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.*, 2007b). 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 an asexual population 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 demonstrated 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) 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. These changes 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. 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 the evolutionary process.

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 our 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. Here, we represent the "niche" implicitly based on the allelic states present in the subpopulation. 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 $l-1$. 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 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:

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

where z is a baseline fitness and $I(a)$ indicates whether a given adaptive allele is non-zero:

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

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 benefits a subpopulation by enabling it to reach 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:

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

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 $\{0\} \cup \{1, 2, \ldots, A\}$.

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 (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 adap-

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

tive 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, in turn, influences 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 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 construction (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. We find that simply increasing selective values does not enable cooperators to persist (Figure 3B). Niche construction therefore plays an important 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. 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 (see Figure 1). 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).

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 challenge is particularly threatening, as they are equally adapted, yet do not incur the cost of cooperation. When isogenic defectors are introduced at a single patch in the center of an 11×11 population of cooperator subpopulations, they quickly spread if no mutations are allowed (Figure 4A). However, when resident cooperators can adapt (mutations occur at adaptive loci), cooperators evade defector invasion in over half of the replicate populations (Figure 4B). Figure 5 depicts one such instance where cooperators gained an adaptation that stopped and eliminated invading defectors. We further highlight this process in Figure 4C, where an adapted cooperator can rapidly 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 (Figure 2C). 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 selective benefits provided 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 adaptive opportunities, adaptation eventually grinds to a halt. Once this occurs, cooperators face the threat of invasion by defectors that arise de novo through mutation. Since these defectors are equally adapted but do not bear the cost of cooperation, they quickly drive cooperators to extinction. 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 confer these adaptations, defectors invade, and the cycle is broken.

TODO: the rate of niche construction is crucial

In our model, cooperation and niche construction are orthogonal, which allows us to focus on hitchhiking. However, the form of cooperation used in this model could itself be seen as a niche constructing behavior. Explicitly modeling this cooperative behavior, which is akin to the production of public goods, would likely yield additional insights into the relationship between cooperation and niche construction. For example, previous work has shown that niche construction can favor deleterious alleles (Laland *et al.*, 1996, 1999). Cooperation, especially in competition against equally-adapted defectors, can be considered deleterious, so introducing selective feedbacks from cooperation could further bolster cooperation. Van Dyken and Wade (2012) showed that

when two cooperative behaviors co-evolve and niche construction feedbacks benefit the other type, niche construction can increasingly favor these traits, which were otherwise disfavored when alone. Arguably, this can be viewed as another instance of hitchhiking: the maladaptive form of cooperation is maintained by association with the adaptive form. However, negative niche construction then reverses these roles and perpetuates the cycle.

By their very nature, public goods benefit populations by making their environment more hospitable (West *et al.*, 2007a). For example, bacteria produce a host of extracellular products that scavage 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 cooperative behaviors such as the production of these public goods, relatively few have examined how the resulting selective feedbacks influence evolution as public goods modify the environment. In these instances, environmental changes are likely to occur on different timescales than reproduction. These differences 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. Lehmann (2007) demonstrated that cooperative, niche constructing behaviors can be favored when they affect selection for future generations. When this occurs, conflict among contemporary kin is reduced. The evolutionary inertia that this creates, however, may ultimately

work against cooperators. 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.

Acknowledgments

We are grateful to TODO for helpful comments on the manuscript and to Anuraag Pakanati for assistance with simulations. This material is based upon work supported by the National Science Foundation Postdoctoral Research Fellowship in Biology under Grant No. DBI-1309318 (to BDC) and under Cooperative Agreement No. DBI-0939454 (BEACON STC). Computational resources were provided by an award from Google Inc. (to BDC and BK).

Figures

Figure 1

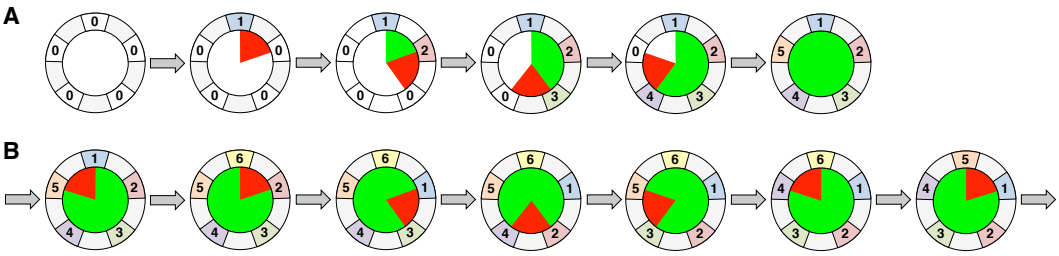

Figure 1: **Adaptation to External and Constructed Environments.** (**A**) We start with the case with five loci ($L = 5$) and five non-zero alleles ($A = 5$). All simulations are initialized with a non-adapted genotype with allele 0 at every locus—the genotype on the far left. Random mutation will introduce a non-zero allele, which is expected to increase in frequency. For simplicity, we assume that 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 an maximal epsilon increment of fitness of due to its 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). Thus, 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/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

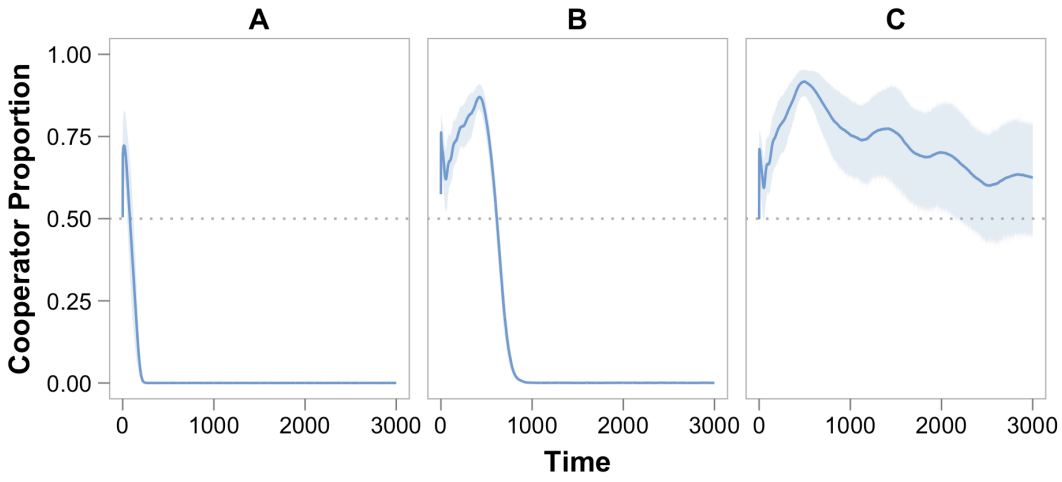

Figure 2: **Adaptation, Hitchhiking, and the Evolution of Cooperation.** The proportion of cooperators present in the population is shown for the duration of simulations. Curves show the average among replicate populations, and shaded areas indicate 95% confidence intervals. Unless otherwise noted, parameter 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 (13/18), cooperation remained the dominant strategy. Individual populations are shown in Figure 3A.

Figure 3

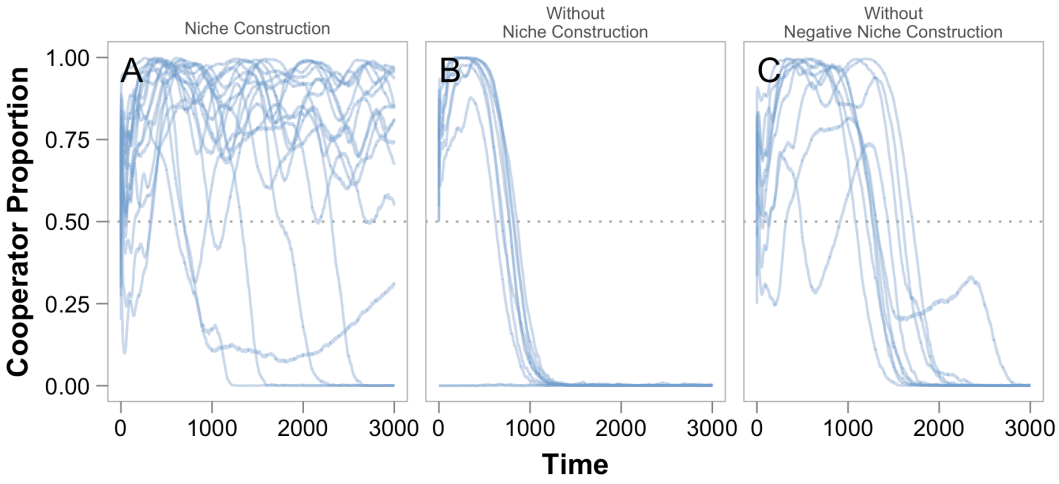

Figure 3: **Niche Construction and the Evolution of Cooperation.** The proportion of cooperators present in each replicate population is shown for the duration of simulations. (**A**) Dispite some oscillations, niche construction enables cooperation to be maintained indefinitely in 14 of 18 populations. (**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

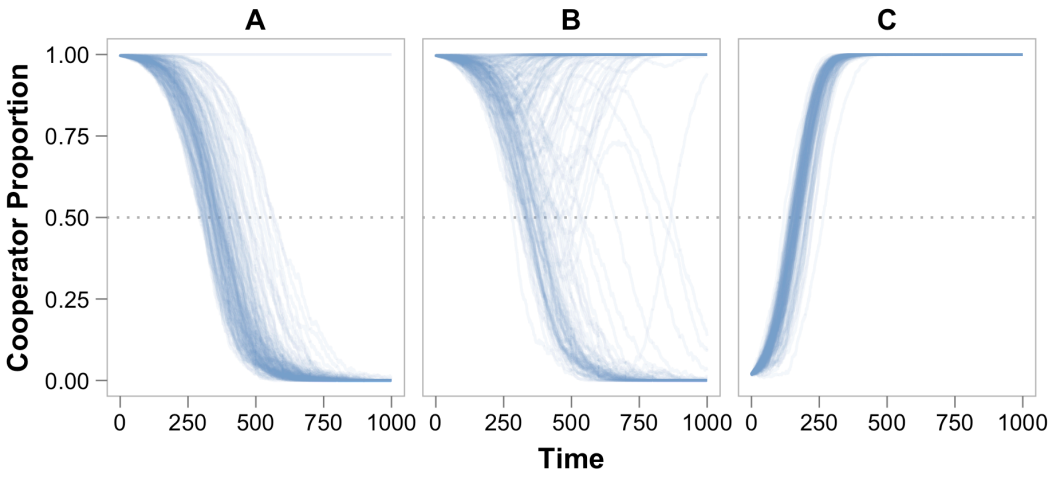

Figure 4: **Niche Construction and Invasion.** Curves trace the proportion of cooperators present in the population for the duration of 160 replicate simulations ($T = 1000$). These experiments examine whether a rare cooperator or defector strategy can invade when initiated at a single patch in the center 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 remoed 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 an 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

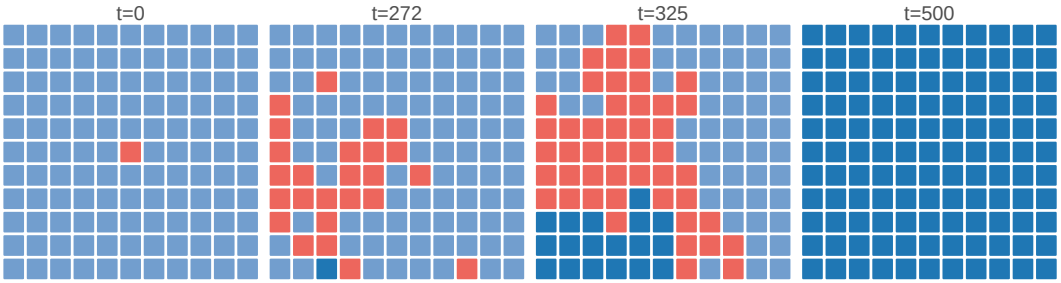

Figure 5: **Defector Invasion Stopped by Cooperator Adaptation.** Here we depict the distribution of dominant types among populations over time for one representative simulation in which isogenic defectors arise. For clarity, mutations occurred at the adaptive loci, but not at the cooperation locus ($\mu_c = 0$) 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

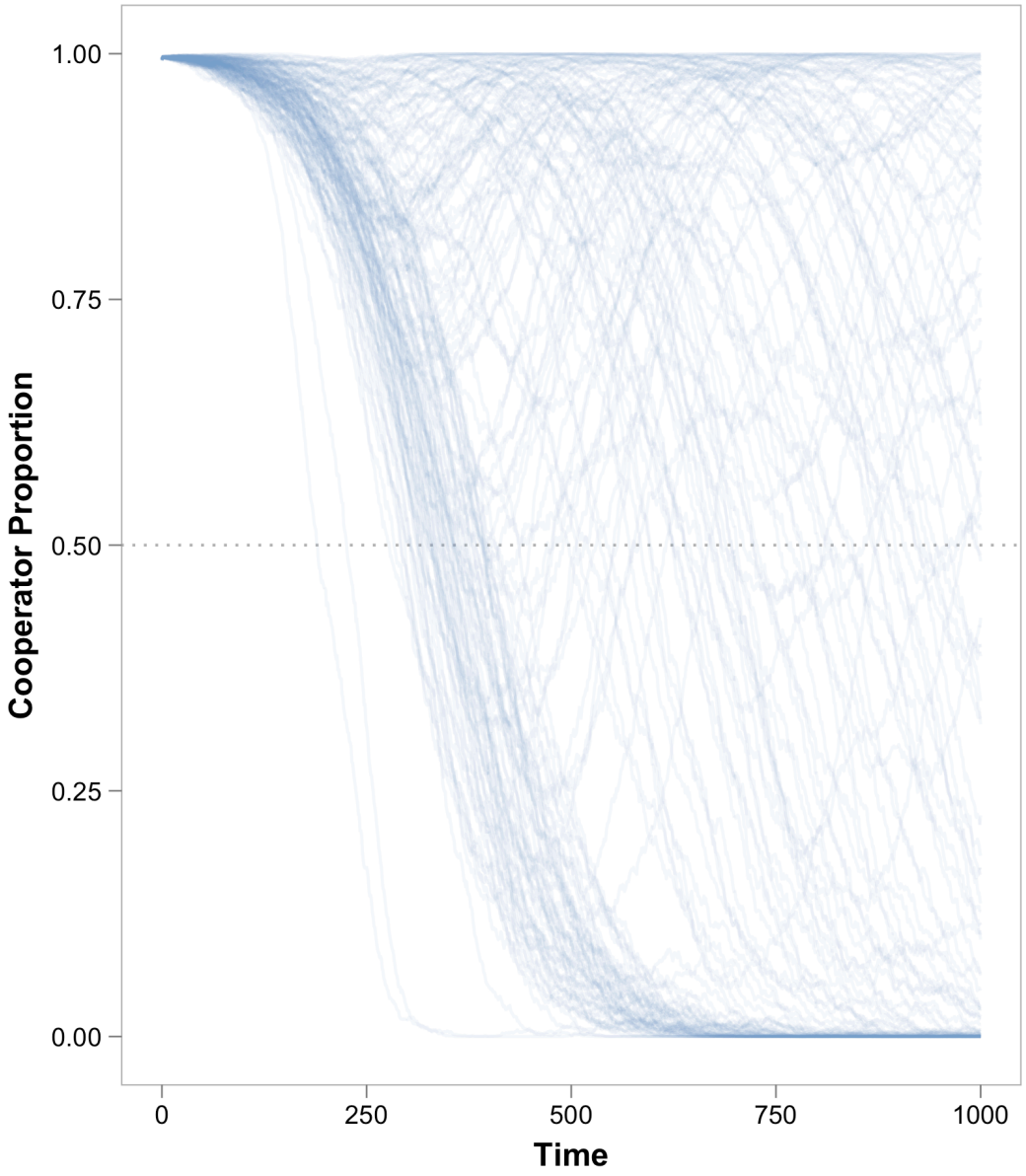

Figure S1: **Defector Invasion with Mutations.** With mutations occurring both at the adaptive loci and the cooperation locus ($\mu_a = \mu c = 0.00005$), cooperation remains the dominant strategy in 58 replicate simulations. Curves trace the proportion of cooperators present in the population for the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