

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?

Materials and Methods

We build upon the model described in Hankshaw and Kerr (2015), in which cooperators and defectors compete and evolve in a spatially-structured metapopulation of populations. Each of these populations grows to carrying capacity, mutates, and migrates to neighboring patches. During this process, populations adapt to their local environments. In our extended model, we allow

89 these individuals to modify their local environment, and these modifications
90 feed back to affect selection.

91 Model Description

92 Our simulated environment consists of N^2 patches arranged as an $N \times N$
93 lattice (see [Table 1](#) for model parameters and their values), where each patch
94 supports a population of zero or more individuals. Each individual in the
95 population has a genotype, which is an ordered list of $L + 1$ integers (loci).
96 The first L loci are *adaptive loci*, and are each occupied by a 0 or an integer
97 from the set $A = \{1, \dots, a_{max}\}$, where a_{max} is the number of potential alleles.
98 At each of these loci, the presence of a non-zero allele represents an adaptation
99 to the environment that confers fitness benefit δ . A binary allele at locus $L + 1$
100 determines whether or not that individual is a cooperator. Individuals with
101 allelic state 1 at this locus are cooperators, carrying a cost c , while individuals
102 with allelic state 0 are defectors. When $\delta \geq c$, an adapted cooperator recoups
103 the cost of cooperation.

104 Organisms also influence their environment, which can feed back to influence
105 selection. We model this as a form of frequency dependent selection. Specif-
106 ically, the selective value of adaptive allele a at locus l increases with the
107 number of individuals in the population that have allele $a - 1$ (modulo a_{max})
108 at locus $l - 1$ (modulo L). The slope of this increase is ϵ , which specifies the
109 intensity of niche construction. As a consequence of this form of frequency
110 dependence, genotypes with sequentially-increasing allelic states will tend to

111 evolve. Because mutations are random, as described later, each population
 112 will evolve sequences that start with different allelic states. These different
 113 sequences represent the unique niches constructed by populations. Under this
 114 model, the fitness of an individual with genotype g in population P is:

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

115 where z is a baseline fitness, $a_{g,l}$ represents the allelic state of genotype g at
 116 locus l , L is the number of adaptive loci, and c is the cost of the cooperative
 117 allele. The function I_A indicates whether allelic state y is in A (i.e., it is non-
 118 zero). The function $n(a_{g,l})$ gives the number of individuals in the population
 119 with allelic state at the previous locus equal to one less than that at the focal
 120 locus $a_{g,l}$, or:

$$n(a_{g,l}) = \sum_{i \in P} I_{a_{g,l}}(1 + a_{\gamma(i), \{1+(l-2 \pmod L)\} \pmod{a_{max}}}) \quad (2)$$

121 Here, $I_x(y)$ indicates whether the allelic state y matches allelic state x (1) or
 122 not (0), and $\gamma(j)$ is the genotype of individual j . Note that the selective value
 123 of an allele at the first locus is affected by the allelic state at locus L .

124 Cooperators produce a public good that is equally accessible to all members
 125 of the population. This public good increases the carrying capacity at that
 126 patch, allowing the population to reach greater density. This benefit increases
 127 linearly with the proportion of cooperators. Thus, if p is the proportion of
 128 cooperators in a population at the beginning of a growth cycle, then that

129 population reaches the following size during the growth phase:

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

130 The function $S(p)$ reflects the benefit of public good production. A population
 131 composed entirely of defectors reaches size S_{min} , while one composed entirely
 132 of cooperators reaches size S_{max} (with $S_{max} \geq S_{min}$). During growth, indi-
 133 viduals compete for inclusion in the resulting population. The composition of
 134 population P with cooperator proportion p after growth is multinomial with
 135 parameters and $S(p)$ and $\{\pi_1, \pi_2, \dots, \pi_{|P|}\}$, where:

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

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

139 For simplicity, we apply mutations after population growth. Mutations occur
 140 independently at each locus and cause the allelic state to change. Mutations
 141 occur at each adaptive locus at rate μ_a , and cause a new allelic state to be
 142 chosen at random from the set $\{0\} \cup A$. At the binary cooperation locus,
 143 mutations occur at rate μ_c . These mutations flip the allelic state, causing
 144 cooperators to become defectors and vice versa. Therefore, the probability
 145 that genotype g mutates into genotype g' is given by:

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

where $H_a(g, g')$ and $H_c(g, g')$ are the Hamming distances between genotypes g and g' at the cooperation locus and adaptive loci, respectively. The Hamming distance is the number of loci at which allelic states differ (Hamming, 1950). Because we define no inherent relationship among alleles, each of the $a_{max} + 1$ allelic states is equally likely to arise via mutation at a given locus.

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

Metapopulations are initiated in a state that follows an environmental change. First, populations are seeded at all patches with cooperator proportion p_0 and grown to density $S(p_0)$. An environmental challenge is then introduced, which subjects the population to a bottleneck. For each individual, the probability of survival is μ_t , which represents the likelihood that a mutation occurs that confers tolerance. Survivors are chosen by binomial sampling. Because individuals have not yet adapted to this new environment, the allelic state of each individual's genotype is set to 0 at each adaptive locus ($\forall i \in P, l \in \{1, \dots, L\} : a_{\gamma(i), l} = 0$). Following initialization, simulations are run for T cycles, where each discrete cycle consists of growth, mutation, and migration. At the end of each cycle, populations are thinned to allow for growth in the next cycle.

167 The individuals that remain are chosen by binomial sampling, where each
168 individual persists with probability d , regardless of allelic state.

169 Source Code and Software Environment

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

174 Results

175 results...

176 Discussion

- 177 • summary of results
- 178 • similarities/differences from previous work
 - 179 – Schwilk and Kerr (2002)
 - 180 – 10.1073/pnas.0812644106
- 181 • public goods as niche construction
- 182 • future QS or other environmental sensing
- 183 • Facultative cooperation

- 184 – Rodrigues (2012)
- 185 – Dumas and Kümmerli (2010)
- 186 – Kümmerli and Brown (2010)
- 187 – Darch/Diggle
- 188 – QS?
- 189 – Environmental Sensing? - (Koestler and Waters, 2014, Bernier et
- 190 al. (2011))

191 Niche construction and selective feedbacks Niche construction and other social
 192 interactions

193 **Public Goods**

194 By their very nature, public goods benefit populations by making their environ-
 195 ment more hospitable. For example, bacteria produce extracellular products
 196 that find soluble iron (Griffin *et al.*, 2004), digest large proteins (Diggle *et al.*,
 197 2007; Darch *et al.*, 2012), and reduce the risk of predation (Cosson *et al.*, 2002),
 198 among many others (West *et al.*, 2007a). While many studies have explored
 199 how the environment affects the evolution of cooperative behaviors, relatively
 200 few have examined how those behaviors affect the environment and the result-
 201 ing change in evolutionary trajectories. Lehmann (2007) demonstrated analyti-
 202 cally that when niche construction act benefits future generations, cooperation
 203 is favored due to reduced competition among kin. When rate-benefitting and
 204 yield-benefitting altruistic acts co-evolve, Van Dyken and Wade (2012) showed
 205 that “reciprocal niche construction”, where the selective feedbacks produced

206 by one act benefitted the other, can lead to increased selection for both traits.
207 While these studies have focused on the niche constructing effects of cooper-
208 ation, we instead focus our attention here on how niche construction enables
209 cooperators to escape defection by hitchhiking along with non-social traits.

210 **Primacy/Recency**

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

222 **Cooperative Niche Construction**

223 While our focus for this work has been on the eco-evolutionary feedbacks cre-
224 ated by non-social traits, it would also be interesting to explore how this system
225 is affected by the timescale at which carrying capacity at a given patch is in-
226 creased by public goods. In natural settings, a multitude of factors including

227 protein durability (Brown, 2007; Kümmerli and Brown, 2010), diffusion (Al-
 228 lison, 2005; Driscoll and Pepper, 2010), and resource availability (Zhang and
 229 Rainey, 2013; Ghoul *et al.*, 2014) influence both the rate and the degree to
 230 which public goods alter the environment (and thereby selection). Lehmann
 231 (2007) demonstrated that a cooperative, niche constructing behavior can be fa-
 232 vored when it only affected selection for future generations, thus reducing the
 233 potential for competition among contemporary kin. The evolutionary inertia
 234 that this creates, however, may ultimately work against cooperators. When
 235 public good accumulates in the environment, cooperators must reduce their in-
 236 vestment in production to remain competitive (Kümmerli and Brown, 2010).
 237 TODO: wrap up. Facultative cooperation requires sensing.

238 **Host-Symbiont**

239 In many instances of cooperation, the environment is itself a biological entity,
 240 which can produce additional evolutionary feedbacks. As the host population
 241 changes, so too will selection on their symbiont populations. Here, evolution-
 242 ary outcomes depend greatly on the degree of shared interest between the host
 243 and symbiont. For example, the cooperative production of virulence factors by
 244 the human pathogen *P. aeruginosa* in lung infections is harmful to those with
 245 cystic fibrosis (Harrison, 2007). Conversely, cooperative light production by *A.*
 246 *fischeri* is vital for the survival of its host, the Hawaiian bobtail squid (Ruby,
 247 1996). It was recently argued that incorporating the effects of niche construc-
 248 tion is critical for improving our understanding of viral evolution (Hamblin *et*

249 *al.*, 2014) and evolution in co-infecting parasites (Hafer and Milinski, 2015).
250 Incorporating host dynamics, co-evolution, and the feedbacks that they pro-
251 duce into models is likely to be equally important for gaining an understanding
252 of how cooperative behaviors evolve in these host-symbiont settings.

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- 254 • TODO: Organizers?
- 255 • TODO: Anu

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263 **Figures**

Table 1: Model parameters and their value

Parameter	Description	Base Value
N^2	Number of metapopulation sites	625
L	Number of adaptive loci	5
a_{max}	Number of alleles	6
δ	Fitness benefit, nonzero alleles	0.3
c	Production cost	0.1
ϵ	Fitness benefit, sequential alleles	0.00015
z	Baseline fitness	1
S_{min}	Minimum population size	800
S_{max}	Maximum population size	2000
μ_c	Mutation rate (cooperation)	10^{-5}
μ_a	Mutation rate (adaptation)	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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