greater the risk, the greater the 530 reward") In humans, leadership also has its perks and costs. Leaders can make decisions that would 532 benefit them and their closest followers the most, while still maintaining group cohesion. However, wrong decision making that would harm the group could result in harm (media, social status, even 534 violent behavior of subjects on certain cases).

Prestige as a type of leadership (Van Vugt and Smith, 2019)

In their paper, Van Vugt and Smith (2019) suggest a different view of leadership. They say that most discussions assume there is one type of leadership, and so they differ in their definition.

Van Vugt and Smith (2019) suggest a way to solve said contradiction by defining two types of leaderships: prestige-based and dominance-based. They present classical views of leaderships by Confucius and Machiavelli. Confucius views leaders as role models who exercise influence through possessing superior knowledge, skills, and (outstanding) personal qualities. This description is very similar to our indicator trait. By contrast, Machiavelli views leaders as rulers who exercise influence by imposing costs through (the threat of) punishment. They say that these two opposing views are both partially supported by the available evidence but each one on its own offers an incomplete view into the complex and dynamic processes of leadership.

Our current model doesn't reflect the model described in this article, but several adjustments could be made in order to match it. If we assume there's a correlation between trait value to a type of leadership (so in our binary model, one trait would be of prestige, and the other of dominance) we can implement their suggested model. For that we would need to add cost-benefit parameters,

so the ones choosing prestige will be rewarded, but pay more, while the ones choosing dominance would pay less, but gain less benefits. It could be interesting to see the dynamics and relations
 between our model parameters and these cost-benefit parameters.

Evidence from Fijian villages for adaptive learning biases (Henrich and Broesch, 2011a)

Henrich and Broesch (2011a) researched Fijian villages, looking for evidence of social learning biases 556 and their origins. They mention that:

- evolutionary theorists propose that natural selection has favored the emergence of psychological biases for learning from those individuals most likely to possess adaptive information.
- Their goal is to bridge from the laboratory to the field by examining if and how these biases emerge in a small-scale society. During their research they found that:
- Fijian villagers (ages 10 and up) are biased to learn from others perceived as more successful/knowledgeable, both within and across domains (prestige effects).
- Their research shows promising evidence for our prestige model, suggesting that copying from others who are *perceived* as successful, rather than actually are successful. In their paper, they show that the social networks representing copier-role-model relationships are centralized, suggesting:
- This degree of centralization is consistent with the prediction that people substantially share notions about who is a good cultural model (network centrality), but that individuals' model selections are influenced by multiple factors.
- 570 We see here support for both our indicator trait and our influence bias in their data.

Using social influence to reverse harmful traditions (Efferson et al., 2020)

572 Social influence could be a powerful tool to end traditions, harmful or advantageous. Harmful traditions can be child marriage, open defecation, and domestic violence, to name a few. Efferson 574 et al. (2020) suggest a mechanism called *spillover*. By their definition, a spillover is when an intervention affect a large enough group in a target population, so that others not included in the 576 intervention starts changing their behavior as well. In their research, they found that there are individuals who act as agents, who are often looked upon, and therefore they are ideal targets for interventions. This is the same concept as our role-models, where a more prestigious individual 578 will be copied more, therefore spreading his trait wider in the population. Their research support 580 therefore in our assumption that there are social biases, conformist influence specifically. They also suggest a way to use this phenomena to change existing traditions in a population. It is very clear 582 however, that just as it can be used to end harmful traditions, the same agents could be used for any negative way that comes to mind.

The evolution of prestige (Henrich and Gil-White, 2001)

One cannot mention prestige and cultural transmission without the work of Henrich and Gil-White 586 (2001). They as well distinguish between prestige and dominance. The latter is is defined by acquiring social status by using aggression, intimidation and violence. They also mention dominance is more common than prestige in non humans. Their definition of prestige is somewhat synonymous 588 with ours. Specifically prestige is composed both of estimation in the eyes of people (our indica-590 tor/success trait) and commanding position in people's minds, i.e number of copiers people think they have, which they define as *influence* (similar to our definition for influence). In their paper, 592 they show that prestige evolved from natural selection, as an efficient process to extract reproductive benefit from the flow of socially transmitted information. Simply put, prestige is a natural step 594 where social learning exists, due to saving costs of individual learning. It could be interesting in the future to enhance our model with this idea, i.e observing the copier trait of evaluation, rather than only observing the evolution of the indicator trait copied. Henrich and Gil-White (2001) suggest 596 that the most skilled role-models will, on average, end up with most copiers. This is consistent with our findings, and in a way our model is a mathematical equivalent to their suggested model. 598

Biased learning may be disfavored when payoffs are uncertain (Takahashi and 600 Ihara, 2019)

Takahashi and Ihara (2019) remind that social learning not only takes the form of random copying 602 of other individuals, but also involves learners' choice of what to learn or from whom to learn. They suggest a best-of-K model where an individual samples k role-models and choose the one he 604 deems most "successful". They mentioned that a previous mathematical analysis has shown that it may sometimes result in maladaptive cultural evolution when the payoffs associated with cultural variants vary stochastically. In such a case, learners may be selectively disfavored and in the long 606 run replaced by unbiased learners, who simply copy someone chosen at random. They develop new 608 mathematical models that are more simplified and mathematically tractable. They found that bestof-k learning, unlike unbiased learning, can facilitate the invasion of an on average inferior variant that sometimes gives a very high payoff. Our model, which includes influence bias, is consistent 610 with that claim. When a maladaptive trait is "piggybacking" a role-model with high influence, said trait could spread in the population, as mentioned. In addition, they show that best-of-k learning 612 can be stable against invasion by unbiased learning if social learning is sometimes combined with 614 individual learning. Our model is based on copying based learning only, but it could be interesting to combine it with individual learning and see how it affects the dynamics of the population.

616 Prestige in medicine (Norredam and Album, 2007)

So far we presented the existence of prestige in nature and in humans. We mostly discussed it in 618 regard of social ranking in animals, and what are the costs and benefits. In humans, we mostly addressed spread of adaptive or maladaptive traits due to the prestige bias. Norredam and Album

(2007) present a specific and important effect of prestige - its significance for medical specialties and 620 diseases. They examined literature from 1950 to 2005 regarding the effects of prestige on medicinal 622 practices. They discovered that active, specialized, biomedical, and high-technological types of medicine on organs in the upper part of the bodies of young and middle-aged people were accorded high levels of prestige, while medicine with opposite characteristics had low levels of prestige. They 624 have concluded that such differences in prestige bear consequences for actual priority setting in healthcare systems. They discovered that surgery counts as the most prestigious specialty, while 626 psychiatry is the less prestigious. In addition, doctors tend to rank practices that require more time 628 to master as more prestigious, while other procedures that are considered easier are less prestigious. Norredam and Album (2007) found that the advance in technology and research was in accordance 630 with the prestige rankings.

Collective brains (Muthukrishna and Henrich, 2016)

632 We showed cultural transmission is a process that manifests in many species, with emphasis on humans. We also displayed similarities between this process and genetic transmission, while presenting differences between them, specifically selective biases such as influence and prestige. Muthukrishna 634 and Henrich (2016) offer a take on prestige as a factor of human physical evolution. They present a concept called *cultural brains* - brains that evolved primarily for the acquisition of adaptive knowl-636 edge. They build on the hypothesis of Dunbar (2009) that shows that larger, more complex brains can store and manage more information and in turn, this information can support the costs of a 638 larger brain. Muthukrishna and Henrich (2016) built a model that predicts a strong relationship 640 between brain size and group size, because group size also provides access to more adaptive knowledge. They later present their *cumulative cultural brain* hypothesis, an approach which proposes 642 that human brains have evolved with an ability and proclivity for selective, high fidelity social learning. As part of this process, there are a variety of strategies and biases that have evolved to hone 644 in on the most adaptive knowledge. These strategies and biases include direct and indirect cues of the popularity of cultural traits (e.g. success and prestige biases). In short, they suggest that some of the reasons for the extreme increase in brain size in humans, are the ability to "cheaply" 646 acquire adaptive knowledge, i.e transmission biases, such as prestige.

Prestige-biased cultural learning (Chudek et al., 2012)

Culture—gene coevolutionary (CGC) researchers theorize that humans should possess several learning biases which robustly enhance the fitness of cultural learners. Chudek et al. (2012) report the 650 first direct tests in children of CGC's prediction of prestige bias, a tendency to learn from individuals to whom others have preferentially attended, learned or deferred. Their definition of prestige 652 is similar to our *influence bias*, and brings concrete proof of its existence and effects. Their study showed that the odds of 3-4 years-old children learning from an adult model to whom bystanders 654 had previously preferentially attended for 10 seconds were over twice those of their learning from a model whom by standers ignored. In addition to this first study, they also discovered prestige 656

effects are domain-sensitive. They saw that prestigious models were listened to by most when demonstrating artifact-use, but not as much as when presenting food preferences. It lead Chudek et al. (2012) to believe that when the trait is costly to learn individually, prestige will have a higher effect. It would be interesting to include costs in our model to try and observe these effects and their dynamics in the simulations of a larger population than this study.

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