Prestige as a Driving Force in Cultural Transmission

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Abstract

Copying role-models can be an efficient method for acquiring knowledge. A common bias when choosing a role-model to copy is success bias: copying whoever appears more successful. This bias depends on the performance of the role-model alone, with no other factors. We propose an additional bias that may be prevalent in cultural transmission: influence bias, in which role-model choice is affected by the number of individuals that have already copied each potential role-model. We combine success and influence bias together to a "prestige bias" and analyze its effects on cultural-evolutionary dynamics using mathematical analysis and stochastic simulations. We find analytic approximations to our stochastic model, facilitating further mathematical analysis and reducing the computational complexity of simulations. We validate these approximations using simulations, and demonstrate their robustness to model assumptions. We also find approximations to the fixation probability and the fixation time of an invading advantageous cultural trait, in both a constant and changing environment, which resemble Kimura's classical formulas. These approximations show that success bias effectively plays the part of natural selection, whereas influence bias effectively reduces the population size. It also accelerates the evolutionary dynamics, as can be expected in a *rich-getting-richer* process. Our model may provide a better description of human cultural transmission, especially in the last years where social networks are very popular. Further work is needed to test if this model could predict various phenomena in human cultural evolution when extended with the effects of selection and innovation.

Introduction

Cultural transmission. In cultural transmission, individuals transmit cultural traits (i.e., behavior, beliefs, norms) to one another, typically via learning and demonstrating (Cavalli-Sforza and Feldman, 1981). Examples for cultural traits in humans are behavioral patterns, such as personalities and habits, transmitted via both verbally and by observations (O'Brien et al., 2010). Although cultural transmission is most common in humans, it is also observed in other animals such as chimpanzees (Horner et al., 2010; Kendal et al., 2015), dolphins and whales (Whitehead, 2017). In elephants, McComb et al. (2001) showed that once a matriarch is removed from the group, the group's survival instincts are inferior and that "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." By playing audio recordings of African elephants, they showed that groups with a matriarch recognize and react better to hostile or friendly calls than groups without a matriarch. Battesti et al. (2012) showed that choice of oviposition site in fruit flies is culturally transmitted: inexperienced flies that spent some time with experienced flies chose the same type of oviposition site even without directly observing this behavior. How the information is transmitted is still an open question, but it has been suggested that flies may use olfactory cues like other animals, such as rodents and bees.

Direction of transmission. Similar to genetic transmission, culturally transmitted traits can be transmitted from parents to offspring, and their effects of can be physiological rather than behavioral. For example, parents can "teach" their children to be strong or tall, within some biological limits, by instructing them to maintain a specific diet and engage in physical activity. Contrary to genetic transmission, cultural transmission can be non-vertical, that is, traits may be transmitted via social learning from non-parental individuals, and even unrelated individuals such as teachers, leaders, media, or any stranger that interacts with the learning individual. Thus, cultural transmission may combine vertical transmission, where parents transmit to their offspring; oblique transmission, where adults transmit traits to unrelated offspring; and horizontal transmission, where peers from the same age cohort transmit to one another. Vertical transmission is also possible in the opposite direction: parents may copy traits from their offspring (Cavalli-Sforza and Feldman, 1981; Creanza et al., 2017).

Transmission biases. In social learning, transmission biases cause a trait to have a disproportionate probability to be transmitted compared to its frequency in the population. Although more common in cultural transmission, transmission biases do occur in genetic transmission. For example, *wtf genes* in yeast bias their transmission to the gamete by secreting a long life-expectancy poison together with a short life-expectancy antidote, so that a gamete without the gene will perish because the poison will outlive the antidote (Eickbush et al., 2019). Importantly, even when a trait is disfavored by natural selection, it may still spread in a population due to transmission biases that are strong enough to overcome selection (Boyd and Richerson, 1988, Ch. 8 pg. 279). Cohen et al. (2021) show that cooperative behavior can evolve in an individual due to horizontal transmission bias even when there is no benefit to it, or when it benefits its competitors.

64 **Success bias.** Rendell et al. (2010) have conducted a tournament between learning strategies. Each strategy defines when individuals observe and copy from others, and when they engage in individual

66 learning, in which an individual learns a cultural trait on his own. The best strategies had a high frequency of social learning relative to individual learning, even when the transmission error was

68 almost 50%. From these results we understand that all the winning strategies were mostly based on success biased social learning, meaning it contributed more to the general success of the population

70 than individual learning. However, all winning strategies included individual learning to some degree, implying that success-biased learning alone isn't the best way to advance, even when success is

72 measured accurately.

Evaluating success. Boyd and Richerson (1988, Ch. 5) suggest that the evaluation of success can

74 be divided into three groups: *direct bias*, *indirect bias* and *frequency-dependent bias*. A direct bias occurs when a variation of a trait is more attractive than others, and is evaluated by *directly* testing the

76 variation of the trait. For example, an individual observing a Ping-Pong match can attempt both of the observed paddle grips to determine which grip is better. An indirect bias occurs when an individual

78 uses the value of one trait to determine the attractiveness of another, so it *indirectly* evaluates the attractiveness of the role-model. For example, an observer may copy the paddle grip of the Ping-Pong

80 player who scored more points in the match, thus indirectly evaluating the grip by the points scored. A frequency-dependent bias occurs when an individual has a probability to copy a variant of the trait that

82 higher or lower than trait's frequency among demonstrators. For 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

84 frequency-biased, or in this case, conformist. Frequency bias could be negative, i.e., non-conformist bias. Conformity and non-conformity are well-known biases in cultural transmission (Molleman et al.,

86 2013), and its effect on cultural evolution have been studied with both models (Denton et al., 2020a,b) and experiments (Aljadeff et al., 2020).

88 **Prestige.** 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, Ch.

90 8) and Fogarty et al. (2017) suggest 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

92 in a population if indirect biases are strong enough. They suggest that such biases could lead to a runaway process caused by a cultural equivalent of sexual selection (Andersson, 1994). On the other

94 hand, Henrich and Broesch (2011) suggest that prestige biases, over generations, can lead to cultural adaptations, and that although prestige can lead to maladaptive traits spreading in the population, it

96 can also accelerate the spread of adaptive traits. Prestige is often mentioned in the cultural-evolution literature, but seldom modeled.

98 **Influence bias.** Today, social media provides an easy way to estimate the influence individuals have over others, and therefore may have an effect on decision making. Online social networks such as

100 *Facebook* or *Instagram* are known to affect the social influence of individuals (Anagnostopoulos et al., 2008; Peng et al., 2018; Diga and Kelleher, 2009). For example, specific marketing practices

102 were invented following this realization (Lee et al., 2012), Here we propose an indirect bias that we

call *influence bias*, in which the choice of a role-model depends on the the choices made by other individuals that have already chosen a role-model. This bias depends on the state of a role-model rather than a trait, in contrast to frequency biases such as conformity, which depend on the frequency of a trait in the population or in a sample of role-models. We define a model for prestige bias that combines both success and influence biases, provide analytic approximations for this model, and analyze its dynamics.

Models and Methods

We begin with a continuous trait model with indirect bias suggested by Boyd and Richerson (1988), develop an extension with influence bias, and then develop a model with a dichotomous trait. We implemented our stochastic models and approximations, performed statistical analyses, and produced figures using Python (Van Rossum et al., 2007) with NumPy (Van Der Walt et al., 2011) and Matplotlib (Hunter, 2007).

Source code is available at this notebook.

Continuous trait

We follow the model of Boyd and Richerson (1988), assuming only oblique transmission of the indicator trait and omitting the indirect trait in order to reduce model complexity. We consider a population of N individuals, described by a single indicator trait with a continuous value. Each generation, N naive individuals, or copiers, choose an individual from the previous generation, or role-models, from which they will copy their trait. Similar to a Wright–Fisher model, we assume non-overlapping generations such that the entire population is replaced in each generation. The population at time t can be described by $\mathbf{A}(t) = \big(A_1(t), \ldots, A_N(t)\big)$ where $A_i(t)$ is trait value of individual i at time t. We assume the initial population is drawn from a standard normal distribution, $\mathbf{A}(0) \sim N(0, 1)$. Cultural transmission is modeled by a function F such that

$$A_i(t + 1) = F_i(\mathbf{A}(t)) \,. \tag{1}$$

Success bias. Boyd and Richerson (1988, Ch.8, p.247-249) describe a blended transmission algorithm by defining F as a weighted average of the traits of all role-models,

$$F_i(\mathbf{A}) = \sum_{j=1}^{N} G_{i,j} \cdot A_j \,, \tag{2}$$

where $G_{i,j}$ is the success bias of role-model j in the eyes of copier i,

$$G_{i,j} = \frac{\beta(A_{i,j})}{\sum_{k=1}^{N} \beta(A_{i,k})} \,, \tag{3}$$

4

$A_{i,j}$ is the absolute indicator trait value copier i estimates for role-model j with some error $e_i \sim N(0, \frac{1}{\eta^2})$,

$$A_{i,j} = A_j + e_i, \tag{4}$$

and $\beta(\cdot)$ is the bias function that quantifies the success bias of a role-model,

$$\beta(A_{i,j}) = b \cdot \exp\left(-\frac{(A_{i,j} - \hat{A})^2}{2J}\right), \tag{5}$$

with $\hat{A}$ as the arbitrary optimal indicator value, and J and b as parameters that control the bias strength. Therefore, $G_{i,j}$ is a relative success score that copier i assigns to role-model j, equivalent to relative fitness in evolutionary-genetic transmission models.

Boyd and Richerson (1988) note that the deterministic blended transmission algorithm they use has alternatives. We thus develop a similar stochastic model with transmission from a single random role-model. Instead of eq. (2), we define the transmission function F as a random variable with its distribution given by

$$\Pr\left(F_i(\mathbf{A}) = A_j\right) = G_{i,j}, \tag{6}$$

such that $G_{i,j}$ gives the probability of copier i to choose to copy the trait of role-model j.

Influence bias. Here we introduce a new element to the model. We assume that in each generation, copiers choose their role-models one by one. We denote $K_{i,j}$ as the number of copiers that chose role-model j after copier i chose a role-model. Thus, i out of N copiers already chose a role-model, $\sum_{j=1}^{N} K_{i,j} = i$, and there are $N - i$ copiers that have yet to choose a role-model. The stochastic process of role-model choice,

$$\left\{\mathbf{K}_i = (K_{i,1}, \ldots, K_{i,N})\right\}_{i=1}^{N}, \tag{7}$$

is described by the recurrence equation

$$K_{i,j} = K_{i-1,j} + S_{i,j}, \quad i, j = 1, 2, \ldots, N, \tag{8}$$

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

Following eq. (6), the probability that the i-th copier chose role-model j is given by the prestige of role-model j in the eyes of copier i,

$$Pr(S_{i,j} = 1 \mid S_{1,j}, S_{2,j}, ..., S_{i-1,j}) = G_{i,j}. \tag{9}$$

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, $K_{i-1,j}$, replacing eq. (3) of Boyd and Richerson (1988) with

$$G_{i,j} = \frac{\alpha_j \cdot \beta(A_{i,j}) + (1 - \alpha_j) \cdot K_{i-1,j}}{W_i}. \tag{10}$$

Here, the bias weight α_j is a characteristic of role-model j that determines the relative significance of success and influence in his prestige, the indicator value of role-model j estimates by copier i, $A_{i,j}$, remains as in eq. (4), and W_i is a normalizing factor that sums the numerator over all role-models $(1 \leq j \leq N)$ to ensure $\sum_{j=1}^{N} G_{i,j} = 1$.

Dichotomous trait

To allow for mathematical analysis of the model, we introduce a simplified version where the indicator trait only has two phenotypes: the optimal phenotype $\hat{A}$ and the sub-optimal phenotype A. All role-models with the same phenotype will contribute to the probability of that phenotype to be transmitted, and thus influence is determined by the number of copiers that have already chosen a role-model with either phenotype. Therefore, the probability of the i-th copier to copy phenotype A is

$$P_{i,A} = \frac{(N-X)\alpha'\beta(A) + K_{i,A}}{i - 1 + (N-X)\alpha'\beta(A) + X\alpha'\beta(\hat{A})} = \frac{(N-X)\alpha'\beta(A) + K_{i,A}}{i - 1 + (N-X)\alpha'\beta(A) + \alpha'X} \tag{11}$$

where X is the number of role-models with trait $\hat{A}$ and $K_{i,A}$ is the number of copiers that already chose A when copier i chooses a role-model and α' is the odd ratio of the indicator trait $\alpha' = \frac{\alpha}{1-\alpha}$.

We prove this equation later, based on the proof of the Dirichlet-Multinomial approximation.

Initially, we assume the population has a single individual with phenotype $\hat{A}$ and $N - 1$ individuals with phenotype A. The rest of the details follow the continuous trait model.

Results

Approximations

Our model is defined by two nested stochastic processes. Change over multiple generations is described by the phenotype distribution at each generation, $\{\mathbf{A}(t)\}_t$. The transition from one generation to the next is described by the number of copiers each role-model has after i copiers have chosen a role-model, $\{\mathbf{K}_i\}_{i=1}^{N}$. We emphasize that the models are nested: $\mathbf{A}(t+1)$ can be computed from $\mathbf{A}(t)$ by evaluating $\mathbf{K}_N$. However, the former requires iterating over eqs. (8) and (9). Thus, we sought to find an equivalent stochastic process that has the same joint distribution as $\mathbf{K}_N$. We found two approximations such processes, summarized here and explained in detail below:

1. The number of copiers of a specific role-model at each step, $K_{i,j}$, follows the *generalized binomial distribution* (Drezner and Farnum, 1993) and therefore,

    (a) 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, that is, $\mathrm{E}[K_{N,j}] = N \cdot G_{1,j}$ if trait estimation error is uniform for all copiers ($e = e_i$ for $i = 1, \ldots, N$).

    (b) the expected number of copiers of each role-model equals its relative biased indicator value, similar to the role of relative fitness in population-genetic models, that is, $\mathrm{E}[K_{N,j}] =$

$\beta(A_j + e)/\overline{\beta}$ if the bias weight is uniform for all role-models ($\alpha = \alpha_j$ for $j = 1, \ldots, N$), where $\overline{\beta} = 1/N \sum_{j=1}^{N} \beta(A_j + e)$ is the population mean estimated indicator value.

2. The role-model choice process, $\{\mathbf{K}_i\}_{i=1}^{N}$, is equivalent to a *Pólya urn* model if trait estimation error is uniform for all copiers ($e = e_i$ for all $i = 1, \ldots, N$), meaning there's no meaning for the order of copiers. Therefore, the number of copiers of all role-models, $\mathbf{K}_i = (K_{i,1}, \ldots, K_{i,N})$, follows a Dirichlet-Multinomial distribution, $\mathbf{K}_i \sim DM(N, \mathbf{G}_1)$, where $\mathbf{G}_1 = (G_{1,1}, \ldots, G_{1,N})$ is the prestige vector of all role-models in the "eyes" of the first copier. Note that here $G_{i,j}$ is only a function of the indicator values A_j and the bias weights α_j, as the estimation error is uniform, meaning all copiers are identical.

Generalized binomial distribution

The generalized binomial distribution (GBD) emerges from a series of dependent Bernoulli trials (in contrast to the standard binomial distribution in which trials are independent) and is parameterized by $GBD(n, p, \theta)$ where n is the number of trials, p is the probability of success of the first trial, and θ is the correlation between trials ($\theta = 0$ gives the standard binomial distribution).

Result 1 (GBD approximation). *The number of copiers of role-model j after i copiers have chosen a role-model follows the GBD, $K_{i,j} \sim GBD(i, \alpha_i \cdot \beta(A_j + e))$ if $e_i = e$ for all role-models $i = 1, \ldots, N$.*

Proof. We denote $Q_j(k, i) = P(K_{i,j} = k \mid K_{i-1,j})$ as the probability that exactly k out of i copiers choose role-model j given $K_{i-1,j}$ our of $i-1$ copiers chose role-model j. Using conditional probability and eq. (8),

$$Q_j(k, i) = P_j(S_{i,j} = 1 | k - 1, i - 1) \cdot Q_j(k - 1, i - 1) + P_j(S_{i,j} = 0 | k, i - 1) \cdot Q_j(k, i - 1), \quad (12)$$

where $S_{i,j} = 1$ when the i-th copier chooses role-model j. Equation (12) is equivalent to eq. (2.1) by Drezner and Farnum (1993), which completes the proof.

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

Proof. Drezner and Farnum (1993, eq. (2.3)) show that $\mathrm{E}[k] = N \cdot Q_j(1, 1)$ (modified from their notation). $Q_j(1, 1)$ is the initial probability to choose role-model j, before any role-model choices are made. $Q_j(1, 1) = G_{1,j}$ by definition, which completes the proof.

Corollary 2. $\mathrm{E}[K_{Nj}] = \alpha_j \cdot \beta(A_j + e)/\overline{\alpha \cdot \beta(A + e)}$, *where the averaging in the denominator is over the role-models index, j.*

Proof. The initial prestige of role-model j based on eq. (10) is

$$G_{1,j} = \frac{\alpha_j \cdot \beta(A_j + e)}{\sum\limits_{m=1}^{N} \alpha_m \cdot \beta(A_m + e)} \ . \tag{13}$$

226 The denominator of eq. (13) can also be formulated as:

$$\sum_{m=1}^{N} \alpha_m \beta(A_m + e) = N \cdot \overline{\alpha \cdot \beta(A + e)} \,, \tag{14}$$

228 where $\overline{\alpha \beta(A + e)}$ is the mean value of $\alpha_m \cdot \beta(A_m + e)$ for all m. Using eq. (14) we get,

$$\mathrm{E}[K_{Nj}] = \alpha_j \cdot \beta(A_j + e) \big/ \overline{\alpha \cdot \beta(A + e)} \,, \tag{15}$$

230 completing the proof.

The special case where the bias weight is uniform for all role-models ($\alpha = \alpha_j$ for $j = 1, \ldots, N$) is
232 interesting, because we can evaluate the expected number of copiers using a linear equation

$$\mathrm{E}[K_{Nj}] = N \cdot \frac{\alpha \cdot \beta(A_j + e)}{\sum\limits_{m=1}^{N} \alpha \cdot \beta(A_m + e)} = \beta(A_j + e) \big/ \overline{\beta(A + e)} \,, \tag{16}$$

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

$$\mathrm{E}[K_{Nj}] = L \cdot \beta(A_j + e) \,. \tag{17}$$

238 **Numerical validation.** To be added

Dirichlet-Multinomial distribution

240 **Pólya urn model.** This stochastic 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
242 additional new ball of the same color. Let $\mathbf{U_i} = \{u_{i,1}, u_{i,2}, ..., u_{i,M}\}$ 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,
244 and 0 otherwise. The probability that $S_{i,j} = 1$ given $\mathbf{U_{i-1}}$ is

$$P(S_{i,j} = 1 \mid \mathbf{U_{i-1}}) = \frac{u_{i-1,j}}{\sum\limits_{m=1}^{M} u_{i-1,m}} = \frac{o_j + w_{i-1,j}}{\sum\limits_{m=1}^{M} o_m + w_{i-1,m}} = \frac{o_j + w_{i-1,j}}{i - 1 + \sum\limits_{m=1}^{M} o_m} \,, \tag{18}$$

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

248

Result 2 (Pólya urn model). *The role-model choice process,* $\{\mathbf{K}_i\}_{i=1}^{N}$, *is equivalent to a Pólya urn*
250 *model if both trait estimation error and bias weight are uniform in the population,* $e = e_j$ *and* $\alpha = \alpha_j$
for all $j = 1, \ldots, N$.

252 *Proof.* Denote $\alpha' = \frac{\alpha}{1-\alpha}$ as the bias weight ratio, and $A'_j = A_j + e$. From eq. (10) and because $\sum_{j=1}^{N} K_{i,j} = i$, we have

$$254 \qquad G_{i,j} = \frac{\alpha'\beta(A'_j) + K_{i-1,j}}{\sum\limits_{m=1}^{N} \alpha'\beta(A'_m) + K_{i-1,m}} = \frac{\alpha'\beta(A'_j) + K_{i-1,j}}{i - 1 + \sum\limits_{m=1}^{N} \alpha'\beta(A'_m)} \ . \tag{19}$$

Substituting $M = N$, $o_j = \alpha'\beta(A'_j)$, and $w_{i,j} = K_{i,j}$ in eq. (18) gives eq. (19), thus completing the
256 proof.

Frigyik et al. (2010, section 2) prove that the proportion of different colored balls in a *Pólya urn*
258 *model* converges to the Dirichlet distribution as the number of draws approaches infinity, based on
the *Martingale Convergence Theorem* (Durrett, 1999). We therefore have an approximation for the
260 relative prestige each role-model has when evaluated by copiers. Thus, choosing the role-models for
all copiers is equivalent to drawing from a Multinomial distribution where the parameters are the
262 modified weights from a Dirichlet distribution and we have the following corollary.

264 **Corollary 3.** *The number of copiers of each role-model follows a Dirichlet-Multinomial distribution,*
$\mathbf{K_i} \sim DM(N, \mathbf{G_1})$, *under the conditions of Result 2.*

266 **Numerical validation.** To validate our analytical result (corollary 3) and test its sensitivity to the
assumptions ($e_i = e$ and $\alpha_i = \alpha$ for $i = 1, \ldots, N$) we compare it to results of stochastic simulations. We
268 executed varying amount of simulations of our original model to compute an observed distribution of
number of copiers from the average empirical distribution. The number of simulation required depended
270 on the parameters used. To compare the observed distribution with the expected DM distribution,
we use a Pearson's chi-squared test of goodness-of-fit to reject or accept the null hypothesis that the
272 simulation results are drawn from a DM distribution.

When testing multiple values of the bias weight parameter α, the test p-value was always 1, meaning
274 that the null hypothesis cannot be rejected.

For high α values (above 0.5), very few simulations are needed to reach p-value of 1 (less than five
276 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. When testing our observed distributions
278 against different distributions, for example the uniform distribution (all role-models have exactly one
copier), the p-value was 0, so that the null hypothesis was rejected, as required.

280 **Model testing**

Here we examine the difference between the dichotomous trait model defined in eq. (11) and the
282 Dirichlet-Multinomial approximation we presented in corollary 3. We focus on the fixation probability
and fixation time of the advantageous phenotype $\hat{A}$ when invading a population of phenotype A.

284 First, we find that the number of simulations needed to sufficiently approximate our model with the DM
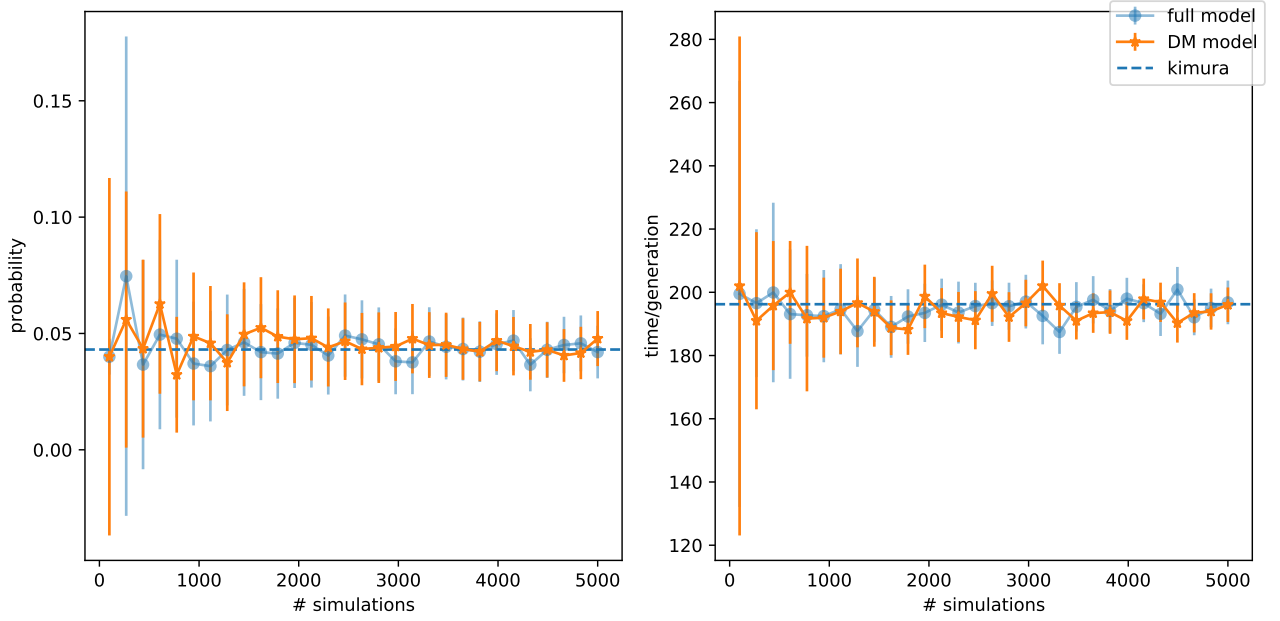approximation is roughly 1000 (Figure 1). Next, we examined the robustness of the DM approximation

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Figure 1: Number of simulations needed to get a good approximation. The approximation (orange) fits simulation results (blue) well when using 1,000 simulations. Markers for average value across simulations, error bars for 95% confidence interval. Here, population size, $N = 1000$; bias weight, $\alpha = 0.5$; phenotype values, $\hat{A} = 1$, $A = 0.7$; success bias value, $\beta(A) = 0.956$.

to relaxing the approximation assumptions First we relaxed our assumption of no estimation error e. Estimation error in the original model was drawn from a normal distribution, and added to the trait value before evaluation of the bias ($A_{ij} = A_j + e_i$).

In the dichotomous model the estimation error will be added by using heterogeneous J parameter, which controls the strength of the bias of eq. (5).

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.

Fixation probability and time

After finding that the DM distribution is a good approximation of the role-model choice process we turn our attention to the evolutionary dynamics. We focus on the effect of the bias weight (α), which determines the relative effects of success and influence on prestige bias, on the fixation probability and fixation time of an advantageous phenotype, similar to analyses in population-genetic models. For simplicity, we do not include mutation in this analysis. As shown above, transmission in our model is
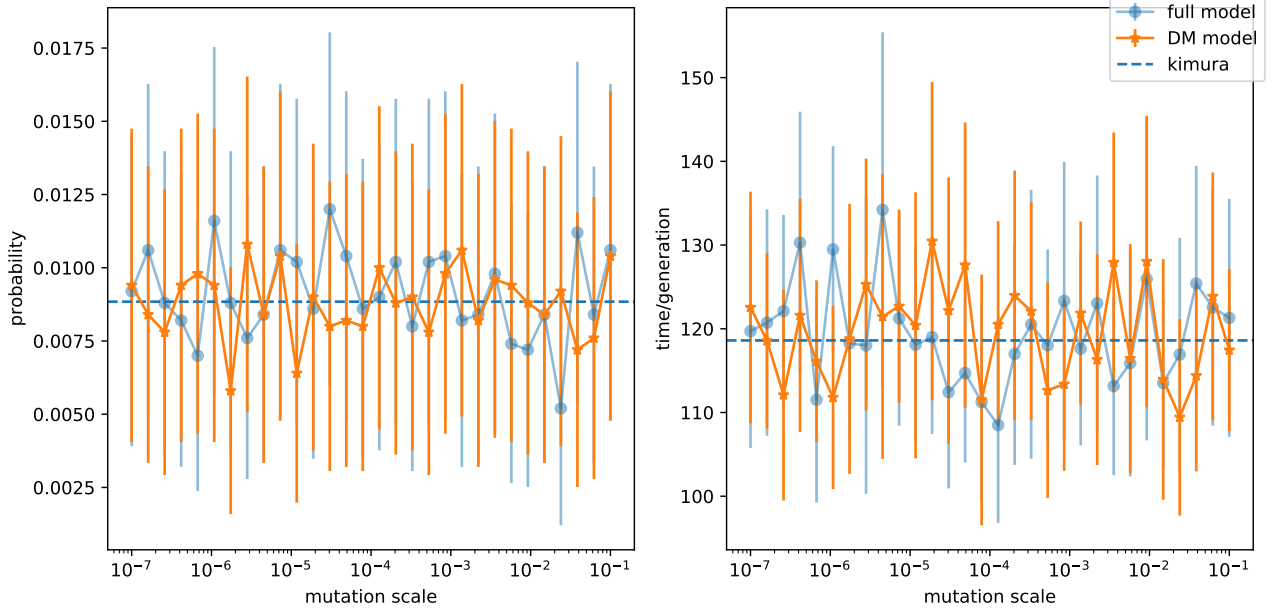
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Figure 2: Robustness of DM approximations to inclusion of mutation. Both the DM approximation (orange) and Kimura's equation (black line) fit the stochastic simulations (blue) well even with a high mutation rate. Markers for average across simulations, error bars for 95% confidence intervals. 5,000 simulations per data point; population size, $N = 1000$; bias weight, $\alpha = 0.1$; phenotype values, $\hat{A} = 1, A = 0.7$; $J \sim N(1, x^2)$ where x is the mutation scale in the x-axis.
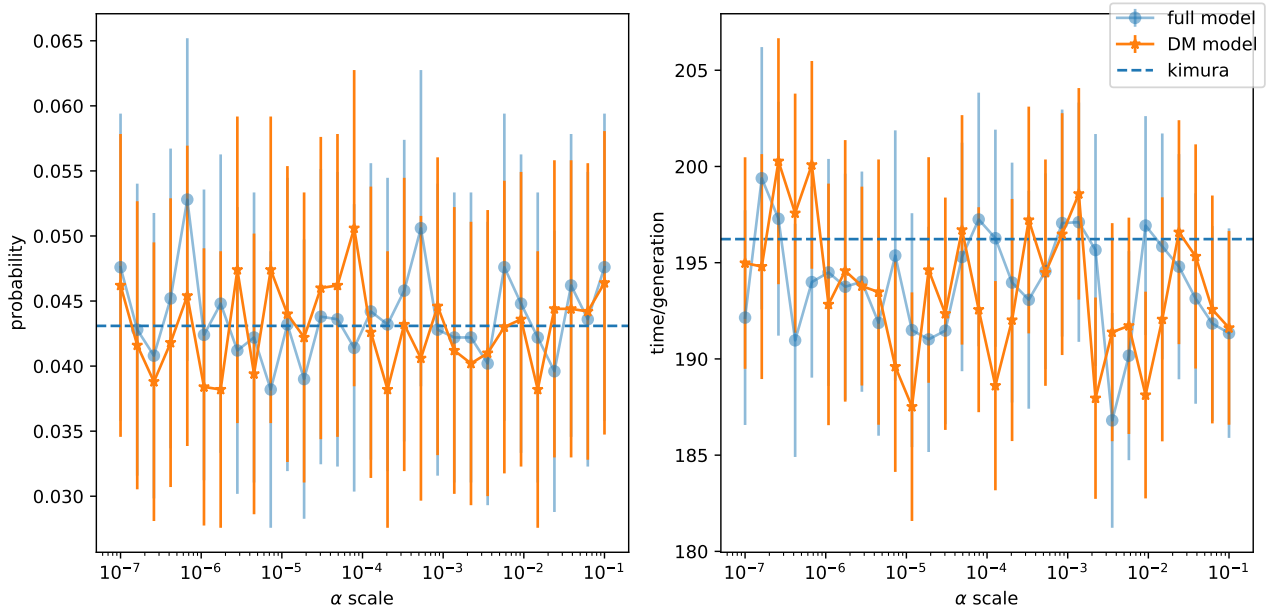

Figure 3: Comparison of the DM approximation and the full model when success weight is heterogeneous. High success weight variance distances the approximation and the full model of generations to fixation from the Kimura's approximation, but not by much (confidence intervals still cover it). Error bars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

approximately DM distributed with a parameters

Result 3 (Effective selection coefficient). $1 - \beta(A)$ *is equivalent to the selection coefficient s in the diffusion-equation approximation of the a classic Wright-Fisher model that approximate the fixation probability and fixation time of an advantageous allele.*

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 $\beta(A) = \beta$ ($\beta < 1$).

The expected number of individuals of a DM distribution is:

$$E[X'] = N\frac{\alpha_1}{\alpha_1 + \alpha_2}, \tag{20}$$

where $\alpha_1 = \alpha'X$ and $\alpha_2 = \alpha'(N - X)\beta$, from eq. (11). We want to use frequencies instead of quantities to follow Durret's process so:

$$E[x'] = E[\frac{X'}{N}] = \frac{1}{N}E[X'] \tag{21}$$

Putting it together we get:

$$\begin{aligned}
E[x'] &= \frac{1}{N}N\frac{\alpha'xN}{\alpha'xN + \alpha'N(1-x)\beta} \\
&= \frac{x}{x + (1-x)\beta}
\end{aligned} \tag{22}$$

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:

$$\begin{aligned}
E[x'] &= \frac{x}{x + (1-x)\beta} = \frac{x}{x + (1-x)(1-s)} = \\
&= x + x(1-x)s + o(s) \\
&= x + x(1-x)(1-\beta) + o(1-\beta)
\end{aligned} \tag{23}$$

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

$$E[x' - x] = E[x'] - E[x] = x(1-x)(1-\beta) + o(1-\beta) \tag{24}$$

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

Result 4 (Effective population size). $N_e = \alpha N + (1 - \alpha)$, *where Ne is the effective population size of our binary model.*

Proof. The variance of a DM distribution is:

$$V(X') = N \frac{\alpha_1}{\alpha_1 + \alpha_2} \left(1 - \frac{\alpha_1}{\alpha_1 + \alpha_2}\right) \left(\frac{N + \alpha_1 + \alpha_2}{1 + \alpha_1 + \alpha_2}\right) \qquad (25)$$

And again, we want to use frequencies so:

$$V\left(\frac{X'}{N}\right) = \frac{1}{N^2} V(x') \qquad (26)$$

Putting it together with our model's notations:

$$V(x') = \frac{1}{N^2} N \frac{x}{x + (1-x)\beta} \left(1 - \frac{x}{x + (1-x)\beta}\right) \left(\frac{N + \alpha' x N + \alpha' N(1-x)\beta}{1 + \alpha' x N + \alpha' N(1-x)\beta}\right) \qquad (27)$$

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

$$\frac{x}{x + (1-x)\beta} \approx x \qquad (28)$$

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

$$V(x') \approx \frac{1}{N} x(1-x) \left(\frac{N + \alpha' x N + \alpha' N - \alpha' x N}{1 + \alpha' x N + \alpha' N - \alpha' x N}\right)$$
$$= x(1-x) \left(\frac{1 + \alpha'}{1 + \alpha' N}\right) \qquad (29)$$

Again following Durrett we want to calculate:

$$V(x' - x) = V(x') - V(x) \approx x(1-x) \left(\frac{1 + \alpha'}{1 + \alpha' N}\right) \qquad (30)$$

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 model as α, so:

$$\alpha' = \frac{\alpha}{1 - \alpha} \qquad (31)$$

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Combining eq. (30) and eq. (31) we get:

$$
\begin{aligned}
V(x'-x) &\approx x(1-x)\left(\frac{1+\frac{\alpha}{1-\alpha}}{1+\frac{\alpha}{1-\alpha}N}\right) \\
&= x(1-x)\left(\frac{\frac{1-\alpha+\alpha}{1-\alpha}}{\frac{1-\alpha+\alpha N}{1-\alpha}}\right) \\
&= x(1-x)\left(\frac{1}{1-\alpha(1-N)}\right) \\
&= x(1-x)\left(\frac{1}{\alpha N+(1-\alpha)}\right) \\
&= x(1-x)\frac{1}{N_e}
\end{aligned}
\tag{32}
$$

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.

The fixation probability derived from Kimura is therefore:

$$
P_{fix} = \frac{1-e^{-2(1-\beta)N_e x}}{1-e^{-2(1-\beta)N_e}}
\tag{33}
$$

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

The time to fixation can be estimated by:

$$
T_{fix} = \frac{1-P_{fix}}{1-\beta}\int_0^x \frac{e^{2(1-\beta)\xi}-1}{\xi(1-\xi)}d\xi + \frac{P_{fix}}{1-\beta}\int_x^1 \frac{1-e^{-2(1-\beta)(1-\xi)}}{\xi(1-\xi)}d\xi
\tag{34}
$$

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. (33) and eq. (34).

Changing environment

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.

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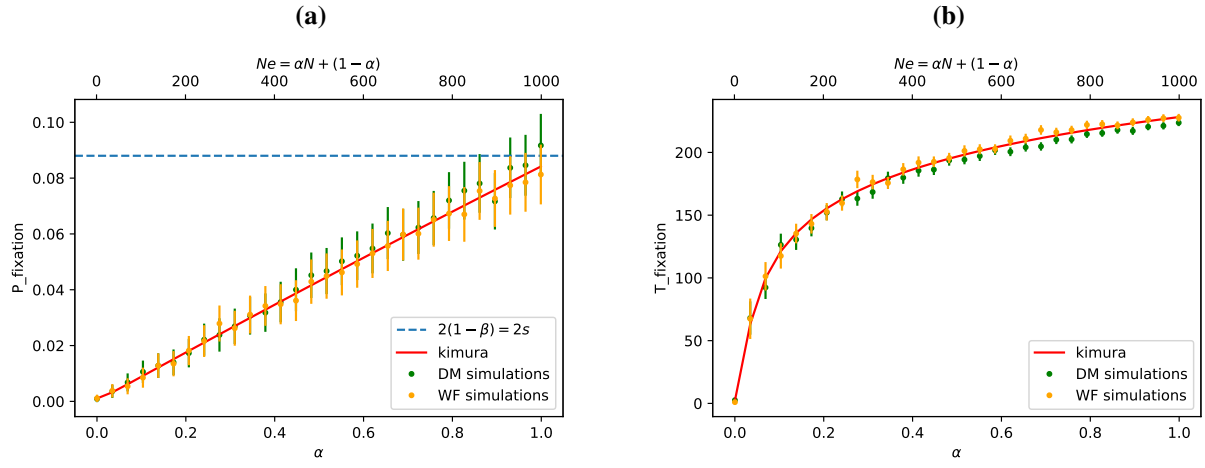
Figure 4: Comparison of the DM approximation and the WF model for different values of the effective population size. The approximation seems very good, and is also condensed around the mathematical equation expectancy. Error bars are 95% confidence intervals. Effective population calculated by $N_e =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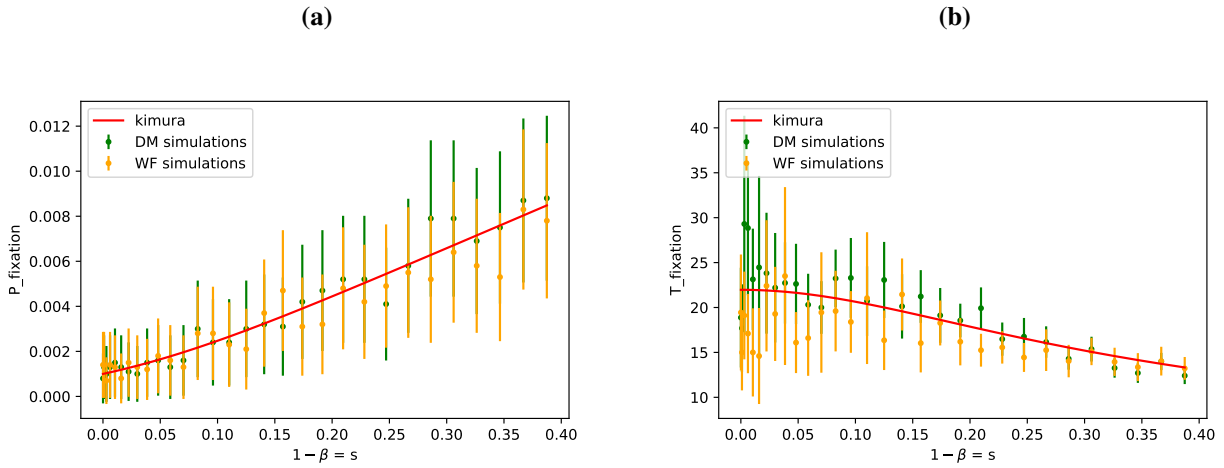

Figure 5: Comparison of the DM approximation and the WF model for different values of the selection coefficient, manifested as success bias in our model. The approximation seems good, and is also condensed around the mathematical equation expectancy. Error bars are 95% confidence intervals. Effective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

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

Proof by induction: From eq. (24) we know that

$$
\begin{aligned}
E\left[\frac{X_{t+1}}{N} - \frac{X_t}{N}\middle|X_t\right] &= \frac{X_t}{N}\left(1 - \frac{X_t}{N}\right)(1 - \beta_{t+1}) \\
&= \frac{1}{N}\frac{X_t}{N}\left(1 - \frac{X_t}{N}\right)s_{t+1}
\end{aligned}
\tag{35}
$$

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

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

$$= E\left[\frac{X_t}{N}\right] - E\left[\left(\frac{X_t}{N}\right)^2\right] \tag{36}$$

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

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

$$E\left[\frac{X_t}{N}\left(1 - \frac{X_t}{N}\right)\right] \simeq E\left[\frac{X_t}{N}\right]\left(1 - E\left[\frac{X_t}{N}\right]\right) - \frac{1}{N_e}tx(1-x) \tag{37}$$

From eq. (35) we know that

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

$$\simeq \frac{1}{N}s_{t+1}\left(E\left[\frac{X_t}{N}\right]\left(1 - E\left[\frac{X_t}{N}\right]\right) - \frac{1}{N_e}tx(1-x)\right) \tag{38}$$

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

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

$$E\left[\frac{X_{t+1}}{N} - \frac{X_t}{N}\right] \simeq \frac{1}{N}s_{t+1} \cdot E\left[\frac{X_t}{N}\right]\left(1 - E\left[\frac{X_t}{N}\right]\right) \tag{39}$$

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

$$E\left[\frac{X_t}{N} - x\right] = E\left[\frac{X_t}{N}\right] - E[x] = E\left[\frac{X_t}{N}\right] - x, \tag{40}$$

so using the assumption we get

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

$$1 - E\left[\frac{X_t}{N}\right] \simeq 1 - \frac{1}{N}S_t x(1-x) + x \tag{41}$$

we'll use both expressions in eq. (39) and get

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

$$\simeq \frac{1}{N}s_{t+1} \cdot x(1-x) \tag{42}$$

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

$$E\left[\frac{X_{t+1}}{N} - x\right] = E\left[\frac{X_{t+1}}{N} - \frac{X_t}{N}\right] + E\left[\frac{X_t}{N} - x\right] \tag{43}$$

so again using the induction assumption, together with eq. (42) we get

$$E\left[\frac{X_{t+1}}{N} - x\right] \simeq \frac{1}{N}s_{t+1} \cdot x(1-x) + \frac{1}{N}S_t \cdot x(1-x)$$
$$\simeq \frac{1}{N}x(1-x)(S_t + s_{t+1}) \tag{44}$$
$$\simeq \frac{1}{N}S_{t+1}x(1-x)$$

which proves the first part of our preposition.

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

$$V\left(\frac{X_{t+1}}{N}\right) = E\left[V\left(\frac{X_{t+1}}{N}\Big|X_t\right)\right] + V\left(E\left[\frac{X_{t+1}}{N}\Big|X_t\right]\right) \tag{45}$$

using eq. (35) we see that:

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

Using eq. (32) we get:

$$V\left(\frac{X_{t+1}}{N}\Big|X_t\right) = \frac{1}{N_e}\frac{X_t}{N}\left(1 - \frac{X_t}{N}\right) \tag{47}$$

and using the equation $y'(1-y') \simeq y(1-y)$ on the first part of eq. (45) we get:

$$E\left[V\left(\frac{X_{t+1}}{N}\Big|X_t\right)\right] = \frac{1}{N_e}E\left[\frac{X_t}{N}\left(1 - \frac{X_t}{N}\right)\right] \simeq \frac{1}{N_e}x(1-x) \tag{48}$$

and moving on to simplify the second part of eq. (45) using eq. (46):

$$V\left(E\left[\frac{X_{t+1}}{N}\Big|X_t\right]\right) = V\left(\frac{X_t}{N} + \frac{1}{N}s_{t+1}\frac{X_t}{N}\left(1 - \frac{X_t}{N}\right)\right) \tag{49}$$

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:

$$V\left(\frac{1}{N}s_{t+1}\frac{X_t}{N}\left(1 - \frac{X_t}{N}\right)\right) \le \frac{1}{4N^2}s_{t+1}^2 \tag{50}$$

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(a) success bias/selection coefficient is: $1 - \beta = s = 0.005$
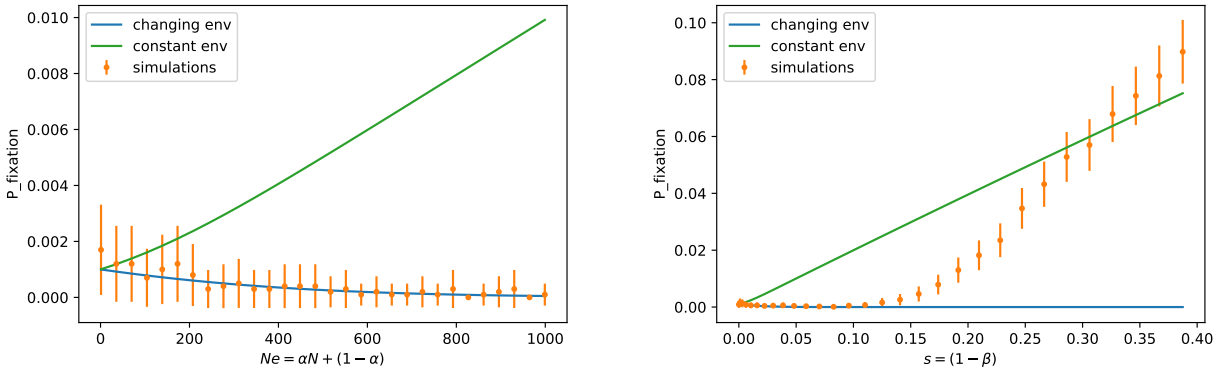**(b)** success weight is: $\alpha = 0.1$

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, $N = 1,000$, $\hat{A} = 1$, $A = 0.9$, $J = 1$.

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

$$V\left(E\left[\frac{X_{t+1}}{N}\Big|X_t\right]\right) = V\left(\frac{X_t}{N}\right) + O\left(\frac{1}{N^2}\right) \simeq V\left(\frac{X_t}{N}\right) \tag{51}$$

Using the induction assumption and eq. (48):

$$V\left(\frac{X_{t+1}}{N}\right) \simeq \frac{1}{N_e}x(1-x) + \frac{1}{N_e}tx(1-x) \simeq \frac{1}{N_e}x(1-x)(t+1) \tag{52}$$

proving the second part of our preposition.

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

$$P_{fix} = \frac{1 - e^{-2\frac{S_n}{n}N_e x}}{1 - e^{-2\frac{S_n}{n}N_e}} \tag{53}$$

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 fig. 6 we can see that α on its own does not cause any deviation for the the estimation. β however affects the results greatly.
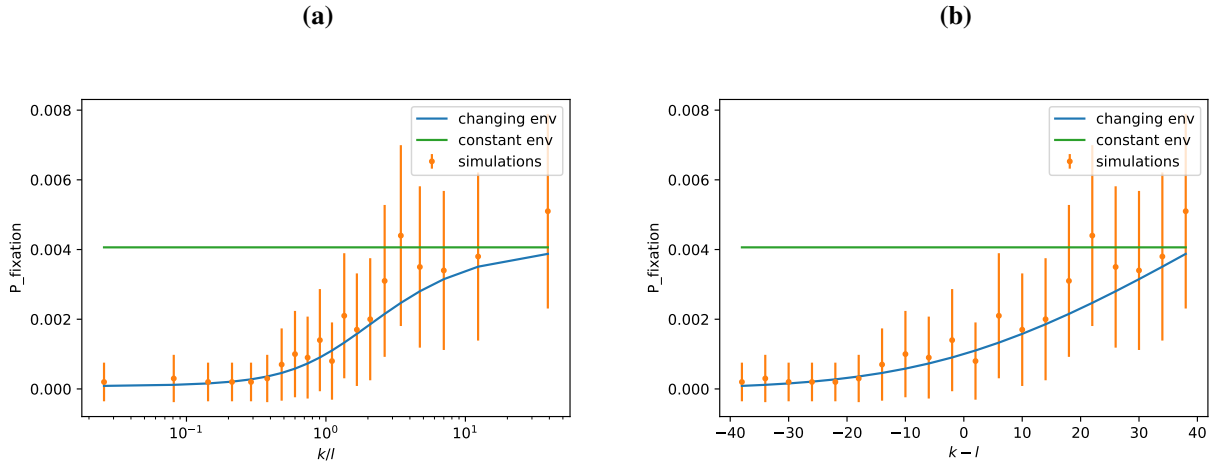
(a)   **(b)**

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, $N = 1,000$, $\hat{A} = 1$, $A = 0.8$, $J = 1$, $1 - \beta = s = 0.02$, $\alpha = 0.1$.

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 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, 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 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

summary

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. 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 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 and estimate

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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 accurately is harder. **We investigated the effects of prestige on a population:** we studied the behavior of an invading trait, analyzed its dynamics mathematically, and extended the basic constant environment to a changing one. 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 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 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 with constant and changing environment in our model, and created a variation of a binary model 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).

Prestige in the literature

So far we discussed prestige as a main bias in humans, and to some extent in non-human species. Here we further base our claims and present additional appearances of prestige in nature and in the literature.

King and Cowlishaw (2009) describe a manifestation of prestige in the form of leadership in animals. According to them, there are two main approaches to decision makings of groups in nature: leadership and consensus. 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 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 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, 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, 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 the rest would prefer to follow, minimizing their costs. In baboons however, King and Cowlishaw (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 reward") What they describe could be the origins of what we know today as prestige. In their paper, they show that in nature, when survival is the main concern at all times, the leaders wouldn't be chosen due to their superior abilities, but because they have the least to lose. When in said position of leadership, there are greater risks, but greater rewards to come with it. In humans, leadership also has its perks and costs. Leaders can make decisions that would 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 violent behavior of subjects on certain cases). In our society it is less common to worry about mere survival, and so the prestigious positions, even though are not without risks and costs, are not as dangerous as for animals in nature. This may be the reason humans strive for the prestigious positions, as they may reap rewards greater than the risk and costs to achieve them. This is in complete contrast to animals, where the weakest/hungriest is driven to lead, compared to humans where leadership positions are mostly competitive.

Van Vugt and Smith (2019) suggest a different view of leadership. They note that most discussions assume there is one type of leadership, as seen above, and so they differ in their definitions. 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.

Henrich and Gil-White (2001) support said claim that there are two types of leadership, and also define the two as prestige based and dominance based leadership types. By their definition, the latter is defined by acquiring social status by using aggression, intimidation and violence. It is also more common than prestige in non humans. Their definition of prestige is somewhat synonymous with

ours. According to their manuscript, prestige is composed both of estimation in the eyes of people (our indicator/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, 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 where social learning exists, due to saving costs of individual learning. It could be interesting in the future to expand our model using this idea: observing the copier trait of *evaluation*, rather than only observing the evolution of the indicator trait copied. Henrich and Gil-White (2001) suggest that the most skilled role-models will, on average, end up with most copiers. Their research, definitions and results, is consistent with ours.

So far we presented the theory behind prestige, and it's appearance in nature. The following will show the appearances of prestige biases in humans, and in recent times.

Chudek et al. (2012), for example, tested the existence of prestige in young children. Chudek et al. (2012) report the first direct tests in children that suggest the existence of *prestige bias*, a tendency to learn from individuals to whom others have preferentially attended, learned or deferred. Their definition of prestige 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 had previously preferentially attended for 10 seconds were over twice those of their learning from a model whom bystanders ignored. In addition to this first study, they also discovered prestige 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.

Henrich and Broesch (2011) researched Fijian villages, looking for evidence of social learning biases 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.

We see here support for both our indicator trait and our influence bias in their data.

Aside from children's learning biases and small villages in a relatively primitive population, we can see prestige in more advanced domains as well, like western medicine.Norredam and Album (2007) present a specific and important effect of prestige - its significance for medical specialties and diseases. They examined literature from 1950 to 2005 regarding the effects of prestige on medicinal 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 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 psychiatry is the less prestigious. In addition, doctors tend to rank practices that require more time to master as more prestigious, while other procedures that are considered *easier* are less prestigious. Simply put, they found that the advance in technology and research was in accordance with the prestige rankings. This means that there may be very important practices that are neglected due to the prestige bias.

As we seen so far, prestige can explain many behaviors and evolution of cultural traits. It is a tool to cheaply estimate and acquire knowledge, which helps an individual to survive and breed. However, it is not always the case, and there could be negative repercussions to this bias, such as invasion of maladaptive traits.

Takahashi and Ihara (2019) mention that social learning not only takes the form of random copying of other individuals, but also involves learners' choice of what to learn and from whom to learn. They suggest a best-of-K model where an individual samples k role-models and choose the one he 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 run replaced by unbiased learners, who simply copy someone chosen at random. They develop new mathematical models that are simpler and mathematically tractable. They found that best-of-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 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 can be stable against invasion

by unbiased learning if social learning is sometimes combined with 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.

We discussed prestige in depth, and provided several proofs for its existence in nature, humans, and even medicine. We saw it could aid invasion of maladaptive traits, or neglect of important medicinal specialities. But, it can also accelerate reversal of harmful traditions. Harmful traditions can be child marriage, open defecation, and domestic violence, to name a few. Efferson 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 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 will be copied more, therefore spreading his trait wider in the population. Their research support 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 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.

Up until now, we showed that 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. We also presented examples of good and bad usages of such biases. All of these are mainly presented as a parallel process to the natural selection process in regards to physical anatomy, or at least have an indirect effect on it. Muthukrishna and Henrich (2016) offer a take on prestige as a factor of human physical evolution directly. They present a concept called *cultural brains* - brains that evolved primarily for the acquisition of adaptive knowledge. 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 larger brain. Muthukrishna and Henrich (2016) built a model that predicts a strong relationship 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 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 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" acquire adaptive knowledge, i.e transmission biases, such as prestige.

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