

TODO title

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Abstract

Through their interactions, their activities, and even their mere presence, organisms change the environment for themselves and others. This “niche construction” process becomes particularly interesting when it creates evolutionary feedback, whereby selective pressures are altered in response to environmental change. Here we consider how niche construction influences the evolution of cooperation, which has been a long-standing challenge to evolutionary theory. We simulate populations of individuals that cooperatively produce a public good that permits increased growth in a stressful environment and investigate how local- and global-scale niche construction affects the ability of these populations to resist invasion by non-producing cheats. We find that niche construction profoundly impacts the evolution of cooperation by creating new opportunities for adaptation. Cooperators are able to escape subversion by cheats as long as niche construction clears these paths of adaptation. This work provides a crucial step towards understanding how evolution occurs in complex environments like those found in nature.

20 Introduction

21 Cooperative behaviors are common across all branches of the tree of life. In-
22 sects divide labor within their colonies, plants and soil bacteria exchange es-
23 sential nutrients, birds care for others' young, and the trillions of cells in the
24 human body restrain their growth and coordinate to provide vital functions.
25 Each instance of cooperation presents an evolutionary challenge: How can in-
26 dividuals that sacrifice their own well-being to help others avoid subversion by
27 those that do not? Over time, we would expect these *defectors* to rise in abun-
28 dance at the expense of others, eventually driving cooperators—and perhaps
29 the entire population—to extinction.

30 Several factors can defer this potential *tragedy of the commons* (Hamilton,
31 1964; Hardin, 1968; Nowak, 2006; West *et al.*, 2007b). For example, coopera-
32 tors must benefit more from the cooperative act than others. This can occur
33 when cooperators are clustered together in spatially-structured populations
34 (Fletcher and Doebeli, 2009; Nadell *et al.*, 2010; Kuzdzal-Fick *et al.*, 2011) or
35 when cooperators use communication (Brown and Johnstone, 2001; Darch *et*
36 *al.*, 2012) or other cues (Sinervo *et al.*, 2006; Gardner and West, 2010; Veelders
37 *et al.*, 2010) to cooperate conditionally with kin. Interestingly, cooperation can
38 also be bolstered by genetic linkage with self-benefitting traits (Foster *et al.*,
39 2004; Dandekar *et al.*, 2012; Asfahl *et al.*, 2015), setting the stage for an “adap-
40 tive race” in which cooperators and defectors vie for the first highly-beneficial
41 non-social adaptation (Waite and Shou, 2012; Morgan *et al.*, 2012).

42 Hankshaw and Kerr (2015) recently showed that in spatially-structured popu-

lations, cooperators can gain a substantial leg up on defectors in an adaptive race when the cooperative behavior increases local population density, thus increasing the likelihood of acquiring beneficial non-social mutations. By hitchhiking along with these adaptations, the cooperative trait can rapidly rise in abundance. Nevertheless, this advantage is fleeting. As soon as the opportunities for adaptation are exhausted, cooperators are once again at a disadvantage against defectors, particularly those that arise from within via mutations that disable cooperation. However, Hankshaw and Kerr (2015) demonstrated that cooperation can be maintained indefinitely when frequent environmental changes produce a steady stream of non-social adaptive opportunities. Although organisms typically find themselves in dynamic environments, change might not occur at a rate that provides sufficient adaptive opportunities to ensure long-term cooperator survival.

Here, we build upon the model presented in Hankshaw and Kerr (2015) to explore whether the selective feedbacks produced as populations modify their environment can act as an additional source of adaptive opportunities. As previously described, this model follows the evolution of cooperation in a metapopulation of populations connected by spatially-limited migration. Cooperators produce a public good that increases the local carrying capacity. Through mutation, individuals gain non-social adaptations that confer fitness benefits. These benefits are large enough so that an adapted cooperator is more fit than a less-adapted defector (note that for simplicity, we refer to all non-cooperators as “defectors”, regardless of their origin). Because of their larger sizes, cooperator populations are more likely to acquire these adaptations. However, coop-

erator populations remain susceptible to invasion by adapted defectors either immigrate from a nearby populaion or arise via social mutation. In our expanded version of this model, populations alter their local environment based on the the presence of different non-social adaptations. Frequency-dependent selection on these adaptations creates an eco-evolutionary feedback that increasingly favors the adaptations present in each population. We focus on two aspects in which the production of public goods is affected by these selective feedbacks. First, the creation of unique niches may diminish the ability of both cooperators and defectors to invade neighboring patches due to maladaptation. However, because cooperator populations are larger, the greater number of emigrants that they produce will allow them to “export” their niche at a higher rate than defectors. As a result, cooperators may be able to expand more quickly. Second, larger cooperator populations will experience more mutations, which better enables these populations to adapt to changing environments. If niche construction produces continual change, can the resulting adaptive opportunities maintain cooperation?

By their very nature, public goods benefit populations by making their environment more hospitable. For example, bacteria produce extracellular products that find soluble iron (Griffin *et al.*, 2004), digest large proteins (Diggle *et al.*, 2007; Darch *et al.*, 2012), and reduce the risk of predation (Cosson *et al.*, 2002), among many others (West *et al.*, 2007a). While many studies have explored how the environment affects the evolution of cooperative bahviors, relatively few have examined how those behaviors affect the environment and the resulting change in evolutionary trajectories. Lehmann (2007) demonstrated analyti-

91 cally that when niche construction act benefits future generations, cooperation
92 is favored due to reduced competition among kin. When rate-benefitting and
93 yield-benefitting altruistic acts co-evolve, Van Dyken and Wade (2012) showed
94 that “reciprocal niche construction”, where the selective feedbacks produced
95 by one act benefitted the other, can lead to increased selection for both traits.
96 While these studies have focused on the niche constructing effects of cooper-
97 ation, we instead focus our attention here on how niche construction enables
98 cooperators to escape defection by hitchhiking along with non-social traits.

99 **Materials and Methods**

100 We build upon the model described in Hankshaw and Kerr (2015), in which co-
101 operators and defectors compete and evolve in a spatially-structured metapop-
102 ulation of populations. Each of these populations grows to carrying capacity,
103 mutates, and migrates to neighboring patches. During this process, popula-
104 tions adapt to their local environments. In our extended model, we allow
105 the presence of these individuals to modify their local environment, and these
106 modifications feed back to affect selection.

107 **Model Description**

108 Our simulated environment consists of N^2 patches arranged as an $N \times N$
109 lattice (see [Table 1](#) for model parameters and their values), where each patch
110 supports a population of zero or more individuals. Each individual in the

111 population has a genotype, which is an ordered list of $L + 1$ integers (loci).
 112 At the first locus, a binary allele determines whether or not that individual
 113 is a cooperator. Individuals with allelic state 1 at this locus are cooperators,
 114 carrying a cost c , while individuals with allelic state 0 are defectors. The
 115 remaining L loci are *stress loci*, and are each occupied by a 0 or an integer
 116 from the set $A = \{1, \dots, a_{max}\}$, where a_{max} is the number of potential alleles.
 117 These alleles represent adaptations to the environment, and the number of loci
 118 determines the number of adaptations that are possible. All non-zero alleles
 119 confer fitness benefit δ . When $\delta \geq c$, an adapted cooperator recoups the cost
 120 of cooperation.

121 Organisms also influence their environment, which can feed back to influence
 122 selection. We model this as a form of frequency dependent selection. Specif-
 123 ically, the selective value of stress allele a at locus l increases with the pro-
 124 portion of the population that has allele $a - 1$ (modulo a_{max}) at locus $l - 1$
 125 (and the first stress locus is affected by the last). The slope of this increase
 126 is ϵ , which specifies the intensity of niche construction. As a consequence of
 127 this form of frequency dependence, genotypes with sequentially increasing al-
 128 lelic states will tend to evolve. Because mutations are random, as described
 129 later, each population will evolve sequences that start with different allelic
 130 states. These different sequences represent the unique niches constructed by
 131 populations. Under this model, the fitness of an individual with genotype g in
 132 population P is:

$$W_g = z + ca_{g,1} + \delta \sum_{l=2}^{L+1} I_A(a_{g,l}) + \epsilon \sum_{l=2}^{L+1} n(a_{g,l}) \quad (1)$$

133 where z is a baseline fitness, c is the cost of the cooperative allele, $a_{g,l}$ represents
 134 the allelic state of genotype g at locus l , L is the number of stress loci, and
 135 $\gamma(j)$ is the genotype of individual j . The function I_A indicates whether allelic
 136 state y is in A (i.e., it is non-zero). The function $n(a_{g,l})$ gives the number of
 137 individuals in the population with allelic state at the previous locus equal to
 138 one less than that at the focal locus $a_{g,l}$, or:

$$n(a_{g,l}) = \sum_{h=1}^N I_{a_{g,l}}(1 + a_{h,l-1}(\text{mod } a_{\max})) \quad (2)$$

139 Here, $I_x(y)$ indicates whether the allelic state y matches allelic state x (1) or
 140 not (0).

141 Cooperators produce a public good that is equally accessible to all members
 142 of the population. This public good increases the carrying capacity at that
 143 patch, allowing the population to reach greater density. This benefit increases
 144 linearly with the proportion of cooperators. Thus, if p is the proportion of
 145 cooperators in a population at the beginning of a growth cycle, then that
 146 population reaches the following size during the growth phase:

$$S(p) = S_{\min} + p(S_{\max} - S_{\min}) \quad (3)$$

147 The function $S(p)$ reflects the benefit of public good production. A population

148 composed entirely of defectors reaches size S_{min} , while one composed entirely
 149 of cooperators reaches size S_{max} (with $S_{max} \geq S_{min}$). During growth, indi-
 150 viduals compete for inclusion in the resulting population. The composition of
 151 population P with cooperator proportion p after growth is multinomial with
 152 parameters and $S(p)$ and $\{\pi_1, \pi_2, \dots, \pi_{|P|}\}$, where:

$$\pi_i = \frac{W_{\gamma(i)}}{\sum_{j \in P} W_{\gamma(j)}} \quad (4)$$

153 Here, $\gamma(i)$ is the genotype of individual i , and $W_{\gamma(i)}$ is its fitness (see Equation
 154 1). π_i therefore reflects that an individual's ability to persist is proportional
 155 to its fitness relative to others'.

156 For simplicity, we apply mutations after population growth. Mutations occur
 157 independently at each locus and cause the allelic state to change. At the binary
 158 cooperation locus, mutations occur at rate μ_c . These mutations flip the allelic
 159 state, causing cooperators to become defectors and vice versa. Mutations
 160 occur at each stress locus at rate μ_s , and cause a new allelic state to be chosen
 161 at random from the set $\{0\} \cup A$. Therefore, the probability that genotype g
 162 mutates into genotype g' is given by:

$$\tau_{g \rightarrow g'} = \mu_c^{H_c(g, g')} (1 - \mu_c)^{\{1 - H_c(g, g')\}} \mu_s^{H_s(g, g')} (1 - \mu_s)^{\{L - H_s(g, g')\}} \quad (5)$$

163 where $H_c(g, g')$ and $H_s(g, g')$ are the Hamming distances between genotypes
 164 g and g' at the cooperation locus and stress loci, respectively. The Hamming
 165 distance is the number of loci at which allelic states differ (Hamming, 1950).

166 Because we define no inherent relationship among alleles, each of the $a_{max} + 1$
167 allelic states is equally likely to arise via mutation at a given locus.

168 After mutation, individuals emigrate to an adjacent patch at rate m . The
169 destination patch is randomly chosen with uniform probability from the source
170 patch's Moore neighborhood, which is composed of the nearest 8 patches on the
171 lattice. Because the metapopulation lattice has boundaries, patches located
172 on an edge have smaller neighborhoods.

173 Metapopulations are initiated in a state that follows the onset of an environ-
174 mental stress. First, populations are seeded at all patches with cooperator
175 proportion p_0 and grown to density $S(p_0)$. Stress is then introduced, which
176 subjects the population to a bottleneck. For each individual, the probability of
177 survival is μ_t , which represents the likelihood that a mutation occurs that con-
178 fers tolerance. Survivors are chosen by binomial sampling. Because individuals
179 have not yet adapted to this new stress, the allelic state of each individual's
180 genotype is set to 0 at each stress locus ($\forall i \in P, l \in \{2, \dots, L+1\} : a_{\gamma(i),l} = 0$).
181 Following initialization, simulations are run for T cycles, where each discrete
182 cycle consists of growth, mutation, and migration. At the end of each cycle,
183 populations are thinned to allow for growth in the next cycle. The individuals
184 that remain are chosen by binomial sampling, where each individual persists
185 with probability d , regardless of allelic state.

186 Source Code and Software Environment

187 The simulation software and configurations for the experiments reported are
188 available online (Us, 2015). Simulations used Python 3.4.0, NumPy 1.9.1,
189 Pandas 0.15.2 (McKinney, 2010), NetworkX 1.9.1 (Hagberg *et al.*, 2008). Data
190 analyses were performed with R 3.1.3 (R Core Team, 2015).

191 Results

192 results...

193 Discussion

- 194 • summary of results
- 195 • similarities/differences from previous work
 - 196 – Schwilk and Kerr (2002)
 - 197 – 10.1073/pnas.0812644106
- 198 • public goods as niche construction
- 199 • future QS or other environmental sensing
- 200 • Facultative cooperation
 - 201 – Rodrigues (2012)
 - 202 – Dumas and Kümmerli (2010)
 - 203 – Kümmerli and Brown (2010)

- 204 – Darch/Diggle
- 205 – QS?
- 206 – Environmental Sensing?

207 Niche construction and selective feedbacks Niche construction and other social
208 interactions

209 **Primacy/Recency**

210 In our model, alterations to the environment were immediately echoed by
211 changes in selection. However, decoupling the timescales on which these pro-
212 cesses occur can have substantial effects (Laland *et al.*, 1996). By integrating
213 past allelic states into Equation 1, we can begin to explore how the cumulative
214 effects of niche construction affect the creation of non-social adaptive oppor-
215 tunities and the benefits that they offer cooperation. Here, how these past
216 allelic states are integrated will play an important role. For example, when
217 the effects of earlier generations are weighted more heavily, the influence of
218 migration may be diminished. While this will reduce the threat of emigration
219 by defectors, cooperator populations will also be less effective at exporting
220 their niche.

221 **Cooperative Niche Construction**

222 While our focus for this work has been on the eco-evolutionary feedbacks cre-
223 ated by non-social traits, it would also be interesting to explore how this

224 system is affected by the timescale at which carrying capacity at a given patch
 225 is increased by public goods. In natural settings, a multitude of factors includ-
 226 ing protein durability (Brown, 2007), diffusion (???), and resource availability
 227 (Zhang and Rainey, 2013; Ghoul *et al.*, 2014) influence both the rate and
 228 the degree to which public goods alter the environment (and thereby selec-
 229 tion). Lehmann (2007) demonstrated that a cooperative, niche constructing
 230 behavior can be favored when it only affected selection for future generations,
 231 thus reducing the potential for competition among contemporary kin. The
 232 evolutionary inertia that this creates, however, may ultimately work against
 233 cooperators. When public good accumulates in the environment, cooperators
 234 must reduce their investment in production to remain competitive (Kümmerli
 235 and Brown, 2010).

236 **Host-Symbiont**

237 In many instances of cooperation, the environment is itself a biological entity,
 238 which can produce additional evolutionary feedbacks. As the host population
 239 changes, so too will selection on their symbiont populations. Here, evolution-
 240 ary outcomes depend greatly on the degree of shared interest between the host
 241 and symbiont. For example, the cooperative production of virulence factors by
 242 the human pathogen *P. aeruginosa* in lung infections is harmful to those with
 243 cystic fibrosis (Harrison, 2007). Conversely, cooperative light production by *A.*
 244 *fisheri* is vital for the survival of its host, the Hawaiian bobtail squid (Ruby,
 245 1996). It was recently argued that incorporating the effects of niche construc-

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

Acknowledgments

- TODO: Organizers?

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260 **Figures**

Table 1: Model parameters and their value

Parameter	Description	Base Value
N^2	Number of metapopulation sites	625
L	Number of stress loci	5
c	Production cost	0.1
a_{max}	Number of alleles	6
δ	Fitness benefit, nonzero alleles	0.3
ϵ	Fitness benefit, sequential alleles	TODO
z	Baseline fitness	1
S_{min}	Minimum population size	800
S_{max}	Maximum population size	2000
μ_c	Mutation rate (cooperation)	10^{-5}
μ_s	Mutation rate (stress)	10^{-5}
m	Migration rate	0.05
p_0	Initial cooperator proportion	0.5
μ_t	Mutation rate (tolerance to new stress)	10^{-5}
T	Number of simulation cycles	1000
d	Population dilution factor	0.1

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