

1 Negative Niche Construction Favors the  
2 Evolution of Cooperation

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## 11 Abstract

12 By benefitting others at a cost to themselves, cooperators face an ever present  
13 threat from defectors—individuals that avail themselves of the cooperative ben-  
14 efit without contributing. A longstanding challenge to evolutionary biology is  
15 to understand the mechanisms that support the many instances of coopera-  
16 tion that nevertheless exist. Hammarlund et al. recently demonstrated that  
17 cooperation can persist by hitchhiking along with beneficial non-social adapta-  
18 tions. Importantly, cooperators play an active role in this process. In spatially-  
19 structured environments, clustered cooperator populations reach greater densi-  
20 ties, which creates more mutational opportunities to gain beneficial non-social  
21 adaptations. Cooperation rises in abundance by association with these adap-  
22 tations. However, once adaptive opportunities have been exhausted, the ride  
23 abruptly ends as cooperators are displaced by adapted defectors. Using an  
24 agent-based model, we demonstrate that the selective feedback that is created  
25 as populations construct their local niches can maintain cooperation indefi-  
26 nitely. Further, we show that cooperator success depends specifically on nega-  
27 tive niche construction. Here, negative niche construction acts as a perpetual  
28 source of adaptive opportunities. As populations adapt, they alter their envi-  
29 ronment in ways that reveal additional opportunities for adaptation. Despite  
30 being independent of niche construction in our model, cooperation feeds this  
31 cycle. We show that by reaching larger densities, populations of cooperators  
32 are better able to adapt to changes in their constructed niche and successfully  
33 respond to the constant threat posed by defectors. We relate these findings to

34 previous studies from the niche construction literature and discuss how this  
35 model could be extended to provide a greater understanding of how coopera-  
36 tion evolves in the complex environments in which it is found.

## 37 Introduction

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

47 Several factors can prevent this *tragedy of the commons* (Hamilton, 1964;  
48 Nowak, 2006; West *et al.*, 2007b). One such factor involves non-random so-  
49 cial interaction, in which cooperators benefit more from the cooperative act  
50 than defectors. This can occur when cooperators are clustered together in  
51 spatially-structured populations (Fletcher and Doebeli, 2009; Nadell *et al.*,  
52 2010; Kuzdzal-Fick *et al.*, 2011) or when cooperators use communication  
53 (Brown and Johnstone, 2001; Darch *et al.*, 2012) or other cues (Sinervo *et*  
54 *al.*, 2006; Gardner and West, 2010; Veelders *et al.*, 2010) to cooperate condi-  
55 tionally with kin. Cooperation can also be bolstered by pleiotropic connections  
56 to personal benefits (Foster *et al.*, 2004; Dandekar *et al.*, 2012) or through as-  
57 sociation with alleles encoding self-benefitting traits (Asfahl *et al.*, 2015). In  
58 the latter case, the associated alleles may provide private benefits that are  
59 completely independent from the public benefits of cooperation. In asexual

60 populations of cooperators and defectors, this sets the stage for an “adaptive  
61 race” in which both types vie for the first highly beneficial adaptation (Waite  
62 and Shou, 2012; Morgan *et al.*, 2012). The tragedy of the commons can be  
63 deferred if a cooperator, by chance, wins the adaptive race.

64 Hammarlund et al. (2015) recently showed that in spatially-structured pop-  
65 ulations, the “Hankshaw effect” can give cooperators a substantial leg up on  
66 defectors in an adaptive race. This advantage is reminiscent of Sissy Han-  
67 kshaw, a fictional character in Tom Robbins’ *Even Cowgirls Get the Blues*,  
68 whose oversized thumbs—which were otherwise an impairment—made her a  
69 prolific hitchhiker. Similarly, cooperation is costly, but it increases local pop-  
70 ulation density. As a result, cooperators are more likely to acquire benefi-  
71 cial mutations. By hitchhiking along with these adaptations, cooperation can  
72 rise in abundance. Nevertheless, this advantage is fleeting. As soon as the  
73 opportunities for adaptation are exhausted, cooperators are once again at a  
74 selective disadvantage against adapted defectors that arise via mutation. How-  
75 ever, Hammarlund et al. (2015) also demonstrated that cooperation can be  
76 maintained when frequent environmental changes produce a steady stream of  
77 new adaptive opportunities. Although organisms typically find themselves in  
78 dynamic environments, the nature and frequency of these changes might not  
79 ensure long-term cooperator survival.

80 However, organisms do more than passively experience changing environments.  
81 Through their activities, their interactions with others, and even their deaths,  
82 organisms constantly modify their environment. This *niche construction* pro-  
83 cess can produce evolutionary feedback loops in which environmental modifi-

84 cation alters selection, which, in turn, alters the distribution of types and their  
85 corresponding influence on the environment (Odling-Smee *et al.*, 2003). The  
86 nature of this feedback can have dramatic evolutionary consequences. One crit-  
87 ical distinction is whether the constructing type is favored in the environment  
88 that it constructs. Under positive niche construction, selection favors the con-  
89 structor, and evolution stagnates as this type fixes. Whereas under negative  
90 niche construction, selection favors a type other than the constructor, which  
91 creates an opportunity for novel adaptation. If the resulting adapted type also  
92 engages in negative niche construction, cycles of construction and adaptation  
93 can ensue, such that populations find themselves continually chasing beneficial  
94 mutations as their adaptive landscape perpetually shifts.

95 Here, we show that the selective feedbacks that result from niche construction  
96 can maintain cooperation indefinitely. We find that it is specifically negative  
97 niche construction that is responsible for this result due to the endless opportu-  
98 nities for adaptation that it produces. These results suggest that cooperators  
99 can ensure their survival by playing an active role in their own evolution.

## 100 **Methods**

101 Building upon Hammarlund *et al.* (2015), we describe an individual-based  
102 model in which cooperators and defectors evolve and compete in a population  
103 of subpopulations (i.e., a metapopulation). Through mutations, individuals  
104 gain adaptations to their environment, which increase reproductive fitness and  
105 allow those lineages to rise in abundance. More successful lineages spread to

106 neighboring subpopulations by migration.

107 In the expanded model here, subpopulations additionally modify their local  
108 environment. As this process occurs, environmental changes feed back to  
109 affect selection. We explore how niche construction affects the evolution of  
110 cooperation; specifically, how cooperative behavior can hitchhike along with  
111 adaptations to modified environments.

## 112 **Model Description**

### 113 **Individual Genotypes and Adaptation**

114 Each individual has a haploid genome with  $L + 1$  loci (see [Table 1](#) for model  
115 parameters and their values). Different alleles at each locus are represented by  
116 different integers. An allele at the *cooperation locus* (locus zero) determines  
117 whether that individual is a cooperator (allele 1), which carries fitness cost  
118  $c$ , or a defector (allele 0). The remaining  $L$  loci are *adaptive loci*, and are  
119 each occupied by 0 or a value from the set  $\{1, 2, \dots, A\}$ . Allele 0 represents  
120 a lack of adaptation, while a non-zero allele represents one of the  $A$  possible  
121 adaptations at that locus.

122 These non-zero alleles signify two types of adaptations, both of which increase  
123 fitness. First, adaptations to the external environment confer a fitness benefit  $\delta$ .  
124 This selective value is the same regardless of which non-zero allele is present  
125 and is not affected by other individuals. We assume  $\delta > c$ , which allows a  
126 minimally adapted cooperator to recoup the cost of cooperation and gain a  
127 fitness advantage.

## 128 Niche Construction and Selective Feedbacks

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

134 In our model, the feedback that results from niche construction takes the form  
135 of density dependent selection, and individuals evolve to better match their  
136 constructed niche. We do not represent this niche explicitly, but rather allow  
137 the allelic composition of the subpopulation to feed back to affect selection.  
138 Specifically, the selective value of non-zero allele  $a$  at adaptive locus  $l$ —and  
139 consequently the fitness of an individual carrying that allele—increases with  
140 the number of individuals in the subpopulation that have allele  $a - 1$  at locus  
141  $l - 1$ . For example, if  $L = 5$  and  $A = 6$ , and allele 4 has fixed at locus 2, then  
142 a genotype with allele 5 at locus 3 is favored. And as allele 5 fixes at locus  
143 3, the niche that this population constructs will favor allele 6 at locus 4 (see  
144 **Box 1**). As a consequence, genotypes with sequentially increasing allelic states  
145 will tend to evolve. We treat both adaptive loci and their non-zero allelic  
146 states as “circular”: the selective value of an allele at locus 1 is affected by the  
147 allelic composition of the subpopulation at locus  $L$ . Similarly, the selective  
148 value of allele 1 at any locus increases with the number of individuals carrying  
149 allele  $A$  at the previous locus. This circularity is represented by the function  
150  $\beta(x, X)$ , which gives the integer that is below an arbitrary value  $x$  in the set



151  $\{1, 2, \dots, X\}$ :

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

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

## 156 **Individual Fitness**

157 Consider a genotype  $g$  with allelic state  $a_{g,l}$  at locus  $l$ ; the fitness of an indi-  
 158 vidual with this genotype is defined as:

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

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

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

162 Thus, an individual's fitness is determined both by adaptations to the exter-

163 nal environment and by adaptations to its constructed environment. **Box 1**  
 164 illustrates the process of adaptation to the constructed environment. While  
 165 cooperation is costly, we assume its effects are independent of the external and  
 166 constructed components of the environment.

## 167 **Subpopulation Growth and the Benefit of Cooperation**

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

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

173 During subpopulation growth, individuals compete through differential repro-  
 174 duction. Each individual's probability of success is determined by its fitness.  
 175 The composition of a subpopulation with size  $P$  and cooperator proportion  $p$   
 176 after growth is multinomial with parameters  $S(p)$  and  $\{\pi_1, \pi_2, \dots, \pi_P\}$ , where  
 177  $\pi_i$  represents the reproductive fitness of individual  $i$  relative to others in the  
 178 subpopulation (using Equation 2).

## 179 **Mutation**

180 For simplicity, we apply mutations after subpopulation growth. Mutations  
181 occur independently at each locus and cause an allelic state change. At the  
182 binary cooperation locus, mutations occur at rate  $\mu_c$ . These mutations flip  
183 the allelic state, causing cooperators to become defectors and vice versa. Mu-  
184 tations occur at rate  $\mu_a$  at each adaptive locus. These mutations replace the  
185 existing allele with a value randomly sampled from the set  $\{0\} \cup \{1, 2, \dots, A\}$ .

## 186 **Migration**

187 Populations are composed of  $N^2$  patches arranged as an  $N \times N$  lattice, where  
188 each patch can support a subpopulation. After mutation, individuals emigrate  
189 to an adjacent patch with probability  $m$ . During each migration event, a single  
190 destination patch is randomly chosen from each source patch's Moore neigh-  
191 borhood, which is composed of the nearest 8 patches on the lattice. Because  
192 the population lattice has boundaries, patches located on the periphery have  
193 smaller neighborhoods.

## 194 **Population Initialization and Simulation**

195 Following Hammarlund et al. (2015), we begin simulations with sparse pop-  
196 ulations. Subpopulations are first seeded at all patches with size  $S(p_0)$  and  
197 cooperator proportion  $p_0$ . The population is then thinned. Each individual  
198 survives this bottleneck with probability  $\sigma$ . Starting from this initial state,  
199 simulations then proceed for  $T$  cycles, where each discrete cycle consists of

subpopulation growth, mutation, migration, and dilution. Dilution reduces each subpopulation to support growth in the next cycle. Each individual remains with probability  $d$ , regardless of its genotype.

## Simulation Source Code and Software Dependencies

The simulation software and configurations for the experiments reported are available online.<sup>1</sup> 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 of subpopulations that are connected by spatially-limited migration. Individuals increase their competitiveness by gaining adaptations. While cooperation does not directly affect the fitness benefits that these adaptations confer, cooperation has indirect effects on the adaptive process. Specifically, cooperation increases subpopulation density. As a result, larger subpopulations of cooperators experience more mutational opportunities. Cooperation can rise in abundance by hitchhiking along with beneficial mutations, which compensate for the cost of cooperation.

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<sup>1</sup>These materials are temporarily available at <https://github.com/briandconnelly/nicheconstruct/> and will be moved to a long-term repository shortly

219 Importantly, subpopulations alter their local environments, which feeds back  
220 to influence selection. Here, we explore how such niche construction affects  
221 the evolution of cooperation.

## 222 Cooperation Persists with Niche Construction

223 Without any opportunity for adaptation ( $L = 0$ ), cooperators are swiftly elim-  
224 inated from populations (Figure 1A). Despite an initial lift in cooperator abun-  
225 dance due to increased productivity, the cost of cooperation becomes disad-  
226 vantageous as migration mixes the initially isolated subpopulations. When  
227 populations can adapt to the external environment ( $L > 0$  and  $\delta > 0$ ), but  
228 niche construction is absent ( $\epsilon = 0$ ), cooperators are maintained only tran-  
229 siently (Figure 1B). Here, larger cooperator subpopulations can more quickly  
230 adapt to their external environment. However, as previously described by  
231 Hammarlund et al. (2015), cooperation is subsequently lost once populations  
232 become fully adapted. This occurs when isogenic defectors (i.e., defectors with  
233 identical adaptive loci) arise via mutation and displace cooperators due to their  
234 selective advantage. However, when niche construction feeds back to influence  
235 selection ( $\epsilon > 0$ ), cooperation persists in the majority of the replicate popula-  
236 tions (Figure 1C). We see in Figure 2A that despite oscillations, cooperation  
237 is maintained at high levels in most of these populations.

## **Fitness Increases Alone do not Support Persisting Cooperation**

Adaptations to both the external environment and the constructed environment contribute to an individual's fitness in this model. Here, we determine whether cooperation is maintained solely due to the larger selective values that result from the contributions of niche construction. We performed simulations in which these contributions were transferred to supplement the benefits conferred by adaptation to the external, non-constructed environment (replacing  $\epsilon = 0.3, \delta = 0.3$  with  $\epsilon = 0, \delta = 0.6$ ). In doing so, we conservatively estimate the selective effects of niche construction. Nevertheless, we find that simply increasing selective values does not enable cooperators to persist (Figure 2B). Niche construction, therefore, plays a decisive role here.

## **Negative Niche Construction is Critical to Cooperator Persistence**

In our model, an adaptation to the constructed environment initiates a new instance of niche construction, leading to sequentially increasing allelic states across the adaptive loci. Under certain conditions, this construction always makes the constructor sub-optimal for the niche it creates (see Box 1). This negative niche construction occurs when the number of adaptive alleles ( $A$ ) does not divide evenly into the number of adaptive loci ( $L$ ). In such a case, any sequence of integers on the circular genome will always contain a break

259 in the sequence; that is, one locus will have an allele that is not one less than  
 260 the allele at the next locus. Given this unavoidable mismatch, any type that  
 261 has fixed will always construct a niche that favors selection for a new type.  
 262 When negative niche construction is removed (by setting  $L = 5$ ,  $A = 5$ ), coop-  
 263 erators are again driven extinct after an initial lift in abundance (Figure 2C).  
 264 These results indicate that the type of niche construction matters. Specifically,  
 265 negative niche construction is crucial for maintaining cooperation.

## 266 **Selective Feedbacks Limit Defector Invasion**

267 The adaptation resulting from selective feedbacks can limit invasion by defec-  
 268 tors, which arise either through migration from neighboring patches or through  
 269 mutation at the cooperation locus. The latter challenge is particularly threat-  
 270 ening, as these isogenic defectors are equally adapted, yet do not incur the  
 271 cost of cooperation. As demonstrated in Figure 3A, isogenic defectors rapidly  
 272 spread when introduced at a single patch in the center of a population of  
 273 cooperators if mutations do not occur. However, when cooperators can gain  
 274 adaptations via mutation, cooperators resist defector invasion in over half of  
 275 the replicate populations (Figure 3B). Figure 4 depicts one such instance. In  
 276 that population, isogenic defectors are seeded at a single patch in an otherwise  
 277 all-cooperator population. These defectors quickly begin to spread. However,  
 278 a neighboring cooperator population gains an adaptation, which increases its  
 279 fitness above that of the defector. This type spreads more quickly, stopping  
 280 the spread of defectors and eventually driving them to extinction. Because

281 this adaption occurs in a cooperator population, cooperation is able to hitch-  
282 hike to safety. Importantly, this new cooperator is favored because of the  
283 niche that its ancestral type—and therefore the defector—constructed. Here,  
284 cooperators can find safety in numbers—because their larger subpopulations  
285 have more mutational opportunities, they are more likely to gain adaptations  
286 that rescue them from invasion. Further, these larger cooperator subpopula-  
287 tions exert greater influence on their niches, which increases selection for an  
288 adapted type. This allows that type to appear and to spread more quickly in  
289 the population. **Figure 3C** shows how quickly an adapted cooperator type can  
290 invade a population of defectors. Importantly, this cooperator type is adapted  
291 to the niche constructed by the defector.

## 292 Discussion

293 Despite their negative effects, deleterious traits can rise in abundance due to  
294 genetic linkage with other traits that are strongly favored by selection (May-  
295 nard Smith and Haigh, 1974). In a process termed the “Hankshaw effect”,  
296 Hammarlund et al. (2015) recently demonstrated that cooperation can ac-  
297 tively prolong its existence by increasing its likelihood of hitchhiking with a  
298 beneficial trait. In that work and here, subpopulations of cooperators grow to a  
299 higher density than those of defectors. These larger cooperator subpopulations  
300 therefore experience more mutations and are consequently more likely to gain  
301 adaptations. While this process does favor cooperation in the short term, it  
302 eventually reaches a dead end: When the opportunities for adaptation are ex-



303 exhausted, and cooperators can no longer hitchhike, they face extinction. Here,  
 304 we have investigated whether niche construction might serve to perpetually  
 305 generate new adaptive opportunities and thus favor cooperation indefinitely.

306 When niche construction occurs, cooperation can indeed persist (Figures 1C  
 307 and 2A). In our model, niche construction introduces additional selective ef-  
 308 fects that influence the evolutionary process, leading to a more pronounced  
 309 Hankshaw effect. However, these fitness benefits alone do not maintain co-  
 310 operation (Figure 2B). Niche construction and the selective feedbacks that it  
 311 produces play a crucial role.

312 We find that it is specifically *negative* niche construction that maintains coop-  
 313 eration (Figure 2C). As cooperator and defector types gain adaptations, they  
 314 alter their environment in ways that favor other types. Thus, negative niche  
 315 construction serves as a perpetual source of adaptation. Here we observe an-  
 316 other facet of the Hankshaw effect: Because subpopulations of cooperators are  
 317 larger, they are better able to respond to the adaptive opportunities that are  
 318 created by negative niche construction. By gaining adaptations more quickly,  
 319 cooperators resist invasion by defectors (Figure 3B). Even in the presence of  
 320 an isogenic defector type, cooperator subpopulations are more likely to pro-  
 321 duce the mutant most adapted to the current niche, which can then displace  
 322 the slower-adapting defectors. These recurring cycles of defector invasion and  
 323 cooperator adaptation underlie the oscillations in cooperator proportion seen  
 324 in Figure 2A. When mutations do not confer these adaptations, cooperators  
 325 lose the adaptive race and are driven to extinction by the defector. This is  
 326 something that we see occur stochastically in Figures 2A and 3B.

## Cooperation as Niche Construction

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

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

When dispersal is limited, competition among kin can undermine cooperation. To separate kin competition from kin selection, Lehmann (2007) developed

350 a model in which a cooperative, niche-constructing behavior only benefitted  
351 future generations. Kin competition was thereby reduced, and cooperation  
352 instead benefitted descendants. This work highlights an important aspect of  
353 niche construction: Often, the rate of selective feedback from niche construc-  
354 tion is different from the rate at which populations grow.

## 355 **Evolution at Multiple Timescales**

356 In our work, the niche is modeled implicitly by the composition of the sub-  
357 population. Any changes in the subpopulation, therefore, produce immediate  
358 effects on the constructed environment and the resulting selective feedbacks.  
359 However, timescales in our model could be de-coupled in two ways. First, co-  
360 operators modify their niche by enabling their subpopulation to reach larger  
361 density (Equation 4). These increased subpopulation sizes play a critical role  
362 by effectively increasing the rate of evolution in these subpopulations. Because  
363 of the importance of this process, it would be very informative to explore how  
364 sensitive our results are to the rate at which cooperators increase subpopulation  
365 sizes and the rate at which this benefit decays in the absence of cooperators.  
366 Similarly, our results could be substantially affected by alterations in the rate  
367 at which the constructed environment changes in response to changes in the  
368 subpopulation.

369 Other studies, while not focused on cooperation, have similarly shown that the  
370 timescales at which niche construction feedbacks occur can strongly influence  
371 evolutionary outcomes (Laland *et al.*, 1996, 1999). This perspective is likely to

372 be crucial for understanding the evolution of cooperative behaviors like the pro-  
 373 duction of public goods. In these instances, environmental changes are likely  
 374 to occur on different timescales than growth, which can have profound effects.  
 375 For example, a multitude of factors, including protein durability (Brown and  
 376 Taddei, 2007; Kümmerli and Brown, 2010), diffusion (Allison, 2005; Driscoll  
 377 and Pepper, 2010), and resource availability (Zhang and Rainey, 2013; Ghoul  
 378 *et al.*, 2014) influence both the rate and the degree to which public goods alter  
 379 the environment. While Lehmann (2007) showed that cooperation was favored  
 380 when selective feedbacks act over longer timescales, niche construction may in  
 381 fact hinder cooperation when selection is more quickly altered. For example,  
 382 when public goods accumulate in the environment, cooperators must decrease  
 383 production to remain competitive (Kümmerli and Brown, 2010; Dumas and  
 384 Kümmerli, 2012). This favors cooperation that occurs facultatively, perhaps  
 385 by sensing the abiotic (Bernier *et al.*, 2011; Koestler and Waters, 2014) or  
 386 biotic environment (Brown and Johnstone, 2001; Darch *et al.*, 2012). In order  
 387 to study how regulatory traits such as these evolve, we could instead represent  
 388 the niche explicitly, allowing it to have its own dynamics.

## 389 **Cooperation and Niche Construction in Host-Symbiont** 390 **Co-Evolution**

391 In many biological systems, the environments modified by organisms are other  
 392 organisms. In these instances, the “niche” becomes a biological entity with its  
 393 own evolutionary process. A logical extension to our model, would be to treat

the environment as an organism. Such a model could be used to explore the evolution of cooperation in host-symbiont systems, where cooperation among symbionts affects host fitness. As the host population changes, either in response to symbiont cooperation or other factors, so too does selection on their symbiont populations. In our model, each patch could become hosts with their own genotypes, and death and reproduction at the host level could be defined in ways that are sensitive to both host and symbiont genotypes. Here, evolutionary outcomes depend greatly on the degree of shared interest between the host and symbiont.

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

More generally, it was recently argued that incorporating the effects of niche construction is critical for improving our understanding of viral evolution (Hamblin *et al.*, 2014) and evolution in co-infecting parasites (Hafer and Milin-

418 ski, 2015). Incorporating host dynamics, co-evolution, and the feedbacks that  
419 they produce is likely to be equally important for gaining a greater understand-  
420 ing of how cooperative behaviors evolve in these host-symbiont settings.

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## Box 1: Description of niche construction in our model

See [Figure B1](#)

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

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

**(C) Niche Construction and Adaptation.** The evolutionary transition shown in Part B is indicated in the dashed box. Here, we depict entire sub-

454 populations fixed for a genotype using a single instance of that genotype. Simi-  
 455 larly, an arrow represents niche construction and adaptation to the constructed  
 456 environment. We start with a case in which there are five alleles ( $A = 5$ ). Sub-  
 457 populations begin with the non-adapted genotype  $[0,0,0,0,0]$ , shown on the far  
 458 left. A non-zero allele is introduced via mutation, which represents an adapta-  
 459 tion to external aspects of the environment. Here, allele 1 arises and fixes at  
 460 locus 1. The remainder of this figure focuses on adaptation to the constructed  
 461 aspects of the environment. This genotype has a “mismatch” (shown by the  
 462 red sector), because  $E_{[1,0,0,0,0]}$  favors  $[1,2,0,0,0]$ . Assuming allele 2 arises at the  
 463 second locus, it will be selected, creating a “match” at the first and second  
 464 loci (green sector). Now there is a mismatch between the second and third  
 465 loci in the resulting environment, which a new round of mutation and selection  
 466 corrects, and so on. The green sector grows as the red sector shifts clockwise.  
 467 When the population reaches  $[1,2,3,4,5]$ , it constructs  $E_{[1,2,3,4,5]}$ . Here, since  
 468 allele 1 follows allele 5, there is no longer a mismatch, so no further adaptation  
 469 occurs.

470 **(D) Negative Niche Construction.** A different case emerges when the  
 471 number of alleles does not evenly divide into the number of loci. Here, we  
 472 change the number of alleles to six ( $A = 6$ ). As shown on the far left, we  
 473 begin with a subpopulation fixed for genotype  $[1,2,3,4,5]$ . This genotype has a  
 474 mismatch, because the niche constructed by allele 5 favors allele 6 (not 1) at the  
 475 next locus (locus 1). A mutant with genotype  $[6,2,3,4,5]$  has a fitness advantage  
 476 and can fix in  $E_{[1,2,3,4,5]}$ . However, as this type constructs  $E_{[6,2,3,4,5]}$ , a new  
 477 mismatch appears. In this instance of negative niche construction, adapting



478 to correct one mismatch generates a new mismatch. This system can never  
479 escape its mismatches—the red sector just shifts clockwise around the genome  
480 perpetually.

## 481 Figures

482 **Figure 1**

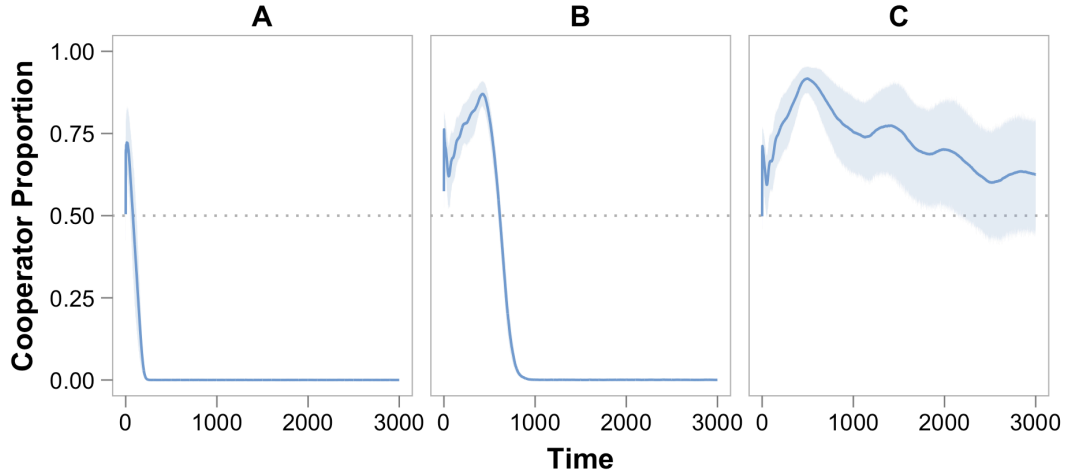
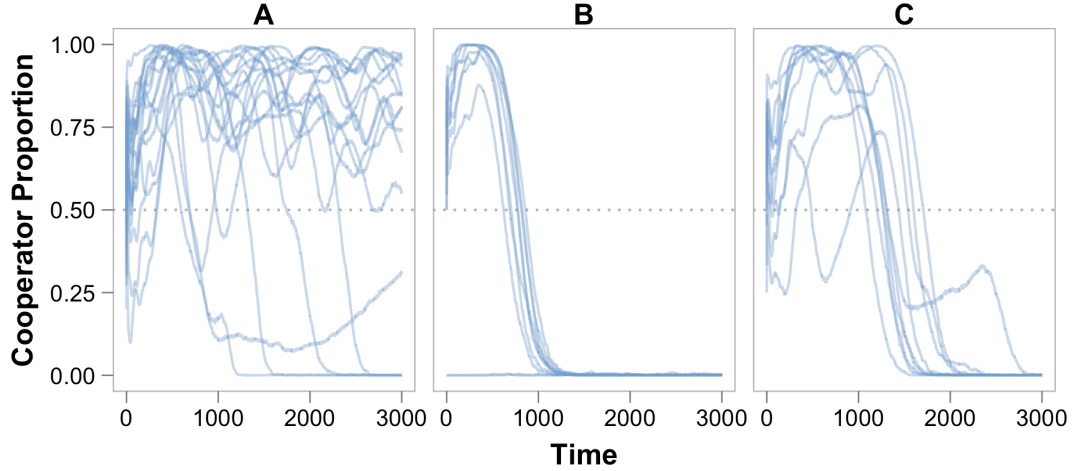
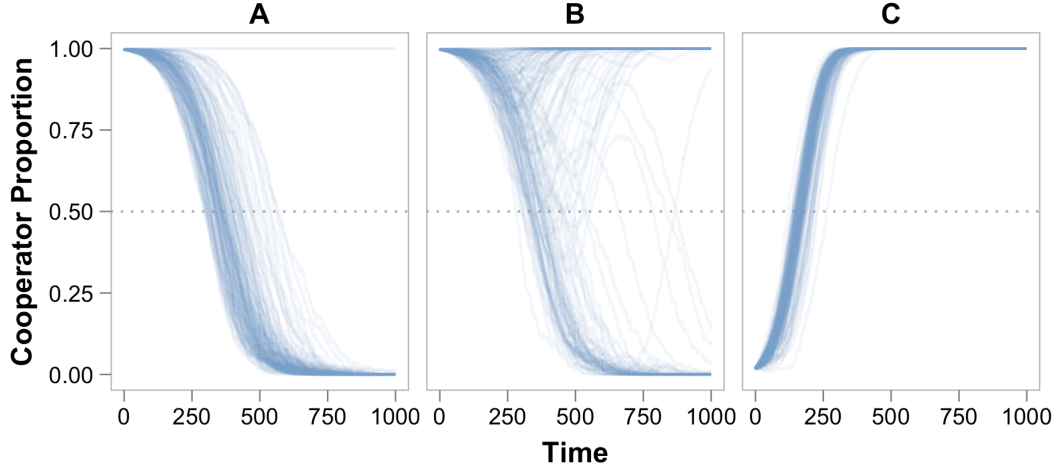


Figure 1: **Adaptation and the Evolution of Cooperation.** Curves show the average cooperator proportion among replicate populations for the duration of simulations, and shaded areas indicate 95% confidence intervals. **(A)** Without any opportunity to adapt ( $L = 0$ ), cooperation is quickly lost. **(B)** When adaptation can occur ( $L = 5$ ,  $\delta = 0.3$ ), but niche construction does not affect selection ( $\epsilon = 0$ ), cooperators rise in abundance by hitchhiking along with adaptations to the external environment. Nevertheless, this effect is transient, and cooperators eventually become extinct. **(C)** Niche construction ( $\epsilon = 0.00015$ ) enables cooperation to be maintained indefinitely. In the majority of populations, cooperation remained the dominant strategy. The trajectories of individual populations are shown in Figure 2A. Parameter values not given here are listed in [Table 1](#).

483 **Figure 2**



**Figure 2: Niche Construction and the Evolution of Cooperation.** The proportion of cooperators present in each replicate population is shown for the duration of simulations. **(A)** Despite some oscillations, cooperation dominates in 13 of 18 populations when niche construction affects selection. **(B)** When the selective effects of niche construction are transferred to supplement the benefits conferred by adaptation to the external, non-constructed environment, cooperators are driven to extinction by defectors (replacing  $\epsilon = 0.3$ ,  $\delta = 0.3$  with  $\epsilon = 0$ ,  $\delta = 0.6$ ). Note that cooperation was not present after initialization in one replicate population. **(C)** Cooperators are also driven to extinction without negative niche construction ( $A = 5$ ).

**Figure 3**

**Figure 3: Niche Construction and Invasion.** Curves trace the proportion of cooperators present in each replicate population for the duration of simulations ( $T = 1000$ ). In each simulation, a rare type was initiated at a single patch in the center of the population lattice ( $N^2 = 121$ ). Unless otherwise noted, mutations are disabled in these ecological simulations to highlight the dynamics of invasion ( $\mu_a = 0, \mu_c = 0$ ). **(A)** When cooperators and defectors are isogenic (i.e., both types have stress alleles [1,2,3,4,5]), 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, negative niche construction creates adaptive opportunities that enable cooperators to resist invasion by isogenic defectors. When adaptive mutations occur ( $\mu_a = 0.00005$ ), cooperation remained dominant in 91 of 160 populations. Results from simulations where mutations also occurred at the cooperation locus are shown in Figure S1. **(C)** In fact, a cooperator (stress alleles [6,2,3,4,5], see Box 1) that is adapted to the niche constructed by the defectors can swiftly displace defectors.

485 **Figure 4**

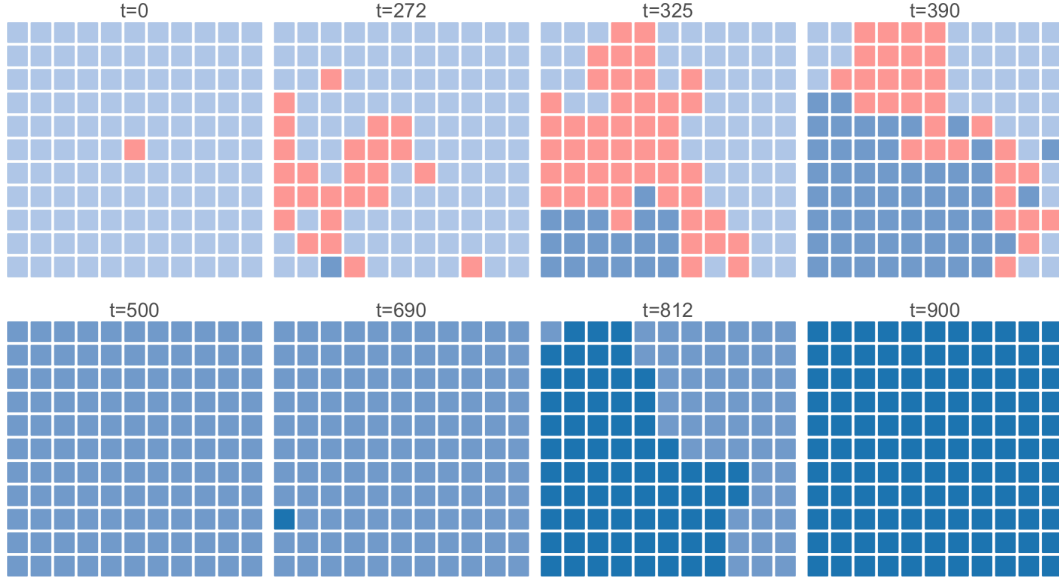


Figure 4: **Cooperator Adaptation Prevents Defector Invasion.** Here we depict the distribution of dominant types among subpopulations over time for one representative simulation in which isogenic defectors arise. To highlight the effects of adaptation, mutations did not occur at the cooperation locus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

486 **Box 1 Figures**

487 **Figure B1**

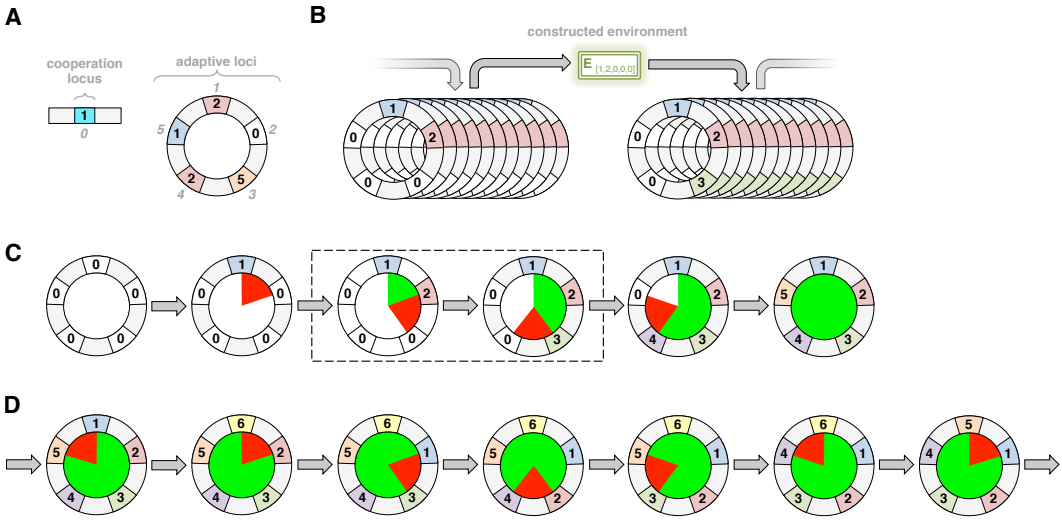


Figure B1: Figure for Box 1

<sup>488</sup> **Supplemental Figures**

<sup>489</sup> **Figure S1**



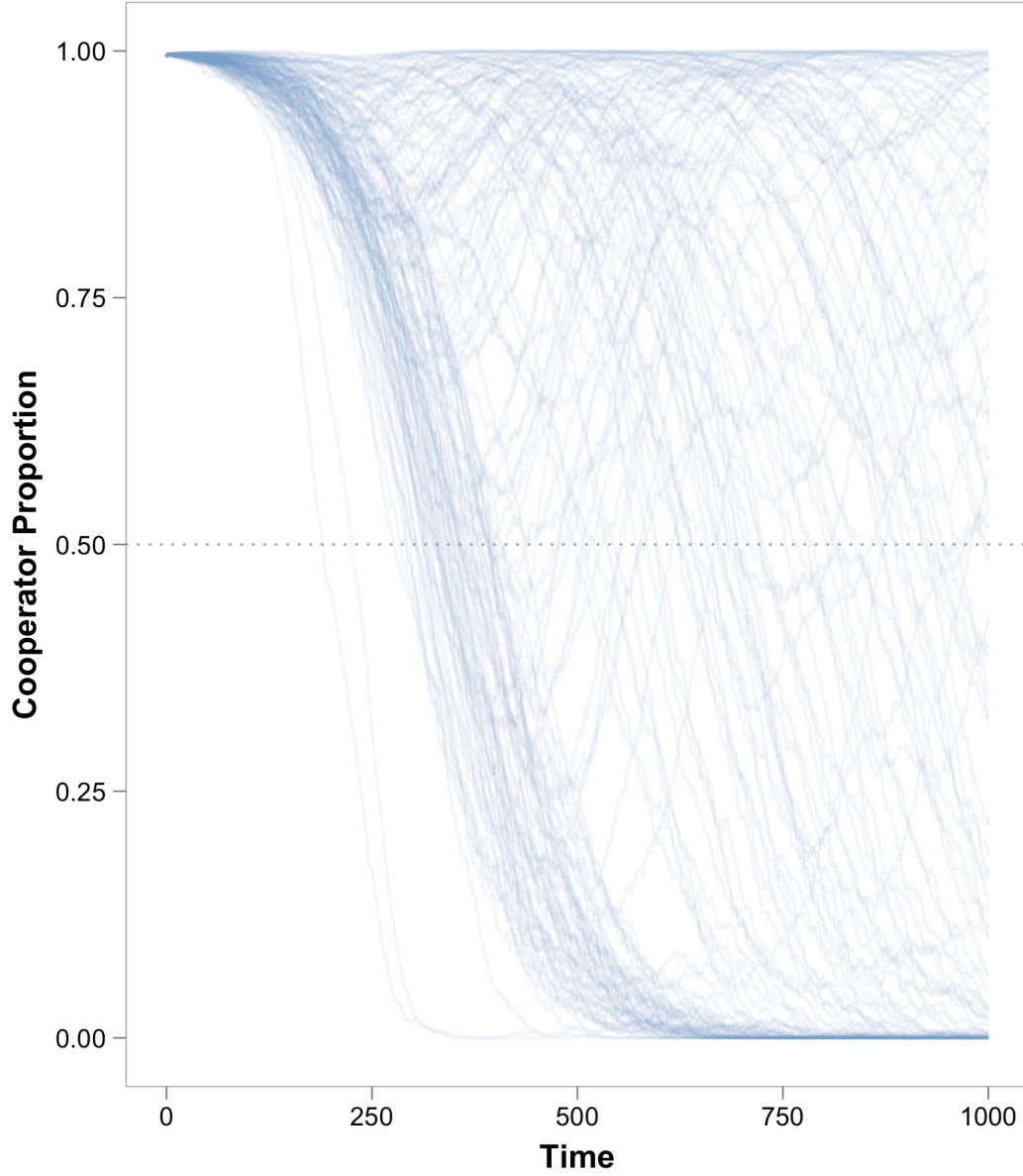


Figure S1: **Defector Invasion with Mutations.** The proportion of co-operators present in each replicate population is shown for the duration of simulations ( $T = 1000$ ). When mutations occur both at the adaptive loci and the cooperation locus ( $\mu_a = \mu_c = 0.00005$ ), cooperation remains dominant in 58 of 160 replicate populations.

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, adaptation to external environment	0.3
$\epsilon$	Fitness benefit, adaptation to constructed environment	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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