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ORIGINAL PAPER

Negative niche construction favors the evolution of cooperation

Brian D. Connelly[1] · Katherine J. Dickinson[1] · Sarah P. Hammarlund[1,2] · Benjamin Kerr[1]

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Abstract By benefitting others at a cost to themselves, cooperators face an ever present threat from defectors—individuals that avail themselves of the cooperative benefit without contributing. A longstanding challenge to evolutionary biology is to understand the mechanisms that support the many instances of cooperation that nevertheless exist. In spatially-structured environments, clustered cooperator populations reach greater densities, which creates more mutational opportunities to gain beneficial non-social adaptations. Hammarlund et al. recently demonstrated that cooperation rises in abundance by hitchhiking with these non-social mutations. However, once adaptive opportunities have been exhausted, the ride abruptly ends as cooperators are displaced by adapted defectors. Using an agent-based model, we demonstrate that the selective feedback that is created as populations construct their local niches can maintain cooperation at high proportions and even allow cooperators to invade. This cooperator success depends specifically on negative niche construction, which acts as a perpetual source of adaptive opportunities. As populations adapt, they alter their environment in ways that reveal additional opportunities for adaptation. Despite being independent of niche construction in our model, cooperation feeds this cycle. By reaching larger densities, populations of cooperators are better able to adapt to changes in their constructed niche and successfully respond to the constant threat posed by defectors. We relate these findings to previous studies from the niche construction literature and discuss how this model could be extended to provide a greater understanding of how cooperation evolves in the complex environments in which it is found.

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✉ Benjamin Kerr
kerrb@uw.edu

Brian D. Connelly
bdcx@uw.edu

[1] Department of Biology and BEACON Center for the Study of Evolution in Action, University of Washington, Seattle, WA 98195, USA

[2] Present Address: Department of Zoology, University of Oxford, Oxford OX1 3PS, UK

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Introduction

Cooperative behaviors are common across all branches of the tree of life. Insects divide labor within their colonies, plants and soil bacteria exchange essential nutrients, birds care for others' young, and the trillions of cells in the human body coordinate to provide vital functions. Each instance of cooperation presents an evolutionary challenge: How can individuals that sacrifice their own well-being to help others avoid subversion by those that do not? Over time, we would expect these *defectors* to rise in abundance at the expense of others, eventually driving cooperators—and perhaps the entire population—to extinction.

Several factors can prevent this *tragedy of the commons* (Hamilton 1964; Nowak 2006; West et al. 2007b). One such factor involves non-random social interaction, in which cooperators benefit more from the cooperative act than do defectors. This can occur when cooperators are clustered together in spatially-structured populations (Fletcher and Doebeli 2009; Nadell et al. 2010; Kuzdzal-Fick et al. 2011), or when cooperators use communication (Brown and Johnstone 2001; Darch et al. 2012) or other cues (Sinervo et al. 2006; Gardner and West 2010; Veelders et al. 2010) to cooperate conditionally with kin. Cooperation can also be bolstered by pleiotropic connections to personal benefits (Foster et al. 2004; Dandekar et al. 2012) or through association with alleles encoding self-benefitting traits (Asfahl et al. 2015). In the latter case, the associated alleles may provide private benefits that are entirely independent from the public benefits of cooperation. In asexual populations of cooperators and defectors, this sets the stage for an "adaptive race" in which both types vie for the first highly beneficial adaptation (Waite and Shou 2012; Morgan et al. 2012). The tragedy of the commons can be deferred if a cooperator, by chance, wins the adaptive race.

Hammarlund et al. (2015) recently showed that in spatially-structured populations, the "Hankshaw effect" can give cooperators a substantial leg up on defectors in an adaptive race. Inspired by a fictional character in Tom Robbins' *Even Cowgirls Get the Blues*, the Hankshaw effect describes how a trait can proliferate by actively creating opportunities to hitchhike along with other highly beneficial traits. In Robbins' novel, Sissy Hankshaw was born with extremely oversized thumbs. Although her thumbs were an impairment to everyday activities, they made her a prolific hitchhiker. Similarly, cooperative behaviors can enjoy increased opportunities to hitchhike, despite their cost, by increasing their local population density. This makes cooperators more likely to acquire beneficial mutations. By hitchhiking along with these adaptations, cooperation can rise in abundance. Nevertheless, this advantage is fleeting. As soon as the opportunities for adaptation are exhausted, cooperators are once again at a selective disadvantage against adapted defectors that arise via mutation. However, cooperators can maintain their advantage when frequent environmental changes produce a steady stream of new adaptive opportunities (Hammarlund et al. 2015). Although organisms typically find themselves in dynamic environments, the frequency and regularity of these changes might not ensure long-term cooperator survival.

Importantly, organisms do more than passively experience changing environments. Through their activities, their interactions with others, and even their deaths, organisms continually modify their environment. This *niche construction* process can produce evolutionary feedback loops in which environmental modification alters selection, which, in

turn, alters the distribution of types and their corresponding influence on the environment (Odling-Smee et al. 2003). This feedback can have dramatic evolutionary consequences. One critical distinction is whether the constructing type is favored in the environment that it constructs. Under *positive niche construction*, selection favors the constructor, and evolution stagnates as this type fixes. Whereas under *negative niche construction*, selection favors a type other than the constructor, which creates an opportunity for novel adaptation. If the adapted type arises and also engages in negative niche construction, cycles of construction and adaptation can ensue, such that populations find themselves endlessly chasing beneficial mutations as their adaptive landscape continually shifts.

Here, we show that the selective feedbacks that result from niche construction can enable the evolution of cooperation. Further, we find that it is specifically negative niche construction that is responsible for this result due to the endless opportunities for adaptation that it produces. Under certain circumstances, we demonstrate that niche construction can even allow cooperators to invade established defector populations. These results suggest that by playing an active role in their own evolution, cooperators can ensure their own survival.

Methods

Building upon Hammarlund et al. (2015), we describe an individual-based model in which cooperators and defectors evolve and compete in a population of subpopulations (i.e., a metapopulation). Through mutation, individuals gain adaptations to their environment, which increase reproductive fitness and allow those lineages to rise in abundance. Adapted lineages then spread throughout the population by migration to neighboring subpopulations.

In the expanded model described here, subpopulations also continually modify their local environment. These environmental changes feed back to affect selection. We use this model to explore how niche construction affects the evolution of cooperation; specifically, how cooperative behavior can hitchhike along with adaptations to modified environments.

Model description

Individual genotypes and adaptation

Each individual has a haploid genome with $L + 1$ loci, where integers represent different alleles at each locus (Table 1 lists all model parameters and their values). An allele at the *cooperation locus* (locus zero) determines whether that individual is a cooperator (allele 1), which carries fitness cost c, or a defector (allele 0). The remaining L loci are *adaptive loci*, and are each occupied by a value from the set $\{0, 1, 2, \ldots, A\}$, where A is the number of different adaptive alleles possible at each locus.

Allele 0 represents a lack of adaptation, while non-zero alleles signify two types of adaptations, both of which increase fitness. First, adaptations to the *external environment* confer a fitness benefit δ. This selective value is the same regardless of which non-zero allele is present. We assume $\delta > c$, which allows a minimally adapted cooperator to recoup the cost of cooperation and gain a fitness advantage.

Table 1 Model parameters and their values

Parameter	Description	Base value	Alternate values
L	Number of adaptive loci	5	0
c	Cost of cooperation	0.1	
A	Number of alleles	6	5
δ	Benefit of adaptation to external environment	0.3	0, 0.6
ϵ	Benefit of adaptation to constructed environment	0.00015	0
z	Baseline fitness	1	
S_{min}	Minimum subpopulation size	800	
S_{max}	Maximum subpopulation size	2000	8000
μ_c	Mutation rate at cooperation locus	10^{-5}	0
μ_a	Mutation rate at adaptive loci	10^{-5}	0
N^2	Number of patches	625	121
m	Migration rate	0.05	
p_0	Initial cooperator proportion	0.5	0, 0.01, 0.1, 0.2, 0.3, 0.9, 0.99, 1[a]
σ_i	Survival rate at population initialization	10^{-5}	
T	Number of simulation cycles	3000	1000, 5000
σ_d	Survival after dilution	0.1	0.01, 0.3, 0.5, 0.7, 0.9[a]
γ	Convexity of cooperative benefit[a]	1.0	0.25, 0.5, 1.5, 2, 4

[a] See Supplementary Material

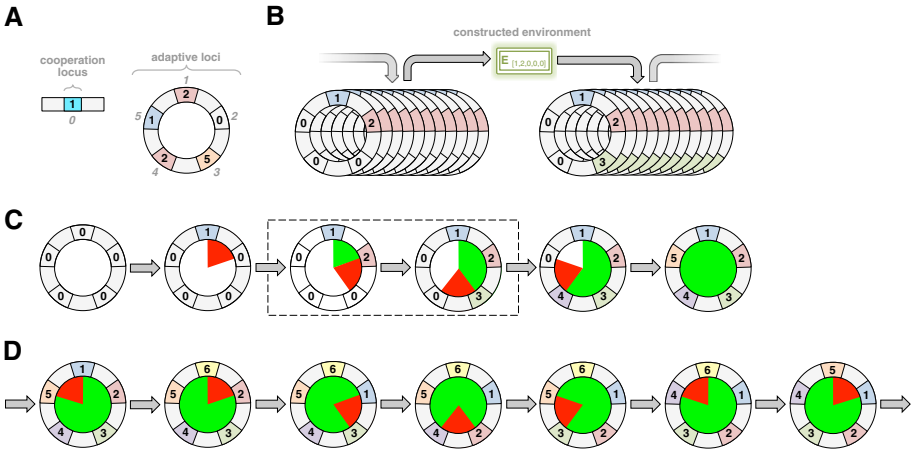
Niche construction and selective feedbacks

Individual fitness is also affected by aspects of the local environment that are modified by organisms. This constructed "niche" depends on the specific allelic states present in the subpopulation. As allelic states change, the subpopulation alters its environment in new ways, creating a unique niche. As described below, the specific alleles at each locus become important.

In our model, the feedback that results from niche construction takes the form of density dependent selection, and populations evolve to better match the constructed niche. We do not represent this niche explicitly, but rather allow the allelic composition of the subpopulation to feed back to affect selection. Specifically, the selective value of non-zero allele a at adaptive locus l—and consequently the fitness of an individual carrying that allele—increases with the number of individuals in the subpopulation that have allele $a-1$ at locus $l-1$. For example, if $L=5, A=6$, and allele 4 has fixed at locus 2, then selection favors genotypes with allele 5 at locus 3. And as allele 5 becomes more abundant at locus 3, the niche that this population constructs will increasingly favor allele 6 at locus 4 (see Box 1). As a consequence, genotypes with sequentially increasing allelic states will tend to evolve.

We treat both adaptive loci and their non-zero allelic states as "circular": the selective value of an allele at locus 1 is affected by the allelic composition of the subpopulation at locus L. Similarly, the selective value of allele 1 at any locus increases with the number of individuals carrying allele A at the previous locus. This circularity is represented by the

Box 1 Description of niche construction in our model



(**A**) *Individuals.* The genome of each individual consists of a single *cooperation locus* and *L adaptive loci* (here, $L = 5$). At the cooperation locus (labeled *0*), this individual has allele 1, making it a cooperator. The adaptive loci (labeled *1–5*) are arranged as a circular chromosome, where each locus has an integer allele between 0 and *A*, inclusive. In the description that follows, we focus exclusively on these adaptive loci. Genotypes are given by their allelic states starting with locus *1*. For instance, the genotype shown here is [2,0,5,2,1]. Because of their circular structure, allele 2 at the first locus follows allele 1 at the fifth locus.

(**B**) *Niche Construction.* Consider a subpopulation fixed for genotype [1,2,0,0,0]. This subpopulation constructs environment $E_{[1,2,0,0,0]}$. Every non-zero allele influences selection at the next locus, favoring sequential allelic states. In this constructed environment, allele 3 at locus *3* is favored. If genotype [1,2,3,0,0] arises via mutation, it is expected to fix. However, genotype [1,2,3,0,0] affects the environment differently. As [1,2,3,0,0] rises in abundance, the constructed environment changes to $E_{[1,2,3,0,0]}$, which favors [1,2,3,4,0].

(**C**) *Niche Construction and Adaptation.* The evolutionary transition shown in Part B is indicated in the dashed box. Here, we depict entire subpopulations fixed for a genotype using a single instance of that genotype. Similarly, an arrow represents niche construction and adaptation to the constructed environment. We start with a case in which there are five alleles ($A = 5$). Subpopulations begin with the non-adapted genotype [0,0,0,0,0], shown on the far left. A non-zero allele is introduced via mutation, which represents an adaptation to external aspects of the environment. Here, allele 1 arises and fixes at locus *1*. The remainder of this figure focuses on adaptation to the constructed aspects of the environment. This genotype has a mismatch (shown by the red sector), because $E_{[1,0,0,0,0]}$ favors [1,2,0,0,0]. Assuming allele 2 arises at the second locus, it will be selected, creating a match at the first and second loci (green sector). Now there is a mismatch between the second and third loci in the resulting environment, which a new round of mutation and selection corrects, and so on. The green sector grows as the red sector shifts clockwise. When the population reaches [1,2,3,4,5], it constructs $E_{[1,2,3,4,5]}$. Here, since allele 1 follows allele 5, there is no longer a mismatch, so no further adaptation occurs.

(**D**) *Negative Niche Construction.* A different case emerges when the number of alleles does not evenly divide into the number of loci. Here, we change the number of alleles to six ($A = 6$). As shown on the far left, we begin with a subpopulation fixed for genotype [1,2,3,4,5]. This genotype has a mismatch, because the niche constructed by allele 5 favors allele 6 (not 1) at the next locus (locus *1*). A mutant with genotype [6,2,3,4,5] has a fitness advantage and can fix in $E_{[1,2,3,4,5]}$. However, as this type constructs $E_{[6,2,3,4,5]}$, a new mismatch appears. In this instance of negative niche construction, adapting to correct one mismatch generates a new mismatch. This system can never escape its mismatches—the red sector just shifts clockwise around the genome perpetually. We call this negative niche construction, as the actions of constructors increase the fitness of a different genotype and thereby lower their own relative fitness.

function $\beta(x, X)$, which gives the integer that is below an arbitrary value x in the set $\{1, 2, \ldots, X\}$:

$$\beta(x, X) = \mathrm{mod}_X(x - 2 + X) + 1 \tag{1}$$

Here, $\mathrm{mod}_X(x)$ is the integer remainder when dividing x by X. For example, $\beta(3, 5)$ is 2, while $\beta(1, 5)$ is 5. Using this function, the selective value of allele a at adaptive locus l increases by ϵ for each individual in the subpopulation that has allele $\beta(a, A)$ at locus $\beta(l, L)$. Thus, ϵ specifies the intensity of selection due to niche construction.

Individual fitness

For an individual with allelic state a_l at locus l, fitness is defined as:

$$W = z - \underbrace{ca_0}_{\substack{\text{cost of} \\ \text{cooperation}}} + \underbrace{\delta \sum_{l=1}^{L} I(a_l)}_{\substack{\text{adaptation to} \\ \text{external environment}}} + \underbrace{\epsilon \sum_{l=1}^{L} n(\beta(a_l, A), \beta(l, L))}_{\substack{\text{adaptation to} \\ \text{constructed environment}}} \tag{2}$$

where z is a baseline fitness, $n(a, l)$ is the number of individuals in the subpopulation with allele a at locus l, and $I(a)$ indicates whether a given allele is non-zero:

$$I(a) = \begin{cases} 1 & \text{if } a \in \{1, 2, \ldots, A\} \\ 0 & \text{otherwise} \end{cases} \tag{3}$$

Thus, an individual's fitness is determined both by adaptations to the external environment and by adaptations to its constructed environment. Box 1 illustrates the process of adaptation to the constructed environment. While the separation between exogenous and endogenous environmental change may not always be as clearly differentiated in natural systems, it allows us to directly explore the effects of niche construction.

Subpopulation growth and the benefit of cooperation

The effects of cooperation are independent of the external and constructed components of the environment and do not provide direct fitness benefits (Eq. 2). Instead, cooperation enables a subpopulation to reach a greater density. If p is the proportion of cooperators present at the beginning of a growth cycle, then that subpopulation reaches size $S(p)$, where:

$$S(p) = S_{min} + p(S_{max} - S_{min}) \tag{4}$$

S_{min} and S_{max} define the sizes reached by all-defector and all-cooperator subpopulations, respectively. This benefit affects all individuals equally and accumulates linearly with the proportion of cooperators in the subpopulation. We also explore non-linear benefit accumulation in the Supplementary Material. Because cooperators improve group productivity (Eq. 4) but decrease in proportion within mixed groups (Eq. 2), this form of cooperation would also qualify as "multi-level altruism" (see Supplementary Material and Kerr et al. 2004).

Individuals compete as subpopulations grow. Each individual's probability of reproductive success is proportional to its fitness. The composition of a subpopulation with size P and cooperator proportion p after growth is multinomial with parameters $S(p)$ and $\{\pi_1, \pi_2, \ldots, \pi_P\}$, where π_i represents the reproductive fitness of individual i relative to others in its subpopulation (Eq. 2).

Mutation

For simplicity, we apply mutations to new offspring after subpopulation growth. Mutations occur independently at each locus and cause an allelic state change. At the binary cooperation locus, mutations occur at rate μ_c. These mutations flip the allelic state, causing cooperators to become defectors and vice versa. Mutations occur at rate μ_a at each adaptive locus. These mutations replace the existing allele with a value randomly sampled from the set $\{0, 1, \ldots, A\}$.

Migration

Populations consist of N^2 patches arranged as an $N \times N$ lattice, where each patch can support a subpopulation. After mutation, individuals emigrate to an adjacent patch. This process is unaffected by fitness. For each source subpopulation, a single destination patch is randomly chosen from the source patch's Moore neighborhood, which encompasses the nearest 8 patches on the lattice. Because the population lattice has boundaries, patches located on the periphery have smaller neighborhoods. Individuals emigrate with probability m, which means larger subpopulations produce more emigrants. Through immigration, subpopulations can exceed S_{max} individuals. As described below, however, this increase in subpopulation size is temporary.

Population initialization, dilution, and simulation

Following Hammarlund et al. (2015), we begin simulations with sparse populations. Subpopulations are first seeded at all patches with cooperator proportion p_0 and size $S(p_0)$. The population is then thinned. Each individual survives this bottleneck with probability σ_i. Starting from this initial state, simulations then proceed for T cycles, where each discrete cycle consists of subpopulation growth, mutation, migration, and dilution. Dilution reduces each subpopulation to support growth in the next cycle. Each individual survives dilution with probability σ_d, regardless of its genotype. Dilution remains the same for each of the simulations described, however we further explore its effects in the Supplementary Material.

Simulation source code and software dependencies

The simulation software and configurations for the experiments reported are available online (Connelly et al. 2015). Simulations used Python 3.4, NumPy 1.9.1, Pandas 0.15.2 (McKinney 2010), and NetworkX 1.9.1 (Hagberg et al. 2008). Data analyses were performed with R 3.2.2 (Core and Team. 2015). Reported 95% confidence intervals were estimated by bootstrapping with 1000 resamples.

Results

Using the model described in the previous section, we perform simulations that follow the evolution of cooperation in a population of subpopulations that are connected by spatially-limited migration. Individuals increase their competitiveness by gaining adaptations. While cooperation does not directly affect the fitness benefits that these adaptations confer, it does have indirect effects on the adaptive process. Specifically, cooperation increases

subpopulation density. As a result, larger subpopulations of cooperators experience more mutational opportunities. Cooperation can rise in abundance by hitchhiking along with beneficial mutations, which compensate for the cost of cooperation. Importantly, subpopulations alter their local environments, which feeds back to influence selection. Here, we explore how such niche construction affects the evolution of cooperation.

Cooperation persists with niche construction

Without any opportunity for adaptation ($L = 0$), cooperators are swiftly eliminated (Fig. 1a). Despite an initial lift in cooperator abundance due to increased productivity, the cost of cooperation becomes disadvantageous as migration mixes the initially isolated subpopulations. When populations can adapt to the external environment ($L > 0$ and $\delta > 0$), but niche construction is absent ($\epsilon = 0$), cooperators are maintained only transiently (Fig. 1b). Here, larger cooperator subpopulations adapt more quickly to their external environment, which allows them to rise in abundance. As previously described by Hammarlund et al. (2015), cooperation is swiftly lost once populations become fully adapted. This occurs when isogenic defectors (i.e., defectors with identical adaptive loci) arise via mutation and displace cooperators due to their selective advantage. However, when niche construction feeds back to influence selection ($\epsilon > 0$), cooperation persists in the majority of replicate populations (Fig. 1c). We see in Fig. 2a that despite some oscillations, cooperation is maintained at high levels in the majority of these populations.

Fitness increases alone do not support persisting cooperation

An individual's fitness is affected in this model by adaptations to both the external environment and to the constructed environment. Here, we determine whether cooperation

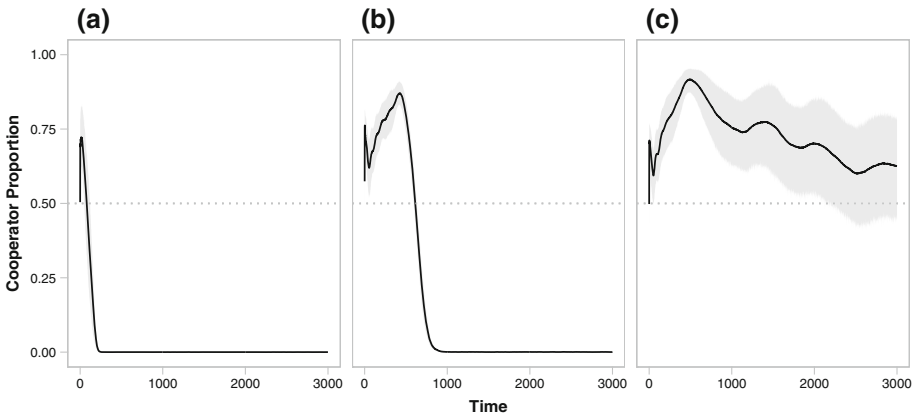


Fig. 1 Adaptation and the evolution of cooperation. The average cooperator proportion among replicate populations for the duration of simulations are shown as *curves*, and *shaded areas* indicate 95 % confidence intervals. (**a**) Without any opportunity to adapt ($L = 0$), cooperation is quickly lost. (**b**) When adaptation can occur ($L = 5$, $\delta = 0.3$), but niche construction does not affect selection ($\epsilon = 0$), cooperators rise in abundance by hitchhiking along with adaptions to the external environment. Nevertheless, this effect is transient, and defectors eventually dominate. (**c**) Selective feedback from niche construction ($\epsilon = 0.00015$) enables cooperation to be maintained in the majority of populations. Figure 2a shows the individual trajectories of these populations

is maintained as we see in Fig. 2a solely due to the larger selective values that result from the contributions of niche construction. For these simulations, the selective contributions of niche construction are transferred to supplement the benefits conferred by adaptation to the external, non-constructed environment (i.e., replacing $S_{max} \times \epsilon = 0.3$, $\delta = 0.3$ with $\epsilon = 0$, $\delta = 0.6$). In doing so, we liberally estimate the selective effects of niche construction. Nevertheless, we find that simply increasing selective values extends the maintenance of cooperation, but does not enable cooperators to persist (Fig. 2b). Niche construction, therefore, plays a decisive role here.

Negative niche construction is critical to cooperator persistence

In our model, an adaptation to the constructed environment initiates a new instance of niche construction, leading to sequentially increasing allelic states across the adaptive loci. Under certain conditions, this construction always makes the constructor sub-optimal for the niche it creates. This form of negative niche construction occurs when the number of adaptive alleles (A) does not divide evenly into the number of adaptive loci (L). In such a case, any sequence of integers on the circular genome will always contain a break in the sequence; that is, one locus will perpetually have an allele that is maladapted to the constructed niche (see Box 1, Part D). Given this unavoidable mismatch, types will always construct a niche in which selection for a different type is enhanced. When negative niche construction is removed (by setting $L = 5, A = 5$; see Box 1, Part C), cooperators are again driven to extinction after an initial lift in abundance (Fig. 2c). Here, a fully-adapted type constructs a niche that favors itself. When this occurs, a fully-adapted cooperator is at a selective disadvantage against a fully-adapted defector, which does not incur the cost of cooperation. These results indicate that the type of niche construction matters. Specifically, negative niche construction is key for maintaining cooperation by the Hankshaw effect. Here, cooperators prevent defector invasion by hitchhiking along with adaptations to the constructed environment.

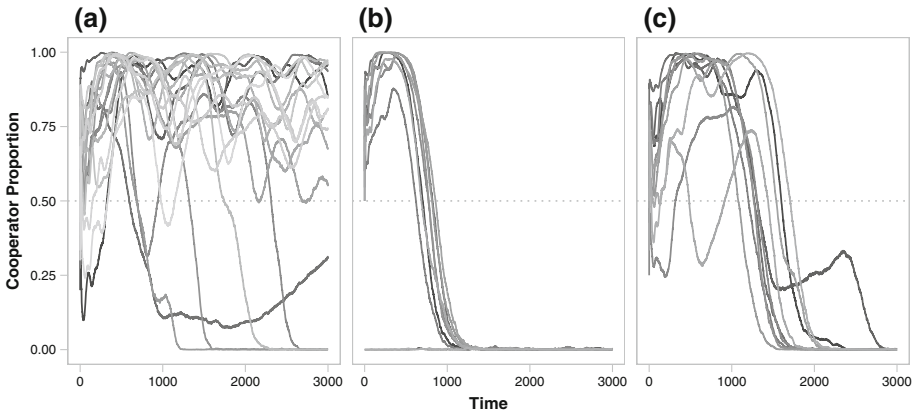


Fig. 2 Niche construction and the evolution of cooperation. The proportion of cooperators present in each replicate population is shown for the duration of simulations. (**a**) Despite some oscillation, cooperators dominate in 13 of 18 populations when niche construction affects selection. (**b**) When the selective effects of niche construction are transferred to supplement the benefits conferred by adaptation to the external, non-constructed environment, cooperators are driven to extinction by defectors (replacing $S_{max} \times \epsilon = 0.3$, $\delta = 0.3$ with $\epsilon = 0$, $\delta = 0.6$). Note that cooperation was not present after initialization in one replicate population. (**c**) Cooperators are also driven to extinction without negative niche construction ($A = 5$)

Selective feedbacks limit defector invasion

The process of adaptation to the constructed niche can limit invasion by defectors, which arise either through migration from neighboring patches or through mutation at the cooperation locus. This latter challenge is particularly threatening, as these isogenic defectors are equally adapted, yet do not incur the cost of cooperation. As demonstrated in Fig. 3a, when adaptation to the environment cannot occur, isogenic defectors rapidly invade when introduced as a single subpopulation in the center of a population of otherwise all-cooperator subpopulations. However, cooperators resist defector invasion in over half of the replicate populations when adaptations can arise through mutation (Fig. 3b). Figure 4 depicts one such instance. In that population, isogenic defectors are seeded at a single patch in an otherwise all-cooperator population. These defectors quickly begin to spread. However, a neighboring cooperator subpopulation gains an adaptation, which increases its fitness above that of the defector. This type spreads more quickly, stopping the spread of defectors and eventually driving them to extinction. Because this adaption arises in a cooperator subpopulation, cooperation is able to hitchhike to safety. Importantly, this new cooperator type is favored because of the niche that its ancestral type—and therefore also the defector—constructed. Here, cooperators can find safety in numbers: because their larger subpopulations have more mutational opportunities, they are more likely to gain adaptations that rescue them from invasion. Further, these larger cooperator subpopulations exert greater influence on their niches, which increases selection for an adapted type. This allows that type to appear and to spread more quickly in the population. Figure 3c shows how quickly an adapted cooperator type can invade a population of defectors.

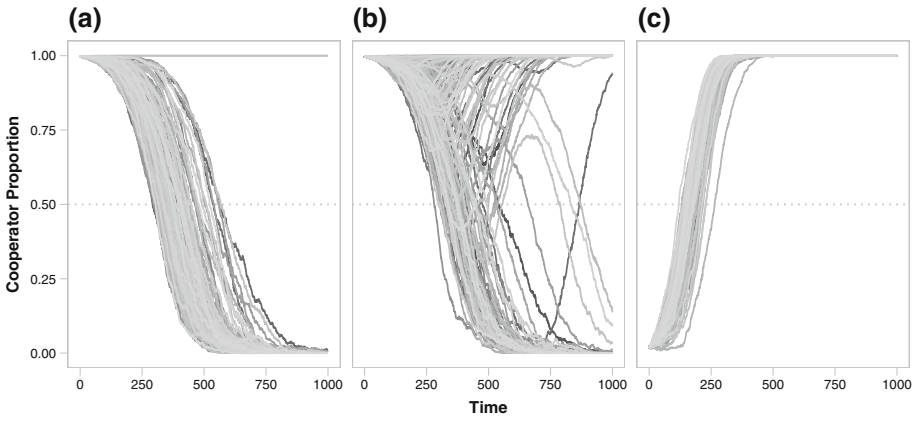


Fig. 3 Niche Construction and Invasion. The proportion of cooperators present in each replicate population is shown for the duration of simulations ($T = 1000$). In each simulation, a rare type was initiated at a single patch in the center of the population lattice ($N^2 = 121$). Unless otherwise noted, mutations are disabled in these ecological simulations to highlight the dynamics of invasion ($\mu_a = 0, \mu_c = 0$). (**a**) When cooperators and defectors are isogenic (i.e., both types have adaptive alleles [1,2,3,4,5]), rare defectors quickly invade and drive cooperators to extinction due to the cost of cooperation. Note that defectors were stochastically eliminated in two replicate populations. (**b**) However, when populations can adapt, negative niche construction creates adaptive opportunities that enable cooperators to resist invasion by isogenic defectors. When adaptive mutations occur ($\mu_a = 0.00005$), cooperation remained dominant in 91 of 160 populations. Results from simulations where mutations also occurred at the cooperation locus are shown in Figure S8. (**c**) In fact, a cooperator (adaptive alleles [6,2,3,4,5], see Box 1) that is adapted to the niche constructed by the defectors can swiftly displace defectors

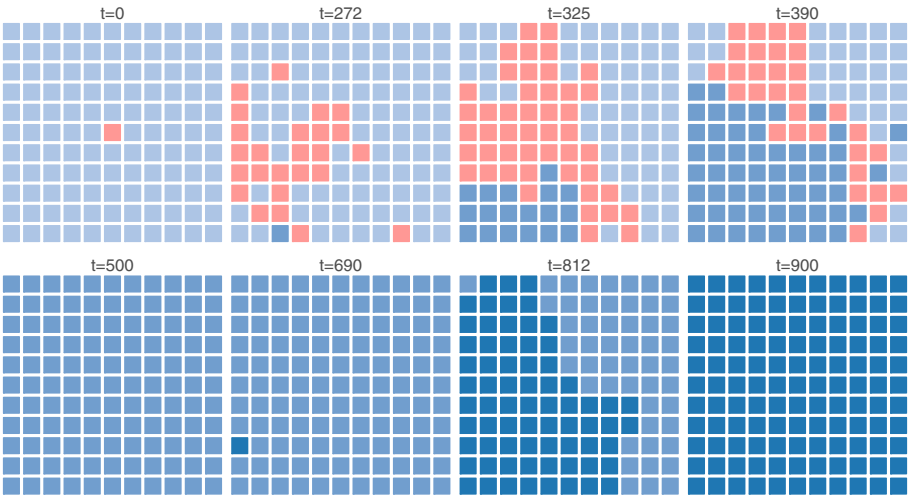


Fig. 4 Cooperator adaptation prevents defector invasion. The spatial distribution of dominant types within each subpopulations is shown at different time points for one representative simulation in which isogenic defectors arise. To highlight the effects of adaptation, mutations did not occur at the cooperation locus ($\mu_c = 0$). At time $t = 0$ (*upper left panel*), a single isogenic defector subpopulation (*red*) is placed within an all-cooperator population (*light blue*). Because these defectors do not bear the cost of cooperation, they quickly spread ($t = 272$). However, cooperators in one subpopulation gain an adaptation that gives them a fitness advantage over defectors (*second panel*, *medium blue*, *lower left*). At $t = 325$, defectors continue to invade cooperator subpopulations. However, the adapted cooperator type spreads more quickly due to its fitness advantage, invading both defector and ancestral cooperator subpopulations ($t = 390$), until it eventually fixes in the population ($t = 500$). At $t = 690$, a new cooperator type emerges that is favored in the constructed niche (*dark blue*). This new type spreads rapidly ($t = 812$) until reaching fixation ($t = 900$). At this point, it becomes susceptible to invasion by the next "adapted" cooperator type, and the cycle continues

Negative niche construction promotes cooperator invasion

The majority of the results shown above have focused on the maintenance of cooperation. Specifically, cooperators have started at—and maintained—reasonably high proportions in their populations. In the previous section, we considered cooperator invasion; however, cooperators began in their own single subpopulation without defectors. It remains to be seen whether cooperators can invade from extreme rarity. In a population in which cooperators are initially absent, can cooperators that arise by mutation increase in frequency? With baseline parameters (Table 1), cooperators tend not to invade over 3000 cycles when the initial cooperator proportion is low (see Supplemental Materials). However, when the benefits of cooperation are increased ($S_{max} = 8000$), cooperators can readily invade and reach high proportions (Fig. 5a). Despite this large benefit, cooperator success still depends on the presence of niche construction. Without the selective effects that negative niche construction continually exerts, cooperators cannot invade (Fig. 5b).

Discussion

Despite their negative effects, deleterious traits can rise in abundance through genetic linkage with other traits that are strongly favored by selection (Hartfield and Otto 2011). The role of hitchhiking in the evolution cooperation has been explored experimentally and

theoretically (Schwilk and Kerr 2002; Santos and Szathmáry 2008; Morgan et al. 2012; Waite and Shou 2012; Asfahl et al. 2015; Wilder et al. 2015). In a process termed the "Hankshaw effect", Hammarlund et al. (2015) recently demonstrated that traits such as cooperation and spite can actively prolong their existence by increasing their likelihood of hitchhiking with a beneficial trait. In that work and here, subpopulations of cooperators grow to a higher density than those of defectors. These larger subpopulations are more likely to gain adaptations as a result of this increase in growth and the corresponding mutational opportunities. Although this process favors cooperation in the short term, it eventually reaches a dead end: When the opportunities for adaptation are exhausted, and cooperators can no longer hitchhike, they face extinction. Here, we have investigated whether niche construction might serve to perpetually generate new adaptive opportunities and thus favor cooperation.

When niche construction occurs, cooperation can indeed persist (Figs. 1c, 2a). In our model, niche construction introduces additional selective effects that influence the evolutionary process, leading to a more pronounced Hankshaw effect. However, these fitness benefits alone do not maintain cooperation (Fig. 2b). Niche construction and the selective feedbacks that it produces play a crucial role.

We find that it is specifically *negative* niche construction that maintains cooperation (Fig. 2c) and can even support invasion by cooperators (Fig. 5a). As cooperator and defector types gain adaptations, they alter their environment in ways that favor other types. Thus, negative niche construction serves as a perpetual source of adaptation. Here we observe another facet of the Hankshaw effect: Because subpopulations of cooperators are

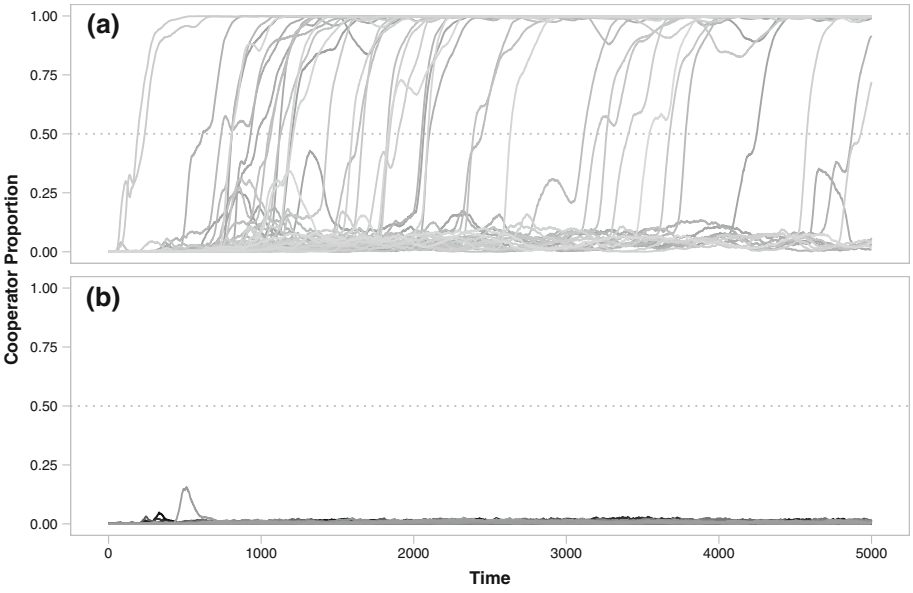


Fig. 5 Niche construction and the invasion of cooperation. The proportion of cooperators present in each of 50 replicate populations is shown for the duration of simulations ($T = 5000$). Baseline parameters are used, except for $S_{max} = 8000$, and the initial proportion of cooperators in each population is zero ($p_0 = 0$), which requires cooperators to arise via mutation. (**a**) Cooperators invade and reach very high proportions in 42 of 50 populations when niche construction affects selection. (**b**) Without selective feedback from niche construction ($\epsilon = 0$), cooperators do not invade (50 replicates shown)

larger, they are better able to respond to the adaptive opportunities that they create through negative niche construction. By gaining adaptations more quickly, cooperators resist invasion by defectors (Fig. 3b). Even in the presence of an isogenic defector type, cooperator subpopulations are more likely to produce the mutant most adapted to the current constructed niche, which can then displace the slower-adapting defectors. These recurring cycles of defector invasion and cooperator adaptation underlie the oscillations in cooperator proportion seen in Fig. 2a. Mutation is still a stochastic process, cooperators lose the adaptive race and are driven to extinction when defectors gain these adaptations first. We see this occur occasionally in Figs. 2a, 3b. However, under other parameter settings within our model, it is possible for cooperators at extremely low abundances to later re-emerge and invade (Fig. 5a). In these instances, negative niche construction provides continual opportunities for cooperators to dominate.

Cooperation as niche construction

In our model, niche construction and adaptation are independent of cooperation, which allows us to focus on hitchhiking. However, individuals often cooperate in ways that alter the environment. These cooperative behaviors, therefore, can themselves be seen as niche construction. For example, bacteria produce a multitude of extracellular products that scavenge soluble iron (Griffin et al. 2004), digest large proteins (Diggle et al. 2007; Darch et al. 2012), and reduce the risk of predation (Cosson et al. 2002), among many others (West et al. 2007a). As in our model, these forms of cooperation are likely to increase local subpopulation density. While many studies have focused on how the environment affects the evolution of these cooperative traits, relatively few have addressed how the environmental changes created by these products feed back to influence evolution.

Perhaps most similar to this study, Van Dyken and Wade (2012) demonstrated that when two negative niche constructing, cooperative behaviors co-evolve, selection can increasingly favor these traits, which are otherwise disfavored when alone. In that model, "reciprocal niche construction" occurred when the negative feedback resulting from one strategy positively influenced selection for the other, creating a perpetually oscillating cycle that maintained both forms of cooperation. Arguably, this can be seen as an instance of hitchhiking: The currently-maladaptive form of cooperation is maintained by association with the adaptive form.

When dispersal is limited, competition among kin can undermine cooperation. To separate kin competition from kin selection, Lehmann (2007) developed a model in which a cooperative, niche-constructing behavior only benefitted future generations. Kin competition was thereby reduced, and cooperation instead benefitted descendants. This work highlights an important aspect of niche construction: Often, the rate of selective feedback from niche construction is different from the rate at which populations grow.

Evolution at multiple timescales

In our work, the niche is modeled implicitly by the composition of the subpopulation. Any changes in the subpopulation, therefore, produce immediate effects on the constructed environment and the resulting selective feedbacks. However, timescales in our model could be de-coupled in two ways. First, cooperators modify their niche by enabling their subpopulation to reach larger density (Eq. 4). These increased subpopulation sizes play a critical role by effectively increasing the rate of evolution in these subpopulations. Because of the importance of this process, it would be very informative to explore how sensitive our

results are to the rate at which cooperators increase subpopulation sizes and the rate at which this benefit decays in the absence of cooperators. Similarly, our results could be substantially affected by alterations in the rate at which the constructed environment changes in response to changes in the subpopulation.

Other studies, while not focused on cooperation, have shown that the timescales at which niche construction feedbacks occur can strongly influence evolutionary outcomes (Laland et al. 1996, 1999). This perspective may be crucial for understanding the evolution of cooperative behaviors like the production of public goods. In these instances, environmental changes are likely to occur on different timescales than growth, which can have profound effects. For example, a multitude of factors, including protein durability (Brown and Taddei 2007; Kümmerli and Brown 2010), diffusion (Allison 2005; Driscoll and Pepper 2010), and resource availability (Zhang and Rainey 2013; Ghoul et al. 2014) influence both the rate and the degree to which public goods alter the environment. While Lehmann (2007) showed that cooperation was favored when selective feedbacks act over longer timescales, niche construction may in fact hinder cooperation when selection is more quickly altered. For example, when public goods accumulate in the environment, cooperators must decrease production to remain competitive (Kümmerli and Brown 2010; Dumas and Kümmerli 2012). This favors cooperation that occurs facultatively, perhaps by sensing the abiotic (Bernier et al. 2011; Koestler and Waters 2014) or biotic environment (Brown and Johnstone 2001; Darch et al. 2012). To study how regulatory traits such as these evolve, we could instead represent the niche explicitly, allowing it to have its own dynamics. A representation in which the "niche" is simultaneously influenced by external forces and the actions of organisms would more closely resemble many natural systems.

Cooperation and niche construction in host-symbiont co-evolution

In many biological systems, the environments modified by organisms are themselves other organisms. In these instances, the "niche" becomes a biological entity with its own evolutionary process. A logical extension to our model would be to treat the environment as an organism. Such a model could be used to explore the evolution of cooperation in host-symbiont systems, where cooperation among symbionts affects host fitness. As the host population changes, either in response to symbiont cooperation or other factors, so too does selection on their symbiont populations. In our model, each patch could become a host with its own genotype, and death and reproduction at the host level could be defined in ways that are sensitive to both host and symbiont genotypes. Here, evolutionary outcomes depend greatly on the degree of shared interest between the host and symbiont.

Of particular importance are cases where the interests of host and symbiont are in conflict. By selecting for new, more resistant host genotypes or by provoking a specific immune response, pathogens make their host environment less hospitable and can therefore be seen as potent negative niche constructors. The results that we have presented here suggest that such negative niche construction can perhaps favor cooperative behavior among these symbiont pathogens. This may be especially relevant when infection is mediated by cooperative behaviors. For example, the cooperative production of several public goods by the pathogenic bacterium *Pseudomonas aeruginosa* facilitate infection in hosts with cystic fibrosis (Harrison 2007). Models such as what we have described may permit exploration into how cooperation and niche construction intersect in these and other medically-relevant instances.

More generally, it was recently argued that incorporating the effects of niche construction is critical for improving our understanding of viral evolution (Hamblin et al.

2014) and evolution in co-infecting parasites (Hafer and Milinski 2015). Incorporating host dynamics, transmission, co-evolution, and the feedbacks that they produce is likely to be equally important for gaining a greater understanding of how cooperative behaviors evolve in these host-symbiont settings.

Summary

We have previously shown that a combination of non-social adaptation and population structure can favor the evolution of cooperation (Hammarlund et al. 2015). However, this "Hankshaw effect" was transient; without continual opportunities for adaptation (e.g., a changing environment), defectors eventually dominate. Here, we explore one source for such continual opportunities: negative niche construction. Specifically, the process of adaptation creates opportunities for further adaptation through selective feedback. In our model, the active role of the organism is paramount; not only does cooperative behavior make hitchhiking more likely given adaptive opportunities, but these adaptive opportunities themselves are continually generated through the effects organisms have on their environment.

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