

Non-Vertical Cultural Transmission, Assortment, and the Evolution of Cooperation

immediate

4

June 19, 2021

This work was carried out under the supervision of **Dr. Yoav Ram** from the School
6 of Zoology, Faculty of Life Sciences, Tel Aviv University.

Abstract

8 Cultural evolution of cooperation under vertical and non-vertical cultural transmission is studied,
and conditions are found for fixation and coexistence of cooperation and defection. The evolution
10 of cooperation is facilitated by its horizontal transmission and by an association between social
interactions and horizontal transmission. The effect of oblique transmission depends on the horizontal
12 transmission bias. Stable polymorphism of cooperation and defection can occur, and when it does,
reduced association between social interactions and horizontal transmission evolves, which leads to a
14 decreased frequency of cooperation and lower population mean fitness. The deterministic conditions
are compared to outcomes of stochastic simulations of structured populations. Parallels are drawn
16 with Hamilton's rule incorporating relatedness and assortment.

Contents

18	1 Introduction	5
	2 Models	9
20	3 Results	13
	3.1 Evolution of cooperation	13
22	3.2 Evolution of interaction-transmission association	20
	3.3 Population structure	22
24	4 Discussion	26
	A Local stability criterion	32
26	B Effect of interaction-transmission association on mean fitness	33
	C Reduction principle	34

28 1 Introduction

Cooperative behavior can reduce an individual's fitness and increase the fitness of its conspecifics or
30 competitors [1]. Nevertheless, cooperative behavior appears to occur in many animals [2], including
humans, primates [3], rats [4], birds [5, 6], and lizards [7]. Evolution of cooperative behavior has
32 been an important focus of research in evolutionary theory since at least the 1930s [8]. Since the work
of Hamilton [9] and Axelrod and Hamilton [1], theories for the evolution of cooperative and altruistic
34 behaviors have been intertwined often under the rubric of *kin selection*. Kin selection theory posits
that natural selection is more likely to favor cooperation between more closely related individuals. The
36 importance of *relatedness* to the evolution of cooperation and altruism was demonstrated by Hamilton
[9], who showed that an allele that determines cooperative behavior will increase in frequency if the
38 reproductive cost to the actor that cooperates, c , is less than the benefit to the recipient, b , times the
relatedness, r , between the recipient and the actor. This condition is known as *Hamilton's rule*:

$$40 \quad c < b \cdot r, \quad (1)$$

where the relatedness coefficient r measures the probability that an allele sampled from the cooperator
42 is identical by descent to one at the same locus in the recipient.

There is an ongoing debate about to what extent kin selection explains evolution of cooperation and
44 altruism. It has been suggested that kin selection to explain the cooperative behaviour of eusocial
insects like the honey bee. The most significant argument against kin selection is that cooperation can
46 evolve with zero relatedness [10]. This makes Hamilton's rule incomplete according to Wilson [10].
Foster et al. [11] reject this claim. They argue that altruism without relatedness can not evolve. They
48 refer us to Hamilton who claimed that relatedness can arise without recent common ancestry. Wilson
also criticises kin selection on the grounds that environmental or ecological factors probably be more
50 important than relatedness in determining social actions. On the other hand, Foster et al. [11] argue
that kin selection does not ignore ecology. Hamilton's rule shows that environmental factors causing
52 a high benefit: cost ratio will favour cooperation.

Beside kin selection, two more major theories were suggested to explain to evolution of coopera-
54 tion.

Reciprocity suggests repeating interactions or individual recognition as key factors for explaining the
56 evolution of cooperation. In *direct reciprocity* there are a repeated encounters between the same two
individuals. In every encounter, each player has a choice between cooperation and defection. If I
58 cooperate now, you may cooperate later. Hence, it may pay off to cooperate. This game-theoretic
framework is known as the repeated Prisoner's Dilemma. Direct reciprocity can only lead to the
60 evolution of cooperation if the cost is smaller than w the probability for another encounter between
the same two individuals multiplied by the benefit.

$$62 \quad c < bw \quad (2)$$

Direct reciprocity assumes that both players are in a position to cooperate. Direct reciprocity can
64 not explain cooperation in asymmetric interactions. In humans, such interactions happen often, for

example humans often donate money. *Indirect reciprocity* has been suggested to explain this behavior.

66 Nowak [12] claims that direct reciprocity is like a barter economy based on the immediate exchange of
67 goods, while indirect reciprocity resembles the invention of money. The money that "fuels the engines"
68 of indirect reciprocity is reputation. However, Reciprocity assume repeating interactions and therefore,
has difficulty explaining evolution of cooperation if the no repeating interactions occurs.

70 **Group Selection** theory posits that cooperation is favoured because of the advantage to the whole
71 group, if selection acts at the group level in addition to the individual level. A common model for group
72 selection work as is: the population is divided into groups. In each groups there are cooperators, which
73 help to other group members and defectors which do not help. Individuals reproduce proportional to
74 their fitness. Offspring are added to the same group. If a group reaches a certain size it can split to two
75 groups. A group that grow faster will split more often. Groups of cooperators are growing faster than
76 group of defectors. Therefore, cooperation can evolve in this model when the ratio between benefit
77 b and cost c is more than one plus the ratio between the maximum group size n and the number of
78 groups m:

$$\frac{b}{c} > 1 + \frac{n}{m} \quad (3)$$

80 All three theories mentioned above assume that cooperation is genetically determined. This raise
the question, is it possible that cooperation is determined by environmental or social influences.
82 Cooperative behavior can be subject to *cultural transmission*, which allows an individual to acquire
attitudes or behavioral traits from other individuals in its social group through imitation, learning, or
84 other modes of communication [13, 14]. Cultural transmission may be modeled as vertical, horizontal,
or oblique: vertical transmission occurs between parents and offspring, horizontal transmission occurs
86 between individuals from the same generation, and oblique transmission occurs to offspring from the
generation to which their parents belong (i.e. from non-parental adults). Evolution under either of these
88 transmission models can be be more rapid than under pure vertical transmission [13, 15, 16].

Here, we study models for the cultural evolution of cooperation that include both vertical and non-
90 vertical transmission. In our models behavioral changes are mediated by cultural transmission that
can occur specifically during social interactions. For instance, there may be an association between
92 the choice of partner for social interaction and the choice of partner for cultural transmission, or when
an individual interacts with an individual of a different phenotype, exposure to the latter may lead the
94 former to convert its phenotype. Our results demonstrate that cultural transmission, when associated
with social interactions, can enhance the evolution of cooperation even when genetic transmission
96 cannot, partly because it facilitates the generation of assortment [17], and partly because it diminishes
the effect of selection (due to non-vertical transmission from non-reproducing individuals [16]).

98 To understand the evolution of cooperation we are going to use *replicator dynamics*. The replicator
in replicator dynamics has the ability to make one or more copies of itself. The replicator can be
100 a gene, a phenotype, a stragey in a game and etc. In cooperation context replicator is a different
stragey in the game, whether the individual is a cooperative or a defector. In replicator dynamics
102 we assume large population of replicators, which interact with respect to their proportion. Those

interactions of different replicator affect the fitness according to some payoff matrix. The payoff
 104 matrix depends on the game which is played. The most common game to describe cooperation is
 prisoner's dilemma[18]. Similar to dominant strategies bringing forth Nash equilibria when games are
 106 repeated, strategies in replicator dynamics can become evolutionary stable. Such strategies are called
 Evolutionarily Stable Strategy (ESS). Such strategies cannot be invaded by any other strategy that is
 108 initially rare. Evolutionarily stable strategy maximizes the expected fitness of its chooser and therefore,
 causing maximum mean fitness. An interesting and fundamental question is to verify whether natural
 110 selection, operating within the framework of known genetical models leads to ESS. Note that usually
 natural selection does not lead to an increase in the average mean fitness of the population. Therefore,
 112 it is not generally true that natural selection does operate as to maximize the average individual fitness
 resulting in an optimal population strategy. Instead of searching evolutionarily stable strategy, we
 114 focus on *Evolutionary Genetic Stability (EGS)*[19]. EGS is when the frequency of types (geneotype,
 phenotype etc.) stays the same. Here, we study both local stability and external stability. Equilibrium
 116 is considered *Local stability* when a system near the equilibrium approaches it. *External stability* is a
 local stability of the equilibrium to invasion by the modifier allele m . One of the main questions in
 118 the evolution of cooperation is under what conditions such invasions are possible, or in other words
 under what conditions the system is external unstable.

120 Eshel and Cavalli-Sforza [20] studied a related model for the evolution of cooperative behavior. Their
 model included *assortative meeting*, or non-random encounters, where a fraction m of individuals in
 122 the population each interact specifically with an individual of the same phenotype, and a fraction $1 - m$
 interacts with a randomly chosen individual. Such assortative meeting may be due, for example, to
 124 population structure or active partner choice. In their model, cooperative behavior can evolve if [20,
 eq. 3.2]

$$c < b \cdot m, \quad (4)$$

where b and c are the benefit and cost of cooperation¹.

128 The role of assortment in the evolution of altruism was emphasized by Fletcher and Doebeli [17].
 They found that in a *public-goods* game, altruism will evolve if cooperative individuals experience
 130 more cooperation, on average, than defecting individuals, and “thus, the evolution of altruism requires
 (positive) assortment between focal *cooperative* players and cooperative acts in their interaction
 132 environment.” With some change in parameters, this condition is summarized by [17, eq. 2.3]

$$c < b \cdot (p_C - p_D), \quad (5)$$

134 where p_C is the probability that a cooperator receives help, and p_D is the probability that a defector
 receives help². Bijma and Aanen [21] obtained a result related to inequality 5 for other games.
 136 Cooperation can also evolve when interactions are determined by population structure. For example,
 Ohtsuki et al. [22] studied populations on graphs with average degree k , that is, the average individual

¹In an extended model, which allows an individual to encounter N individuals before choosing a partner, the right hand side is multiplied by $E[N]$, the expected number of encounters [20, eq. 4.6].

²Inequality 5 generalizes inequalities 1 and 4 by substituting $p_C = r + p$, $p_D = p$ and $p_C = m + (1-m)p$, $p_D = (1-m)p$, respectively, where p is the frequency of cooperators.

138 has k potential interaction partners. Assuming that selection is weak and that the population size is
much larger than k (i.e. sparse structure), they found that cooperative behaviour can evolve if [22]

140

$$c < b \cdot \frac{1}{k}. \quad (6)$$

They thus interpret $1/k$ as *social relatedness* or *social viscosity* [22].

142 Feldman et al. [23] introduced the first model for the evolution of altruism by cultural transmission
with kin selection and demonstrated that if the fidelity of cultural transmission of altruism is φ , then
144 the condition for evolution of altruism in the case of sib-to-sib altruism is [23, Eq. 16]

$$c < b \cdot \varphi - \frac{1 - \varphi}{\varphi}. \quad (7)$$

146 In inequality 7, φ replaces relatedness (r in inequality 1) or assortment (m in inequality 4), but the ef-
fective benefit $b \cdot \varphi$ is reduced by $(1 - \varphi)/\varphi$. This shows that under a cultural transmission, the condition
148 for the evolutionary success of altruism entails a modification of Hamilton's rule (inequality 1).

Both Woodcock [24] and Lewin-Epstein et al. [25] demonstrated that non-vertical transmission can help
150 explain the evolution of cooperative behavior, the former using simulations with cultural transmission,
the latter using a model where cooperation is mediated by host-associated microbes. Indeed, models
152 in which microbes affect their host's behavior [25, 26, 27] are mathematically similar to models of
cultural transmission, and they also emphasize the role of non-vertical transmission [13].

154 Handley and Mathew [28] showed the importance of culture on the human behavior. They showed that
the probability of individual to cooperate with unrelated strangers from a different group in transient
156 interactions corresponds to the degree of cultural similarity between those groups. Therefore, they have
suggested that group-level selection on culturally differentiated populations can explain cooperation
158 between unrelated humans from different groups.

2 Models

- 160 Consider a large population whose members can be one of two phenotypes: $\phi = A$ for cooperators or $\phi = B$ for defectors. An offspring inherits its phenotype from its parent via vertical transmission
 162 with probability v or from a random individual in the parental population via oblique transmission with probability $(1 - v)$ (Figure 1a). Following Ram et al. [16], given that the parent's phenotype is
 164 ϕ and assuming uni-parental inheritance [29], the conditional probability that the phenotype ϕ' of the offspring is A is

$$166 \quad P(\phi' = A | \phi) = \begin{cases} v + (1 - v)p, & \text{if } \phi = A \\ (1 - v)p, & \text{if } \phi = B \end{cases}, \quad (8)$$

where $p = P(\phi = A)$ is the frequency of A among all adults in the parental generation.

- 168 Not all adults become parents, and we denote the frequency of phenotype A among parents by \dot{p} . Therefore, the frequency \hat{p} of phenotype A among juveniles (after selection and vertical and oblique
 170 transmission) is

$$\hat{p} = \dot{p}[v + (1 - v)p] + (1 - \dot{p})[(1 - v)p] = v\dot{p} + (1 - v)p. \quad (9)$$

- 172 Individuals are assumed to interact according to a *prisoner's dilemma*. Specifically, individuals interact in pairs; a cooperator suffers a fitness cost $0 < c < 1$, and its partner gains a fitness benefit b , where we
 174 assume $c < b$. Figure 1a shows the payoff matrix, i.e. the fitness of an individual with phenotype ϕ_1 when interacting with a partner of phenotype ϕ_2 . The choice of prisoner's dilemma as the interaction
 176 model was motivated by the fact that prisoner's dilemma is a common game used to study evolution of cooperation[1], [30], [18]. Although we decided to focus on prisoner's dilemma, other games such
 178 as stag hunt[31] may be a better explanation of cooperation behavior in humans[32].

- Social interactions occur randomly: two juvenile individuals with phenotype A interact with probability
 180 \hat{p}^2 , two juveniles with phenotype B interact with probability $(1 - \hat{p})^2$, and two juveniles with different phenotypes interact with probability $2\hat{p}(1 - \hat{p})$. Horizontal cultural transmission occurs
 182 between pairs of individuals from the same generation. It occurs between socially interacting partners with probability α , or between a random pair with probability $1 - \alpha$ (see Figure 1b). However,
 184 horizontal transmission is not always successful, as one partner may reject the other's phenotype. The probability of successful horizontal transmission of phenotypes A and B are T_A and T_B , respectively
 186 (Table 1, Figure 1d). Thus, the frequency p' of phenotype A among adults in the next generation, after horizontal transmission, is

$$188 \quad p' = \hat{p}^2[\alpha + (1 - \alpha)(\hat{p} + (1 - \hat{p})(1 - T_B))] + \\ \hat{p}(1 - \hat{p})[\alpha(1 - T_B) + (1 - \alpha)(\hat{p} + (1 - \hat{p})(1 - T_B))] + \\ (1 - \hat{p})\hat{p}[\alpha T_A + (1 - \alpha)\hat{p}T_A] + (1 - \hat{p})^2[(1 - \alpha)\hat{p}T_A] \\ = \hat{p}^2(T_B - T_A) + \hat{p}(1 + T_A - T_B). \quad (10)$$

The frequency of A among parents (i.e. after selection) follows a similar dynamic, but also includes

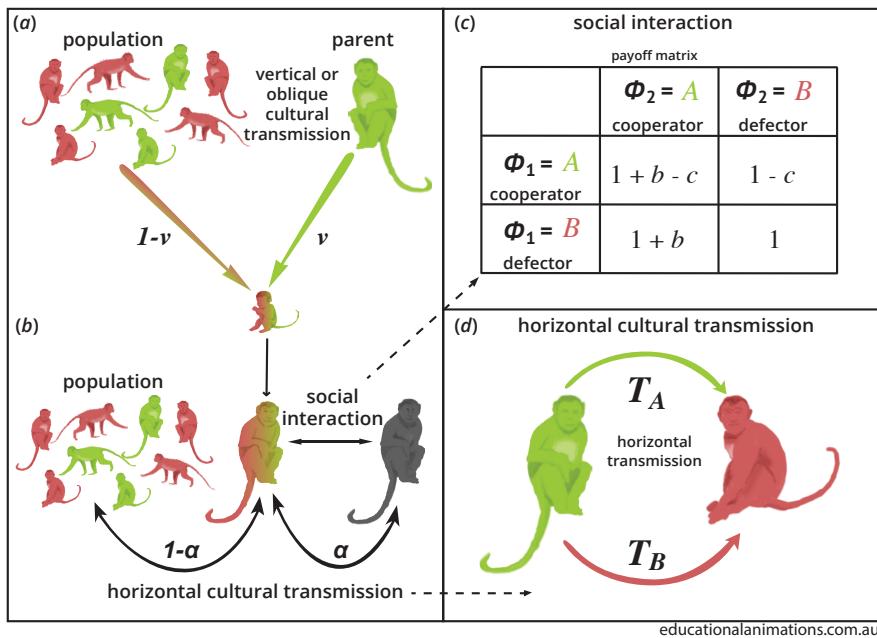


Figure 1: Model illustration. **(a)** First, offspring inherit their parent's phenotype via vertical cultural transmission with probability v , or the phenotype of a random non-parental adult via oblique cultural transmission with probability $1 - v$. **(b)** Second, adults socially interact in pairs in a prisoner's dilemma game. Horizontal cultural transmission occurs from a random individual in the population, with probability $1 - \alpha$, or from the social partner, with probability α , where α is the interaction-transmission association parameter. **(c)** The prisoner's dilemma payoff matrix shows the fitness of phenotype ϕ_1 when interacting with phenotype ϕ_2 . **(d)** The probabilities of successful horizontal cultural transmission of phenotypes A (cooperator) and B (defector) are T_A and T_B , respectively.

190 the effect of natural selection, and is therefore

$$\begin{aligned}\bar{w}\dot{p}' = & \hat{p}^2(1+b-c)\left[\alpha + (1-\alpha)(\hat{p} + (1-\hat{p})(1-T_B))\right] + \\ & \hat{p}(1-\hat{p})(1-c)\left[\alpha(1-T_B) + (1-\alpha)(\hat{p} + (1-\hat{p})(1-T_B))\right] + \\ & (1-\hat{p})\hat{p}(1+b)\left[\alpha T_A + (1-\alpha)\hat{p}T_A\right] + (1-\hat{p})^2\left[(1-\alpha)\hat{p}T_A\right],\end{aligned}\quad (11)$$

192 where fitness values are taken from Figure 1c and Table 1, and the population mean fitness is
 $\bar{w} = 1 + \hat{p}(b - c)$. Starting from Eq. 9 with $\hat{p}' = v\dot{p}' + (1-v)p'$, we substitute p' from Eq. 10 and \dot{p}'
194 from Eq. 11 and obtain

$$\begin{aligned}\hat{p}' = & \frac{v}{\bar{w}}\left[\hat{p}^2(1+b-c)\left(1 - (1-\hat{p})(1-\alpha)T_B\right)\right] + \\ & \frac{v}{\bar{w}}\left[\hat{p}(1-\hat{p})(1-c)\left(\hat{p}(1-\alpha)T_B + 1 - T_B\right)\right] + \\ & \frac{v}{\bar{w}}\left[\hat{p}(1-\hat{p})(1+b)\left(\hat{p}(1-\alpha) + \alpha\right)T_A\right] + \\ & \frac{v}{\bar{w}}(1-\hat{p})^2\hat{p}(1-\alpha)T_A + (1-v)\hat{p}^2(T_B - T_A) + (1-v)\hat{p}(1+T_A - T_B).\end{aligned}\quad (12)$$

196 Table 2 lists the model variables and parameters.

Table 1: Interaction frequency, fitness, and transmission probabilities.

Phenotype ϕ_1	Phenotype ϕ_2	Frequency	Fitness of ϕ_1	$P(\phi_1 = A)$ via horizontal transmission:	
				from partner, α	from population, $(1 - \alpha)$
A	A	\hat{p}^2	$1 + b - c$	1	$\hat{p} + (1 - \hat{p})(1 - T_B)$
A	B	$\hat{p}(1 - \hat{p})$	$1 - c$	$1 - T_B$	$\hat{p} + (1 - \hat{p})(1 - T_B)$
B	A	$\hat{p}(1 - \hat{p})$	$1 + b$	T_A	$\hat{p}T_A$
B	B	$(1 - \hat{p})^2$	1	0	$\hat{p}T_A$

Table 2: Model variables and parameters.

Symbol	Description	Values
A	Cooperator phenotype	
B	Defector phenotype	
p	Frequency of phenotype A among adults	$[0, 1]$
\dot{p}	Frequency of phenotype A among parents	$[0, 1]$
\hat{p}	Frequency of phenotype A among juveniles	$[0, 1]$
v	Vertical transmission rate	$[0, 1]$
c	Cost of cooperation	$(0, 1)$
b	Benefit of cooperation	$c < b$
α	Probability of interaction-transmission association	$[0, 1]$
T_A, T_B	Horizontal transmission rates of phenotype A and B	$(0, 1)$

3 Results

198 We determine the equilibria of the model in Eq. 12 and analyze their local stability. We then analyze
 the evolution of a modifier of interaction-transmission association, α . Finally, we compare derived
 200 conditions to outcomes of stochastic simulations with a structured population.

3.1 Evolution of cooperation

202 To learn about the evolution of cooperation we investigate local stability of the equilibria of the model
 in Eq. 12. The equilibria are the solutions of $\hat{p}' - \hat{p} = 0$. Note that Eq. 12 may look simple cubic
 204 polynomial. However, because \bar{w} is a function of \hat{p} , Eq. 12 is not simple polynomial but a fractional
 polynomial. The solution of fractional polynomial is not trivial and that is why it is better to find the
 206 equilibria and analyze the stability. Let $f(\hat{p}) = \bar{w}(\hat{p}' - \hat{p})$. Then, using *SymPy* [33], a Python library
 for symbolic mathematics, this simplifies to

$$208 \quad f(\hat{p}) = \bar{w}(\hat{p}' - \hat{p}) = \beta_1 \hat{p}^3 + \beta_2 \hat{p}^2 + \beta_3 \hat{p} , \quad (13)$$

where

$$\begin{aligned} \beta_1 &= [c(1 - v) - b(1 - \alpha v)](T_A - T_B) , \\ 210 \quad \beta_2 &= -\beta_1 - \beta_3 , \\ \beta_3 &= \alpha b v T_A - c v (1 - T_B) + (T_A - T_B) . \end{aligned} \quad (14)$$

If $T = T_A = T_B$ then $\beta_1 = 0$ and $\beta_3 = -\beta_2 = \alpha b v T - c v (1 - T)$, and $f(\hat{p})$ becomes a quadratic
 212 polynomial,

$$f(\hat{p}) = \hat{p}(1 - \hat{p})[\alpha b v T - c v (1 - T)] . \quad (15)$$

214 Clearly the only two equilibria are the fixations $\hat{p} = 0$ and $\hat{p} = 1$, which are locally stable if
 $f'(\hat{p}) < 0$ near the equilibrium (see Appendix A), where $f'(\hat{p}) = (1 - 2\hat{p})[\alpha b v T - c v (1 - T)]$, so
 216 that

$$\begin{aligned} f'(0) &= \alpha b v T - c v (1 - T) , \\ 218 \quad f'(1) &= -\alpha b v T + c v (1 - T) . \end{aligned} \quad (16)$$

In the general case where $T_A \neq T_B$, the coefficient β_1 is not necessarily zero, and $f(\hat{p})$ is a cubic
 220 polynomial. Therefore, three equilibria may exist, two of which are $\hat{p} = 0$ and $\hat{p} = 1$, and the third
 is

$$\hat{p}^* = \frac{\beta_3}{\beta_1} = \frac{\alpha b v T_A - c v (1 - T_B) + (T_A - T_B)}{[c(1 - v) - b(1 - \alpha v)](T_A - T_B)} . \quad (17)$$

222 Note that the sign of the cubic (Eq. 13) at positive (negative) infinity is equal (opposite) to the sign of
 β_1 . If $T_A > T_B$, then

$$224 \quad \beta_1 < [c(1 - \alpha v) - b(1 - \alpha v)](T_A - T_B) = (1 - \alpha v)(c - b)(T_A - T_B) < 0 , \quad (18)$$

since $c < b$ and $\alpha v < 1$. Hence the signs of the cubic at positive and negative infinity are negative and
 226 positive, respectively. First, if $\beta_3 < \beta_1$ then $1 < \hat{p}^*$. Also, $f'(0) < 0$ and $f'(1) > 0$; that is, fixation

of the defector phenotype B is the only locally stable feasible equilibrium. Second, if $\beta_1 < \beta_3 < 0$ then $0 < \hat{p}^* < 1$ and therefore $f'(0) < 0$ and $f'(1) < 0$ so that both fixations are locally stable and \hat{p}^* separates the domains of attraction. Third, if $0 < \beta_3$ then $\hat{p}^* < 0$ and therefore $f'(0) > 0$ and $f'(1) < 0$; that is, fixation of the cooperator phenotype A is the only locally stable legitimate equilibrium.

Similarly, if $T_A < T_B$, then

$$\beta_1 > [c(1 - \alpha v) - b(1 - \alpha v)](T_A - T_B) = (1 - \alpha v)(c - b)(T_A - T_B) > 0 , \quad (19)$$

since $c < b$ and $\alpha v < 1$, and the signs of the cubic at positive and negative infinity are positive and negative, respectively. First, if $\beta_3 < 0$ then $\hat{p}^* < 0$ and therefore $f'(0) < 0$ and $f'(1) > 0$; that is, fixation of the defector phenotype A is the only locally stable legitimate equilibrium. Second, if $0 < \beta_3 < \beta_1$ then $0 < \hat{p}^* < 1$ and therefore $f'(0) > 0$ and $f'(1) > 0$; that is, both fixations are locally unstable and \hat{p}^* is a stable polymorphic equilibrium. Third, if $\beta_1 < \beta_3$ then $\hat{p}^* > 1$ and therefore $f'(0) > 0$ and $f'(1) < 0$, and fixation of the cooperator phenotype A is the only locally stable feasible equilibrium.

This analysis can be summarized as follows:

1. *Fixation of cooperation:* if (i) $T = T_A = T_B$ and $c < b \cdot \frac{\alpha T}{1-T}$; or if (ii) $T_A > T_B$ and $0 < \beta_3$; or if (iii) $T_A < T_B$ and $\beta_1 < \beta_3$.
2. *Fixation of the defection:* if (iv) $T = T_A = T_B$ and $c > b \cdot \frac{\alpha T}{1-T}$; or if (v) $T_A > T_B$ and $\beta_3 < \beta_1 < 0$; or if (vi) $T_A < T_B$ and $\beta_3 < 0$.
3. *polymorphism of both phenotypes at \hat{p}^* :* if (vii) $T_A < T_B$ and $0 < \beta_3 < \beta_1$.
4. *Fixation of either phenotype depending on initial frequency:* if (viii) $T_A > T_B$ and $\beta_1 < \beta_3 < 0$.

We now proceed to use the cost thresholds, γ_1 and γ_2 , and the vertical transmission threshold, \hat{v} (??). First, assume $T_A < T_B$. $\beta_3 < 0$ requires $\gamma_1 < c$. For $\beta_3 < \beta_1$ we need $c[v(1 - T_B) + (1 - v)(T_A - T_B)] > b\alpha v T_B + (1 + b)(T_A - T_B)$. Note that the expression in the square brackets is positive if and only if $v > \hat{v}$. Thus, for $\beta_3 < \beta_1$ we need $v > \hat{v}$ and $\gamma_2 < c$ or $v < \hat{v}$ and $c < \gamma_2$, and for $0 < \beta_3 < \beta_1$ we need $v > \hat{v}$ and $\gamma_2 < c < \gamma_1$, or $v < \hat{v}$ and $c < \min(\gamma_1, \gamma_2)$. For $\beta_1 < \beta_3$ we need $v > \hat{v}$ and $c < \gamma_2$ or $v < \hat{v}$ and $\gamma_2 < c$. However, some of these conditions cannot be met, since $v < \hat{v}$ implies $c < 1 < \gamma_2$.

Second, assume $T_A > T_B$. $\beta_3 > 0$ requires $\gamma_1 > c$. For $\beta_1 < \beta_3$ we need $c[v(1 - T_B) + (1 - v)(T_A - T_B)] < b\alpha v T_B + (1 + b)(T_A - T_B)$. Thus for $\beta_1 < \beta_3$ we need $v > \hat{v}$ and $c < \gamma_2$ or $v < \hat{v}$ and $c > \gamma_2$. But $\hat{v} < 0$ when $T_A > T_B$, and therefore we have $\beta_1 < \beta_3$ if $c < \gamma_2$. Similarly, we have $\beta_3 < \beta_1$ if $c > \hat{\gamma}_2$.

Then we have the following result.

258

Result 1. *With vertical, horizontal, and oblique transmission, the cultural evolution of a cooperation follows one of the following scenarios in terms of the cost thresholds γ_1 and γ_2 and the vertical transmission threshold \hat{v} (??):*

- 262 1. Fixation of cooperation: if (i) $T_A \geq T_B$ and $c < \gamma_1$; or if (ii) $T_A < T_B$ and $v > \hat{v}$ and $c < \gamma_2$.

263 2. Fixation of defection: if (iii) $T_A \geq T_B$ and $\gamma_2 < c$; or if (iv) $T_A < T_B$ and $\gamma_1 < c$.

264 3. Stable polymorphism: if (v) $T_A < T_B$ and $v < \hat{v}$ and $c < \gamma_1$; or if (vi) $T_A < T_B$ and $v > \hat{v}$ and $\gamma_2 < c < \gamma_1$.

265 4. Unstable polymorphism: if (vii) $T_A > T_B$ and $\gamma_1 < c < \gamma_2$.

Thus, cooperation can take over the population if it has either a horizontal transmission advantage, or
 268 if it has a horizontal transmission disadvantage, but the vertical transmission rate is high enough. In
 either case, the cost of cooperation must be small enough. A stable polymorphism can exist between
 270 cooperation and defection only if defection has a horizontal transmission advantage. In this case,
 the existence of a stable polymorphism depends on the interplay between the benefit and cost of
 272 cooperation and the vertical transmission rate. These conditions are illustrated in Figures 2a, 2b, 3a,
 and 3b. Note that *stable* and unstable polymorphism are also called, respectively, *coexistence* and
 274 *bistable competition*.

Much of the literature on evolution of cooperation focuses on conditions for an initially rare cooperative phenotype to invade a population of defectors. The following remarks address this condition.

278 **Remark 1.** If the initial frequency of cooperation is very close to zero, then its frequency will increase if the cost of cooperation is low enough,

$$c < \gamma_1 = \frac{bv\alpha T_A + (T_A - T_B)}{v(1 - T_B)}. \quad (20)$$

This unites the conditions for fixation of cooperation and for stable polymorphism, both of which entail instability of the state where defection is fixed, $\hat{p} = 0$.

Importantly, increasing interaction-transmission association α increases the cost threshold ($\partial\gamma_1/\partial\alpha > 0$), making it easier for cooperation to increase in frequency when initially rare. Similarly, increasing the horizontal transmission of cooperation, T_A , increases the threshold ($\partial\gamma_1/\partial T_A > 0$), facilitating the evolution of cooperation ((Figure 3a and 3b). However, increasing the horizontal transmission of defection, T_B , can increase or decrease the cost threshold, but it increases the cost threshold when the threshold is already above one ($c < 1 < \gamma_1$): $\partial\gamma_1/\partial T_B$ is positive when $T_A > \frac{1}{1+\alpha bv}$, which gives $\gamma_1 > 1/v$. Therefore, increasing T_B decreases the cost threshold and limits the evolution of cooperation, but only if $T_A < \frac{1}{1+\alpha bv}$.

Increasing the vertical transmission rate, v , can either increase or decrease the cost threshold, depending on the horizontal transmission bias, $T_A - T_B$, because $\text{sign}(\partial\gamma_1/\partial v) = -\text{sign}(T_A - T_B)$. When $T_A < T_B$ we have $\partial\gamma_1/\partial v > 0$, and as the vertical transmission rate increases, the cost threshold increases, making it easier for cooperation to increase when rare (Figure 2b). In contrast, when $T_A > T_B$ we get $\partial\gamma_1/\partial v < 0$, and therefore as the vertical transmission rate increases, the cost threshold decreases, making it harder for cooperation to increase when rare (Figure 2a).

In general, this condition cannot be formulated in the form of Hamilton's rule due to the bias in horizontal transmission, represented by $T_A - T_B$. If $T_A = T_B$, then, from Result 1 and inequality 20, cooperation will take over the population from any initial frequency if the cost is low enough,

$$c < b \cdot \frac{\alpha T}{1 - T}, \quad (21)$$

and regardless of the vertical transmission rate, v . This condition can be interpreted as a version of Hamilton's rule ($c < b \cdot r$, inequality 1) or as a version of inequality 5, where $\alpha T/(1 - T)$ can be regarded as the *effective relatedness* or *effective assortment*, respectively. Note that the right-hand side of inequality 21 equals γ_1 when $T = T_A = T_B$.

From inequality 20, without interaction-transmission association ($\alpha = 0$), cooperation will increase when it is rare if there is horizontal transmission bias for cooperation, $T_A > T_B$, and

$$c < \frac{T_A - T_B}{v(1 - T_B)}. \quad (22)$$

Figure 3a illustrates this condition (for $v = 1$), which is obtained by setting $\alpha = 0$ in inequality 20. In this case, the benefit of cooperation, b , does not affect the evolution of cooperation, and the outcome is determined only by cultural transmission. Further, inequality 20 shows that with perfect interaction-transmission association ($\alpha = 1$), cooperation will increase when rare if

$$c < \frac{bvT_A + (T_A - T_B)}{v(1 - T_B)}. \quad (23)$$

In the absence of oblique transmission, $v = 1$, the only equilibria are the fixation states, $\dot{p} = 0$ and $\dot{p} = 1$, and cooperation will evolve from any initial frequency (i.e. $\dot{p}' > \dot{p}$) if inequality 23 applies (Figure 3). This is similar to case of microbe-induced cooperation studied by Lewin-Epstein et al. [25]; therefore when $v = 1$, this remark is equivalent to their eq. 1.

It is interesting to examine the general effect of interaction-transmission association α on the evolution of cooperation. Define the interaction-transmission association thresholds, a_1 and a_2 , as

$$a_1 = \frac{c \cdot v(1 - T_A) - (T_A - T_B)(1 + b - c)}{b \cdot v \cdot T_B}, \quad a_2 = \frac{c \cdot v(1 - T_B) - (T_A - T_B)}{b \cdot v \cdot T_A}. \quad (24)$$

Remark 2. Cooperation will increase when rare if interaction-transmission association is high enough, specifically if $a_2 < \alpha$.

Figures 2c and 2d illustrate this condition. With horizontal transmission bias for cooperation, $T_A > T_B$, cooperation can fix from any initial frequency if $a_2 < \alpha$ (green area in the figures). With horizontal bias favoring defection, $T_A < T_B$, cooperation can fix from any frequency if α is large enough, $a_1 < \alpha$ (green area with $T_A < T_B$), and can reach stable polymorphism if α is intermediate, $a_2 < \alpha < a_1$ (yellow area). Without horizontal bias, $T_A = T_B$, fixation of cooperation occurs if α is high enough, $\frac{c}{b} \cdot \frac{1-T}{T} < \alpha$ (inequality 21; in this case $a_1 = a_2$).

Interestingly, because $\text{sign}(\partial a_2 / \partial v) = \text{sign}(T_A - T_B)$, the effect of the vertical transmission rate v on a_1 and a_2 depends on the horizontal transmission bias. That is, if $T_A > T_B$, then evolution of

330 cooperation is facilitated by oblique transmission, whereas if $T_A < T_B$, then evolution of cooperation
is facilitated by vertical transmission (Figures 2c and 2d).

332

Next, we examine the roles of vertical and oblique transmission in the evolution of cooperation.
334 Fixation of cooperation is possible only if the vertical transmission rate is high enough,

$$v > \hat{v} = \frac{T_B - T_A}{1 - T_A}. \quad (25)$$

336 This condition is necessary for fixation of cooperation, but it is not sufficient. If horizontal transmission
is biased for cooperation, $T_A > T_B$, cooperation can fix with any vertical transmission rate (because
338 $\hat{v} < 0$). In contrast, if horizontal transmission is biased for defection, $T_A < T_B$, cooperation can fix
only if the vertical transmission rate is high enough: in this case oblique transmission can prevent
340 fixation of cooperation (see Figures 2b and 2d).

With only vertical transmission ($v = 1$), from inequality 20, cooperation increases when rare if

342

$$c < \frac{baT_A + (T_A - T_B)}{1 - T_B}, \quad (26)$$

which can also be written as

344

$$\frac{c(1 - T_B) - (T_A - T_B)}{bT_A} < \alpha. \quad (27)$$

In the absence of vertical transmission ($v = 0$), from recursion 12 we see that the frequency of the
346 cooperator phenotype among adults increases every generation, i.e. $p' > p$, if there is a horizontal
transmission bias in favor of cooperation, namely $T_A > T_B$. That is, if $v = 0$, then selection plays no
348 role in the evolution of cooperation (i.e. b and c do not affect \hat{p}'). The dynamics are determined solely
by differential horizontal transmission of the two phenotypes. With no bias in horizontal transmission,
350 $T_A = T_B$, phenotype frequencies do not change, $\hat{p}' = \hat{p}$.

Cooperation and defection can coexist at frequencies \hat{p}^* and $1 - \hat{p}^*$ (??). When it is feasible, this
352 equilibrium is stable or unstable under the conditions of Result 1, parts 3 and 4, respectively. The
yellow and blue areas in Figures 3 and 2 show cases of stable and unstable polymorphism, respectively.
354 When \hat{p}^* is unstable, cooperation will fix if its initial frequency is $\hat{p} > \hat{p}^*$, and defection will fix if
 $\hat{p} < \hat{p}^*$. \hat{p}^* is unstable when there is horizontal transmission bias for cooperation, $T_A > T_B$, and the
356 cost is intermediate, $\gamma_1 < c < \gamma_2$. Figure 3d shows $\hat{p}' - \hat{p}$ as a function of \hat{p} .

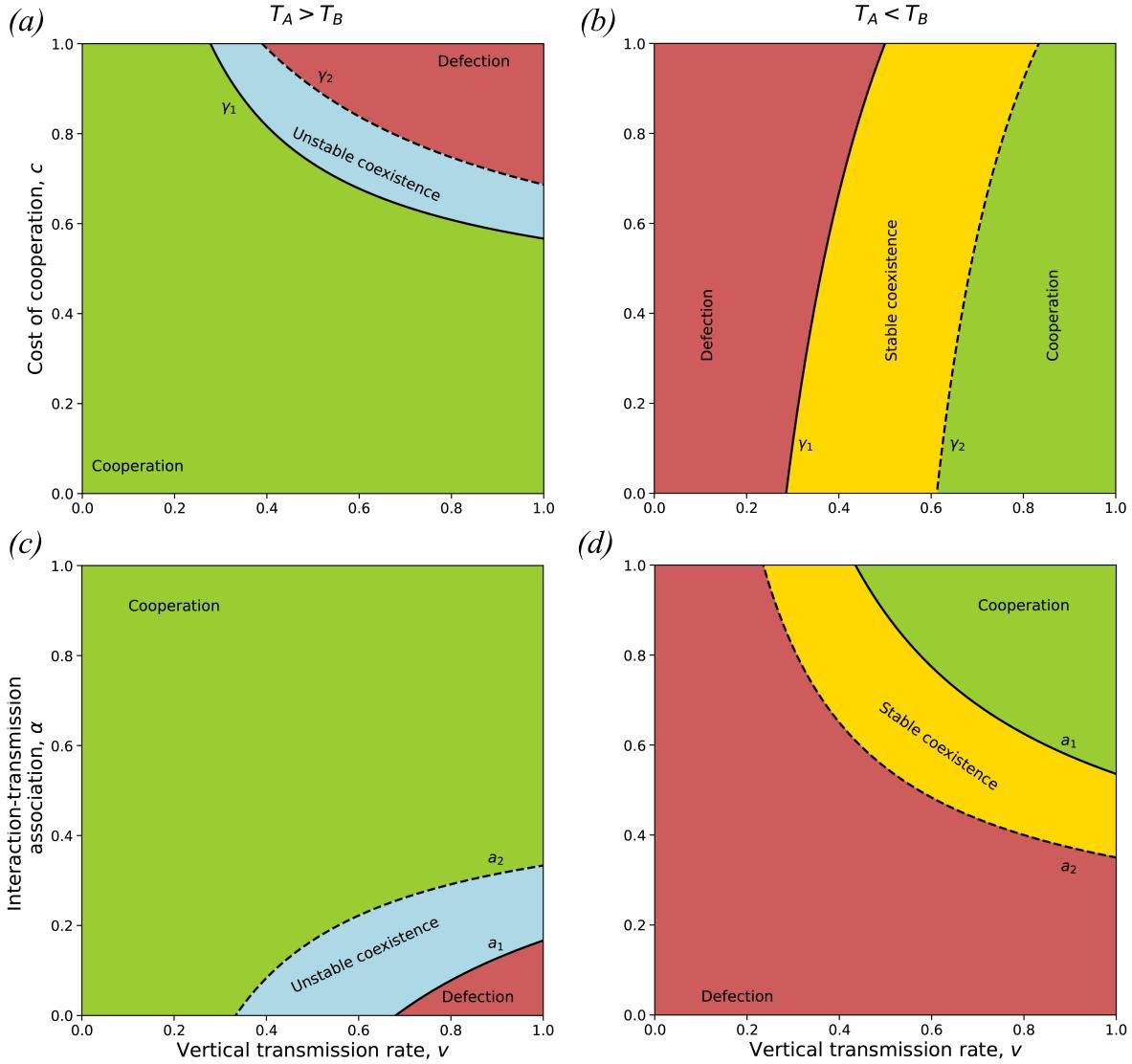


Figure 2: Evolution of cooperation under vertical, oblique, and horizontal cultural transmission.

The figure shows parameter ranges for global fixation of cooperation (green), global fixation of defection (red), fixation of either cooperation or defection depending on the initial conditions, i.e. unstable polymorphism (blue), and stable polymorphism of cooperation and defection (yellow). In all cases the vertical transmission rate v is on the x-axis. **(a-b)** Cost of cooperation c is on the y-axis and the cost thresholds γ_1 and γ_2 (Eqs. ??) are represented by the solid and dashed lines, respectively. **(c-d)** Interaction-transmission association α is on the y-axis and the interaction-transmission association thresholds a_1 and a_2 (Eqs. 24) are represented by the solid and dashed lines, respectively. Horizontal transmission is biased in favor of cooperation, $T_A > T_B$, in **(a)** and **(c)**, or defection, $T_A < T_B$, in **(b)** and **(d)**. Here, $T_A = 0.5$, and **(a)** $b = 1.2$, $T_B = 0.4$, $\alpha = 0.4$; **(b)** $b = 2$, $T_B = 0.7$, $\alpha = 0.7$; **(c)** $b = 1.2$, $T_B = 0.4$, $c = 0.5$; **(d)** $b = 2$, $T_B = 0.7$, $c = 0.5$.

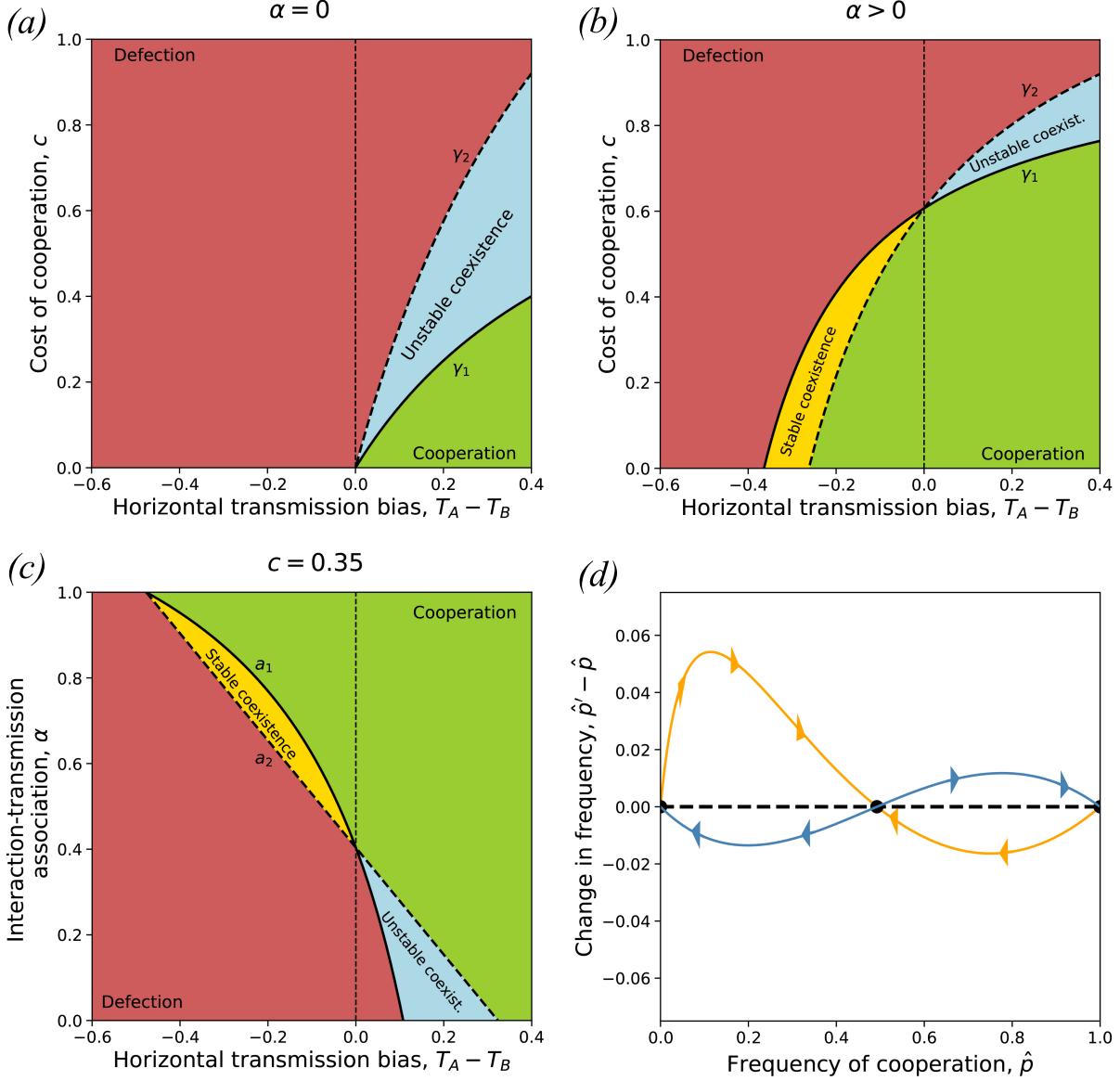


Figure 3: Evolution of cooperation under vertical and horizontal cultural transmission ($v=1$).

The figure shows parameter ranges for global fixation of cooperation (green), global fixation of defection (red), fixation of either cooperation or defection depending on the initial conditions, i.e. unstable polymorphism (blue), and stable polymorphism of cooperation and defection (yellow). **(a-c)** The horizontal transmission bias ($T_A - T_B$) is on the x-axis. In panels **(a)** and **(b)**, the cost of cooperation c is on the y-axis and the cost thresholds γ_1 and γ_2 (??) are the solid and dashed lines, respectively. In panel **(c)**, interaction-transmission association α is on the y-axis and the interaction-transmission association thresholds a_1 and a_2 (Eqs. 24) are the solid and dashed lines, respectively. Here, $b = 1.3$, $T_A = 0.4$, $v = 1$, (a) $\alpha = 0$, (b) $\alpha = 0.7$, (c) $c = 0.35$. **(d)** Change in frequency of cooperation among juveniles ($\hat{p}' - \hat{p}$) as a function of the frequency (\hat{p}), see Eq. 12. The orange curve shows convergence to a stable polymorphism ($T_A = 0.4$, $T_B = 0.9$, $b = 12$, $c = 0.35$, $v = 1$, and $\alpha = 0.45$). The blue curve shows fixation of either cooperation or defection, depending on the initial frequency ($T_A = 0.5$, $T_B = 0.1$, $b = 1.3$, $c = 0.904$, $v = 1$, and $\alpha = 0.4$). Black circles show the three equilibria.

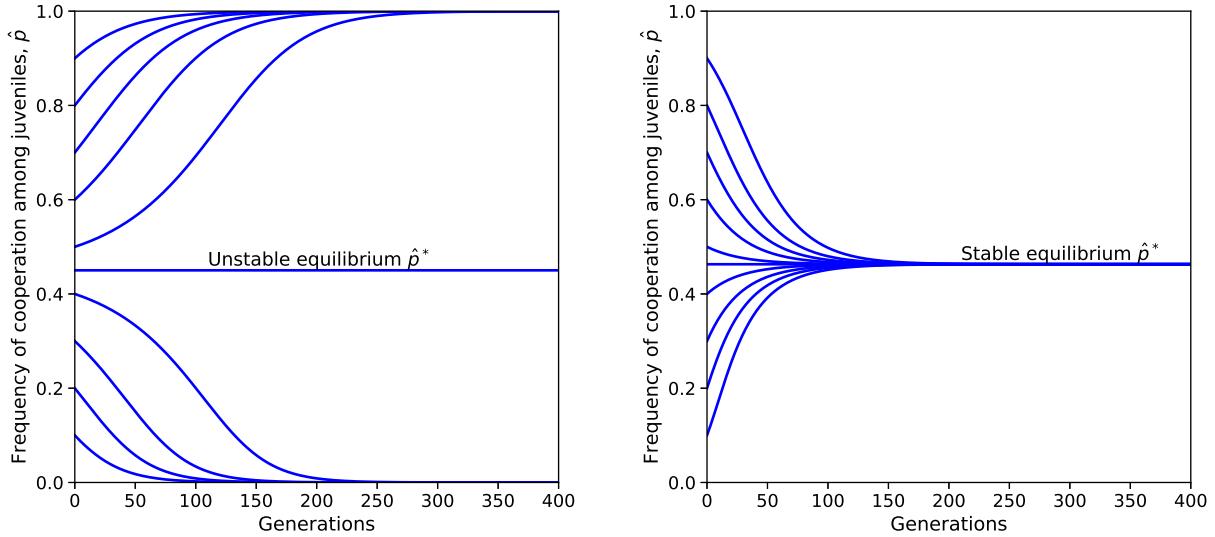


Figure 4: Stable and unstable equilibrium dynamics The figure shows the frequency of cooperation over time (generations) starting from different initial frequencies. In (a) the system has unstable equilibrium and therefore, all the curves are moving away from it. In (b) the system has stable equilibrium and therefore, all the curves moves towards it. Here, $T_A = 0.5$, $c = 0.5$, $v = 0.59$ and (a) $T_B = 0.4$, $\alpha = 0.1$ and $b = 1.2$. (b) $T_B = 0.7$, $\alpha = 0.6$ and $b = 2.3$.

3.2 Evolution of interaction-transmission association

358 We now focus on the evolution of interaction-transmission association under perfect vertical transmission, $v = 1$, assuming that the population is initially at a stable polymorphism of the two phenotypes,
 360 cooperation A and defection B , where the frequency of A among juveniles is \hat{p}^* (??). Note that for
 362 a stable polymorphism, there must be horizontal bias for defection, $T_A < T_B$, and an intermediate
 364 cost of cooperation, $\gamma_2 < c < \gamma_1$ (??), see Figure 3b. The equilibrium population mean fitness is
 $\bar{w}^* = 1 + \hat{p}^*(b - c)$, which is increasing in \hat{p}^* , and \hat{p}^* is increasing in α (Appendix B). Therefore, \bar{w}^*
 increases as α increases. But can this population-level advantage lead to the evolution of α ?

To answer this question, we add a “modifier locus” [34, 35, 36, 37] that determines the value of α
 366 but has no direct effect on fitness. This locus has two alleles, M and m , which induce interaction-
 transmission associations α_1 and α_2 , respectively. Suppose that the population has evolved to a stable
 368 equilibrium \hat{p}^* when only allele M is present. We study the local stability of this equilibrium to in-
 vasion by the modifier allele m ; this is called “external stability” [36, 38] and obtain the following result.
 370

Result 2. *From a stable polymorphism between cooperation and defection, a modifier allele can
 372 successfully invade the population if it decreases the interaction-transmission association α .*

The analysis is in Appendix C. This reduction principle entails that successful invasions will reduce
 374 the frequency of cooperation, as well as the population mean fitness (Figure 5). Furthermore, if we a
 modifier allele that decreases α appears and invades the population from time to time, then the value

376 of α will continue to decrease, further reducing the frequency of cooperation and the population mean
 378 fitness. This evolution will proceed as long as there is a stable polymorphism, that is, as long as
 $a_2 < \alpha < a_1$ (Remark 2, Figure 3c). Thus, we can expect the value of α to approach a_2 , the frequency
 380 of cooperation to fall to zero, and the population mean fitness to decrease to one (Figure 5).

382 We can do similiar analysis for the general case where $0 < v < 1$ as we did in Appendix C. First we
 found the value of the characteristic polynomial at 1, $R(1)$ using *SymPy* [33], a Python library for
 382 symbolic mathematics.

$$R(1) = \frac{cv\hat{p}^*[T_A b\hat{p}^{*2}(v\alpha_2 - 1) - 2T_A b\hat{p}^*v\alpha_2 + T_A b\hat{p}^*(1 + v\alpha_2)]}{b\hat{p}(b\hat{p}^* - 2\hat{p}^* + 2) + c\hat{p}^*(c\hat{p}^* - 2) + 1} \\ + \frac{cv\hat{p}^*[T_A c\hat{p}^{*2}(1 - v) + T_A c\hat{p}^*(v - 1) - T_A \hat{p}^*(1 - c) + T_A]}{b\hat{p}(b\hat{p}^* - 2\hat{p}^* + 2) + c\hat{p}^*(c\hat{p}^* - 2) + 1} \\ + \frac{cv\hat{p}^*[T_B b\hat{p}^{*2}(1 - v\alpha_2) + T_B b\hat{p}^2(v\alpha_2 - 1) + T_B c\hat{p}^{*2}(v - 1)]}{b\hat{p}(b\hat{p}^* - 2\hat{p}^* + 2) + c\hat{p}^*(c\hat{p}^* - 2) + 1} \\ + \frac{cv\hat{p}^*[T_B c\hat{p}^*(1 - v) + T_B cv(1 - \hat{p}^* + T_B(\hat{p}^* - 1) + cv(\hat{p}^* - 1))]}{b\hat{p}(b\hat{p}^* - 2\hat{p}^* + 2) + c\hat{p}^*(c\hat{p}^* - 2) + 1} \quad (28)$$

384 Similarly to what we did in Appendix C, we should find when $R(1) < 0$. However, when adding the
 vertical transmission into the system the math is more complicated. Therefore, we will use a different
 386 technique. We know that when $\alpha_1 = \alpha_2$, $R(1) = 0$. Now, let's assume that $\alpha_2 = \alpha_1 + \epsilon$. The derivative
 of $R(1)$ by ϵ will give us better understand about the sign of $R(1)$ when $\alpha_2 < \alpha_1$.

$$388 \quad \frac{\partial R(1)}{\partial \epsilon} = \frac{cbv^2\hat{p}^*[T_A \hat{p}^{*2} - 2T_A \hat{p}^* + T_A - T_B \hat{p}^{*2} + T_B \hat{p}^*]}{b\hat{p}(b\hat{p}^* - 2\hat{p}^* + 2) + c\hat{p}^*(c\hat{p}^* - 2) + 1} \quad (29)$$

We can simplify Eq. 29

$$390 \quad \frac{\partial R(1)}{\partial \epsilon} = \frac{cbv^2\hat{p}^*[(T_A - T_B)\hat{p}^{*2} + (T_B - 2T_A)\hat{p}^* + T_A]}{(b\hat{p}^* - c\hat{p}^*)^2 + 2\hat{p}^*(b - c) + 1} \quad (30)$$

Since $b > c$ the denominator is always positive. The numerator is quadratic polynomial of \hat{p}^* with
 392 the following roots:

$$\hat{p}_1^* = \frac{T_A}{T_A - T_B}\hat{p}_2^* = 1 \quad (31)$$

394 $\hat{p}_1^* < 0$ since $T_B > T_A$ and since the quadratic polynomial has negative leading coefficient then the
 numerator is positive for every $\hat{p}_1^* < \hat{p}^* < 1$ which is always true. We found that derivative of $R(1)$
 396 by ϵ is positive for every ϵ . Therefore, $R(1)$ grows as ϵ grows, or in other words as $\alpha_2 - \alpha_1$ grows.
 Thus, $R(1) < 0$ if and only if $\alpha_2 < \alpha_1$. Therefore, different value of vertical transmission does not
 398 change the evolution of social-interaction association and invasion is possible when $\alpha_2 < \alpha_1$.

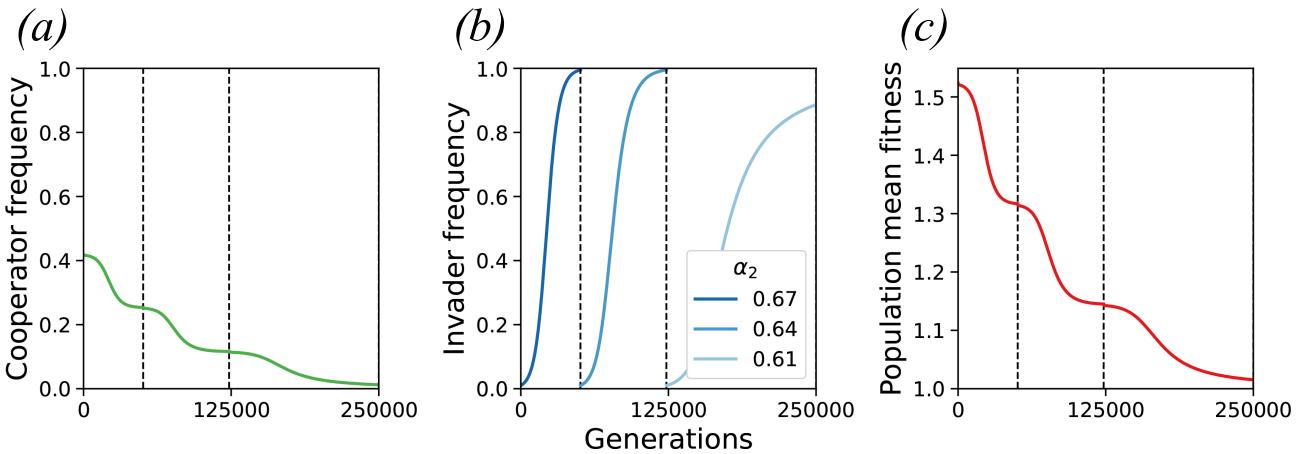


Figure 5: Reduction principle for interaction-transmission association. Consecutive fixation of modifier alleles that reduce interaction-transmission association α in numerical simulations of evolution with two modifier alleles (Eq. D1). When an invading modifier allele is established in the population (frequency $> 99.95\%$), a new modifier allele that reduces interaction-transmission association by 5% is introduced (at initial frequency 0.5%). **(a)** The frequency of the cooperative phenotype A over time. **(b)** The frequency of the invading modifier allele m over time. **(c)** The population mean fitness (\bar{w}) over time. Here, $v = 1$, $c = 0.05$, $b = 1.3$, $T_A = 0.4 < T_B = 0.7$, initial interaction-transmission association $\alpha_1 = 0.7$, lower interaction-transmission association threshold $\alpha_2 = 0.605$.

3.3 Population structure

- 400 All the simulations in this section were made by Ohad Lewin-Epstein from Tel Aviv University.
 Interaction-transmission association may also emerge from population structure. Consider a pop-
 402 ulation colonizing a two-dimensional grid of size 100-by-100, where each site is inhabited by one
 individual, similarly to the model of Lewin-Epstein and Hadany [26]. Each individual is characterized
 404 by its phenotype: either cooperator, A , or defector, B . Initially, each site in the grid is randomly
 colonized by either a cooperator or a defector, with equal probability. In each generation, half of the
 406 individuals are randomly chosen to "initiate" interactions, and these initiators interact with a random
 neighbor (i.e. individual in a neighboring site) in a prisoners' dilemma game (Figure 1c) and a random
 408 neighbor (with replacement) for horizontal cultural transmission (Figure 1b). The expected number of
 each of these interactions per individual per generation is one, but the realized number of interactions
 410 can be zero, one, or even more than one, and in every interaction both individuals are affected, not just
 the initiator. The effective interaction-transmission association α in this model is the probability that
 412 the same neighbor is picked for both interactions, or $\alpha = 1/M$, where M is the number of neighbors.
 On an infinite grid, $M = 8$ (i.e. Moore neighbourhood [39]), but on a finite grid M can be lower
 414 in neighbourhoods close to the grid border. As before, T_A and T_B are the probabilities of successful
 horizontal transmission of phenotypes A and B , respectively.
- 416 The order of the interactions across the grid at each generation is random. After all interactions take
 place, an individual's fitness is determined by $w = 1 + b \cdot n_b - c \cdot n_c$, where n_b is the number of interactions

418 that individual had with cooperative neighbors, and n_c is the number of interactions in which that
419 individual cooperated (note that the phenotype may change between consecutive interactions due to
420 horizontal transmission). Then, a new generation is produced, and the sites can be settled by offspring
421 of any parent, not just the neighboring parents. Selection is global, rather than local, in accordance
422 with our deterministic model: The parent is randomly drawn with probability proportional to its
423 fitness, divided by the sum of the fitness values of all potential parents. Offspring are assumed to have
424 the same phenotype as their parents (i.e. $v = 1$).

The outcomes of stochastic simulations with such a structured population are shown in Figure 6, which
426 demonstrates that the highest cost of cooperation c that permits the evolution of cooperation agrees
427 with the conditions derived above for our model without population structure or stochasticity. An
428 example of stochastic stable polymorphism is shown in Figure 6c. Changing the simulation so that
429 selection is local (i.e. sites can only be settled by offspring of neighboring parents) had only a minor
430 effect on the agreement with the derived conditions (Figure 7).

These comparisons between the deterministic unstructured model and the stochastic structured model
432 show that the conditions derived for the deterministic model can be useful for predicting the dynamics
433 under complex scenarios. Moreover, this structured population model demonstrates that our parameter
434 for interaction-transmission association, α , can represent local interactions between individuals.

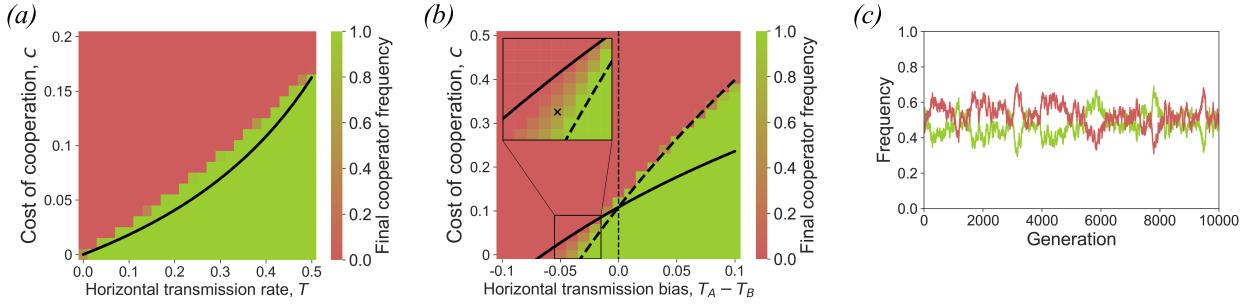


Figure 6: Evolution of cooperation in a structured population. (a-b) The expected frequency of cooperators in a structured population after 10,000 generations is shown (red for 0%, green for 100%) as a function of both the cost of cooperation, c , on the y-axis, and either the symmetric horizontal transmission rate, $T = T_A = T_B$, on the x-axis of panel (a), or the transmission bias, $T_A - T_B$, on the x-axis of panel (b). Black curves represent the cost thresholds for the evolution of cooperation in a well-mixed population with interaction-transmission association, where $\alpha = 1/8$ in inequality 21 for panel (a) and in Eqs. ?? for panel (b). The inset in panel (b) focuses on an area of the parameter range in which neither phenotype is fixed throughout the simulation, maintaining a stochastic locally stable polymorphism [40]. This stochastic polymorphism is illustrated in panel (c), which shows the frequency of cooperators (green) and defectors (red) over time for the parameter set marked by an x in panel (b). In all cases, the population evolves on a 100-by-100 grid. Cooperation and horizontal transmission are both local between neighbouring sites, and each site has 8 neighbours. Selection operates globally (see Figure S2 for results from a model with local selection). Simulations were stopped at generation 10,000 or if one of the phenotypes fixed. 50 simulations were executed for each parameter set. Benefit of cooperation, $b = 1.3$; perfect vertical transmission $v = 1$. (a) Symmetric horizontal transmission, $T = T_A = T_B$; (b) Horizontal transmission rate T_A is fixed at 0.4, and T_B varies, $0.3 < T_B < 0.5$. (c) Horizontal transmission rates $T_A = 0.4 < T_B = 0.435$ and cost of cooperation $c = 0.02$.

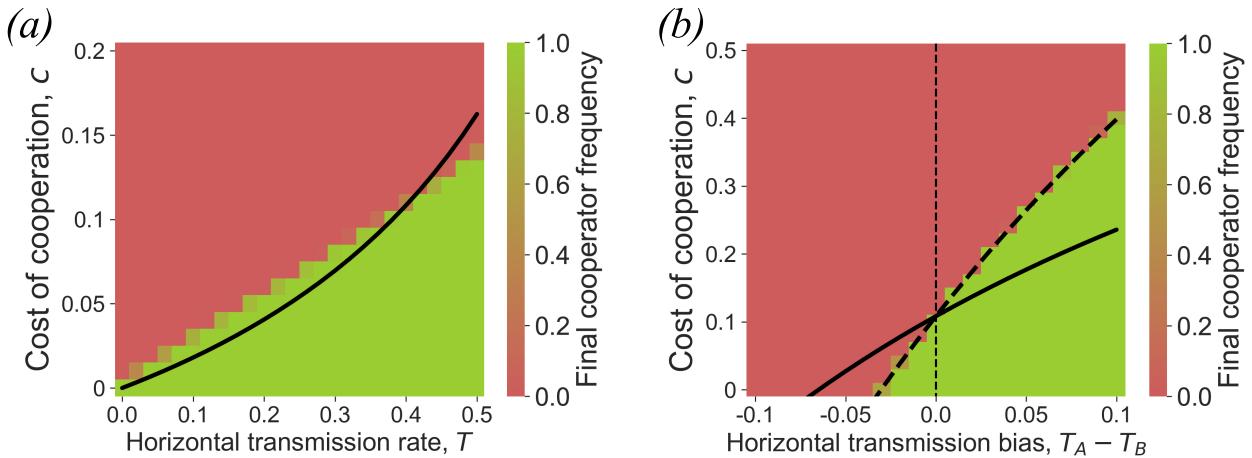


Figure 7: Evolution of cooperation in a structured population with local selection. The expected frequency of cooperators in a structured population after 10,000 generations is shown (red for 0%, green for 100%) as a function of both the cost of cooperation (c) on the y-axis, and the symmetric horizontal transmission rate ($T = T_A = T_B$) on the x-axis of panel (a), or the transmission bias $T_A - T_B$ on the x-axis of panel (b). Cooperation and horizontal transmission are both local between neighbouring sites, and each site had 8 neighbours. Selection operates locally (see Figure 4 for results from a model with global selection). The black curves represent the cost thresholds for the evolution of cooperation in a well-mixed population with interaction-transmission association, where $\alpha = 1/8$ in inequality 14 for panel (a) and in Eqs. 12 for panel (b). The population evolves on a 100-by-100 grid. Simulations were stopped at generation 10,000 or if one of the phenotypes fixed. 50 simulations were executed for each parameter set. Here, benefit of cooperation, $b = 1.3$; perfect vertical transmission $v = 1$. (a) Symmetric horizontal transmission, $T = T_A = T_B$. (b) Horizontal transmission rate T_A is fixed at 0.4, and T_B varies, $0.3 < T_B < 0.5$.

4 Discussion

436 Under a combination of vertical, oblique, and horizontal transmission with payoffs in the form
437 of a prisoner’s dilemma game, cooperation or defection can either fix or coexist, depending on
438 the relationship between the cost and benefit of cooperation, the horizontal transmission bias, and
439 the association between social interaction and horizontal transmission (Result 1, Figures 2 and 3).
440 Importantly, cooperation can increase when initially rare (i.e. invade a population of defectors) if and
441 only if, rewriting inequality 20, $c \cdot v(1 - T_B) < b \cdot v\alpha T_A + (T_A - T_B)$, namely, the effective cost of
442 cooperation (left-hand side) is smaller than the effective benefit plus the horizontal transmission bias
443 (right-hand side). This condition cannot be formulated in the form of Hamilton’s rule, $c < b \cdot r$, due to
444 the effect of biased horizontal transmission, represented by $(T_A - T_B)$. Remarkably, a polymorphism
445 of cooperation and defection can be stable if horizontal transmission is biased in favor of defection
446 ($T_A < T_B$) and both c and α are intermediate (yellow areas in Figures 2 and 3).

We find that stronger interaction-transmission association α leads to evolution of higher frequency
448 of cooperation and increased population mean fitness. Nevertheless, when cooperation and defection
449 coexist, α is expected to be reduced by natural selection, leading to extinction of cooperation and
450 decreased population mean fitness (Result 2, Figure 5). With $\alpha = 0$, the benefit of cooperation cannot
facilitate its evolution; it can only succeed if horizontal transmission is biased in its favor.

452 Indeed, in our model, horizontal transmission plays a major role in the evolution of cooperation:
increasing the transmission of cooperation, T_A , or decreasing the transmission of defection, T_B , facilitates
454 the evolution of cooperation. However, the effect of oblique transmission is more complicated.
When there is horizontal transmission bias in favor of cooperation, $T_A > T_B$, increasing the rate of
456 oblique transmission, $1 - v$, will facilitate the evolution of cooperation. In contrast, when the bias is
in favor of defection, $T_A < T_B$, higher rates of vertical transmission, v , are advantageous for cooperation,
458 and the rate of vertical transmission must be high enough ($v > \hat{v}$) for cooperation to fix in the
population.

460 Our deterministic model provides a good approximation to outcomes of simulations of a complex
stochastic model with population structure in which individuals can only interact with and transmit
462 to their neighbors. In these structured populations interaction-transmission association arises due to
both social interactions and horizontal cultural transmission being local (Figure 6 and Figure 7). We
464 did not find any significant difference between local and global selection.

Feldman et al. [23] studied the dynamics of an altruistic phenotype with vertical cultural transmission
466 and a gene that modifies the transmission of the phenotype. Their results are very sensitive to
this genetic modification: without it, the conditions for invasion of the altruistic phenotype reduce
468 to Hamilton’s rule. Further work is needed to incorporate such genetic modification of cultural
transmission into our model. Woodcock [24] stressed the significance of non-vertical transmission for
470 the evolution of cooperation and carried out simulations with prisoner’s dilemma payoffs but without
horizontal transmission or interaction-transmission association ($\alpha = 0$). Nevertheless, his results
472 demonstrated that it is possible to sustain altruistic behavior via cultural transmission for a substantial

length of time. He further hypothesized that horizontal transmission can play an important role in the
474 evolution of cooperation, and our results provide strong evidence for this hypothesis.

To understand the role of horizontal transmission, we first review the role of *assortment*. Eshel and
476 Cavalli-Sforza [20] showed that altruism can evolve when the tendency for *assortative meeting*, i.e.
478 for individuals to interact with others of their own phenotype, is strong enough. Fletcher and Doebeli
480 [17] further argued that a general explanation for the evolution of altruism is given by *assortment*: the
482 correlation between individuals that carry an altruistic trait and the amount of altruistic behavior in
484 their interaction group (see also Bijma and Aanen [21]). They suggested that to explain the evolution of
486 altruism, we should seek mechanisms that generate assortment, such as population structure, repeated
488 interactions, and individual recognition. Our results highlight another mechanism for generating
490 assortment: an association between social interactions and horizontal transmission that creates a
492 correlation between one's partner for interaction and the partner for transmission. This mechanism
494 does not require repeated interactions, population structure, or individual recognition. We show that
496 high levels of such interaction-transmission association greatly increase the potential for evolution of
498 cooperation. With enough interaction-transmission association, cooperation can increase in frequency
500 when initially rare even when there is horizontal transmission bias against it ($T_A < T_B$).

How does non-vertical transmission generate assortment? Lewin-Epstein et al. [25] and Lewin-Epstein
490 and Hadany [26] suggested that microbes that induce their hosts to act altruistically can be favored
492 by selection, which may help to explain the evolution of cooperation. Indeed, it has been shown
494 that microbes can mediate behavioral changes in their hosts [41, 42]. Therefore, natural selection
496 on microbes may favor manipulation of the host so that it cooperates with others. From the kin
498 selection point-of-view, if microbes can be transmitted *horizontally* from one host to another during
500 host interactions, then following horizontal transmission the recipient host will carry microbes that
502 are closely related to those of the donor host, even when the two hosts are (genetically) unrelated.
From the assortment point-of-view, infection by behavior-determining microbes during interactions
504 effectively generates assortment because a recipient of help may be infected by a behavior-determining
506 microbe and consequently become a helper. Cultural horizontal transmission can similarly generate
508 assortment between cooperators and enhance the benefit of cooperation if cultural transmission and
510 helping interactions occur between the same individuals, i.e. when there is interaction-transmission
512 association, so that the recipient of help may also be the recipient of the cultural trait for cooperation.
Thus, with horizontal transmission, “assortment between focal cooperative players and cooperative
514 acts in their interaction environment” [17] is generated not because the helper is likely to be helped,
516 but rather because the helped is likely to become a helper.

506 Another mechanism that was suggested by Traxler and Spichtig [43] showed that *conditional coop-*
508 *eration* based on norm-dependent relational utilities, i.e. individual will only cooperate if it knows
510 that others will cooperate too, can sustain cooperation in a community – provided that cooperation is
512 already at a high level. Unlike *conditional cooperation*, interaction-transmission association can sus-
514 tain cooperation in community even if cooperation is initially rare. Morsky and Akcay [44] suggested
516 that false beliefs on the frequencies of the cooperator can affect the individual decision whether to

512 cooperate or not. If the individual over estimate the number of cooperators it will be more likely to cooperate. This flase belief can help sustain cooperation even if cooperartion is initially rare.

514 **References**

- [1] Robert Axelrod and William D Hamilton. The evolution of cooperation. *Science*, 211(4489):1390–1396, 1981.
- [2] Lee Alan Dugatkin. *Cooperation among Animals: An Evolutionary Perspective*. Oxford University Press on Demand, 1997.
- [3] Adrian V Jaeggi and Michael Gurven. Natural cooperators: food sharing in humans and other primates. *Evolutionary Anthropology: Issues, News, and Reviews*, 22(4):186–195, 2013.
- [4] George E Rice and Priscilla Gainer. “Altruism” in the albino rat. *Journal of Comparative and Physiological Psychology*, 55(1):123, 1962.
- [5] Peter B Stacey and Walter D Koenig, editors. *Cooperative breeding in birds: long term studies of ecology and behaviour*. Cambridge University Press, 1990.
- [6] Indrikis Kramps, Tatjana Krama, Kristine Igaune, and Raivo Mänd. Experimental evidence of reciprocal altruism in the pied flycatcher. *Behavioral Ecology and Sociobiology*, 62(4):599–605, 2008.
- [7] Barry Sinervo, Alexis Chaine, Jean Clobert, Ryan Calsbeek, Lisa Hazard, Lesley Lancaster, Andrew G McAdam, Suzanne Alonzo, Gwynne Corrigan, and Michael E Hochberg. Self-recognition, color signals, and cycles of greenbeard mutualism and altruism. *Proceedings of the National Academy of Sciences*, 103(19):7372–7377, 2006.
- [8] J. B. S. Haldane. *The Causes of Evolution*. Longmans, London, 1932.
- [9] William D Hamilton. The genetical evolution of social behaviour. ii. *Journal of Theoretical Biology*, 7(1):17–52, 1964.
- [10] Edward O Wilson. Kin selection as the key to altruism: its rise and fall. *Social Research*, pages 159–166, 2005.
- [11] Kevin R Foster, Tom Wenseleers, and Francis LW Ratnieks. Kin selection is the key to altruism. *Trends in Ecology & Evolution*, 21(2):57–60, 2006.
- [12] Martin A Nowak. Five rules for the evolution of cooperation. *Science*, 314(5805):1560–1563, 2006.
- [13] Luigi Luca Cavalli-Sforza and Marcus W Feldman. *Cultural transmission and evolution: A quantitative approach*. Number 16. Princeton University Press, 1981.
- [14] Peter J Richerson and Robert Boyd. *Not by Genes Alone: How Culture Transformed Human Evolution*. University of Chicago Press, 2008.
- [15] Stephen J Lycett and John AJ Gowlett. On questions surrounding the acheulean ‘tradition’. *World Archaeology*, 40(3):295–315, 2008.
- [16] Yoav Ram, Uri Liberman, and Marcus W Feldman. Evolution of vertical and oblique transmission under fluctuating selection. *Proceedings of the National Academy of Sciences*, 115(6):E1174–E1183, 2018.
- [17] Jeffrey A. Fletcher and Michael Doebeli. A simple and general explanation for the evolution of altruism. *Proc. R. Soc. B Biol. Sci.*, 276(1654):13–19, 2009.
- [18] Martin A Nowak and Karl Sigmund. Tit for tat in heterogeneous populations. *Nature*, 355(6357):250–253, 1992.
- [19] Sabin Lessard et al. Evolutionary stability: one concept, several meanings. *Theoretical population biology*, 37(1):159–170, 1990.
- [20] Ilan Eshel and Luigi Luca Cavalli-Sforza. Assortment of encounters and evolution of cooperativeness. *Proceedings of the National Academy of Sciences*, 79(4):1331–1335, 1982.
- [21] Piter Bijma and Duur K. Aanen. Assortment, Hamilton’s rule and multilevel selection. *Proc. R. Soc. B Biol. Sci.*, 277(1682):673–675, 2010.
- [22] Hisashi Ohtsuki, Christoph Hauert, Erez Lieberman, and Martin A. Nowak. A simple rule for

- the evolution of cooperation on graphs and social networks. *Nature*, 441(7092):502–505, 2006.
- [23] Marcus W Feldman, Luca L Cavalli-Sforza, and Joel R Peck. Gene-culture coevolution: models for the evolution of altruism with cultural transmission. *Proceedings of the National Academy of Sciences*, 82(17):5814–5818, 1985.
- [24] Scott Woodcock. The significance of non-vertical transmission of phenotype for the evolution of altruism. *Biology and Philosophy*, 21(2):213–234, 2006.
- [25] Ohad Lewin-Epstein, Ranit Aharonov, and Lilach Hadany. Microbes can help explain the evolution of host altruism. *Nature Communications*, 8:14040, 2017.
- [26] Ohad Lewin-Epstein and Lilach Hadany. Host-microbiome coevolution can promote cooperation in a rock-paper-scissors dynamics. *Proc. R. Soc. B Biol. Sci.*, 287(1920):20192754, feb 2020.
- [27] Yael Gurevich, Ohad Lewin-Epstein, and Lilach Hadany. The evolution of paternal care: a role for microbes? *Philos. Trans. R. Soc. B Biol. Sci.*, 375(1808):20190599, sep 2020.
- [28] Carla Handley and Sarah Mathew. Human large-scale cooperation as a product of competition between cultural groups. *Nature communications*, 11(1):1–9, 2020.
- [29] Matthew R. Zefferman. Mothers teach daughters because daughters teach granddaughters: the evolution of sex-biased transmission. *Behav. Ecol.*, 27(4):1172–1181, 2016.
- [30] Manfred Milinski. Tit for tat in sticklebacks and the evolution of cooperation. *nature*, 325(6103):433–435, 1987.
- [31] Brian Skyrms. *The stag hunt and the evolution of social structure*. Cambridge University Press, 2004.
- [32] Michael Tomasello, Alicia P Melis, Claudio Tennie, Emily Wyman, Esther Herrmann, Ian C Gilby, Kristen Hawkes, Kim Sterelny, Emily Wyman, Michael Tomasello, et al. Two key steps in the evolution of human cooperation: The interdependence hypothesis. *Current anthropology*, 53(6):000–000, 2012.
- [33] Aaron Meurer, Christopher P Smith, Mateusz Paprocki, Ondřej Čertík, Sergey B Kirpichev, Matthew Rocklin, AMiT Kumar, Sergiu Ivanov, Jason K Moore, Sartaj Singh, et al. Sympy: symbolic computing in python. *PeerJ Computer Science*, 3:e103, 2017.
- [34] Marcus W. Feldman. Selection for linkage modification: I. Random mating populations. *Theor. Popul. Biol.*, 3:324–346, 1972.
- [35] Uri Liberman and Marcus W. Feldman. A general reduction principle for genetic modifiers of recombination. *Theor. Popul. Biol.*, 30(3):341–71, dec 1986.
- [36] Uri Liberman and Marcus W. Feldman. Modifiers of mutation rate: A general reduction principle. *Theor. Popul. Biol.*, 30:125–142, 1986.
- [37] Uri Liberman. External stability and ESS: criteria for initial increase of new mutant allele. *J. Math. Biol.*, 26(4):477–485, 1988.
- [38] Lee Altenberg, Uri Liberman, and Marcus W. Feldman. Unified reduction principle for the evolution of mutation, migration, and recombination. *Proc. Natl. Acad. Sci. U. S. A.*, 114(12):E2392–E2400, mar 2017.
- [39] Edward F Moore. Machine models of self-reproduction. In *Proceedings of symposia in applied mathematics*, volume 14, pages 17–33. American Mathematical Society New York, 1962.
- [40] Samuel Karlin, Uri Lieberman, and Uri Liberman. Random temporal variation in selection intensities: One-locus two-allele model. *J. Math. Biol.*, 6(3):1–17, 1975.
- [41] Andrew P Dobson. The population biology of parasite-induced changes in host behavior. *The Quarterly Review of Biology*, 63(2):139–165, 1988.
- [42] Robert Poulin. Parasite manipulation of host behavior: an update and frequently asked questions. In *Advances in the Study of Behavior*, volume 41, pages 151–186. Elsevier, 2010.
- [43] Christian Traxler and Mathias Spichtig. Social norms and the indirect evolution of conditional

- 608 cooperation. *Journal of Economics*, 102(3):237–262, 2011.
- [44] Bryce Morsky and Erol Akcay. False beliefs can bootstrap cooperative communities through
610 social norms. 2020.

Appendices

612 Appendix A Local stability criterion

Let $f(p) = \lambda \cdot (p' - p)$, where $\lambda > 0$, and 0 and 1 are equilibria, that is, $f(0) = 0$ and $f(1) = 0$.

614 Set $p > p^* = 0$. Using a linear approximation for $f(p)$ near 0, we have

$$p' < p \Leftrightarrow f(p)/p < 0 \Leftrightarrow \frac{f'(0) \cdot p + O(p^2)}{p} < 0 \Leftrightarrow f'(0) + O(p) < 0. \quad (\text{A1})$$

616 Therefore, by definition of big-O notation, if $f'(0) < 0$ then there exists $\epsilon > 0$ such that for any local perturbation $0 < p < \epsilon$, it is guaranteed that $0 < p' < p$; that is, p' is closer to zero than p .

618 Set $p < p^* = 1$ Using a linear approximation for $f(p)$ near 1, we have

$$1 - p' < 1 - p \Leftrightarrow -\frac{f(p)}{1 - p} < 0 \Leftrightarrow \frac{f'(1)(p - 1) + O((p - 1)^2)}{p - 1} < 0 \Leftrightarrow f'(1) - O(1 - p) < 0. \quad (\text{A2})$$

620 Therefore, if $f'(1) < 0$ then there exists $\epsilon > 0$ such that for any $1 - \epsilon < 1 - p < 1$ we have $1 - p' < 1 - p$; that is, p' is closer to one than p .

622 **Appendix B Effect of interaction-transmission association on mean fitness**

624 To determine the effect of increasing α on the stable population mean fitness, $\bar{w}^* = 1 + (b - c)\hat{p}^*$, we must analyze its effect on \hat{p}^* ,

$$626 \quad \frac{\partial \hat{p}^*}{\partial \alpha} = \frac{bT_A - c(1 - T_B) + (T_A - T_B)}{b(1 - \alpha)^2(T_B - T_A)}. \quad (\text{B1})$$

Note that stable polymorphism implies $c < \gamma_1$, and because $\alpha < 1$, we have

$$628 \quad c < \gamma_1 = \frac{b\alpha T_A + (T_A - T_B)}{1 - T_B} < \frac{bT_A + (T_A - T_B)}{1 - T_B}. \quad (\text{B2})$$

Therefore, the numerator in Eq. B1 is positive. Since $T_A < T_B$, the denominator in Eq. B1 is also
630 positive, and hence the derivative $\partial \hat{p}^*/\partial \alpha$ is positive. Thus, the population mean fitness increases as interaction-transmission association α increases.

632 Appendix C Reduction principle

We assume here that $v = 1$, i.e. no oblique transmission, and therefore $\hat{p} = \dot{p}$. Denote the frequencies 634 of the pheno-genotypes AM , BM , Am , and Bm by $\mathbf{p} = (\dot{p}_1, \dot{p}_2, \dot{p}_3, \dot{p}_4)$. The frequencies of the pheno-genotypes in the next generation are defined by the recursion system,

$$\begin{aligned}
\bar{w}\dot{p}'_1 &= \dot{p}_1x(1+b-c)(1-(1-\alpha_1)(1-x)T_B) + \\
&\quad \dot{p}_1(1-x)(1-c)(1-\alpha_1T_Bx-T_B(1-x)) + \\
&\quad \dot{p}_2x(1+b)T_A(x+\alpha_1(1-x)) + \\
&\quad \dot{p}_2(1-x)x(1-\alpha_1)T_A, \\
\bar{w}\dot{p}'_2 &= \dot{p}_1x(1+b-c)(1-\alpha_1)(1-x)T_B + \\
&\quad \dot{p}_1(1-x)(1-c)(\alpha_1T_B+(1-\alpha_1)(1-x)T_B) + \\
&\quad \dot{p}_2x(1+b)(1-\alpha_1T_A(1-x)-T_AX) + \\
&\quad \dot{p}_2(1-x)(1-(1-\alpha_1)xT_A), \\
\bar{w}\dot{p}'_3 &= \dot{p}_3x(1+b-c)(1-(1-\alpha_2)(1-x)T_B) + \\
&\quad \dot{p}_3(1-x)(1-c)(1-\alpha_2T_Bx-T_B(1-x)) + \\
&\quad \dot{p}_4x(1+b)T_A(x+\alpha_2(1-x)) + \\
&\quad \dot{p}_4(1-x)x(1-\alpha_2)T_A, \\
\bar{w}\dot{p}'_4 &= \dot{p}_3x(1+b-c)(1-\alpha_2)(1-x)T_B + \\
&\quad \dot{p}_3(1-x)(1-c)(\alpha_2T_B+(1-\alpha_2)(1-x)T_B) + \\
&\quad \dot{p}_4x(1+b)(1-\alpha_2T_A(1-x)-T_AX) + \\
&\quad \dot{p}_4(1-x)(1-(1-\alpha_2)xT_A),
\end{aligned} \tag{C1}$$

636

where $x = \dot{p}_1 + \dot{p}_3$ is the total frequency of the cooperative phenotype A , and $\bar{w} = 1 + (b - c)x$ is the 638 population mean fitness.

The equilibrium where only allele M is present is $\mathbf{p}^* = (\dot{p}^*, 1 - \dot{p}^*, 0, 0)$, where

$$640 \quad \dot{p}^* = \frac{c(1 - T_B) - b\alpha_1 T_A - (T_A - T_B)}{b(1 - \alpha_1)(T_A - T_B)}, \tag{C2}$$

setting $\alpha = \alpha_1$ and $v = 1$ in ???. When $v = 1$, \dot{p}^* is a feasible polymorphism ($0 < \dot{p}^* < 1$) if $T_A < T_B$ 642 and $\gamma_2 < c < \gamma_1$ (Result 1).

The local stability of \mathbf{p}^* to the introduction of allele m is determined by the linear approximation \mathbf{L}^* 644 of the transformation in Eq. C1 near \mathbf{p}^* (i.e. the Jacobian of the transformation at the equilibrium).

\mathbf{L}^* is known to have a block structure, with the diagonal blocks occupied by the matrices \mathbf{L}_{in}^* and \mathbf{L}_{ex}^* 646 [36, 38]. The latter is the external stability matrix: the linear approximation to the transformation near \mathbf{p}^* involving only the pheno-genotypes Am and Bm , derived from Eq. C1, with $\bar{w}^* = 1 + (b - c)\dot{p}^*$ as

648 the stable population mean fitness,

$$\mathbf{L}_{ex}^* = \frac{1}{\bar{w}^*} \begin{bmatrix} l_{11} & l_{12} \\ l_{21} & l_{22} \end{bmatrix} = \frac{1}{\bar{w}^*} \begin{bmatrix} \frac{\partial \bar{w} \dot{p}_3'}{\partial \dot{p}_3}(\mathbf{p}^*) & \frac{\partial \bar{w} \dot{p}_3'}{\partial \dot{p}_4}(\mathbf{p}^*) \\ \frac{\partial \bar{w} \dot{p}_4'}{\partial \dot{p}_3}(\mathbf{p}^*) & \frac{\partial \bar{w} \dot{p}_4'}{\partial \dot{p}_4}(\mathbf{p}^*) \end{bmatrix} = \frac{1}{\bar{w}^*} \begin{bmatrix} (1 + b\dot{p}^* - c)(1 - T_B(1 - \dot{p}^*)) + b\dot{p}^*\alpha_2 T_B(1 - \dot{p}^*) & (1 + b\dot{p}^*)T_A \dot{p}^* + b\dot{p}^*\alpha_2 T_A(1 - \dot{p}^*) \\ (1 + b\dot{p}^* - c)T_B(1 - \dot{p}^*) - b\dot{p}^*\alpha_2 T_B(1 - \dot{p}^*) & (1 + b\dot{p}^*)(1 - T_A \dot{p}^*) - b\dot{p}^*\alpha_2 T_A(1 - \dot{p}^*) \end{bmatrix}. \quad (\text{C3})$$

650 Because we assume that \mathbf{p}^* is internally stable (i.e. locally stable to small perturbations in the frequencies of AM and BM), the stability of \mathbf{p}^* is determined by the eigenvalues of the external
652 stability matrix \mathbf{L}_{ex}^* . This is a positive matrix, and due to the Perron-Frobenius theorem, the leading
654 eigenvalue of \mathbf{L}_{ex}^* is real and positive. Thus, if the leading eigenvalue is less (greater) than one, then the
656 equilibrium \mathbf{p}^* is externally stable (unstable) and allele m cannot (can) invade the population of allele
658 M . The eigenvalues of \mathbf{L}_{ex}^* are the roots of the characteristic polynomial, $R(\lambda)$, which is a quadratic
with a positive leading coefficient. Therefore, $\lim_{\lambda \rightarrow \pm\infty} R(\lambda) = \infty$, and the leading eigenvalue is less
than one (implying stability) if and only if $R(1) > 0$ and $R'(1) > 0$. Thus, a sufficient condition for
external instability of \mathbf{p}^* is $R(1) < 0$.

$R(\lambda)$ is defined as a determinant, $R(\lambda) = \det(\mathbf{L}_{ex}^* - \lambda\mathbf{I})$, where \mathbf{I} is the 2-by-2 identity matrix. Since
660 multiplication by a positive factor doesn't change the sign, and using the properties of the determinant,
we have

$$662 \quad \begin{aligned} \text{sign } R(1) &= \text{sign } \det(\mathbf{L}_{ex}^* - \mathbf{I}) = \text{sign } (\bar{w}^*)^2 \det(\mathbf{L}_{ex}^* - \mathbf{I}) = \\ \text{sign } \det(\bar{w}^* \mathbf{L}_{ex}^* - \bar{w}^* \mathbf{I}) &= \text{sign } \det \begin{bmatrix} l_{11} - \bar{w}^* & l_{12} \\ l_{21} & l_{22} - \bar{w}^* \end{bmatrix}, \end{aligned} \quad (\text{C4})$$

where l_{ij} are defined in Eq. C3. Adding the second row in Eq. C4 to the first row, which does not
664 change the determinant, and substituting $\bar{w}^* = 1 + (b - c)\dot{p}^*$, we get

$$\begin{aligned} \text{sign } R(1) &= \text{sign } \det \begin{bmatrix} -c(1 - \dot{p}^*) & c\dot{p}^* \\ (1 - \dot{p}^*)[(1 + b\dot{p}^* - c)T_B - b\alpha_2 T_B \dot{p}^*] & \dot{p}^*[-(1 + b\dot{p}^*)T_A - b\alpha_2 T_A(1 - \dot{p}^*) + c] \end{bmatrix} = \\ \text{sign } &\left[c\dot{p}^*(1 - \dot{p}^*) \cdot \det \begin{bmatrix} -1 & 1 \\ (1 + b\dot{p}^* - c)T_B - b\alpha_2 T_B \dot{p}^* & -(1 + b\dot{p}^*)T_A - b\alpha_2 T_A(1 - \dot{p}^*) + c \end{bmatrix} \right] = \\ \text{sign } \det &\begin{bmatrix} -1 & 1 \\ (1 + b\dot{p}^* - c)T_B - b\alpha_2 T_B \dot{p}^* & -(1 + b\dot{p}^*)T_A - b\alpha_2 T_A(1 - \dot{p}^*) + c \end{bmatrix}, \end{aligned} \quad (\text{C5})$$

666 since $c > 0, 0 < \dot{p}^* < 1$. That is,

$$\begin{aligned} \text{sign } R(1) &= \text{sign } \left[(1 + b\dot{p}^*)T_A + b\alpha_2 T_A(1 - \dot{p}^*) - c - (1 + b\dot{p}^* - c)T_B + b\dot{p}^*\alpha_2 T_B \right] = \\ \text{sign } &\left[(1 + b(1 - \alpha_2)\dot{p}^*)(T_A - T_B) + b\alpha_2 T_A - c(1 - T_B) \right]. \end{aligned} \quad (\text{C6})$$

668 Substituting \dot{p}^* from Eq. C2, we get

$$\begin{aligned}
R(1) < 0 \Leftrightarrow & [c(1 - T_B) - b\alpha_1 T_A - (T_A - T_B)] \frac{1 - \alpha_2}{1 - \alpha_1} - c(1 - T_B) + b\alpha_2 T_A + (T_A - T_B) < 0 \Leftrightarrow \\
& (1 - \alpha_2)[c(1 - T_B) - b\alpha_1 T_A - (T_A - T_B)] < (1 - \alpha_1)[c(1 - T_B) - b\alpha_2 T_A - (T_A - T_B)] \Leftrightarrow \\
& -b\alpha_1 T_A - \alpha_2 c(1 - T_B) + \alpha_2 (T_A - T_B) < -b\alpha_2 T_A - \alpha_1 c(1 - T_B) + \alpha_1 (T_A - T_B) \Leftrightarrow \\
& \alpha_1[c(1 - T_B) - bT_A - (T_A - T_B)] < \alpha_2[c(1 - T_B) - bT_A - (T_A - T_B)] \Leftrightarrow \\
& \alpha_1[bT_A + (T_A - T_B) - c(1 - T_B)] > \alpha_2[bT_A + (T_A - T_B) - c(1 - T_B)] .
\end{aligned} \tag{C7}$$

670 We assumed $c < \gamma_1$, and since $0 \leq \alpha_1 \leq 1$,

$$\begin{aligned}
c < \gamma_1 = \frac{b\alpha_1 T_A + (T_A - T_B)}{1 - T_B} \Leftrightarrow \\
0 < b\alpha_1 T_A + (T_A - T_B) - c(1 - T_B) \Rightarrow \\
0 < bT_A + (T_A - T_B) - c(1 - T_B) .
\end{aligned} \tag{C8}$$

672 Combining inequalities C7 and C8, we find that $R(1) < 0$ if and only if $\alpha_1 > \alpha_2$, which is a sufficient condition for external instability. Therefore, if α_2 , the interaction-transmission association of the 674 invading modifier allele m , is less than α_1 , the interaction-transmission association of the resident allele M , then invasion will be successful.

676 Determining a necessary and sufficient condition for successful invasion is more complicated, requiring analysis of the sign of $R'(1)$. However, we have numerically validated that the leading eigenvalue is 678 greater than one if and only if $\alpha_1 > \alpha_2$.