

1 Negative Niche Construction Favors the  
2 Evolution of Cooperation

3  
4 **Abstract**

5 TODO

6 **Introduction**

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

16 Several factors can prevent this *tragedy of the commons* (Hamilton, 1964;  
17 Nowak, 2006; West *et al.*, 2007). One such factor involves non-random so-  
18 cial interaction, in which cooperators benefit more from the cooperative act  
19 than defectors. This can occur when cooperators are clustered together in  
20 spatially-structured populations (Fletcher and Doebeli, 2009; Nadell *et al.*,  
21 2010; Kuzdzal-Fick *et al.*, 2011) or when cooperators use communication  
22 (Brown and Johnstone, 2001; Darch *et al.*, 2012) or other cues (Sinervo *et al.*,  
23 2006; Gardner and West, 2010; Veelders *et al.*, 2010) to cooperate condition-  
24 ally with kin. Cooperation can also be bolstered by pleiotropic connections to  
25 personal benefits (Foster *et al.*, 2004; Dandekar *et al.*, 2012) or through associ-  
26 ation with alleles encoding self-benefitting traits (Asfahl *et al.*, 2015). In these  
27 cases, the alleles may provide private benefits that are completely independent  
28 from the public benefits of cooperation. In asexual populations of cooperators  
29 and defectors, this sets the stage for an “adaptive race” in which both types  
30 vie for the first highly beneficial adaptation (Waite and Shou, 2012; Morgan  
31 *et al.*, 2012). The tragedy of the commons can be deferred if a cooperator, by  
32 chance, wins the adaptive race.

33 Hammarlund *et al.* (2015) recently showed that in spatially-structured pop-  
34 ulations, the “Hankshaw effect” can give cooperators a substantial leg up on  
35 defectors in an adaptive race. This advantage is reminiscent of Sissy Han-  
36 kshaw, a fictional character in Tom Robbins’ *Even Cowgirls Get the Blues*,  
37 whose oversized thumbs—which were otherwise an impairment—made her a  
38 prolific hitchhiker. Similarly, cooperation is costly, but it increases local pop-  
39 ulation density. As a result, cooperators are more likely to acquire beneficial

40 mutations. By hitchhiking along with these adaptations, cooperation can then  
41 rise in abundance. Nevertheless, this advantage is fleeting. As soon as the  
42 opportunities for adaptation are exhausted, cooperators are once again at a  
43 selective disadvantage against equally-adapted defectors that arise via muta-  
44 tion. However, Hammarlund et al. (2015) also demonstrated that cooperation  
45 can be maintained indefinitely when frequent environmental changes produce  
46 a steady stream of new adaptive opportunities. Although organisms typically  
47 find themselves in dynamic environments, the nature and frequency of these  
48 changes might not ensure long-term cooperator survival.

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

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

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

## 70 **Methods**

71 Building upon Hammarlund et al. (2015), we develop an individual-based  
72 model in which cooperators and defectors evolve and compete in a population  
73 of subpopulations (i.e., a metapopulation). Through mutations, individuals  
74 gain adaptations to their environment, which increase reproductive fitness,  
75 and allow those lineages to rise in abundance. More successful lineages spread  
76 to neighboring subpopulations by migration.

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

## 81 **Model Description**

### 82 **Individual Genotypes and Adaptation**

83 Each individual has a haploid genome with  $L + 1$  loci (see [Table 1](#) for model  
84 parameters and their values). Different alleles at each locus are represented by  
85 different integers. A binary allele at the first locus (here, locus zero) determines  
86 whether that individual is a cooperator (1), which carries fitness cost  $c$ , or a  
87 defector (0). Cooperation is independent from adaptation to the environment.  
88 The remaining  $L$  loci are *adaptive loci*, and are each occupied by 0 or a value  
89 from the set  $\{1, 2, \dots, A\}$ . Allele 0 represents a lack of adaptation, while  
90 a non-zero allele represents one of the  $A$  possible adaptations at that locus.  
91 These non-zero alleles signify adaptations to the external environment that  
92 are not affected by other individuals or the local niche. Adaptations confer  
93 a fitness benefit  $\delta$ , regardless of which non-zero allele is present. We assume  
94  $\delta > c$ , which allows a minimally adapted cooperator to recoup the cost of  
95 cooperation and gain a fitness advantage.

### 96 **Niche Construction and Selective Feedbacks**

97 Individual fitness is also affected by the current state of the local environ-  
98 ment. We represent the “niche” implicitly based on the specific allelic states  
99 present in the subpopulation. Here, the specific alleles that are present at each  
100 locus matter. As allelic states change, subpopulations alter aspects of their  
101 environment, creating a unique niche.

102 Niche construction takes the form of density dependent selection, and indi-  
 103 viduals evolve to better match their niche by a second form of adaptation.  
 104 Specifically, the selective value of adaptive allele  $a$  at locus  $l$  increases with  
 105 the number of individuals in the subpopulation that have allele  $a - 1$  at locus  
 106  $l - 1$ . Once allele  $a$  has fixed in the subpopulation at locus  $l$ , allele  $a + 1$   
 107 becomes the only allele that confers fitness benefits at locus  $l + 1$ . As a conse-  
 108 quence, genotypes with sequentially increasing allelic states will tend to evolve.  
 109 We treat both adaptive loci and allelic states as “circular”: the selective value  
 110 of an allele at locus 1 is affected by the allelic composition of the subpopula-  
 111 tion at locus  $L$ . Similarly, the selective value of allele 1 at any locus increases  
 112 with the number of individuals carrying allele  $A$  at the previous locus. This  
 113 circularity is represented by the function  $\beta(x, X)$ , which gives the integer that  
 114 is below an arbitrary value  $x$  in the set  $\{1, 2, \dots, X\}$ :

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

115 Here,  $\text{mod}_X(x)$  is the integer remainder when dividing  $x$  by  $X$ . The selective  
 116 value of adaptive allele  $a$  at locus  $l$  is increased by  $\epsilon$  for each individual in the  
 117 subpopulation that has allele  $\beta(a, A)$  at locus  $\beta(l, L)$ . Thus,  $\epsilon$  specifies the  
 118 intensity of selection due to niche construction.

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

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

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:

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

Thus, an individual's fitness is determined both by adaptations to the external environment ( $\delta$ ) and adaptations to its constructed environment ( $\epsilon$ ). **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

134 of a subpopulation with size  $P$  and cooperator proportion  $p$  after growth is  
135 multinomial with parameters  $S(p)$  and  $\{\pi_1, \pi_2, \dots, \pi_P\}$ , where  $\pi_i$  represents  
136 an individual's reproductive fitness relative to others in the subpopulation.

## 137 **Mutation**

138 For simplicity, we apply mutations after growth. Mutations occur indepen-  
139 dently at each locus and cause an allelic state change. At the binary coopera-  
140 tion locus, mutations occur at rate  $\mu_c$ . These mutations flip the allelic state,  
141 causing cooperators to become defectors and vice versa. Mutations occur at  
142 rate  $\mu_a$  at each adaptive locus. These mutations replace the existing allele  
143 with a random selection from the set  $\{0\} \cup \{1, 2, \dots, A\}$ . Because mutations  
144 are stochastic, the allelic sequences that evolve depend on which allele arises  
145 first and at which locus.

## 146 **Migration**

147 Populations are composed by  $N^2$  patches arranged as an  $N \times N$  lattice, where  
148 each patch can support a subpopulation. After mutation, individuals emigrate  
149 to an adjacent patch with probability  $m$ . During each migration event, a  
150 single destination patch is randomly chosen with uniform probability from  
151 each source patch's Moore neighborhood, which is composed of the nearest 8  
152 patches on the lattice. Because the population lattice has boundaries, patches  
153 located on the periphery have smaller neighborhoods.



## 154 Population Initialization and Simulation

155 Following Hammarlund et al. (2015), we begin simulations with sparse pop-  
156 ulations. Subpopulations are first seeded at all patches with size  $S(p_0)$  and  
157 cooperator proportion  $p_0$ . The population is then thinned to create empty  
158 patches. Each individual survives this bottleneck with probability  $\sigma$ . Starting  
159 from this state, simulations then proceed for  $T$  cycles, where each discrete cycle  
160 consists of subpopulation growth, mutation, migration, and dilution. Dilution  
161 thins the population to support growth in the next cycle. Each individual  
162 remains with probability  $d$ , regardless of allelic state.

## 163 Simulation Source Code and Software Dependencies

164 The simulation software and configurations for the experiments reported are  
165 available online.<sup>1</sup> Simulations used Python 3.4, NumPy 1.9.1, Pandas 0.15.2  
166 (McKinney, 2010), and NetworkX 1.9.1 (Hagberg *et al.*, 2008). Data analy-  
167 ses were performed with R 3.1.3 (R Core Team, 2015). Reported confidence  
168 intervals were estimated by bootstrapping with 1000 resamples.

## 169 Results

170 Using the model described in the previous section, we perform simulations  
171 that follow the evolution of cooperation in a population consisting of subpopu-

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<sup>1</sup>These materials will be made public at the time of publication, and a reference will be placed here.

172 lations that are connected by spatially-limited migration. Individuals compete  
 173 in these subpopulations by gaining a limited number of adaptations that con-  
 174 fer fitness benefits. While cooperation does not directly affect the selective  
 175 value of these adaptations, cooperation can have indirect effects on the adap-  
 176 tive process. Specifically, cooperation increases subpopulation density. As a  
 177 result, larger subpopulations of cooperators experience more mutational op-  
 178 portunities to gain adaptations. Cooperation can hitchhike along with these  
 179 adaptations, which compensate for the cost of cooperation. During this process,  
 180 subpopulations alter their local environments, which feeds back to influence  
 181 selection. Here, we explore how niche construction affects the evolution of  
 182 cooperation in the simulation environment defined by the parameter values  
 183 listed in [Table 1](#).

## 184 **Cooperation Persists with Niche Construction**

185 Without any opportunity for adaptation ( $L = 0$ ), cooperators are swiftly elim-  
 186 inated in competition with defectors ([Figure 2A](#)). Despite an initial lift in  
 187 cooperator abundance due to increased productivity, the cost of cooperation  
 188 becomes disadvantageous as migration mixes the initially isolated subpopula-  
 189 tions. When populations can adapt to the external environment ( $L = 5$ ), but  
 190 niche construction is absent ( $\epsilon = 0$ ), cooperators are maintained only tran-  
 191 siently ([Figure 2B](#)). Here, larger cooperator subpopulations can more quickly  
 192 adapt to their external environment as before. As previously described by  
 193 Hammarlund et al. (2015), however, cooperation is subsequently lost once

194 populations become fully adapted to their environment. Once this has oc-  
195 curred, isogenic defectors (i.e., defectors with identical adaptive loci) arise via  
196 mutation and displace cooperation due to their selective advantage. However,  
197 when niche construction creates selective feedbacks, cooperation persists in  
198 over 2/3 of the replicate populations (Figure 2C). We see in Figure 3A that  
199 despite oscillations, cooperation is maintained at high levels in these popula-  
200 tions.

## 201 **Fitness Increases Alone do not Support Persisting Coop-** 202 **eration**

203 In the model, adaptations to both the external environment and the con-  
204 structed environment contribute to an individual's fitness. To determine  
205 whether cooperation is maintained solely due to the larger selective values  
206 that result from the contributions of niche construction ( $\epsilon$ ), we performed  
207 simulations in which these contributions were removed ( $\epsilon = 0$ ), and we in-  
208 stead increased the fitness benefits conferred by adaptation to the external,  
209 non-constructed environment ( $\delta = 0.6$ ). In doing so, we conservatively esti-  
210 mate the selective effects of niche construction by supplementing the selective  
211 benefits of adaptations to the external environment by the maximum possible  
212 selective benefit that results from niche construction. Nevertheless, we find  
213 that simply increasing selective values does not enable cooperators to persist  
214 (Figure 3B). Niche construction, therefore, plays a decisive role here.

## 215 Negative Niche Construction is Critical to Cooperator 216 Persistence

217 Negative niche construction can occur in our model due to the selection for  
218 sequentially-increasing allelic states and the circular arrangement of these al-  
219 leles (see [Figure 1](#)). This occurs when the number of adaptive alleles ( $A$ ) does  
220 not divide evenly into the number of adaptive loci ( $L$ ). In such a case, any  
221 sequence of integers on the circular genome will always contain a break in the  
222 sequence; that is, one locus with an allele that is not one less than the allele at  
223 the next locus. Given this unavoidable mismatch, any type that has fixed will  
224 always favor selection for a new type. However, if this negative niche construc-  
225 tion is removed (by setting  $L = 5$ ,  $A = 5$ ), cooperators are again driven extinct  
226 after an initial lift in abundance ([Figure 3C](#)). These results indicate that the  
227 type of niche construction matters. Specifically, negative niche construction  
228 is crucial for maintaining cooperation.

## 229 Selective Feedbacks Limit Defector Invasion

230 The adaptation resulting from selective feedbacks can limit invasion by de-  
231 fectors, which arise either through immigration from neighboring patches or  
232 through mutation from a cooperator ancestor. The latter challenge is partic-  
233 ularly threatening, as these isogenic defectors are equally adapted, yet do not  
234 incur the cost of cooperation. As demonstrated in [Figure 4A](#), these isogenic  
235 defectors rapidly spread when introduced at a single patch in the center of an  
236  $11 \times 11$  population of cooperators if mutations do not occur. However, when

237 resident cooperators can gain adaptations via mutation, cooperators evade de-  
238 fector invasion in over half of the replicate populations (Figure 4B). Figure  
239 5 depicts one such instance where cooperation survived. In that population,  
240 defectors quickly began to spread. However, an adaptation arose at a neighbor-  
241 ing cooperator population that was more fit. This type spread more quickly,  
242 halting defectors and eventually driving them to extinction. Because this adap-  
243 tion occurred in a cooperator population, cooperation was able to hitchhike to  
244 safety. Figure 4C shows how quickly an adapted cooperator type can invade  
245 a population of defectors.

## 246 Negative Niche Construction Must Follow a Path

247 **TODO:** Sorry, results will be here for the morning meeting!

## 248 Discussion

249 Despite their negative effects, deleterious traits can rise in abundance due to  
250 genetic linkage with other traits that are strongly favored by selection (May-  
251 nard Smith and Haigh, 1974). In a process termed the “Hankshaw effect”,  
252 Hammarlund et al. (2015) recently demonstrated that cooperation can pro-  
253 long its existence by increasing the likelihood of hitchhiking with a beneficial  
254 trait. While this process does favor cooperation in the short term, it eventually  
255 reaches a dead end; when the opportunities for adaptation are exhausted, and  
256 cooperators can no longer hitchhike, they face extinction. In this work, we

257 have considered whether niche construction might serve to perpetually gener-  
258 ate new adaptive opportunities, and thus favor cooperation indefinitely.

259 When niche construction occurs, cooperation can indeed persist (Figures 2C  
260 and 3A). In our model, niche construction introduces additional selective ef-  
261 fects that could influence the evolutionary process, leading to a more pro-  
262 nounced Hankshaw effect. However, simply raising the fitness benefits con-  
263 ferred by adaptations does not prolong cooperation (Figure 3B), which indi-  
264 cates that niche construction and the selective feedbacks that it produces play  
265 a crucial role.

266 Further, we find that it is specifically negative niche construction that main-  
267 tains cooperation (Figure 3C). Here we observe another facet of the Hankshaw  
268 effect: because populations of cooperators are larger, they are better able to  
269 respond to the adaptive opportunities that result from negative niche construc-  
270 tion. Without these adaptive opportunities, adaptation eventually grinds to a  
271 halt. Once this occurs, cooperators face the threat of invasion by isogenic de-  
272 fectors that arise through mutation. Since these defectors are equally adapted  
273 but do not bear the cost of cooperation, they quickly drive cooperators to ex-  
274 tinction. Importantly, because every type constructs an environment in which  
275 a different type is more fit, negative niche construction creates continual adap-  
276 tive opportunities. These opportunities can allow cooperators to resist invasion  
277 by defectors, even when defectors are equally adapted (Figure 4B). It is these  
278 recurring cycles of invasion and adaptation that underlie the oscillations in  
279 cooperator populations that we see in Figure 3A. When stochastic mutations  
280 do not engender these adaptations, defectors invade, and the cycle is broken.

281 **TODO: the rate of niche construction is crucial**

## 282 **Niche Construction and the Evolution of Cooperation**

283 In our model, cooperation is orthogonal to niche construction, which allows us  
284 to focus on hitchhiking. However, by increasing the size of the local patch, this  
285 form of cooperation could itself be seen as a niche constructing behavior. Pre-  
286 vious studies have more directly explored how niche construction and coopera-  
287 tion interact. Lehmann (2007) showed that cooperation can be favored when  
288 niche construction decoupled kin competition from kin selection in spatially-  
289 structured populations. Perhaps most similar our work, Van Dyken and Wade  
290 (2012) demonstrated that when two negative niche constructing cooperative  
291 behaviors co-evolve, selection can increasingly favor these traits, which were  
292 disfavored when alone. In that model, “reciprocal niche construction” occurred  
293 when the negative feedback created by one strategy positively influenced se-  
294 lection on the other, creating a perpetual cycle that maintained both forms of  
295 cooperation. Arguably, this can be viewed as an instance of hitchhiking: the  
296 currently-maladaptive form of cooperation is maintained by association with  
297 the adaptive form. Outside of the context of cooperation, studies have shown  
298 that niche construction can allow deleterious alleles to be maintained (Laland  
299 *et al.*, 1996, 1999). However, cooperation, especially in competition against  
300 equally-adapted defectors, can be considered deleterious.

## 301 Evolution at Multiple Timescales

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

310 For example, bacteria produce a host of extracellular products that scavenge  
311 soluble iron (Griffin *et al.*, 2004), digest large proteins (Diggle *et al.*, 2007;  
312 Darch *et al.*, 2012), and reduce the risk of predation (Cosson *et al.*, 2002).  
313 While many studies have focused on how the environment affects the evolu-  
314 tion of these cooperative public goods, relatively few have addressed how the  
315 environmental changes created by public goods feed back to influence evolu-  
316 tion. In these instances, environmental changes are likely to occur on different  
317 timescales than reproduction, which can have profound effects. For exam-  
318 ple, a multitude of factors including protein durability (Brown and Taddei,  
319 2007; Kümmerli and Brown, 2010), diffusion (Allison, 2005; Driscoll and Pep-  
320 per, 2010), and resource availability (Zhang and Rainey, 2013; Ghoul *et al.*,  
321 2014) influence both the rate and the degree to which public goods alter the  
322 environment. While Lehmann (2007) showed that cooperation was favored  
323 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).

## Cooperation and Niche Construction in Host-Symbiont Co-Evolution

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

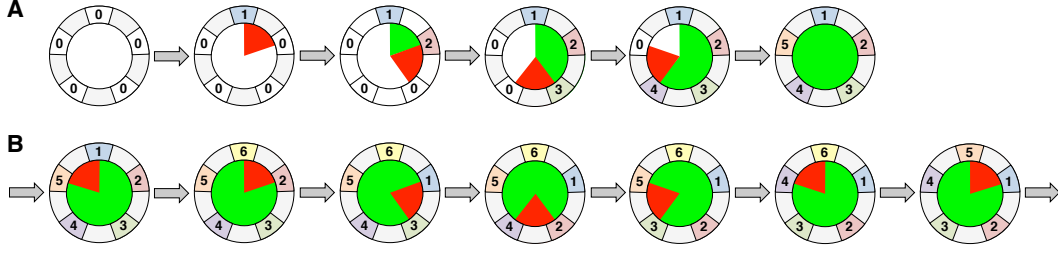
346 settings.

## 347 **Acknowledgments**

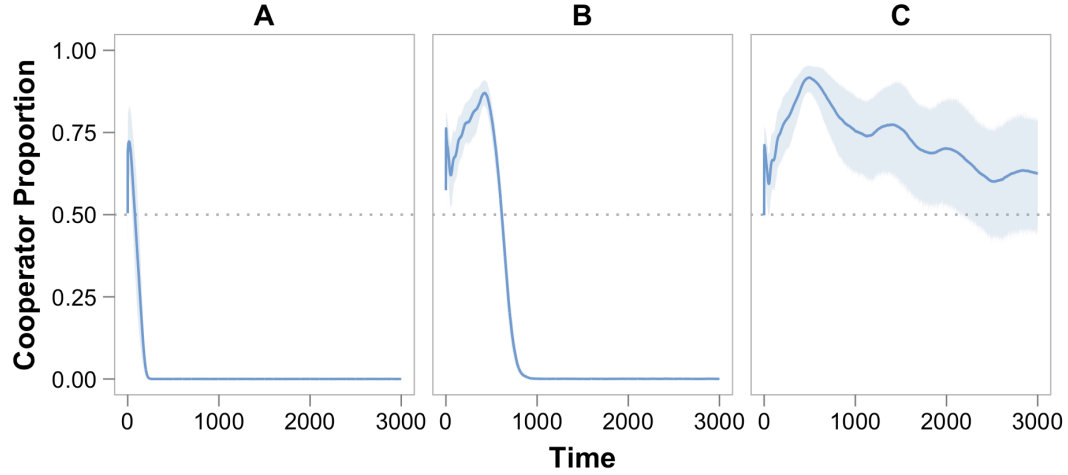
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355 **Figures**

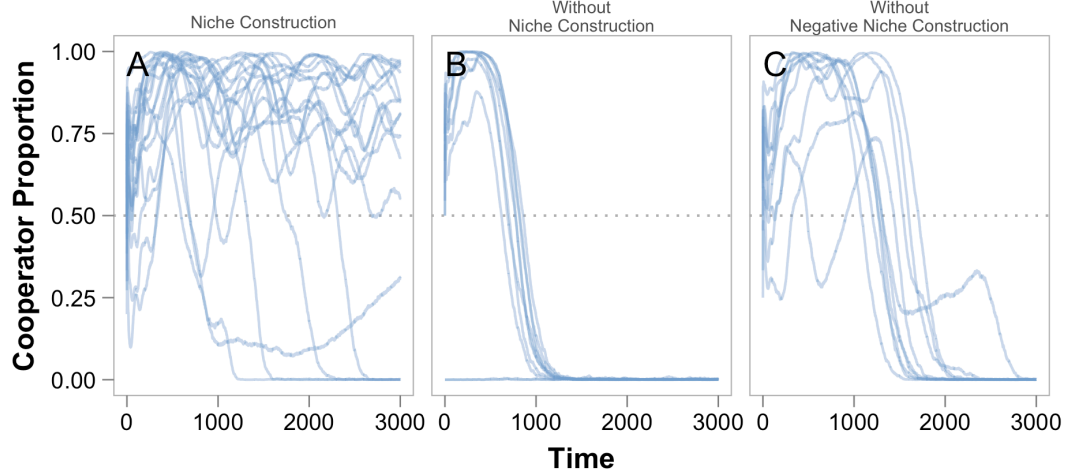
356 **Figure 1**



**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 ( $\epsilon$ , the intensity of niche construction, is zero), cooperation hitchhikes along with adaptations, 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 ( $A = 5$ ).

359 **Figure 4**

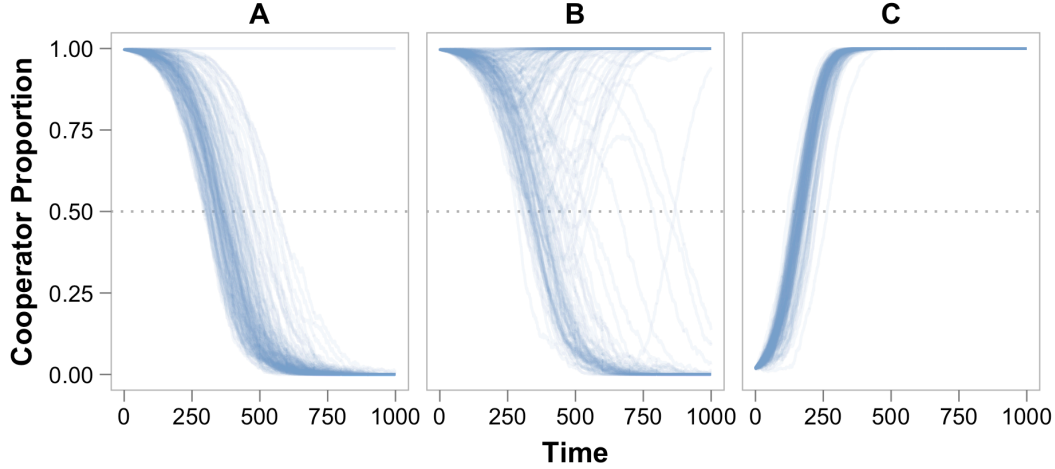


Figure 4: **Niche Construction and Invasion.** Curves trace the proportion of cooperators present in the population for the duration of 160 replicate simulations ( $T = 1000$ ). These experiments examine whether a rare cooperator or defector strategy can invade when initiated at a single patch in the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

360 **Figure 5**

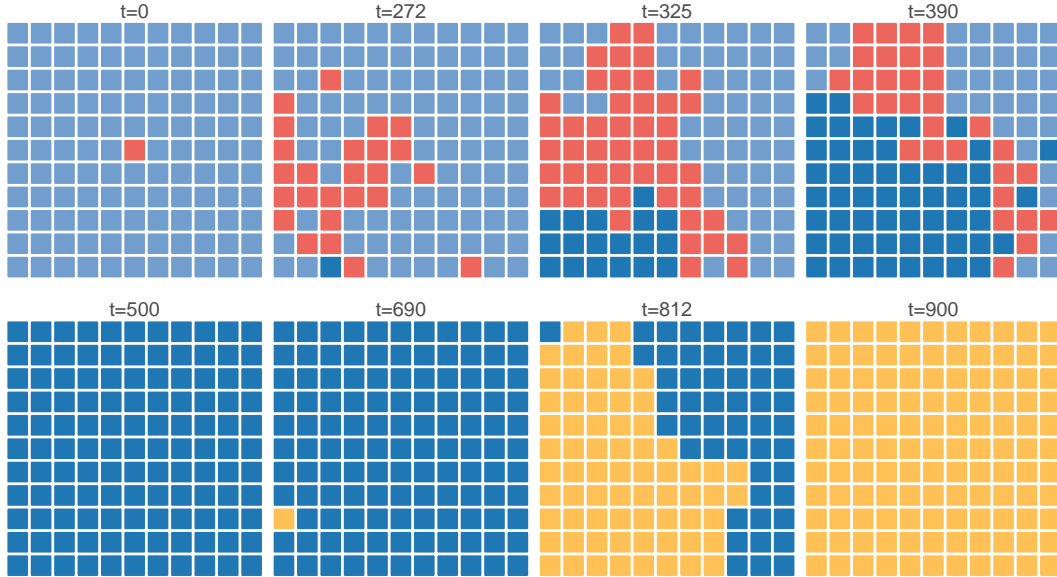


Figure 5: **Defector Invasion Stopped by Cooperator Adaptation.** Here we depict the distribution of dominant types among populations over time for one representative simulation in which isogenic defectors arise. For clarity, mutations occurred at the adaptive loci, but not at the cooperation locus ( $\mu_c = 0$ ) 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 ( $t = 272$ ). However, cooperators in a single population gain an adaptation that give them a fitness advantage over defectors (dark blue, lower left). At  $t = 325$ , 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 ( $t = 390$ ), and eventually fixes in the population ( $t = 500$ ). TODO 690, 812, 900.



361 **Figure 6**

362 **Not All Niches are Created Equally.** (A) TODO: All negative niche  
363 construction (B) TODO: Increased mutation rate

364 Coming soon...

365 **Supplemental Figure 1**

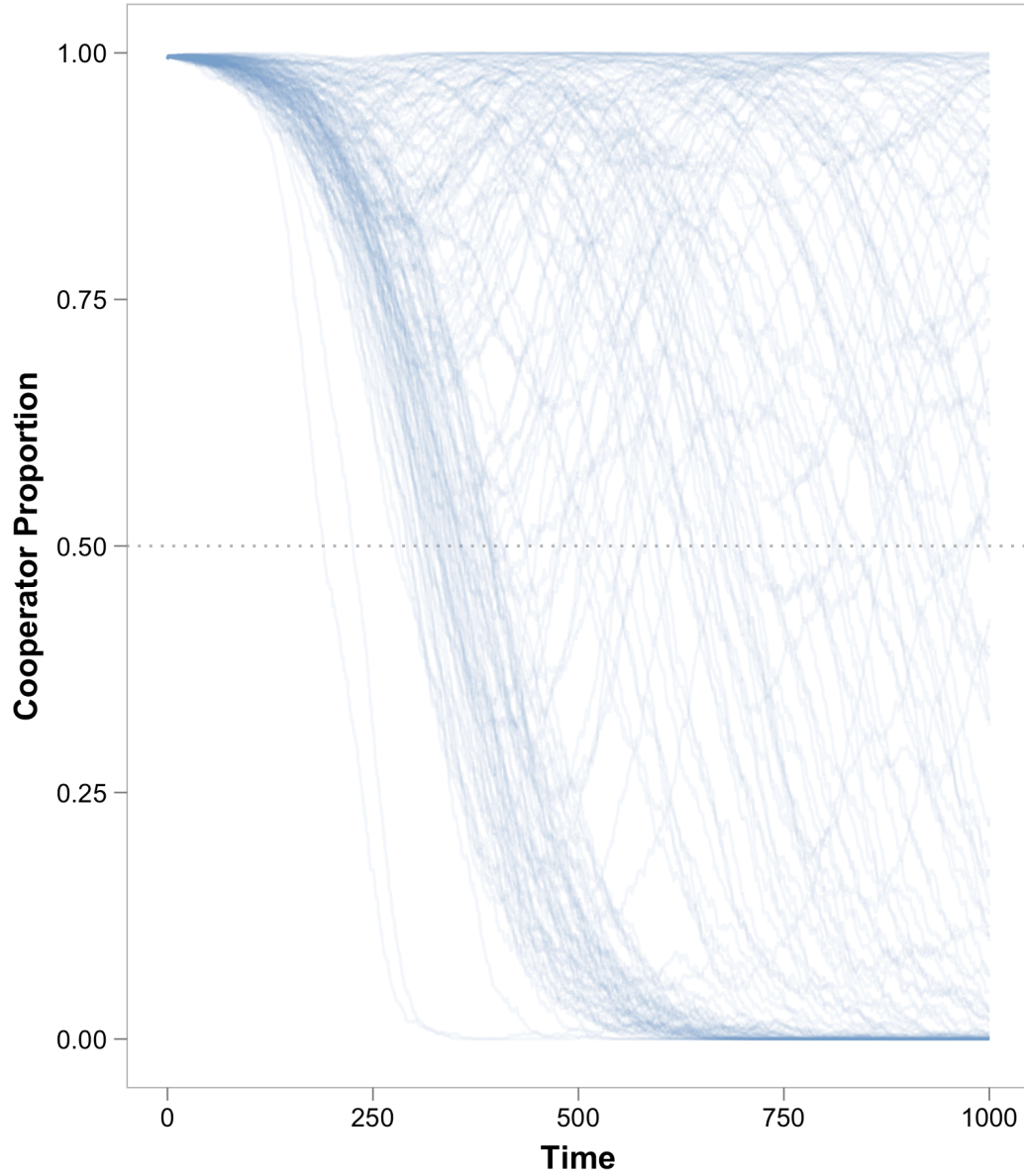


Figure S1: **Defector Invasion with Mutations.** With mutations occurring both at the adaptive loci and the cooperation locus ( $\mu_a = \mu_c = 0.00005$ ), cooperation remains the dominant strategy in 58 replicate simulations. Curves trace the proportion of cooperators present in the population for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

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
$\delta$	Fitness benefit, nonzero alleles	0.3
$\epsilon$	Fitness benefit, sequential alleles	0.00015
$z$	Baseline fitness	1
$S_{min}$	Minimum subpopulation size	800
$S_{max}$	Maximum subpopulation size	2000
$\mu_a$	Mutation rate (adaptation)	$10^{-5}$
$\mu_c$	Mutation rate (cooperation)	$10^{-5}$
$N^2$	Number of patches	625
$m$	Migration rate	0.05
$p_0$	Initial cooperator proportion	0.5
$\sigma$	Survival rate at population initialization	$10^{-5}$
$T$	Number of simulation cycles	3000
$d$	Subpopulation dilution factor	0.1

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