

Prestige as a Driving Force in Cultural Transmission

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Introduction

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

48 to negate the selection bias (Boyd and Richerson, 1988, Ch. 8 pg. 279).

50 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
52 of yeast called *wtf genes*, that bias their transmission to the gametes. They secrete a long life ex-
pectancy poison, together with a short life expectancy antidote, so a gamete without the gene will
54 perish (the poison will outlive the antidote). Transmission biases, though exist in genetic transmis-
sion, are probably more common in cultural transmission. Much like mutation in genetic evolution,
56 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
58 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
60 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,
62 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
64 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.
66 (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.
68 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:
70 *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
72 example, an individual observing a Ping-Pong match between two others can try both of the pad-
dle grips it observed, and decide what grip is better for it. An indirect bias is when an individual
74 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
76 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
78 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
80 to be frequency-biased.

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

Prestige means having a good reputation or high-esteem, therefore does not directly signify
84 success (although it may imply it), making it an *indirect bias*. Both Boyd and Richerson (1988,
Ch. 8) and Fogarty et al. (2017) claim that prestige biases are probably more common in humans
86 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 other hand, Henrich and Broesch (2011) 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 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 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

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 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.

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
 124 a second trait called **Indirectly biased trait** to lower complexity. The model's state at time t
 can be described by:

$$126 \quad \vec{A}_t = (A_{t,1}, \dots, A_{t,N}) \quad (1)$$

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

$$A'_i = F_i(\vec{A}_t) \quad (2)$$

130 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 $t \rightarrow t + 1$. F is a
 132 function over the t generation traits vector, and is defined differently for every implementation of
 the **Generic model**.

134 **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
 136 average of the role-models' traits in a single generation:

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

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

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

140 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:

$$142 \quad A_{i,j} = A_j + e_i, \quad (5)$$

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

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

146 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
 148 *relative fitness* in genetic transmission models.

Random choice transmission. Boyd and Richerson (1988) note that the method of transmis-
 150 sion 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
 152 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
 154 role-model generation:

$$A'_i = A_{i,j} \quad (7)$$

156 **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 $\sum_{j=1}^N K_{i,j} =$
 158 i . The stochastic process of role-model choice,

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

160 is described by the recurrence equation

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

162 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

$$164 \quad G_{i,j} = P(S_{i,j} = 1 | S_{1,j}, S_{2,j}, \dots, S_{i-1,j}) \quad (10)$$

is the prestige of role-model j in the eyes of copier i . This prestige $G_{i,j}$ is determined as follows.
 166 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
 168 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 , $K_{i-1,j}$,

$$170 \quad G_{i,j} = \frac{\alpha_j \cdot \beta(A_{i,j}) + (1 - \alpha_j) \cdot K_{i-1,j}}{W_i}, \quad (11)$$

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

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

174

176 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 distri-
 178 bution 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. 9,10).

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

1. $K_{i,j}$ follows the general binomial distribution defined by Drezner and Farnum (1993). Moreover, $\mathbb{E}[K_{N,j}] = N \cdot G_{1,j}$ 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, $\alpha_l = \alpha_m$ for all l, m , then $\mathbb{E}[K_{N,j}] = \beta(A'_j) / \overline{\beta(A')}$, where A'_j is the estimated indicator value $A'_j = A_j + e$, and $\overline{\beta(A')}$ 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 (equation 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,

$$\vec{K}_i \sim DM(N, \vec{G}_1), \quad (13)$$

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 .

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 $e_l = e_m$ 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 equation 9:

$$Q_j(k, i) = P_j(S_{i,j} = 1 | k-1, i-1) \cdot Q_j(k-1, i-1) + P_j(S_{i,j} = 0 | k, i-1) \cdot Q_j(k, i-1) \quad (14)$$

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

Equation 14 is equivalent to equation 2.1 that Drezner and Farnum (1993) define. $Q_j(k, N)$ 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 equation 8 as (Drezner and Farnum, 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:

210 $\mathbb{E}[k] = N \cdot Q_j(1, 1)$ (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
 212 $\mathbb{E}[K_{N,j}] = N \cdot G_{1,j}$.

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

Proof: The initial prestige of role-model j based on equation 11 is:

$$216 \quad G_{1,j} = \frac{\alpha_j \cdot \beta(A'_j)}{\sum_{m=1}^N \alpha_m \cdot \beta(A'_m)} \quad (15)$$

The denominator of equation 15 can also be formulated as:

$$218 \quad \sum_{m=1}^N \alpha_m \beta(A'_m) = N \cdot \overline{\alpha \cdot \beta(A')} \quad (16)$$

where $\overline{\alpha \beta(A')}$ is the mean value of $\alpha_m \cdot \beta(A'_m)$ for all m . Using equation 16 we get:

$$220 \quad \mathbb{E}[K_{N,j}] = \alpha_j \cdot \beta(A'_j) / \overline{\alpha \cdot \beta(A')} \quad (17)$$

, completing our proof.

222 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:

$$224 \quad \mathbb{E}[K_{N,j}] = N \cdot \frac{\alpha \cdot \beta(A'_j)}{\sum_{m=1}^N \alpha \cdot \beta(A'_m)} = \beta(A'_j) / \overline{\beta(A')} \quad (18)$$

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

$$228 \quad \mathbb{E}[K_{N,j}] = L \cdot \beta(A'_j) \quad (19)$$

Dirichlet-Multinomial Distribution Approximation

230 **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
 232 is drawn, it is then placed back in the urn together with an additional new ball of the same colour.
 Let $\vec{U}_i = \{u_{i,1}, u_{i,2}, \dots, u_{i,M}\}$ where $u_{i,j}$ is the number of balls of the j -th color in the urn after

234 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 U_{i-1}^{\rightarrow} is:

$$\begin{aligned}
 P(S_{i,j} = 1 | U_{i-1}^{\rightarrow}) &= \frac{u_{i-1,j}}{\sum_{m=1}^M u_{i-1,m}} = \frac{o_j + w_{i-1,j}}{\sum_{m=1}^M o_m + w_{i-1,m}} \\
 &= \frac{o_j + w_{i-1,j}}{i - 1 + \sum_{m=1}^M o_m}
 \end{aligned}
 \tag{20}$$

236 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 .

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 $i, j \in N$.

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

$$\begin{aligned}
 G_{i,j} &= \frac{\alpha \cdot \beta(A'_j) + (1 - \alpha) \cdot K_{i-1,j}}{W_i} \cdot \frac{1 - \alpha}{1 - \alpha} \\
 &= \frac{\alpha' \beta(A'_j) + K_{i-1,j}}{\sum_{m=1}^N \alpha' \beta(A'_m) + K_{i-1,m}} \\
 &= \frac{\alpha' \beta(A'_j) + K_{i-1,j}}{i - 1 + \sum_{m=1}^N \alpha' \beta(A'_m)}
 \end{aligned}
 \tag{21}$$

244 Equations 20 and 21 are equivalent when setting $M = N$, $o_j = \alpha' \beta(A'_j)$, $w_{i,j} = K_{i,j}$, completing the proof.

246 **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

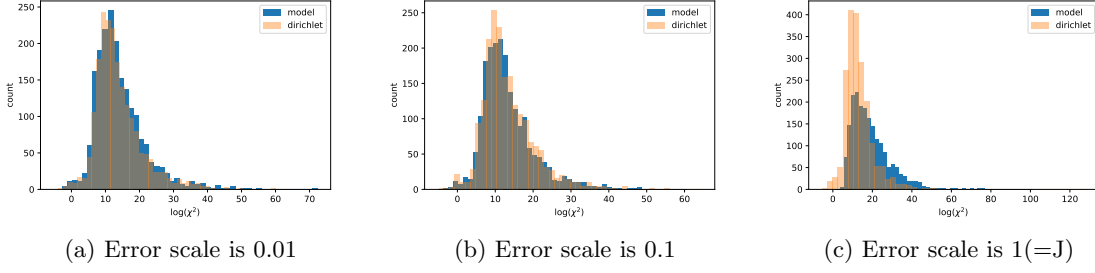


Figure 1: DM approximation vs iterative process. Population size 2000. Aggregated values of 400 simulations

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 test if our process is still DM distributed when there are copying errors in the population, we ran 400 simulations for three different error scales (η). To check the resemblance of the DM approximation to the original iterative process, we measure the χ^2 statistic of both processes and the results can be seen in Fig. 1. Even with a relatively high error scales (1% and 10% of J) we can see the distributions of the χ^2 metrics are very similar. With a higher η (e.g $\eta = J$), which is approximately 100% error when evaluating a trait, the distributions are noticeably different.

In addition to the chi-squared test, we want to observe how well does the Dirichlet-Multinomial distribution approximates our entire model, and not just one generation in it. To do that we simplify our model to a binary model, so the simulations of the model to run in a feasible amount of time.

The 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.21 is:

$$P_{i,A} = \frac{(N - X)\alpha'\beta(A) + K_A}{i - 1 + (N - X)\alpha'\beta(A) + X\alpha'\beta(\hat{A})} = \frac{(N - X)\alpha'\beta(A) + K_A}{i - 1 + (N - X)\alpha'\beta(A) + \alpha'X} \quad (22)$$

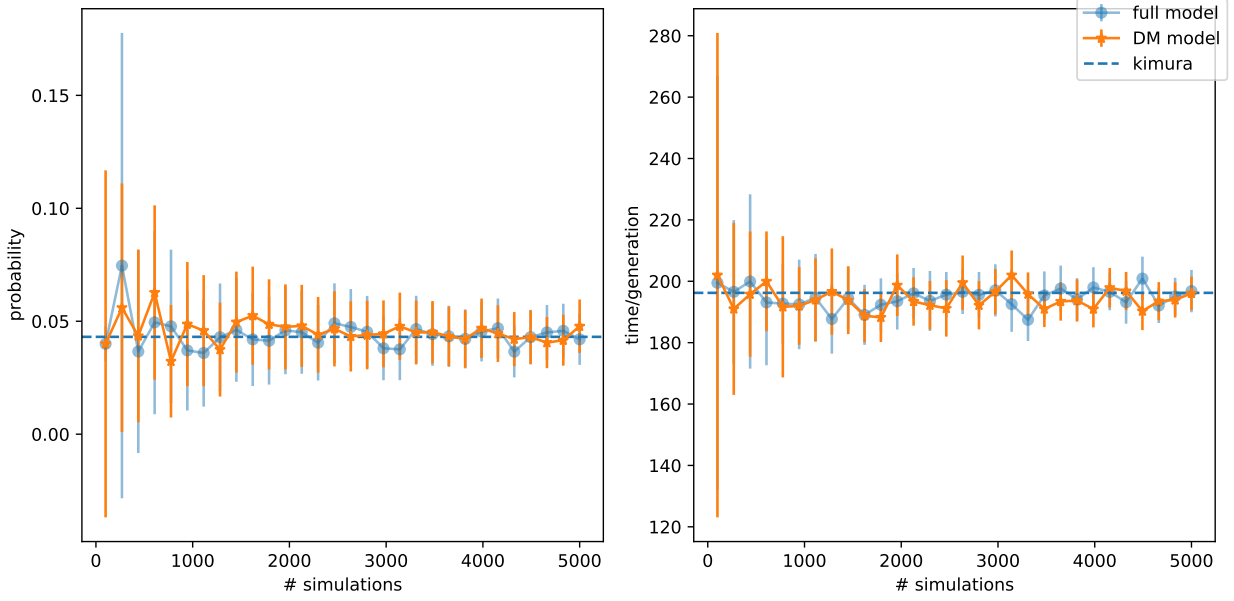


Figure 2: Population size $N = 1000$, $\alpha = 0.5$, $J = 1$, $\hat{A} = 1$, $A = 0.7$, $\beta(A) = 0.956$

where X is the number of role-models with trait \hat{A} and K_A is the number of copiers that already chose A .

In the first generation there is a single individual with \hat{A} , and the rest have A . We're interesting in studying the difference between the real binary model as we defined in eq. 22, 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. 2 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 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 (see eq. 6).

In fig. 3 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. 4 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

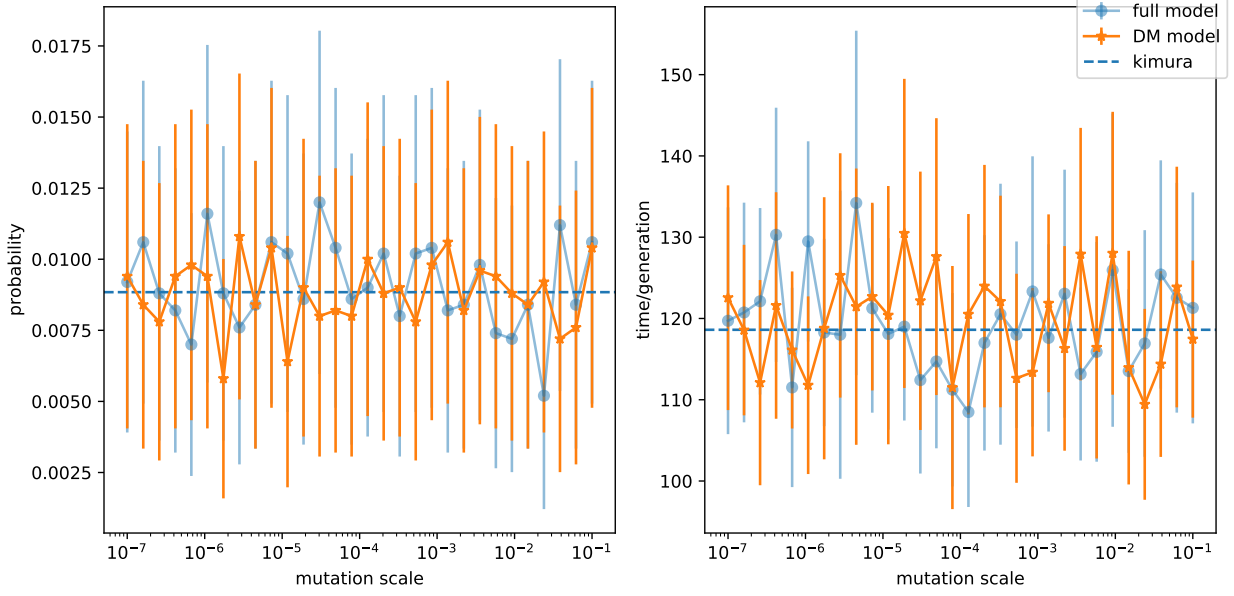


Figure 3: 5000 simulations per data point, $N = 1000$, $\alpha = 0.1, \hat{A} = 1, A = 0.7$, $J \sim N(1, x^2)$ where x is the mutation scale in the x-axis

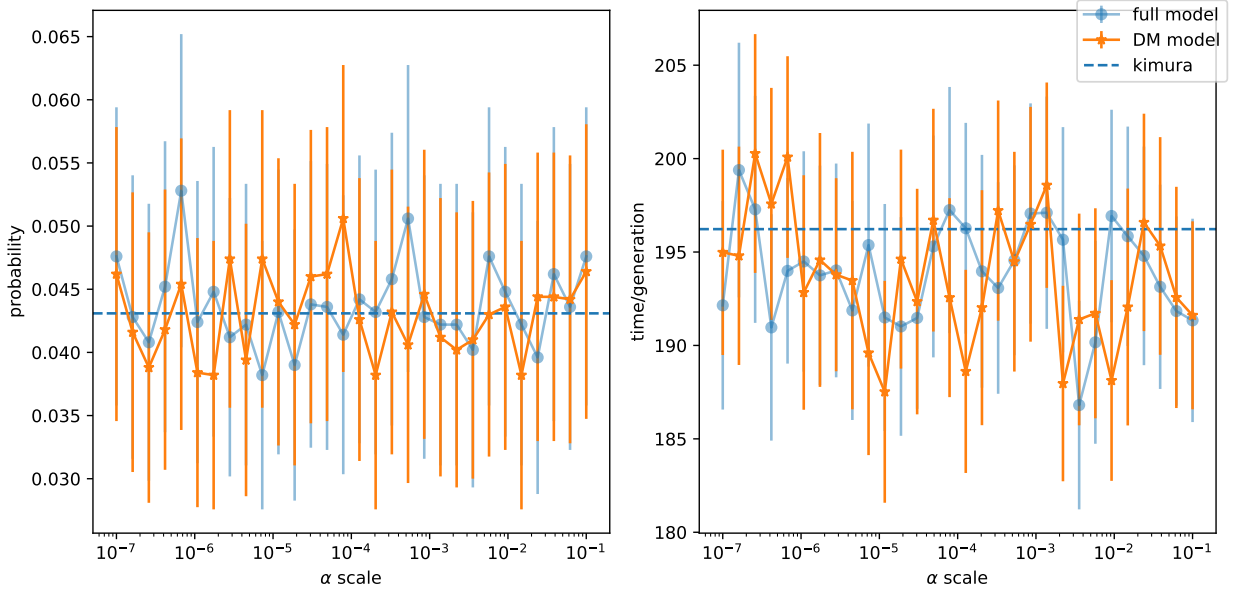


Figure 4: 5000 simulations per data point, $N = 1000$, $\alpha \sim N(0.5, x^2), \hat{A} = 1, A = 0.7$, $J = 1$, $\beta(A) = 0.956$

298 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.

300 Fixation probability and time - binary model

Kimura's approximation: After establishing a case in the favor of our DM approximation, we
302 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
304 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
306 effective population size. From eq. 22 we can derive that the process of inheritance in our binary
model is DM distributed with a parameters vector of size two: $\vec{V} = (\alpha'X, (N - X)\alpha'\beta(A))$.

308 **Proposition:** $1 - \beta(A)$ 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-
310 geous trait.

Proof: Let x be the frequency of type \hat{A} in the population with N individuals. Let X be the
312 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 $\beta(A) = \beta$
314 ($\beta < 1$).

The expected number of individuals of a DM distribution is:

$$316 \quad E[X'] = N \frac{\alpha_1}{\alpha_1 + \alpha_2}, \quad (23)$$

where $\alpha_1 = \alpha'X$ and $\alpha_2 = \alpha'(N - X)\beta$, from eq. 22. We want to use frequencies instead of
318 quantities to follow Durrett's process so:

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

320 Putting it together we get:

$$\begin{aligned} E[x'] &= \frac{1}{N}N \frac{\alpha'xN}{\alpha'xN + \alpha'N(1-x)\beta} \\ &= \frac{x}{x + (1-x)\beta} \end{aligned} \quad (25)$$

322 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:

$$\begin{aligned}
 E[x'] &= \frac{x}{x + (1-x)\beta} = \frac{x}{x + (1-x)(1-s)} = \\
 324 \quad &= x + x(1-x)s + o(s) \\
 &= x + x(1-x)(1-\beta) + o(1-\beta)
 \end{aligned} \tag{26}$$

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

$$326 \quad E[x' - x] = E[x'] - E[x] = x(1-x)(1-\beta) + o(1-\beta) \tag{27}$$

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

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

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

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

332 And again, we want to use frequencies so:

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

334 Putting it together with our model's notations:

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

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

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

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

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

340 Again following Durrett we want to calculate:

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

342 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
 344 model as α , so:

$$\alpha' = \frac{\alpha}{1 - \alpha} \quad (34)$$

346 And combining with the previous equations we get:

$$\begin{aligned} V(x' - x) &\approx x(1 - x) \left(\frac{1 + \frac{\alpha}{1 - \alpha}}{1 + \frac{\alpha}{1 - \alpha} N} \right) \\ &= x(1 - x) \left(\frac{\frac{1 - \alpha + \alpha}{1 - \alpha}}{\frac{1 - \alpha + \alpha N}{1 - \alpha}} \right) \\ &= x(1 - x) \left(\frac{1}{1 - \alpha(1 - N)} \right) \\ &= x(1 - x) \left(\frac{1}{\alpha N + (1 - \alpha)} \right) \\ &= x(1 - x) \frac{1}{N_e} \end{aligned} \quad (35)$$

348 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.

350 The fixation probability derived from Kimura is therefore:

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

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

The time to fixation can be estimated by:

$$354 \quad T_{fix} = \frac{1 - P_{fix}}{1 - \beta} \int_0^x \frac{e^{2(1 - \beta)\xi} - 1}{\xi(1 - \xi)} d\xi + \frac{P_{fix}}{1 - \beta} \int_x^1 \frac{1 - e^{-2(1 - \beta)(1 - \xi)}}{\xi(1 - \xi)} d\xi \quad (37)$$

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

356 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
 358 and N_e for the WF simulations. In fig 5 we changed α (and N_e accordingly) and used a constant
 β . In fig 6 we changed β and used a constant α . In both cases we can see that the two models
 360 behave similarly, and both are approximated well by the Kimura's equations (eq. 36 and 37)

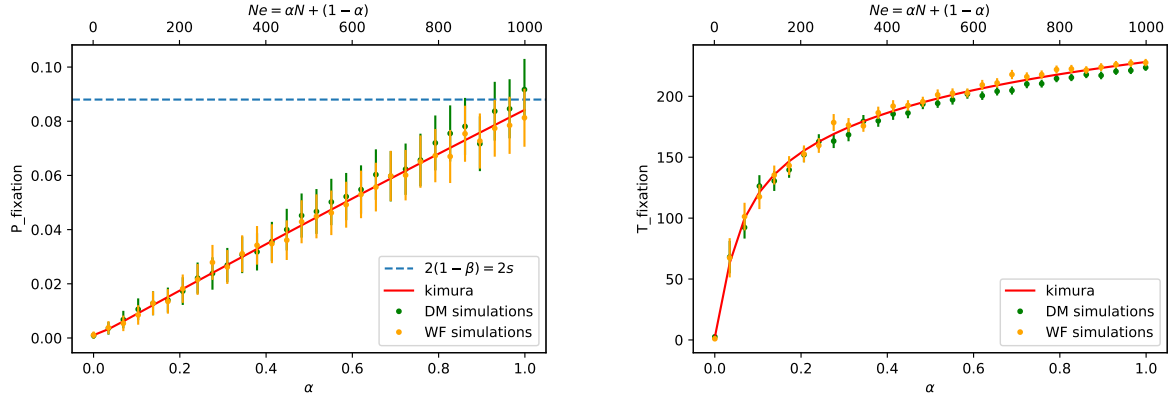


Figure 5: 5,000 simulations per data point, $N = 1,000$, $\hat{A} = 1$, $A = 0.7, J = 1, 1 - \beta = 0.044$

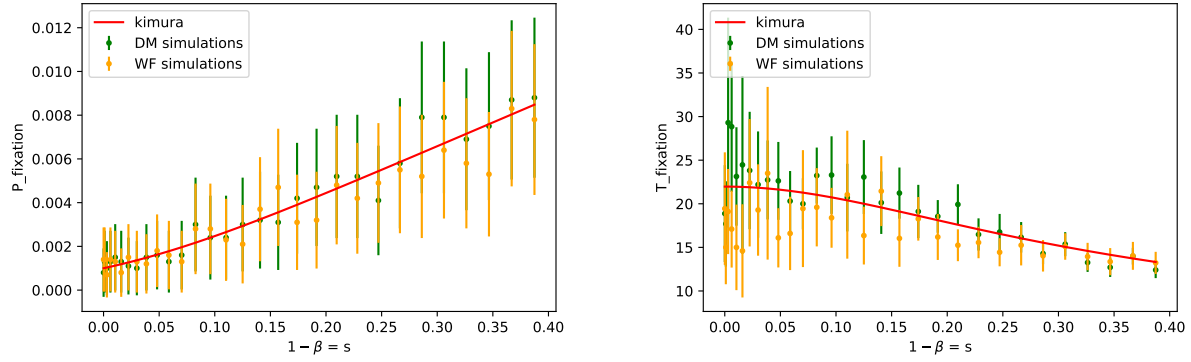


Figure 6: 5,000 simulations per data point, $N = 1,000$, $\hat{A} = 1$, $A = 0.7, J = 1, \alpha = 0.01$

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/invasive 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.

Proof by induction: From eq. 27 we know that

$$\begin{aligned} E \left[\frac{X_{t+1}}{N} - \frac{X_t}{N} \middle| X_t \right] &= \frac{X_t}{N} \left(1 - \frac{X_t}{N} \right) (1 - \beta_{t+1}) \\ &= \frac{1}{N} \frac{X_t}{N} \left(1 - \frac{X_t}{N} \right) s_{t+1} \end{aligned} \quad (38)$$

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

$$\begin{aligned} E \left[\frac{X_t}{N} \left(1 - \frac{X_t}{N} \right) \right] &= E \left[\frac{X_t}{N} - \left(\frac{X_t}{N} \right)^2 \right] \\ &= E \left[\frac{X_t}{N} \right] - E \left[\left(\frac{X_t}{N} \right)^2 \right] \end{aligned} \quad (39)$$

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

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

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

From eq. 38 we know that

$$\begin{aligned} E \left[\frac{X_{t+1}}{N} - \frac{X_t}{N} \right] &= \frac{1}{N} s_{t+1} E \left[\frac{X_t}{N} \left(1 - \frac{X_t}{N} \right) \right] \\ &\simeq \frac{1}{N} s_{t+1} \left(E \left[\frac{X_t}{N} \right] \left(1 - E \left[\frac{X_t}{N} \right] \right) - \frac{1}{N_e} tx(1-x) \right) \\ &\simeq \frac{1}{N} s_{t+1} \cdot E \left[\frac{X_t}{N} \right] \left(1 - E \left[\frac{X_t}{N} \right] \right) - \frac{1}{N_e N} s_{t+1} tx(1-x) \end{aligned} \quad (41)$$

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

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

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

$$E \left[\frac{X_t}{N} - x \right] = E \left[\frac{X_t}{N} \right] - E[x] = E \left[\frac{X_t}{N} \right] - x, \quad (43)$$

so using the assumption we get

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

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

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

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

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

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

$$\begin{aligned}
E \left[\frac{X_{t+1}}{N} - x \right] &\simeq \frac{1}{N} s_{t+1} \cdot x (1 - x) + \frac{1}{N} S_t \cdot x (1 - x) \\
&\simeq \frac{1}{N} x (1 - x) (S_t + s_{t+1}) \\
&\simeq \frac{1}{N} S_{t+1} x (1 - x)
\end{aligned} \tag{47}$$

which proves the first part of our preposition.

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

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

using eq.38 we see that:

$$\begin{aligned}
E \left[\frac{X_{t+1}}{N} \middle| X_t \right] - E \left[\frac{X_t}{N} \middle| X_t \right] &= \frac{1}{N} s_{t+1} \frac{X_t}{N} \left(1 - \frac{X_t}{N} \right) \\
E \left[\frac{X_{t+1}}{N} \middle| X_t \right] &= \frac{X_t}{N} + \frac{1}{N} s_{t+1} \frac{X_t}{N} \left(1 - \frac{X_t}{N} \right)
\end{aligned} \tag{49}$$

Using eq.35 we get:

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

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

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

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

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

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

$$402 \quad V \left(\frac{1}{N} s_{t+1} \frac{X_t}{N} \left(1 - \frac{X_t}{N} \right) \right) \leq \frac{1}{4N^2} s_{t+1}^2 \quad (53)$$

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

$$404 \quad V \left(E \left[\frac{X_{t+1}}{N} \middle| X_t \right] \right) = V \left(\frac{X_t}{N} \right) + O \left(\frac{1}{N^2} \right) \simeq V \left(\frac{X_t}{N} \right) \quad (54)$$

Using the induction assumption and eq.51:

$$406 \quad V \left(\frac{X_{t+1}}{N} \right) \simeq \frac{1}{N_e} x(1 - x) + \frac{1}{N_e} tx(1 - x) \simeq \frac{1}{N_e} x(1 - x)(t + 1) \quad (55)$$

proving the second part of our preposition.

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

$$410 \quad P_{fix} = \frac{1 - e^{-2\frac{S_n}{n}N_ex}}{1 - e^{-2\frac{S_n}{n}N_e}} \quad (56)$$

where $\frac{S_n}{n} = \frac{k-l}{k+l}(1 - \text{beta})$, $n = k + l$. 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.

416 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.

418 We again tried different variations of the parameters, changing only one of them at a time. In fig.7 we can see that α on it's own doesn't cause any deviation for the the estimation. β however affects the results greatly.

420 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.

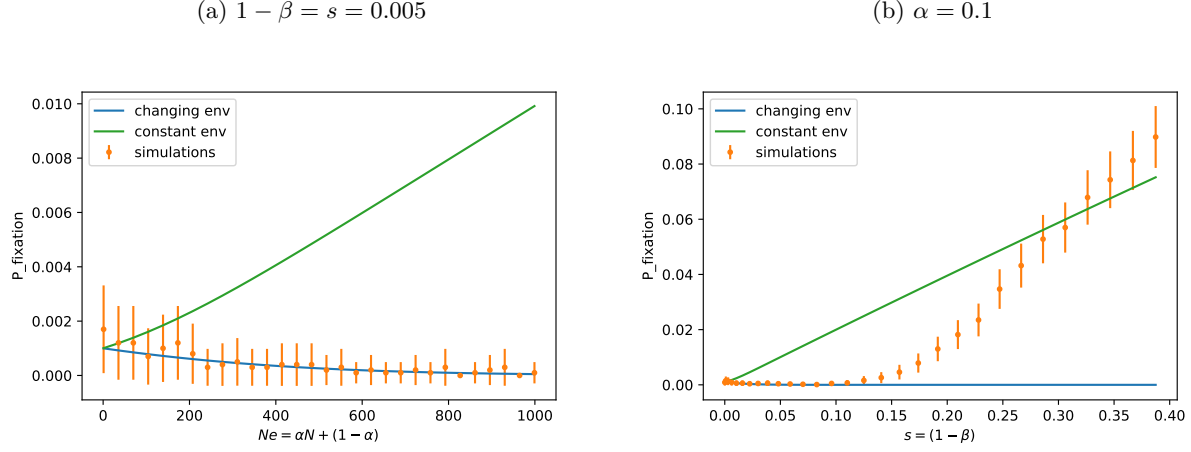


Figure 7: 10,000 simulations per data point, $N = 1,000$, $\hat{A} = 1$, $A = 0.9$, $J = 1$

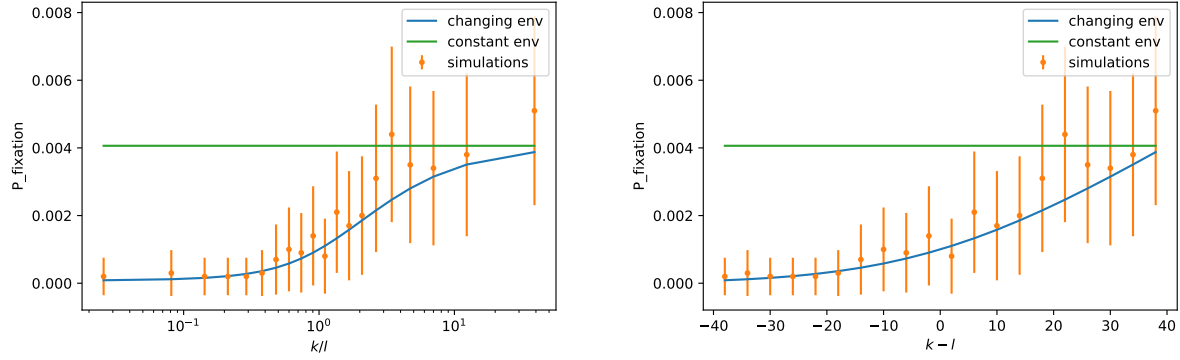


Figure 8: 10,000 simulations per data point, $N = 1,000$, $\hat{A} = 1$, $A = 0.8$, $J = 1$, $1 - \beta = s = 0.02$, $\alpha = 0.1$

In the larger values of β 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.8 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.

Conclusion

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.

438 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
440 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
442 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 probably
444 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
446 individuals already chose him as a role-model. We suggest a model for *prestige bias*, following the
indirect bias model Boyd and Richerson (1988) have suggested, and added the *influence trait* to it.
448 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
450 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
452 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
454 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),
456 and analyzing the differences when including several optimal values for the indicator trait (multiple
preference traits in the population).

458 **Appendix A - Time table**

Today - Oct 2021: Find approximation replacing Durrett's equations for the time to fixation.

460 **Nov - Mar 2021:** Combining the findings to a paper and a thesis.

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