Negative Niche Construction Favors the Evolution of Cooperation

Abstract

TODO

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.*, 2007). One such factor involves non-random social interaction, in which cooperators benefit more from the cooperative act than 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 these cases, the alleles may provide private benefits that are completely 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. This advantage is reminiscent of Sissy Hankshaw, a fictional character in Tom Robbins' *Even Cowgirls Get the Blues*, whose oversized thumbs—which were otherwise an impairment—made her a prolific hitchhiker. Similarly, cooperation is costly, but it increases local population density. As a result, cooperators are more likely to acquire beneficial

mutations. By hitchhiking along with these adaptations, cooperation can then 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 equally-adapted defectors that arise via mutation. However, Hammarlund et al. (2015) also demonstrated that cooperation can be maintained indefinitely when frequent environmental changes produce a steady stream of new adaptive opportunities. Although organisms typically find themselves in dynamic environments, the nature and frequency of these changes might not ensure long-term cooperator survival.

Importantly, however, organisms do more than simply experience changing environments passively. Through their activities, their interactions with others, and even their deaths, organisms constantly modify their environment. This niche construction process can produce evolutionary feedback loops in which environmental change alters selection, which, in turn, alters the distribution of types and their corresponding influence on the environment (Odling-Smee *et al.*, 2003). The nature of this feedback can have dramatic evolutionary consequences. One critical distinction is whether the constructing type or some other type is most adapted in the resulting environment. Under positive niche construction, selection favors the constructor, and evolution stagnates as this type fixes. Under negative niche construction, selection favors a type other than the constructor. In this latter case, populations find themselves continually chasing beneficial mutations as their adaptive landscape perpetually shifts.

Here, we show that the selective feedbacks that result from niche construction

can maintain cooperation indefinitely. We find that it is specifically negative niche construction that is responsible for this result because of the adaptive opportunities that it produces. Furthermore, we show that the rate at which niche construction occurs is also crucial. These results indicate that cooperators can ensure their survival when they play an active role in their own evolution.

Methods

Building upon Hammarlund et al. (2015), we develop an individual-based model in which cooperators and defectors evolve and compete in a population of subpopulations (i.e., a metapopulation). Through mutations, individuals gain adaptations to their environment, which increase reproductive fitness, and allow those lineages to rise in abundance. Migration among neighboring subpopulations allows more successful lineages to spread.

In this expanded model, subpopulations additionally modify their local environment. As this process occurs, environmental changes feed back to affect selection. We explore how niche construction affects this process of adaptation and whether cooperation can be maintained because of selective feedbacks.

Model Description

Individual Genotypes and Adaptation

Each individual has a haploid genome with $L + 1$ loci (see Table 1 for model parameters and their values). Different alleles at each locus are represented by different integers. A binary allele at the first locus (here, locus zero) determines whether that individual is a cooperator (1), which carries fitness cost c, or a defector (0). Cooperation is independent from adaptation to the environment. The remaining L loci are *adaptive loci*, and are each occupied by 0 or a value from the set $\{1, 2, \ldots, A\}$. Allele 0 represents a lack of adaptation, while a non-zero allele represents one of the A possible adaptations at that locus. These non-zero alleles signify adaptations to the external environment that are not affected by other individuals or the local niche. Adaptations confer a fitness benefit δ, 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.

Niche Construction and Selective Feedbacks

Individual fitness is also affected by the current state of the local environment. We represent the "niche" implicitly based on the specific allelic states present in the subpopulation. Here, the specific alleles that are present at each locus matter. As allelic states change, subpopulations alter aspects of their environment, creating a unique niche.

Niche construction takes the form of density dependent selection, and individuals evolve to better match their niche by a second form of adaptation. Specifically, the selective value of adaptive allele a at locus l increases with the number of individuals in the subpopulation that have allele $a-1$ at locus $l-1$. Once allele a has fixed in the subpopulation at locus l, allele $a+1$ becomes the only allele that confers fitness benefits at locus $l+1$. As a consequence, genotypes with sequentially increasing allelic states will tend to evolve. We treat both adaptive loci and 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 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. The selective value of adaptive allele a at locus l is increased 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.

Consider a genotype g with the allelic state at locus l given by $a_{g,l}$; the fitness of an individual with this genotype is defined as:

$$W_g = z - \underbrace{ca_{g,0}}_{\substack{\text{cost of} \\ \text{cooperation}}} + \underbrace{\delta \sum_{l=1}^{L} I(a_{g,l})}_{\substack{\text{adaptation to} \\ \text{external environment}}} + \underbrace{\epsilon \sum_{l=1}^{L} n(\beta(a_{g,l}, A), \beta(l, L))}_{\substack{\text{adaptation to} \\ \text{constructed environment}}} \quad (2)$$

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

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

Thus, an individual's fitness is determined both by adaptations to the external environment (δ) and adaptations to its constructed environment (ϵ). Figure 1 illustrates the effects of these two components.

Population Growth and the Benefit of Cooperation

Cooperation enables a subpopulation to reach a greater density. This benefit affects all individuals equally and accumulates linearly with the proportion of cooperators in the subpopulation. If p is the proportion of cooperators present at the beginning of a growth cycle, then that subpopulation reaches the following size:

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

During growth, individuals compete through differential reproduction. Each individual's probability of success is determined by 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:

$$\pi_i = \frac{W_{\gamma(i)}}{\sum_{j=1}^{P} W_{\gamma(j)}} \tag{5}$$

Here, $W_{\gamma(i)}$ is the fitness of an individual i with genotype $\gamma(i)$ (see Equation 2). The value π_i represents an individual's reproductive fitness relative to others in the subpopulation.

Mutation

For simplicity, we apply mutations after 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 random selection from the set $\{0\} \cup \{1, 2, \ldots, A\}$. Because mutations are stochastic, the allelic sequences that evolve depend on which allele arises first and at which locus.

Migration

Populations are composed by 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 with probability m. During each migration event, a

single destination patch is randomly chosen with uniform probability from each source patch's Moore neighborhood, which is composed of the nearest 8 patches on the lattice. Because the population lattice has boundaries, patches located on the periphery have smaller neighborhoods.

Population Initialization and Simulation

At the beginning of each simulation, subpopulations are seeded at all patches with cooperator proportion p_0 and grown to density $S(p_0)$. An environmental challenge is then introduced, which subjects all subpopulations to a bottleneck. For each individual, the probability of survival is μ_t, which represents the likelihood that tolerance arises via mutation. Because individuals have not yet adapted to this new environment, the allelic state of each individual's genotype is 0 at each adaptive locus. Following initialization, simulations are run for T cycles, where each discrete cycle consists of subpopulation growth, mutation, migration, and dilution. Dilution thins the population to support growth in the next cycle. Each individual remains with probability d, regardless of allelic state.

Simulation Source Code and Software Dependencies

The simulation software and configurations for the experiments reported are available online.[1] Simulations used Python 3.4, NumPy 1.9.1, Pandas 0.15.2

[1] These materials will be made public at the time of publication, and a reference will be placed here.

(McKinney, 2010), and NetworkX 1.9.1 (Hagberg *et al.*, 2008). Data analyses were performed with R 3.1.3 (R Core Team, 2015). Reported 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 consisting of subpopulations that are connected by spatially-limited migration. Individuals compete in these subpopulations by gaining a limited number of adaptations that confer fitness benefits. While cooperation does not directly affect the selective value of these adaptations, cooperation can have indirect effects on the adaptive process. Specifically, cooperation increases subpopulation density. As a result, larger subpopulations of cooperators experience more mutational opportunities to gain adaptations. Cooperation can hitchhike along with these adaptations, which compensate for the cost of cooperation. During this process, subpopulations alter their local environments, which feeds back to influence selection. Here, we explore how niche construction affects the evolution of cooperation in the simulation environment defined by the parameter values listed in Table 1.

Cooperation Persists with Niche Construction

Without any opportunity for adaptation ($L = 0$), cooperators are swiftly eliminated in competition with defectors (Figure 2A). 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 ($L = 5$), but niche construction is absent ($\epsilon = 0$), cooperators are maintained only transiently (Figure 2B). Here, larger cooperator subpopulations can more quickly adapt to their external environment as before. As previously described by Hammarlund et al. (2015), however, cooperation is subsequently lost once populations become fully adapted to their environment. Once this has occurred, isogenic defectors (i.e., defectors with identical adaptive loci) arise via mutation and displace cooperation due to their selective advantage. However, when niche construction creates selective feedbacks, cooperation persists in over 2/3 of the replicate populations (Figure 2C). We see in Figure 3A that despite oscillations, cooperation is maintained at high levels in these populations.

Fitness Increases Alone do not Support Persisting Cooperation

In the model, both adaptation and niche construction contribute to an individual's fitness. To determine whether cooperation is maintained solely due to the larger selective values that result from the contributions of niche construc-

tion (ϵ), we performed simulations in which these contributions were removed ($\epsilon = 0$), and we instead increased the fitness benefits conferred by adaptation to the external, non-constructed environment ($\delta = 0.6$). In doing so, we conservatively estimate the selective effects of niche construction by supplementing the selective benefits of adaptations to the external environment by the maximum possible selective benefit that results from niche construction. Nevertheless, we find that simply increasing selective values does not enable cooperators to persist (Figure 3B). Niche construction, therefore, plays a decisive role here.

Negative Niche Construction is Critical to Cooperator Persistence

Negative niche construction can occur in our model due to the selection for sequentially-increasing allelic states and the circular arrangement of these alleles (see Figure 1). This 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 with an allele that is not one less than the allele at the next locus. Given this unavoidable mismatch, any type that has fixed will always favor selection for a new type. However, if this negative niche construction is removed (by setting $L = 5$, $A = 5$), cooperators are again driven extinct after an initial lift in abundance (Figure 3C). These results indicate that the type of niche construction matters. Specifically, negative niche construction is crucial for maintaining cooperation.

Selective Feedbacks Limit Defector Invasion

The adaptation resulting from selective feedbacks can limit invasion by defectors, which arise either through immigration from neighboring patches or through mutation from a cooperator ancestor. The latter challenge is particularly threatening, as these isogenic defectors are equally adapted, yet do not incur the cost of cooperation. As demonstrated in Figure 4A, these isogenic defectors rapidly spread when introduced at a single patch in the center of an 11×11 population of cooperators if mutations do not occur. However, when resident cooperators can gain adaptations via mutation, cooperators evade defector invasion in over half of the replicate populations (Figure 4B). Figure 5 depicts one such instance where cooperation survived. In that population, defectors quickly began to spread. However, an adaptation arose at a neighboring cooperator population that was more fit. This type spread more quickly, halting defectors and eventually driving them to extinction. Because this adaption occurred in a cooperator population, cooperation was able to hitchhike to safety. Figure 4C shows how quickly an adapted cooperator type can invade a population of defectors.

The Rate of Niche Construction Matters

TODO: Sorry, results coming soon!

Discussion

Despite their negative effects, deleterious traits can rise in abundance due to genetic linkage with other traits that are strongly favored by selection (Maynard Smith and Haigh, 1974). In a process termed the "Hankshaw effect", Hammarlund et al. (2015) recently demonstrated that cooperation can prolong its existence by increasing the likelihood of hitchhiking with a beneficial trait. While this process does favor 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. In this work, we have considered whether niche construction might serve to perpetually generate new adaptive opportunities, and thus favor cooperation indefinitely.

When niche construction occurs, cooperation can indeed persist (Figures 2C and 3A). In our model, niche construction introduces additional selective effects that could influence the evolutionary process, leading to a more pronounced Hankshaw effect. However, simply raising the fitness benefits conferred by adaptations does not prolong cooperation (Figure 3B), which indicates that niche construction and the selective feedbacks that it produces play a crucial role.

Further, we find that it is specifically negative niche construction that maintains cooperation (Figure 3C). Here we observe another facet of the Hankshaw effect: because populations of cooperators are larger, they are better able to respond to the adaptive opportunities that result from negative niche construction. Without these adaptive opportunities, adaptation eventually grinds to a

halt. Once this occurs, cooperators face the threat of invasion by isogenic defectors that arise through mutation. Since these defectors are equally adapted but do not bear the cost of cooperation, they quickly drive cooperators to extinction. Importantly, because every type constructs an environment in which a different type is more fit, negative niche construction creates continual adaptive opportunities. These opportunities can allow cooperators to resist invasion by defectors, even when defectors are equally adapted (Figure 4B). It is these recurring cycles of invasion and adaptation that underlie the oscillations in cooperator populations that we see in Figure 3A. When stochastic mutations do not engender these adaptations, defectors invade, and the cycle is broken.

TODO: the rate of niche construction is crucial

In our model, cooperation is orthogonal to niche construction, which allows us to focus on hitchhiking. However, by increasing the size of the local patch, this form of cooperation could itself be seen as a niche constructing behavior. Previous studies have more directly explored how niche construction and cooperation interact. Lehmann (2007) showed that cooperation can be favored when niche construction decoupled kin competition from kin selection in spatially-structured populations. Perhaps most similar our work, Van Dyken and Wade (2012) demonstrated that when two negative niche constructing cooperative behaviors co-evolve, selection can increasingly favor these traits, which were disfavored when alone. In that model, "reciprocal niche construction" occurred when the negative feedback created by one strategy positively influenced selection on the other, creating a perpetual cycle that maintained both forms of cooperation. Arguably, this can be viewed as an instance of hitchhiking: the

currently-maladaptive form of cooperation is maintained by association with the adaptive form. Outside of the context of cooperation, studies have shown that niche construction can allow deleterious alleles to be maintained (Laland *et al.*, 1996, 1999). However, cooperation, especially in competition against equally-adapted defectors, can be considered deleterious.

In the model described by Lehmann (2007), the selective feedbacks produced by the cooperative, niche-constructing behavior only affected future generations. Because of this, kin competition was reduced, and cooperation instead benefitted descendants. Other studies, while not focusing on cooperation, have similarly shown that the timescales at which niche construction feedbacks occur can strongly influence evolutionary outcomes (Laland *et al.*, 1996, 1999). This perspective is likely to be crucial for understanding the evolution of cooperative behaviors like the production of public goods.

For example, bacteria produce a host 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). While many studies have focused on how the environment affects the evolution of these cooperative public goods, relatively few have addressed how the environmental changes created by public goods feed back to influence evolution. In these instances, environmental changes are likely to occur on different timescales than reproduction, 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).

In many instances where cooperation occurs, the environment is itself a biological entity, which can introduce additional evolutionary feedbacks. As the host population changes, so too does selection on their symbiont populations. Here, evolutionary outcomes depend greatly on the degree of shared interest between the host and symbiont. For example, the cooperative production of virulence factors by the human pathogen *P. aeruginosa* in lung infections is harmful to hosts with cystic fibrosis (Harrison, 2007). Conversely, cooperative light production by *A. fischeri* is vital for the survival of its host, the Hawaiian bobtail squid (Ruby, 1996). 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, co-evolution, and the feedbacks that they produce into models is likely to be equally important for gaining an understanding of how cooperative behaviors evolve in these host-symbiont settings.

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Figures

Figure 1

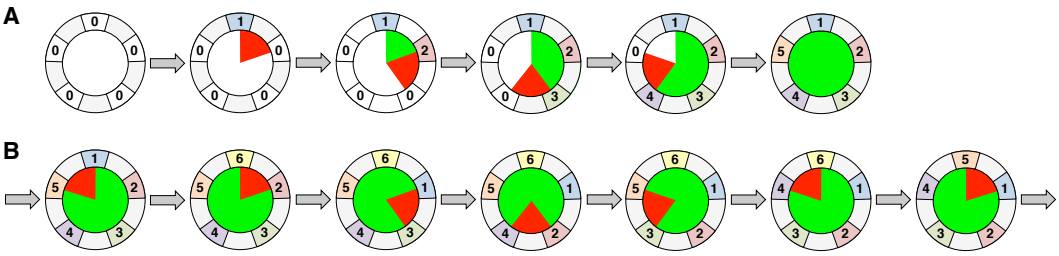

Figure 1: **Adaptation to External and Constructed Environments.** (**A**) We begin with the case with five adaptive loci ($L = 5$) and five non-zero alleles ($A = 5$). All simulations are initialized with a non-adapted genotype with allele 0 at every locus (far left). Random mutation will introduce a non-zero allele, which will increase in frequency. In this example, allele 1 arises at the first locus (in the "12 o'clock" position). The rest of this schematic focuses on niche construction. Every non-zero allele at any locus influences selection at the next locus in the clockwise direction. There is a "mismatch" in this genotype (highlighted by the red sector), because the niche constructed by allele 1 at the first locus favors allele 2, not 0, at its immediate clockwise neighbor (the second locus). Once the appropriate allele arises, it will be selected. In this case, the genotype [1,2,0,0,0] receives an epsilon effect in addition to the extra delta. The "match" at the first and second locus is highlighted as a green sector. However, now there is a new mismatch between the second and third locus, which a new round of mutation and selection corrects, and so on. The green sector grows as the red sector ticks clockwise. Importantly, because A divides evenly into L, this genotype can evolve into a perfectly reinforcing sequence [1,2,3,4,5], which enjoys a maximal epsilon increment of fitness due to niche construction. (**B**) The case of negative niche construction is illustrated for the case of five loci ($L = 5$) and six non-zero alleles ($A = 6$). Here we start with a population fixed for the genotype on the far left [1,2,3,4,5]. There is a single mismatch in this genotype (highlighted by the red sector), because the niche constructed by allele 5 favors allele 6, not 1, at its immediate clockwise neighbor. If the fitter mutant [6,2,3,4,5] arises (see next genotype to the right), it will fix. (We note that the strength of selection will drop as its frequency increases). However, now there is a new mismatch in the genotype (highlighted again with a red sector). We see that correcting one mismatch generates a new mismatch. Thus, this system will never escape its mismatches—-the red sector just clicks clockwise around the genome. Indeed, after six (or A) rounds of mismatch correction and generation, we have ended back where we started with the original genotype turned clockwise by one locus. Here, the adaptation to previous niche construction generates further niche construction that leads to novel adaptation.

Figure 2

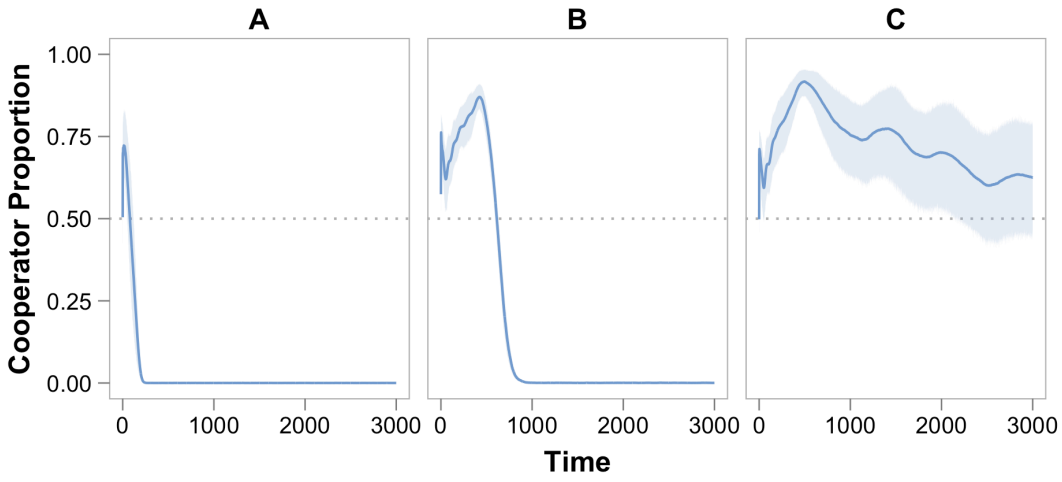

Figure 2: **Adaptation, Hitchhiking, and the Evolution of Cooperation.** The proportion of cooperators present in the population is shown for the duration of simulations. Curves show the average among replicate populations, and shaded areas indicate 95% confidence intervals. Unless otherwise noted, parameter values are listed in Table 1. (**A**) Without any opportunity to adapt (L, the number of adaptive loci, is zero), cooperation is quickly lost. (**B**) When adaptation can occur ($L = 5$), but populations do not alter their environment (ϵ, the intensity of niche construction, is zero), cooperation hitchhikes along with adaptions, allowing cooperators to temporarily rise in abundance before eventually going extinct. (**C**) Niche construction enables cooperation to be maintained indefinitely. In the majority of populations, cooperation remained the dominant strategy. Individual populations are shown in Figure 3A.

Figure 3

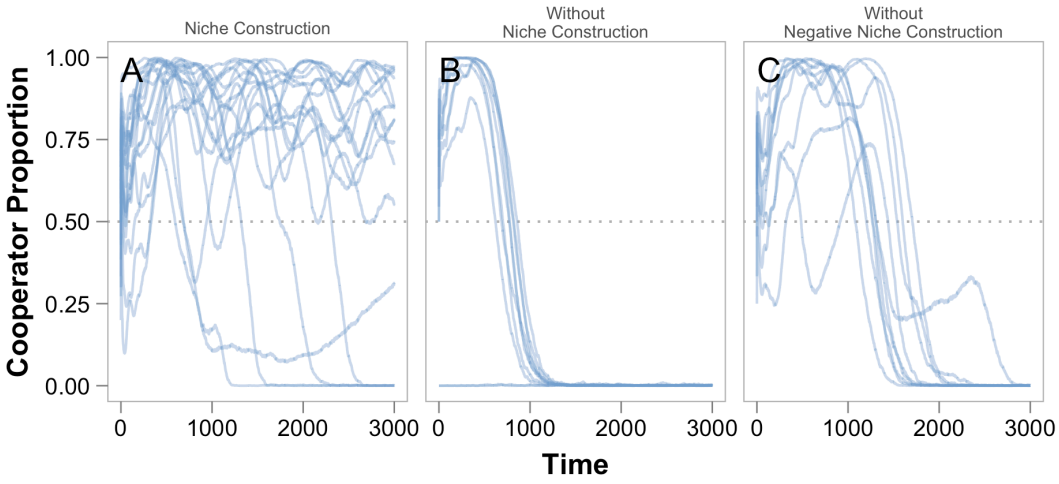

Figure 3: **Niche Construction and the Evolution of Cooperation.** The proportion of cooperators present in each replicate population is shown for the duration of simulations. (**A**) Despite some oscillations, niche construction enables cooperation to be maintained indefinitely in 14 of 18 populations. (**B**) When niche construction is removed and the fitness benefit of adaptation is increased to compensate ($\epsilon = 0$, $\delta = 0.6$), adapted defectors arise and drive cooperators to extinction. (**C**) Without negative niche construction, cooperation is not maintained ($A = 5$).

Figure 4

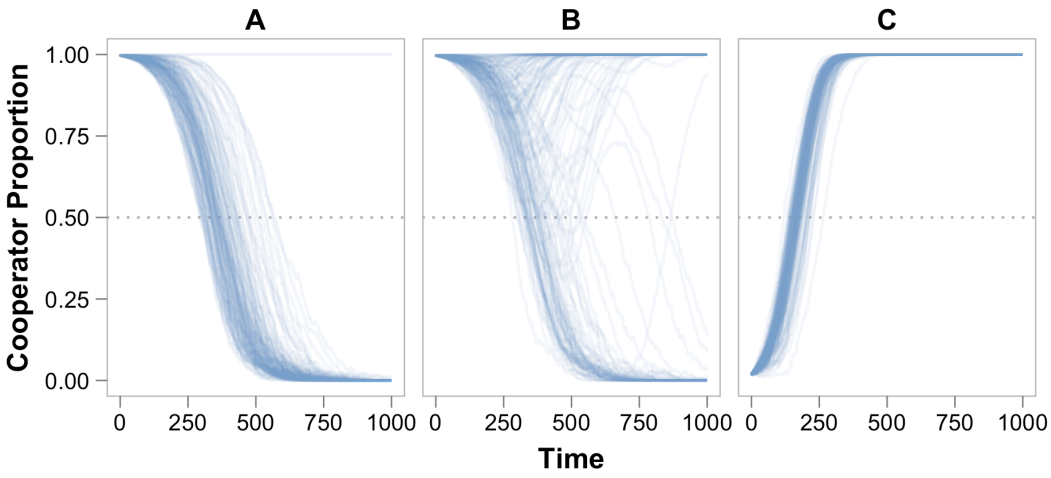

Figure 4: **Niche Construction and Invasion.** Curves trace the proportion of cooperators present in the population for the duration of 160 replicate simulations ($T = 1000$). These experiments examine whether a rare cooperator or defector strategy can invade when initiated at a single patch in the center of the population lattice ($N^2 = 121$). Unless otherwise noted, mutations ($\mu_a = 0, \mu_c = 0$) are disabled in these ecological simulations to highlight the dynamics of invasion. The results from simulations where this limitation is removed are shown in Figure S1. (**A**) When cooperators and defectors are isogenic (i.e., both types have stress alleles [1,2,3,4,5]) and mutation cannot occur, rare defectors quickly invade and drive cooperators to extinction due to the cost of cooperation. Defectors were stochastically eliminated in 2 replicate populations. (**B**) However, the adaptive opportunities produced by negative niche construction can allow cooperators to resist invasion by isogenic defectors. Here, cooperation persisted in the majority of populations ($\mu_a = 0.00005$, the base mutation rate). (**C**) We demonstrate that adaptations such as these can enable a cooperator (stress alleles [6,2,3,4,5], see Figure 1) to displace a population of defectors when defectors cannot arise or adapt via mutation.

Figure 5

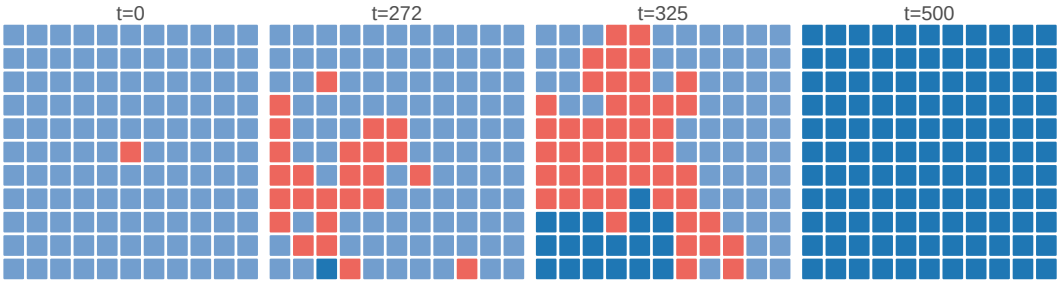

Figure 5: **Defector Invasion Stopped by Cooperator Adaptation.** Here we depict the distribution of dominant types among populations over time for one representative simulation in which isogenic defectors arise. For clarity, mutations occurred at the adaptive loci, but not at the cooperation locus ($\mu_c = 0$) during this ecological simulation. A time $t = 0$ (leftmost panel), a single matched defector population (red) is placed among cooperator populations (light blue). Because these defectors do not bear the costs of cooperation, they spread ($t = 272$, second panel). However, cooperators in a single population gain an adaptation that give them a fitness advantage over defectors (dark blue, lower left). At $t = 325$ (third panel), defectors continue to invade cooperator populations. However, the adapted cooperator type, which can invade both defector populations and ancestral cooperator populations, can spread more quickly due to its greater fitness. Eventually, this strategy spreads and fixes in all populations (rightmost panel) until this strategy itself is replaced by the next adaptation.

Figure 6

TODO Yep. Almost ready.

Supplemental Figure 1

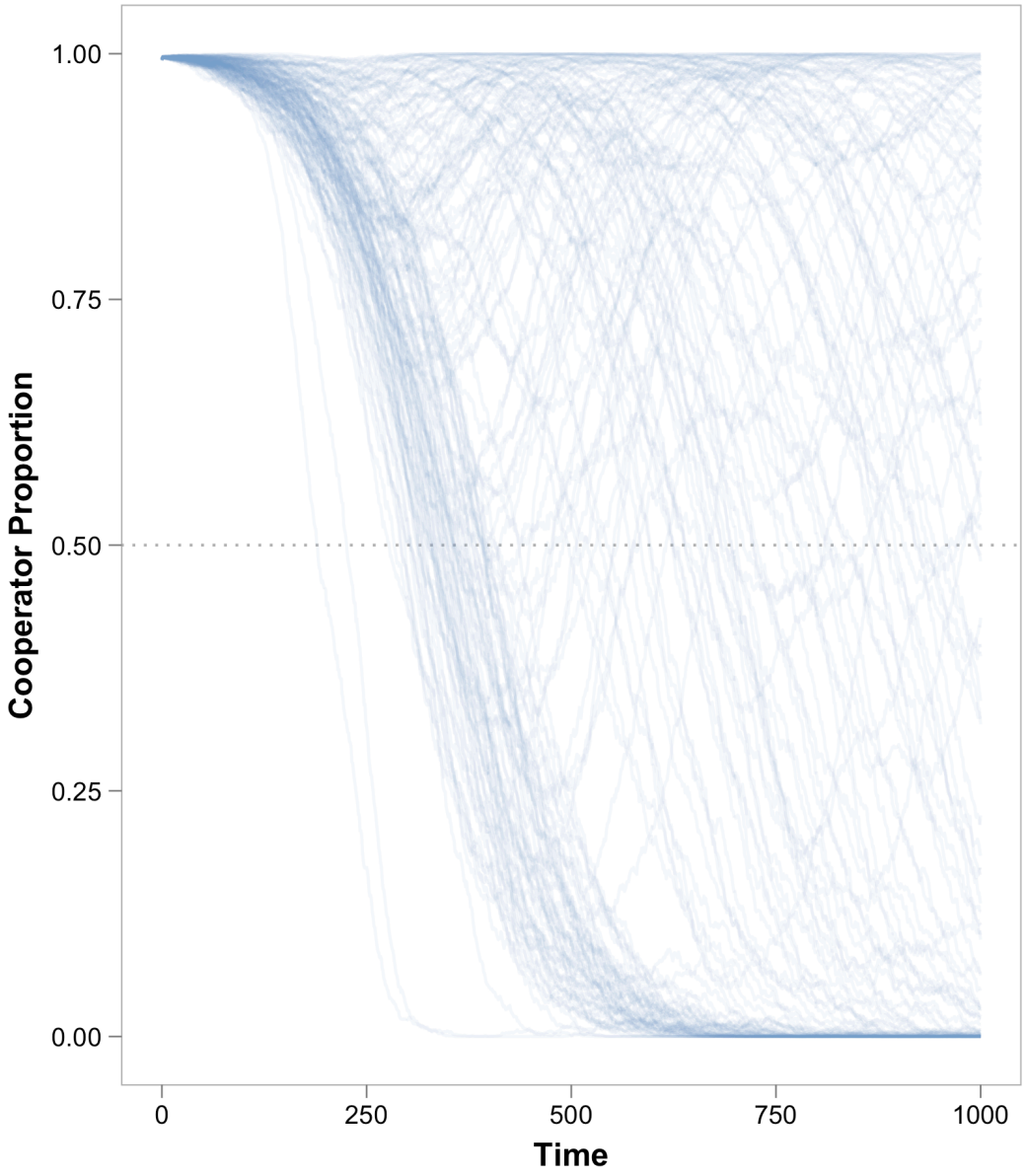

Figure S1: **Defector Invasion with Mutations.** With mutations occurring both at the adaptive loci and the cooperation locus ($\mu_a = \mu c = 0.00005$), cooperation remains the dominant strategy in 58 replicate simulations. Curves trace the proportion of cooperators present in the population for the duration of 160 replicate simulations ($T = 1000$)

Tables

Table 1: Model parameters and their value

Parameter	Description	Base Value
L	Number of adaptive loci	5
c	Fitness cost of cooperation	0.1
A	Number of alleles	6
δ	Fitness benefit, nonzero alleles	0.3
ϵ	Fitness benefit, sequential alleles	0.00015
z	Baseline fitness	1
S_{min}	Minimum subpopulation size	800
S_{max}	Maximum subpopulation size	2000
μ_a	Mutation rate (adaptation)	10^{-5}
μ_c	Mutation rate (cooperation)	10^{-5}
N^2	Number of patches	625
m	Migration rate	0.05
p_0	Initial cooperator proportion	0.5
μ_t	Mutation rate (tolerance to new environment)	10^{-5}
T	Number of simulation cycles	3000
d	Subpopulation dilution factor	0.1

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