

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 rise  
41 in abundance. Nevertheless, this advantage is fleeting. As soon as the oppor-  
42 tunities for adaptation are exhausted, cooperators are once again at a selective  
43 disadvantage against equally-adapted defectors that arise via mutation. How-  
44 ever, Hammarlund et al. (2015) also demonstrated that cooperation can be  
45 maintained when frequent environmental changes produce a steady stream of  
46 new adaptive opportunities. Although organisms typically find themselves in  
47 dynamic environments, the nature and frequency of these changes might not  
48 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 of  
54 types and their corresponding influence on the environment (Odling-Smee *et*  
55 *al.*, 2003). The nature of this feedback can have dramatic evolutionary conse-  
56 quences. One critical distinction is whether the constructing type is favored in  
57 the resulting environment. Under positive niche construction, selection favors  
58 the constructor, and evolution stagnates as this type fixes. Under negative  
59 niche construction, selection favors a type other than the constructor. In this  
60 latter case, populations find themselves continually chasing beneficial muta-  
61 tions as their adaptive landscape perpetually shifts.

62 Here, we show that the selective feedbacks that result from niche construction  
63 can maintain cooperation indefinitely. We find that it is specifically negative

64 niche construction that is responsible for this result because of the endless  
65 opportunities for adaptation that it produces. These results indicate that  
66 cooperators can ensure their survival when they play an active role in their  
67 own evolution.

## 68 **Methods**

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

75 In this expanded model, subpopulations additionally modify their local envi-  
76 ronment. As this process occurs, environmental changes feed back to affect  
77 selection. We explore how niche construction affects this process hitchhiking  
78 along with adaptations, and whether cooperation can be maintained by selec-  
79 tive feedbacks.

## 80 **Model Description**

### 81 **Individual Genotypes and Adaptation**

82 Each individual has a haploid genome with  $L + 1$  loci (see [Table 1](#) for model  
83 parameters and their values). Different alleles at each locus are represented by

different integers. A binary allele at the first locus (here, locus zero) determines whether that individual is a cooperator (1), which carries fitness cost  $c$ , or a defector (0). Cooperation is independent from adaptation to the environment. The remaining  $L$  loci are *adaptive loci*, and are each occupied by 0 or a value from the set  $\{1, 2, \dots, A\}$ . Allele 0 represents a neutral allele, while a non-zero allele represents one of the  $A$  possible adaptations at that locus.

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

## Niche Construction and Selective Feedbacks

Individual fitness is also affected by aspects of the local environment that are affected by organisms. We represent this constructed “niche” implicitly based on the specific allelic states present in the subpopulation. As allelic states change, subpopulations alter their environment, creating a unique niche. As described below, the specific alleles that are present at each locus matter.

In our model, niche construction takes the form of density dependent selection, and individuals evolve to better match their niche by an additional form of adaptation. Non-zero alleles that are more common will improve fitness by a larger selective value (beyond  $\delta$ ). Specifically, the selective value of non-zero

106 allele  $a$  at adaptive locus  $l$ , and consequently the fitness of an individual carry-  
 107 ing that allele, increases with the number of individuals in the subpopulation  
 108 that have allele  $a - 1$  at locus  $l - 1$ . For example, when  $L = 5$  and  $A = 6$ , and  
 109 allele 4 has fixed at locus 2, a genotype with allele 5 at locus 3 will be favored.  
 110 And once allele 5 has fixed at locus 3, the niche that this population creates  
 111 will favor allele 6 at locus 4. As a consequence, genotypes with sequentially  
 112 increasing allelic states will tend to evolve. We treat both adaptive loci and  
 113 allelic states as “circular”: the selective value of an allele at locus 1 is affected  
 114 by the allelic composition of the subpopulation at locus  $L$ . Similarly, the se-  
 115 lective value of allele 1 at any locus increases with the number of individuals  
 116 carrying allele  $A$  at the previous locus. This circularity is represented by the  
 117 function  $\beta(x, X)$ , which gives the integer that is below an arbitrary value  $x$  in  
 118 the set  $\{1, 2, \dots, X\}$ :

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

119 Here,  $\text{mod}_X(x)$  is the integer remainder when dividing  $x$  by  $X$ . Using this  
 120 function, the selective value of allele  $a$  at adaptive locus  $l$  is increased by  $\epsilon$  for  
 121 each individual in the subpopulation that has allele  $\beta(a, A)$  at locus  $\beta(l, L)$ .  
 122 Thus,  $\epsilon$  specifies the intensity of selection due to niche construction.  
 123 Consider a genotype  $g$  with allelic state  $a_{g,l}$  at locus  $l$ ; the fitness of an indi-  
 124 vidual with this genotype is defined as:

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

where  $z$  is a baseline fitness,  $n(a, l)$  is the number of individuals in the subpopulation with allele  $a$  at locus  $l$ , and  $I(a)$  indicates whether a given 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 by 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 of a subpopulation with size  $P$  and cooperator proportion  $p$  after growth is multinomial with parameters  $S(p)$  and  $\{\pi_1, \pi_2, \dots, \pi_P\}$ , where  $\pi_i$  represents individual  $i$ 's reproductive fitness relative to others in the subpopulation.

## Mutation

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

## Migration

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



## 159 Population Initialization and Simulation

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

## 168 Simulation Source Code and Software Dependencies

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

## 174 Results

175 Using the model described in the previous section, we perform simulations  
176 that follow the evolution of cooperation in a population consisting of subpopu-  
177 lations that are connected by spatially-limited migration. Individuals increase

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<sup>1</sup>These materials will be made public prior to publication.

178 their competitiveness by gaining adaptations. While cooperation does not di-  
 179 rectly affect the fitness benefits that these adaptations confer, cooperation has  
 180 indirect effects on the adaptive process. Specifically, cooperation increases  
 181 subpopulation density. As a result, larger subpopulations of cooperators ex-  
 182 perience more mutational opportunities. Cooperation can rise in abundance  
 183 by hitchhiking along with beneficial mutations, which compensate for the cost  
 184 of cooperation. Importantly, subpopulations alter their local environments,  
 185 which feeds back to influence selection. Here, we explore how niche construc-  
 186 tion affects the evolution of cooperation.

## 187 **Cooperation Persists with Niche Construction**

188 Without any opportunity for adaptation ( $L = 0$ ), cooperators are swiftly elim-  
 189 inated in competition with defectors (Figure 2A). Despite an initial lift in  
 190 cooperator abundance due to increased productivity, the cost of cooperation  
 191 becomes disadvantageous as migration mixes the initially isolated subpopula-  
 192 tions. When populations can adapt to the external environment ( $L = 5$ ), but  
 193 niche construction is absent ( $\epsilon = 0$ ), cooperators are maintained only tran-  
 194 siently (Figure 2B). Here, larger cooperator subpopulations can more quickly  
 195 adapt to their external environment. However, as previously described by  
 196 Hammarlund et al. (2015), cooperation is subsequently lost once populations  
 197 become fully adapted to their environment. This occurs when isogenic de-  
 198 fectors (i.e., defectors with identical adaptive loci) arise via mutation and  
 199 displace cooperators due to their selective advantage. However, when niche

200 construction feeds back to influence selection ( $\epsilon > 0$ ), cooperation persists in  
201 the majority of the replicate populations (Figure 2C). We see in Figure 3A that  
202 despite oscillations in the proportion of cooperators, cooperation is maintained  
203 at high levels in these populations.

## 204 **Fitness Increases Alone do not Support Persisting Coop-** 205 **eration**

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

## 218 **Negative Niche Construction is Critical to Cooperator** 219 **Persistence**

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

## 232 **Selective Feedbacks Limit Defector Invasion**

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

tors can gain adaptations via mutation, cooperators resist defector invasion in over half of the replicate populations (Figure 4B). Figure 5 depicts one such instance. In that population, defectors quickly began to spread. However, an adaptation arose in a neighboring cooperator population. This type spreads more quickly, stopping the spread of defectors and eventually driving them to extinction. Because this adaptation occurred in a cooperator population, cooperation was able to hitchhike to safety. Importantly, this adaptation was favored because of the niche that its ancestor created. Here, cooperators can find safety in numbers—because the effective mutation rate is increased in their larger populations, they are more likely to gain adaptations that rescue them from invasion. Further, the larger number of cooperators more strongly construct their niche, and thus more strongly favoring an adapted type. This allows that type to spread more quickly in the population. Figure 4C shows how quickly an adapted cooperator type can invade a population of defectors.

## Discussion

Despite their negative effects, deleterious traits can rise in abundance due to genetic linkage with other traits that are strongly favored by selection (Maynard Smith and Haigh, 1974). In a process termed the “Hankshaw effect”, Hammarlund et al. (2015) recently demonstrated that cooperation can actively prolong its existence by increasing its likelihood of hitchhiking with a beneficial trait. In that work and in ours, cooperation enables populations to grow to a higher density. Because of this, these cooperator populations experienced

262 more mutations and were therefore more likely to gain adaptations. While this  
263 process does favor cooperation in the short term, it eventually reaches a dead  
264 end; when the opportunities for adaptation are exhausted and cooperators can  
265 no longer hitchhike, they face extinction. In our current model, we have con-  
266 sidered whether niche construction might serve to perpetually generate new  
267 adaptive opportunities and thus favor cooperation indefinitely.

268 When niche construction occurs, cooperation can indeed persist (Figures 2C  
269 and 3A). In our model, niche construction introduces additional selective ef-  
270 fects that influence the evolutionary process, leading to a more pronounced  
271 Hankshaw effect. However, simply raising the fitness benefits conferred by  
272 adaptations does not prolong cooperation (Figure 3B). This indicates that  
273 niche construction does play a crucial role.

274 Further, we find that it is specifically *negative* niche construction that main-  
275 tains cooperation (Figure 3C). As cooperator and defector types gain adapta-  
276 tions in our model, they change the environment in a way that favors other  
277 types. In this way, negative niche construction serves as a perpetual source of  
278 adaptation. Here we observe another facet of the Hankshaw effect: because  
279 populations of cooperators are larger, they are better able to respond to the  
280 adaptive opportunities that result from negative niche construction. These op-  
281 portunities can allow cooperators to resist invasion by defectors (Figure 4B).  
282 Although defectors initially have an advantage by saving on the cost of co-  
283 operation, populations of cooperators can quickly gain an advantage because  
284 they are bigger. It is these recurring cycles of invasion and adaptation that  
285 underlie the oscillations in cooperator populations that we see in Figure 3A.

286 When an adaptation to the constructed environment does not occur in the co-  
287 operator background before the defector dominates, the defector can drive the  
288 cooperator to extinction. This is something that we see occur stochastically  
289 in Figures 3A and 4B.

## 290 Cooperation as Niche Construction

291 In our model, cooperation is orthogonal to niche construction, which allows  
292 us to focus on hitchhiking. However, by increasing the size of the local patch,  
293 this form of cooperation can itself be seen as form of niche construction. Co-  
294 operative benefits often take similar forms in natural systems. For example,  
295 bacteria produce a host of extracellular products that scavenge soluble iron  
296 (Griffin *et al.*, 2004), digest large proteins (Diggle *et al.*, 2007; Darch *et al.*,  
297 2012), and reduce the risk of predation (Cosson *et al.*, 2002). While many  
298 studies have focused on how the environment affects the evolution of these co-  
299 operative traits, relatively few have addressed how the environmental changes  
300 created by public goods feed back to influence evolution.

301 Perhaps most similar to our work, Van Dyken and Wade (2012) demonstrated  
302 that when two negative niche constructing, cooperative behaviors co-evolve,  
303 selection can increasingly favor these traits, which were disfavored when alone.  
304 In that model, “reciprocal niche construction” occurred when the negative feed-  
305 back created by one strategy positively influenced selection on the other, cre-  
306 ating a perpetual cycle that maintained both forms of cooperation. Arguably,  
307 this can be viewed as an instance of hitchhiking: the currently-maladaptive

308 form of cooperation is maintained by association with the adaptive form.  
309 When dispersal is limited, competition among kin can undermine cooperation.  
310 To separate kin competition from kin selection, Lehmann (2007) developed  
311 a model in which the selective feedbacks produced by a cooperative, niche-  
312 constructing behavior only benefitted future generations. Kin competition  
313 thereby was reduced, and cooperation instead benefitted descendants. This  
314 work highlights an important aspect of niche construction—often, the rates at  
315 which niche construction affects selection are different from the rates at which  
316 populations grow.

## 317 **Evolution at Multiple Timescales**

318 In our work, the niche is modeled implicitly by the composition of the popula-  
319 tion. Any changes, therefore, in the population produce immediate effects on  
320 the constructed environment and the resulting feedbacks. However, timescales  
321 in our model could be de-coupled in two ways. First, cooperators modify their  
322 niche by enabling their population to reach larger density (Equation 4). These  
323 increased population sizes play a critical role in this work by effectively increas-  
324 ing the rate of evolution in these populations. Because of the importance of  
325 this process, it would be very informative to explore how sensitive our results  
326 are to changes in how long the increases in population size are upheld. Simi-  
327 larly, changes in the timescale at which the niche at a patch change also have  
328 potential to dramatically change our results. Not only would it affect the se-  
329 lective values of alleles as the population changed, but it could also influence



330 whether or not populations were able to evolve adapted types and if, so, how  
331 well those adapted types can propagate through the population to address the  
332 threat of a defector.

333 Other studies, while not focused on cooperation, have similarly shown that the  
334 timescales at which niche construction feedbacks occur can strongly influence  
335 evolutionary outcomes (Laland *et al.*, 1996, 1999). This perspective is likely to  
336 be crucial for understanding the evolution of cooperative behaviors like the pro-  
337 duction of public goods. In these instances, environmental changes are likely  
338 to occur on different timescales than growth, which can have profound effects.  
339 For example, a multitude of factors including protein durability (Brown and  
340 Taddei, 2007; Kümmerli and Brown, 2010), diffusion (Allison, 2005; Driscoll  
341 and Pepper, 2010), and resource availability (Zhang and Rainey, 2013; Ghoul  
342 *et al.*, 2014) influence both the rate and the degree to which public goods alter  
343 the environment. While Lehmann (2007) showed that cooperation was favored  
344 when selective feedbacks act over longer timescales, niche construction may in  
345 fact hinder cooperation when selection is more quickly altered. For example,  
346 when public goods accumulate in the environment, cooperators must decrease  
347 production to remain competitive (Kümmerli and Brown, 2010; Dumas and  
348 Kümmerli, 2012). This favors cooperation that occurs facultatively, perhaps  
349 by sensing the abiotic (Bernier *et al.*, 2011; Koestler and Waters, 2014) or  
350 biotic environment (Brown and Johnstone, 2001; Darch *et al.*, 2012). To allow  
351 our model to address how traits such as these evolve, we would first need to  
352 de-couple the niche from the composition of the population by representing  
353 the niche explicitly.

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

As niche construction becomes more independent, it develops its own state and dynamics. A logical next step, then, could be to treat the environment as a biological entity itself, which could 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. Future models should explicitly capture the environment as a biological entity to explore the rich possibilities that these systems might offer.

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). Similarly to what we have shown in this work, these antagonistic, negative niche constructing behaviors might actually work to maintain these infections. If this is the case, however, perhaps the case could be made for developing treatments that target the selective feedback loop that is created by niche construction. If these populations do indeed perpetually benefit from adaptations that are created by niche construction, as we have shown, case could perhaps be made for developing treatments that target the selective feedback loop that provides adaptive opportunities in these spatial environments. While the idea of removing negative selective feedbacks and supporting stability may seem counterintuitive, if it makes the population more susceptible, then perhaps pairing such a treatment with ones in which mutants

are introduced (e.g., Rumbaugh et al. (2009)), could significantly improve host fitness. Expanding models such as ours to address the additional dynamics present in host-symbiont systems such as these could be quite productive.

Or conversely, cooperative light production by *A. fischeri* is vital for the survival of its host, the Hawaiian bobtail squid (Ruby, 1996). While our current model and that of Van Dyken and Wade (2012) have showed that negative niche construction can play a decisive role in the evolution of cooperation, this instance of positive niche construction is a textbook example of cooperation and mutualism. Therefore, a greater understanding of the additional feedbacks created in symbioses such as these could be gained from modeling. Similar to our model, these host-symbiont systems likely have a many other traits that are orthogonal to cooperation. Perhaps combinations of certain types of behaviors are important for maintaining cooperation, similar to what was shown by Van Dyken and Wade (2012).

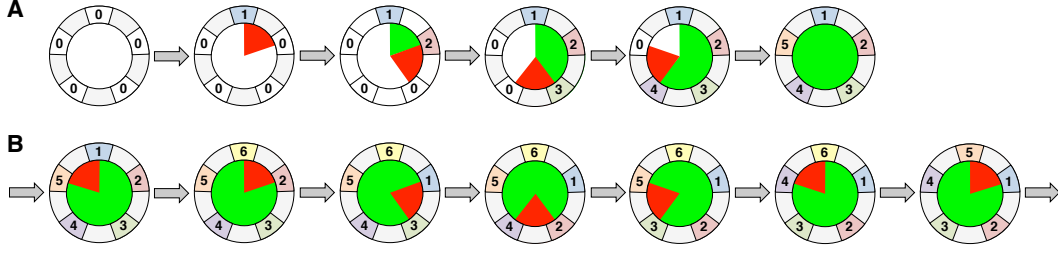
It was recently argued that incorporating the effects of niche construction is critical for improving our understanding of viral evolution (Hamblin *et al.*, 2014) and evolution in co-infecting parasites (Hafer and Milinski, 2015). Incorporating host dynamics, co-evolution, and the feedbacks that they produce into models is likely to be equally important for gaining an understanding of how cooperative behaviors, both positive and negative, evolve in these host-symbiont settings.

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## 406 **Figures**

### 407 **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.

408 **Figure 2**

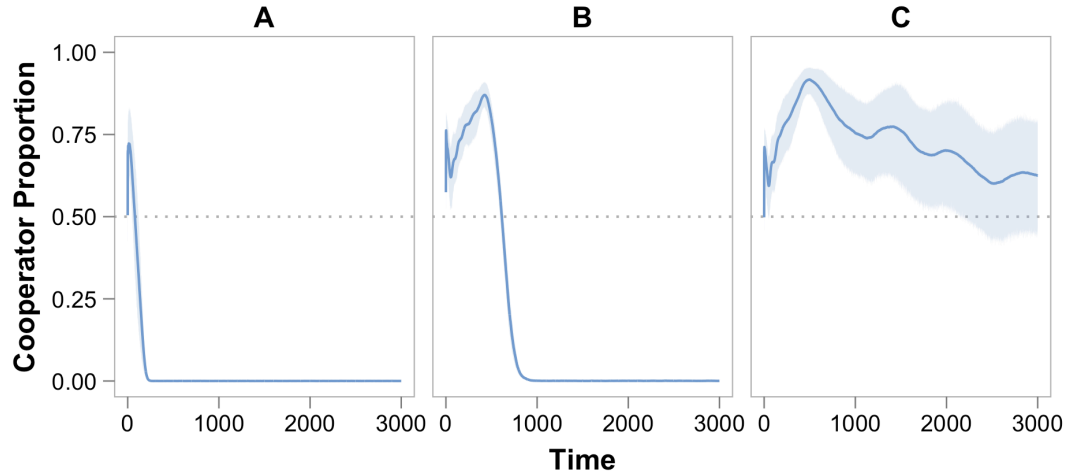
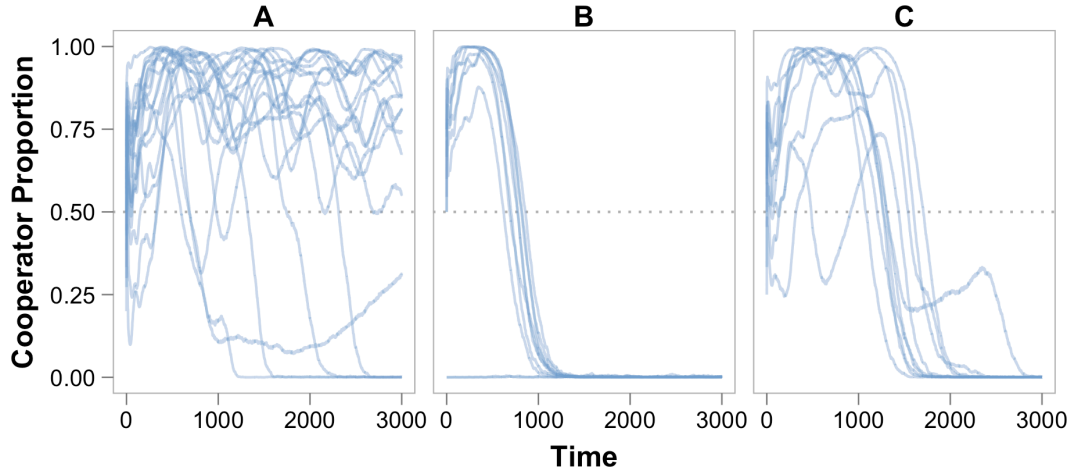


Figure 2: **Adaptation, Hitchhiking, and the Evolution of Cooperation.** Curves show the average cooperator proportion among replicate populations for the duration of simulations, and shaded areas indicate 95% confidence intervals. Unless otherwise noted, parameter values are listed in [Table 1](#). **(A)** Without any opportunity to adapt ( $L = 0$ ), cooperation is quickly lost. **(B)** When adaptation can occur ( $L = 5$ ), 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 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 3A.

409 **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, cooperation dominates in 13 of 18 populations when niche construction affects selection. **(B)** When the selective effects of niche construction ( $\epsilon$ ) are removed, and the selective benefit of adaptation to the external environment ( $\delta$ ) is increased to compensate, cooperators are driven to extinction by isogenic defectors that arise by mutation ( $\epsilon = 0$ ,  $\delta = 0.6$ ). Note that cooperation was not present after initialization in one replicate population. **(C)** Cooperators are also driven to extinction without negative niche construction ( $A = 5$ ).



410 **Figure 4**

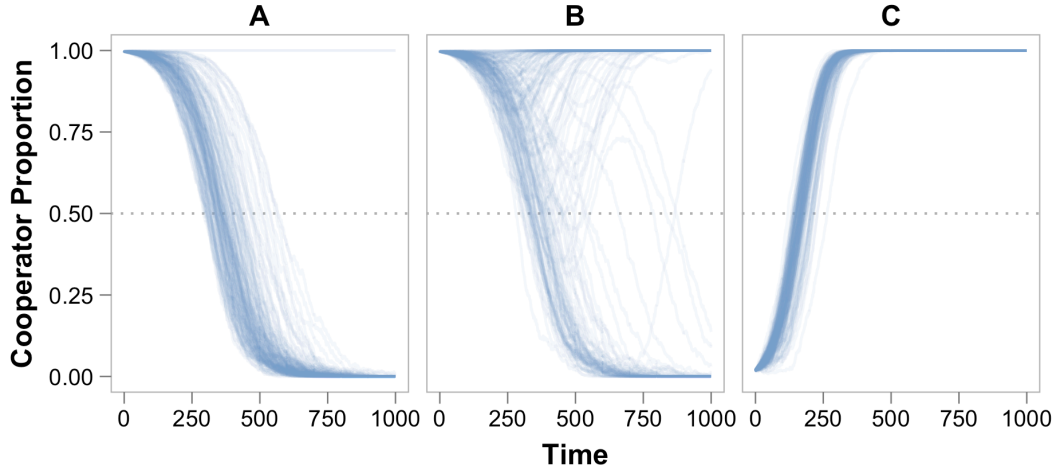


Figure 4: **Niche Construction and Invasion.** Curves trace the proportion of cooperators present in each replicate population for the duration of simulations ( $T = 1000$ ). In each simulation, a rare type was initiated at a single patch in the center of the population lattice ( $N^2 = 121$ ). Unless 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. Here, cooperation remained the dominant in 91 of 160 populations ( $\mu_a = 0.00005$ ). Results from simulations where mutations also occurred at the cooperation locus are shown in Figure S1. **(C)** In fact, an adapted cooperator type (stress alleles [6,2,3,4,5], see Figure 1) can swiftly displace defectors when isogenic defectors cannot arise or adapt via mutation.

<sup>411</sup> **Figure 5**

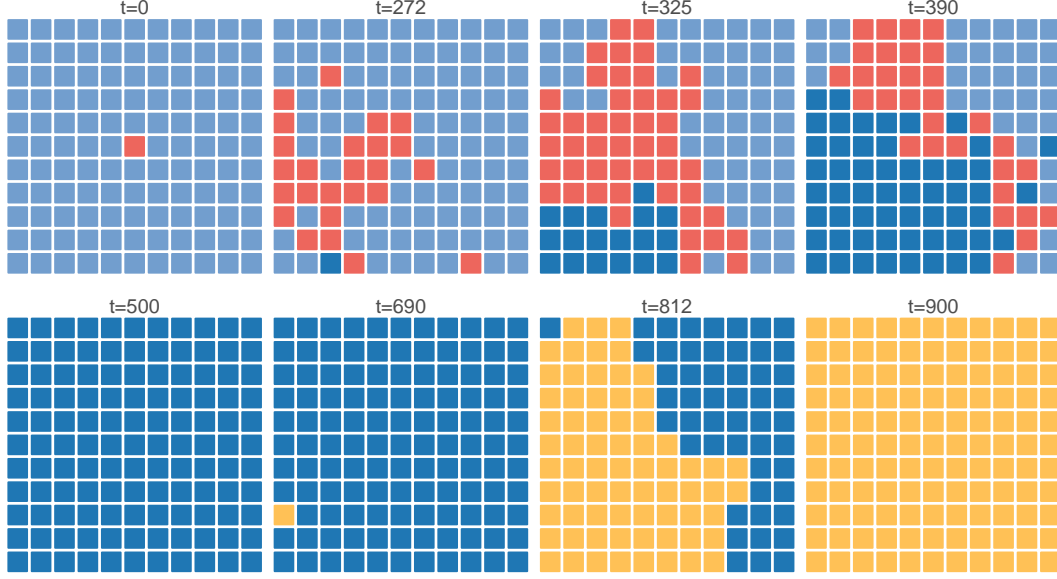


Figure 5: **Cooperator Adaptation Prevents Defector Invasion.** Here we depict the distribution of dominant types among subpopulations over time for one representative simulation in which isogenic defectors arise. To highlight the effects of adaptation, mutations did not occur at the cooperation locus ( $\mu_c = 0$ ). At time  $t = 0$  (upper left panel), a single isogenic defector 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 one population gain an adaptation that gives them a fitness advantage over defectors (second panel, dark blue, lower left). At  $t = 325$ , defectors continue to invade cooperator populations. However, the adapted cooperator type spreads more quickly due to its fitness advantage, invading both defector populations and ancestral cooperator populations ( $t = 390$ ), 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 (orange). 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.

<sup>412</sup> **Supplemental Figure 1**

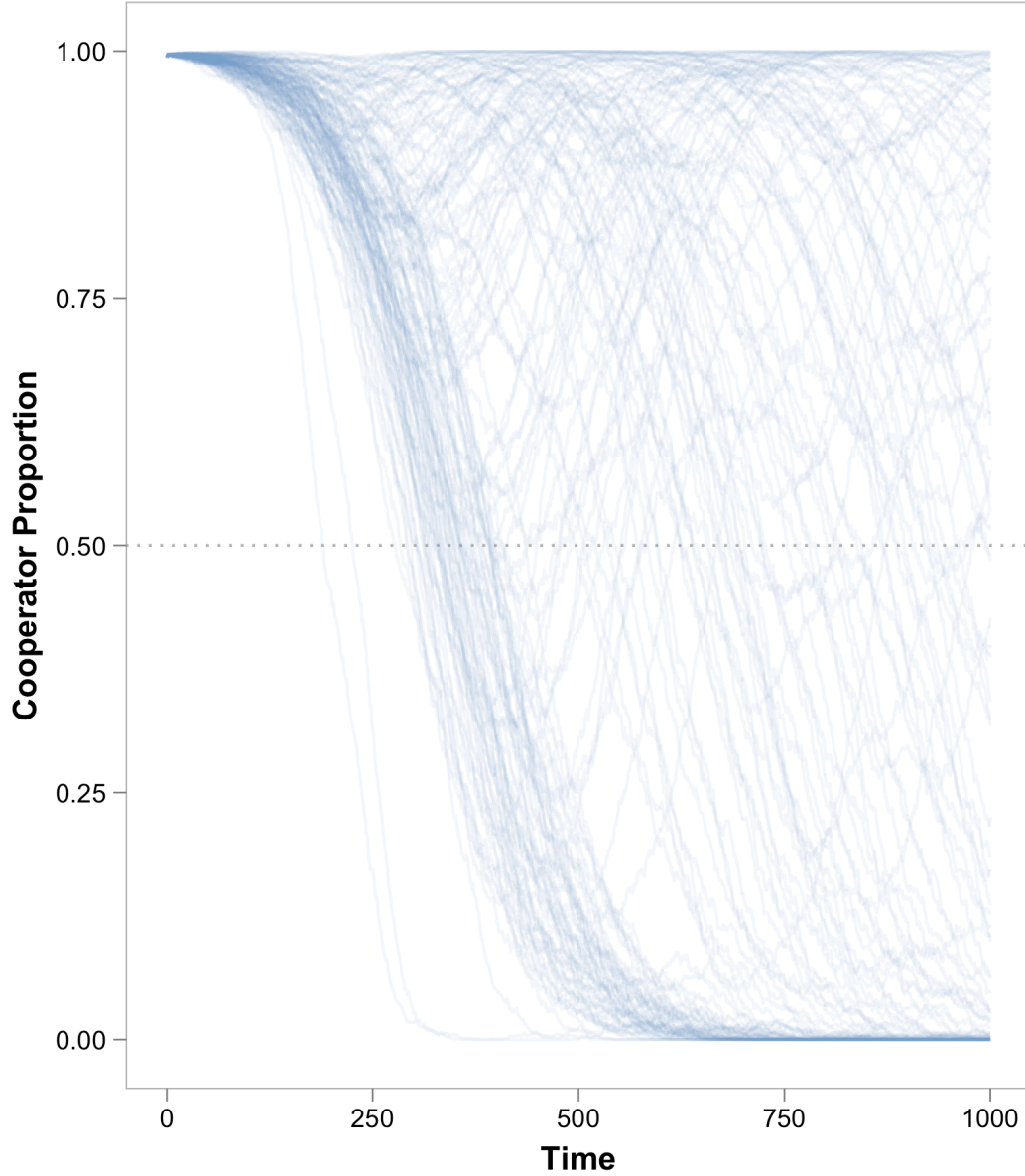


Figure S1: **Defector Invasion with Mutations.** The proportion of co-operators present in each replicate population is shown for the duration of simulations ( $T = 1000$ ). When mutations occur both at the adaptive loci and the cooperation locus ( $\mu_a = \mu_c = 0.00005$ ), cooperation remains dominant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