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 association with alleles encoding self-benefitting traits (Asfahl *et al.*, 2015). In the latter case, 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. When cooperation increases local population density, the likelihood of acquiring beneficial mutations is also increased. The cooperative trait can rise in abundance by hitchhiking along with these adaptations. Nevertheless, this advantage is fleeting. As soon as the opportunities for adaptation are exhausted, cooperators are once again at a selective disad-

vantage 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 not simply play passive roles in environmental change and in evolution. 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 phenotypes and their corresponding influence on the environment (Odling-Smee *et al.*, 2003). Because of these feedback loops, populations may find themselves continually chasing beneficial mutations as their adaptive landscape perpetually shifts beneath them.

Here, we explore whether the selective feedbacks that arise niche construction can prolong cooperation. We build upon the model presented by Hammarlund et al. (2015) to allow populations to modify their local environments in ways that affect fitness. We first use this model to address whether niche construction can extend the Hankshaw effect, allowing cooperation to keep hitchhiking as populations continually adapt. We then focus on how niche construction influences outcomes when isolated cooperator populations encounter populations of defectors, either through migration or through mutations that inevitably produce defectors that share the same adaptations. Finally, niche

construction has frequently been shown to increase diversity (**???**). We explore whether this diversity helps or hinders the evolution of cooperation.

Methods

We develop an individual-based model in which populations of cooperators and defectors evolve and compete in a spatially-structured metapopulation (a collection of populations). Through mutations, individuals gain adaptations to their environment, which increase reproductive fitness, and allow those lineages to rise in abundance. Migration among neighboring populations allows more successful lineages to spread.

We expand upon the model described by Hammarlund et al. (2015) to allow populations to modify their local environment. As this process occurs, environmental changes feed back to affect selection. We perform simulations using this model to explore how niche construction affects this adaptation process, and whether selective feedbacks allow cooperation to be maintained.

Model Description

Individual Genotypes and Adaptation

Each individual in a population has a genotype, which is an ordered list of $L+1$ integers, or *loci* (see Table 1 for model parameters and their values). Different values at these loci represent different alleles. A binary allele at locus $L+1$

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 first L loci are *adaptive loci*, and are each occupied by 0 or an integer 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. Adaptations confer a fitness benefit δ, regardless of which non-zero allele is present. We choose $\delta > c$, which allows a minimally adapted cooperator to recoup the cost of cooperation and gain a fitness advantage. The benefits that these adaptations engender are purely endogenous, and are not affected by the other individuals or the state of the environment.

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 population. As allelic states change, populations alter their environment in different ways, creating a unique niche.

We use a form of density dependent selection to favors individuals that better match their niche. Specifically, the selective value of adaptive allele a at locus l increases with the number of individuals in the population 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 L is affected by the allelic composition of the population at locus 1. Similarly, the selective value of allele

A at any locus increases with the number of individuals carrying allele 1 at the next locus. This circularity is represented by the function $\beta(x, X)$, which gives the integer that follows an arbitrary value x in the set $\{1, 2, \ldots, X\}$:

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

Here, $\mathrm{mod}_X(x)$ is the integer remainder when dividing x by X. Thus, the selective value of adaptive allele a at locus l increases with the number of individuals that have allele $\beta(a, A)$ at locus $\beta(l, L)$. The slope of this increase is ϵ, which 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:

$$W_g = z + \delta \sum_{l=1}^{L} I(a_{g,l}) + \epsilon \sum_{l=1}^{L} n(\beta(a_{g,l}, A), \beta(l, L)) - c a_{g,L+1} \tag{2}$$

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 endogenously by adaptation (δ) and exogenously by its niche (ϵ).

Because mutations occur randomly (see below), each population will evolve

different consecutive sequences. These different sequences represent the unique niches constructed by populations.

Population Growth and the Benefit of Cooperation

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

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

During growth, individuals compete for inclusion in the resulting population. Each individual's probability of success is determined by its fitness. The composition of a population with size P and cooperator proportion p after growth is multinomial with parameters and $S(p)$ 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 population.

Mutation

For simplicity, we apply mutations after population growth. Mutations occur independently at each locus and cause an allelic state change. At each adaptive locus, mutations occur at rate μ_a. These mutations replace the existing allele with a random selection from the set $\{0\} \cup \{1, 2, \ldots, A\}$. Note that this allows for the possibility of an allele replacing itself, thus slightly reducing the effective mutation rate. 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.

Migration

Our simulated environment consists of N^2 patches arranged as an $N \times N$ lattice, where each patch can support a population. After mutation, individuals emigrate to an adjacent patch at rate 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 metapopulation lattice has boundaries, patches located on an edge have smaller neighborhoods.

Metapopulation Initialization and Simulation

Metapopulations are initiated in a state that follows an environmental change, which leaves most patches empty. First, populations 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 the population 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 population growth, mutation, and migration. At the end of each cycle, populations are thinned to allow for growth in the next cycle. Each individual persists with probability d, regardless of allelic state.

Source Code and Software Environment

The simulation software and configurations for the experiments reported are available online. 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 metapopulation consisting of populations connected by spatially-limited migration. Individuals compete in these populations by gaining a limited number of adaptations that confer fit-

ness 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 population density. As a result, larger populations 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, populations 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 1A). Despite an initial lift due to increased productivity, the cost of cooperation becomes disadvantageous as migration mixes the initially isolated populations. When there are opportunities for adaptation ($L = 5$) but no niche construction ($\epsilon = 0$), cooperators are maintained transiently (Figure 1B). Here, larger cooperator populations can more quickly adapt to their environment. 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, adapted defectors that arise via mutation at the cooperation locus have a selective advantage and drive cooperators from the population. However, when niche construction creates selective feedbacks, cooperation persists in 13 of 18 repli-

cate populations (Figure 2A).

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 because to the larger selective values, we performed simulations in which the selective contributions of niche construction were removed ($\epsilon = 0$), and we instead increased the fitness benefits conferred by adaptation ($\delta = 0.6$). In doing so, we conservatively represent the selective effects of niche construction, as fitness benefits of this magnitude would only be given for sequential allelic states that are fixed in full populations. We find that simply increasing selective values does not allow cooperators to persist (Figure 2B).

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. When this occurs, adaptations at one locus reduce the selective effects at another locus, and thus negatively affect fitness. This occurs when when the genome length (L) is not evenly divided by the number of adaptive alleles (A), which makes it impossible to evolve sequentially increasing allelic states.

When negative niche construction is removed ($L = 5$, $A = 5$), cooperators are again driven to extinction after an initial lift in abundance (Figure 2C).

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 pose a particularly challenging threat, as they are equally adapted, yet do not incur the cost of cooperation. When homologous defectors (i.e., defectors with identical stress loci) are introduced as a single population in the center of an $11x11$ metapopulation of cooperators, they quickly spread (Figure 3A). However, when resident cooperators can adapt and respond to defector invasion, the situation improves dramatically, allowing cooperation to evade defector invasion in 91 of 160 replicate populations (Figure 3B). Figure 4 depicts one such instance where cooperators gained an adaptation that stopped and eliminated invading defectors. We further highlight this process in Figure 3C, where an adapted cooperator genotype can rapidly invade a population of defectors.

Diversity Hampers the Evolution of Cooperation

TODO: defector can invade a diverse population of cooperators, while adaptation to a matching defector can't spread to stop invasion.

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 cooperative behaviors can prolong their existence by actively increasing their 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 can maintain cooperation indefinitely.

When niche construction occurs, cooperation can indeed persist (Figure 2A). 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 2B), and indicates that niche construction plays an important role.

We find that cooperator success is due to negative niche construction. Without adaptive opportunities, adaptation eventually slows. 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 are favored by selection, and quickly drive cooperators to extinction. By reducing fitness, negative niche construction creates adaptive

opportunities. These opportunities can allow cooperators to resist invasion by defectors, even when defectors are equally adapted (Figure 3B). 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.

TODO: diversity results TODO: references about diversity

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 envi-

ronment more hospitable (West *et al.*, 2007a). For example, bacteria produce a host of extracellular products that find 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 explored 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, the timescale at which the environment is likely to be decoupled from the timescale at which reproduction occurs. 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 good accumulates 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 of cooperation, the environment is itself a biological entity,

which can produce 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 those 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 thank 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 (to BDC and BK).

Figures

Figure 1

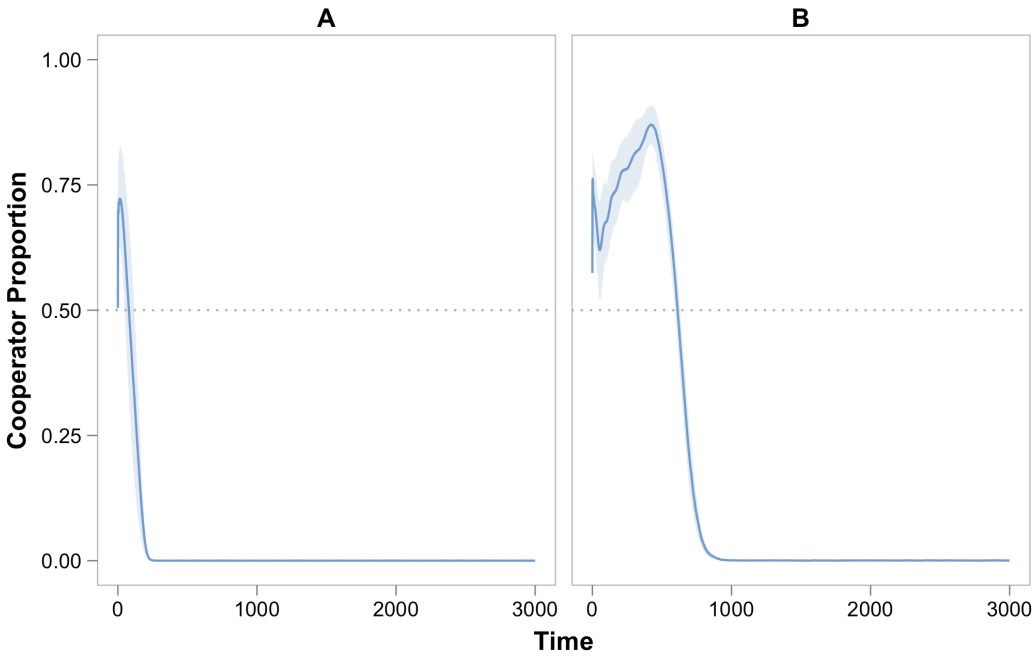

Figure 1: **Adaptation, Hitchhiking, and the Evolution of Cooperation.** The proportion of cooperators present in the population is shown for the duration of simulations. Curves show the average among replicate populations, and shaded areas indicate 95% confidence intervals. Unless otherwise noted, parameter 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.

Figure 2

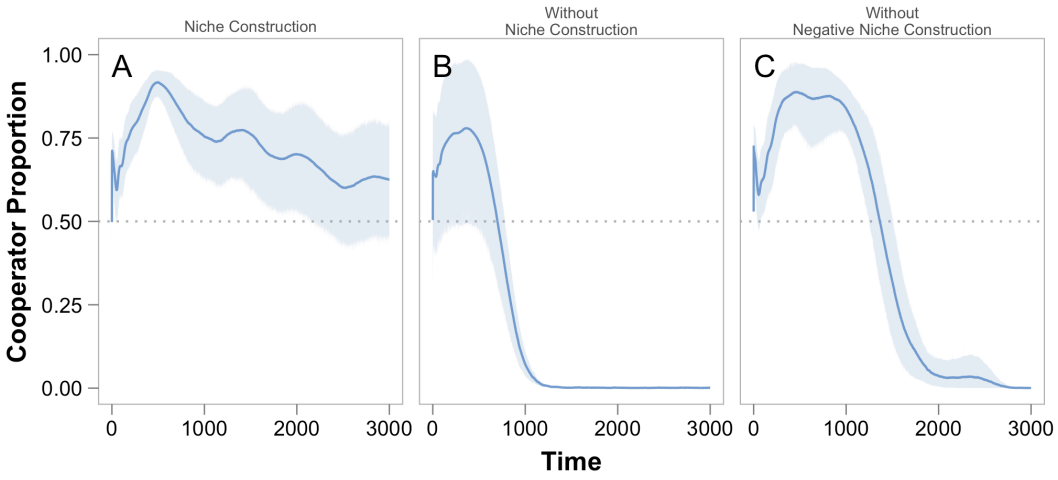

Figure 2: **Niche Construction and the Evolution of Cooperation.** The proportion of cooperators present in the population is shown for the duration of simulations. Curves show the average among replicate populations, and shaded areas indicate 95% confidence intervals. (**A**) Niche construction enables cooperation to be maintained indefinitely. In the majority of populations, cooperation remained as the dominant strategy. (**B**) When niche construction is removed and the fitness benefit of adaptation is increased as compensation ($\epsilon = 0$, $\delta = 0.6$), adapted defectors arise and drive cooperators to extinction. (**C**) Without negative niche construction, cooperation is not maintained ($A = 5$). Here again, cooperators are at a selective disadvantage against equally-adapted defectors.

Figure 3

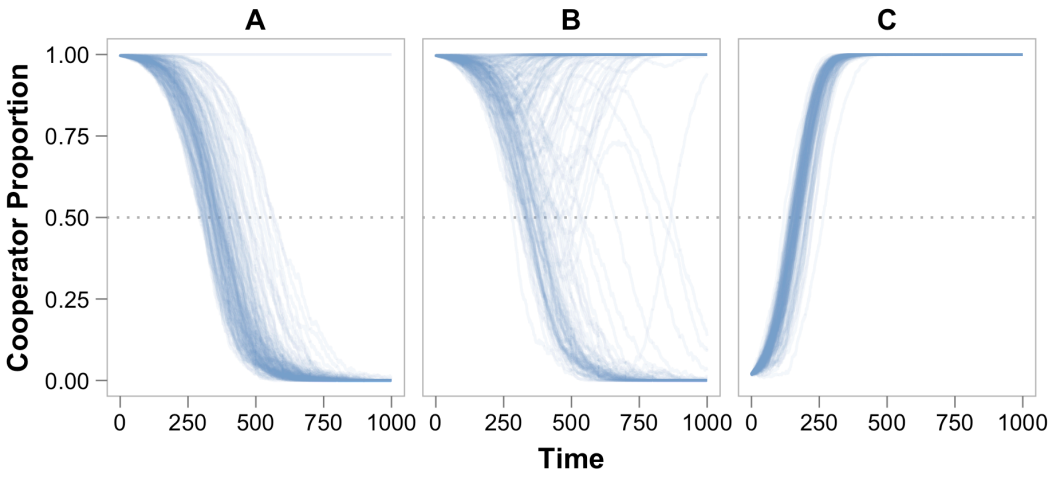

Figure 3: **Niche Construction and Invasion.** Curves trace the proportion of cooperators present in the population for the duration of 160 replicate simulations. These experiments examine whether a strategy initiated at a single population in the center of the metapopulation lattice ($N^2 = 121$) can invade. Unless otherwise noted, we disable mutations ($\mu_a = 0, \mu_c = 0$) to focus on the dynamics of invasion. This limitation is removed in the results shown in Figure SX. (**A**) When cooperators and defectors are matched (i.e., genotypes [1,2,3,4,5]) and adaptation can not occur, defectors quickly 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 initially-matching defectors. Here, cooperation persisted in the majority of populations ($\mu_a = 0.00005$, the base mutation rate). (**C**) Here we demonstrate that these adaptations can enable an adapted cooperator (genotype [1,2,3,4,*6*]) to displace a population of defectors when defectors can not arise or adapt via mutation.

Figure 4

TODO: snapshots of cooperators adapting to thwart defector invasion

Figure 5

TODO: A: defector invading diverse C popuation, B: Adapted cooperators can not spread to resist defector invasion.

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 population size	800
S_{max}	Maximum population size	2000
μ_a	Mutation rate (adaptation)	10^{-5}
μ_c	Mutation rate (cooperation)	10^{-5}
N^2	Number of metapopulation sites	625
m	Migration rate	0.05
p_0	Initial cooperator proportion	0.5
μ_t	Mutation rate (tolerance to new environment)	10^{-5}
d	Population dilution factor	0.1
T	Number of simulation cycles	1000

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