

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-

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

56 Here, we build upon the model presented in Hankshaw and Kerr (2015) to
57 explore whether the selective feedbacks produced as populations modify their
58 environment can act as an additional source of adaptive opportunities. As pre-
59 viously described, this model follows the evolution of cooperation in a metapop-
60 ulation of populations connected by spatially-limited migration. Cooperators
61 produce a public good that increases the local carrying capacity. Through
62 mutation, individuals gain non-social adaptations that confer fitness benefits.
63 These benefits are large enough so that an adapted cooperator is more fit than
64 a less-adapted defector (note that for simplicity, we refer to all non-cooperators
65 as “defectors”, regardless of their origin). Because of their larger sizes, cooper-
66 ator 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

the presence of these individuals to modify their local environment, and these modifications feed back to affect selection.

Model Description

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

Organisms also influence their environment, which can feed back to influence selection. We model this as a form of frequency dependent selection. Specifically, the selective value of adaptive allele a at locus l increases with the proportion of the population that has allele $a - 1$ (modulo a_{max}) at locus $l - 1$ (and the first adaptive locus is affected by the last). The slope of this increase is ϵ , which specifies the intensity of niche construction. As a consequence of

111 this form of frequency dependence, genotypes with sequentially increasing al-
 112 lelic states will tend to evolve. Because mutations are random, as described
 113 later, each population will evolve sequences that start with different allelic
 114 states. These different sequences represent the unique niches constructed by
 115 populations. Under this model, the fitness of an individual with genotype g in
 116 population P is:

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

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

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

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

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

cooperators in a population at the beginning of a growth cycle, then that population reaches the following size during the growth phase:

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

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

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

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

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

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

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

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

157 Metapopulations are initiated in a state that follows the onset of an environ-
 158 mental stress. First, populations are seeded at all patches with cooperator
 159 proportion p_0 and grown to density $S(p_0)$. Stress is then introduced, which
 160 subjects the population to a bottleneck. For each individual, the probability of
 161 survival is μ_t , which represents the likelihood that a mutation occurs that con-
 162 fers tolerance. Survivors are chosen by binomial sampling. Because individuals
 163 have not yet adapted to this new stress, the allelic state of each individual's
 164 genotype is set to 0 at each adaptive locus ($\forall i \in P, l \in \{1, \dots, L\} : a_{\gamma(i), l} = 0$).
 165 Following initialization, simulations are run for T cycles, where each discrete
 166 cycle consists of growth, mutation, and migration. At the end of each cycle,
 167 populations are thinned to allow for growth in the next cycle. The individuals

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

170 Source Code and Software Environment

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

175 Results

176 results...

177 Discussion

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

- 185 – Rodrigues (2012)
- 186 – Dumas and Kümmerli (2010)
- 187 – Kümmerli and Brown (2010)
- 188 – Darch/Diggle
- 189 – QS?
- 190 – Environmental Sensing?

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-

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

Host-Symbiont

In many instances of cooperation, the environment is itself a biological entity, which can produce additional evolutionary feedbacks. As the host population changes, so too will selection on their symbiont populations. Here, evolutionary outcomes depend greatly on the degree of shared interest between the host and symbiont. For example, the cooperative production of virulence factors by the human pathogen *P. aeruginosa* in lung infections is harmful to those with cystic fibrosis (Harrison, 2007). Conversely, cooperative light production by *A. fischeri* is vital for the survival of its host, the Hawaiian bobtail squid (Ruby, 1996). It was recently argued that incorporating the effects of niche construction is critical for improving our understanding of viral evolution (Hamblin *et al.*, 2014) and evolution in co-infecting parasites (Hafer and Milinski, 2015).

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
c	Production cost	0.1
a_{max}	Number of alleles	6
δ	Fitness benefit, nonzero alleles	0.3
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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