

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 Using a model of public goods production, Hankshaw and Kerr (2015) recently

43 showed that in spatially-structured populations, cooperators gain a substan-
44 tial leg up on defectors in an adaptive race when the cooperative behavior
45 increases local population density, thus increasing the likelihood of acquiring
46 beneficial non-social mutations. By hitchhiking along with these adaptations,
47 cooperators can rapidly rise in abundance. Nevertheless, this advantage is
48 fleeting. As soon as the opportunities for adaptation are exhausted, cooper-
49 ators are once again at a disadvantage against defectors, particularly those
50 that arise from within via mutations that disable cooperation. However, coop-
51 eration can be maintained indefinitely when frequent environmental changes
52 produce a steady stream of non-social adaptive opportunities. Although or-
53 ganisms typically find themselves in dynamic environments, change might not
54 occur rapidly enough to provide the adaptive opportunities that cooperators
55 need to ensure their long-term survival.

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

67 ator populations remain susceptible to invasion by adapted defectors either
 68 immigrate from a nearby populaion or arise via social mutation. In our ex-
 69 panded version of this model, populations alter their local environment based
 70 on the the presence of different non-social adaptations. Frequency-dependent
 71 selection on these adaptations creates an eco-evolutionary feedback that in-
 72 creasingly favors the adaptations present in each population. We focus on
 73 two aspects in which the production of public goods is affected by these selec-
 74 tive feedbacks. First, the creation of unique niches may diminish the ability
 75 of both cooperators and defectors to invade neighboring patches due to mal-
 76 adaptation. However, because cooperator populations are larger, the greater
 77 number of emigrants that they produce will allow them to “export” their niche
 78 at a higher rate than defectors. As a result, cooperators may be able to ex-
 79 pand more quickly. Second, larger cooperator populations will experience more
 80 mutations, which better enables these populations to adapt to changing envi-
 81 ronments. If niche construction produces continual change, can the resulting
 82 adaptive opportunities maintain cooperation?

83 By their very nature, public goods benefit populations by making their environ-
 84 ment more hospitable. For example, bacteria produce extracellular products
 85 that find soluble iron (Griffin *et al.*, 2004), digest large proteins (Diggle *et al.*,
 86 2007; Darch *et al.*, 2012), and reduce the risk of predation (Cosson *et al.*, 2002),
 87 among many others (West *et al.*, 2007a). While many studies have explored
 88 how the environment affects the evolution of cooperative bahviors, relatively
 89 few have examined how those behaviors affect the environment and the result-
 90 ing 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 develop a computational model to observe the evolution of public goods
101 cooperation in a spatially-structured metapopulation of populations. As de-
102 scribed below, populations grow to carrying capacity, mutate, and migrate
103 to neighboring patches. During this process, populations adapt to their lo-
104 cal environments. The environments are, in turn, modified by the presence
105 of these adapted individuals, allowing each population to construct a unique
106 niche along its evolutionary trajectory. Model parameters and their values are
107 listed in [Table 1](#).

108 **Individuals and Fitness**

109 Each individual has a length $L + 1$ genotype. A binary allele at the first locus
110 determines whether or not the individual is a cooperator, which carries cost

111 *c.* Note that we refer to all individuals with allelic state 0 at this locus as
 112 a “defector”, regardless of origin. The remaining L loci are *stress loci*, and
 113 are each occupied by a 0 or an integer from the set $A = \{1, \dots, a_{max}\}$, where
 114 a_{max} is the number of possible alleles. These alleles represent adaptations to
 115 the environment, and the number of loci determines the number of possible
 116 adaptations. All non-zero alleles carry fitness benefit δ . When $\delta \geq c$, adapted
 117 cooperators recoup the cost of cooperation. Organisms also influence their
 118 environment, which can feed back to influence selection. We model this as a
 119 form of frequency dependent selection. Specifically, the selective value of stress
 120 allele a at locus i increases with the proportion of the population that has allele
 121 $a - 1$ (modulo a_{max}) at locus $i - 1$. The slope of this increase is ϵ (which gauges
 122 the intensity of niche construction). As a consequence of this form of frequency
 123 dependence, genotypes with sequentially increasing allelic states will tend to
 124 evolve. Because mutations are random, as described below, each population
 125 will evolve sequences that start with different allelic states. These different
 126 sequences represent the unique niches constructed by populations. Under this
 127 model, the fitness of an individual with genotype g is:

$$W_g = z + a_{g,1}c + \delta \sum_{l=2}^{L+1} I_A(a_{g,l}) + \epsilon \sum_{h=1}^N I_{a_{h,1}}(a_{g,1}) + \epsilon \sum_{l=2}^L n(a_{g,l})$$

128 where $a_{g,l}$ represents the allelic state of genotype g at locus l , z is a baseline
 129 fitness, L is the number of stress loci, N is the population size at that patch,
 130 and c is the cost of the cooperative allele. $I_x(y)$ indicates whether the allelic
 131 state y matches allelic state x (1) or not (0). $n(a_{g,l})$ is the number of individuals

132 in the population with allelic state at the previous locus equal to one less than
 133 that at the focal locus $a_{g,l}$, or:

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

134 Population Growth

135 If p is the proportion of cooperators in a population at the beginning of a
 136 growth cycle, then that population reaches the following size during the growth
 137 phase:

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

138 Therefore, a population composed entirely of defectors reaches size S_{min} , while
 139 one composed entirely of cooperators reaches size S_{max} (with $S_{max} \geq S_{min}$).
 140 The function $S(p)$ gauges the benefit of public good production, as popula-
 141 tion size increases linearly with the proportion of cooperators. During growth,
 142 competition occurs. Consider an arbitrary genotype g . Let n_g be the num-
 143 ber of individuals with genotype g , and let W_g be the fitness of genotype g
 144 (see equation [1]). The composition of genotypes after population growth is
 145 multinomial with parameters $S(p)$ and $\{\pi_1, \pi_2, \dots, \pi_{|G|}\}$, where:

$$\pi_g = \frac{n_g W_g}{\sum_{i=1}^G n_i W_i}$$

146 Thus, π_g is the probability that an individual in the population after growth

147 has genotype g (such that $\sum \pi_g = 1$). G represents the set of all $(a_{max} + 1)^L$
 148 genotypes.

149 **Mutation**

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

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

157 where $H_s(g, g')$ and $H_p(g, g')$ are the Hamming distances between genotypes
 158 g and g' at the stress loci and cooperation locus, respectively. The Hamming
 159 distance is the number of loci at which allelic states differ. Because there is
 160 no inherent relationship among alleles, each of the $a_{max} + 1$ alleles is equally
 161 likely to arise via mutation at a given locus.

162 **Migration and Metapopulation Structure**

163 The metapopulation consists of N^2 patches arranged in a $N \times N$ lattice. After
 164 mutation, individuals emigrate to an adjacent patch with probability m . This

adjacent 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.

Initialization and Simulation

Metapopulations are initiated in a state that follows the onset of an environmental stress. First, populations are seeded at each patch with cooperator proportion p_0 and grown to density $S(p_0)$. Stress is then introduced by subjecting the population to a bottleneck. The number of survivors with each genotype g is sampled from a binomial distribution, where the number of trials is n_g . The probability of success is μ_t , which represents the likelihood that a mutation occurs that enables survival. Because individuals have not yet adapted to this new stress, the allelic state of each genotype is set to 0 at each stress locus ($\forall g \in G, l \in \{2, \dots, L + 1\} : a_{g,l} = 0$). Following initialization, simulations are run for T discrete cycles, where each cycle consists of growth, mutation, and migration. After migration, populations are thinned to allow for growth in the next cycle. The number of survivors for each genotype g is sampled from a binomial distribution, where the number of trials is n_g and the probability of success is d .

184 Source Code and Software Environment

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

189 Results

190 results...

191 Discussion

- 192 • summary of results
- 193 • similarities/differences from previous work
 - 194 – Schwilk and Kerr (2002)
- 195 • future primacy/recency
 - 196 – Laland et al. (1996)
 - 197 – Lehmann (2007)
- 198 • public goods as niche construction
- 199 • Host symbiont - many instances of cooperation occur among pathogens.
- 200 • future QS or other environmental sensing
- 201 • Facultative cooperation

- 202 – Rodrigues (2012)
- 203 – Dumas and Kümmerli (2010)
- 204 – Kümmerli and Brown (2010)
- 205 – Darch/Diggle
- 206 – QS?
- 207 – Environmental Sensing?

208 Niche construction and selective feedbacks

209 Niche construction and other social interactions # Acknowledgments

- 210 • PRFB
- 211 • BEACON
- 212 • Google
- 213 • Organizers?

²¹⁴ **Figures**

Table 1: Model parameters and their value.

Parameter	Description	Base Value
L	Number of Stress Loci	5
a_{max}	Number of alleles	6
δ	Fitness benefit, nonzero alleles	0.3
ϵ	Fitness benefit, sequential alleles	TODO
c	Production Cost	0.1
z	Baseline fitness	1
S_{min}	Minimum Population Size	800
S_{max}	Maximum Population Size	2000
μ_s	Mutation Rate (Stress)	10^{-5}
μ_c	Mutation Rate (Cooperation)	10^{-5}
μ_t	Mutation Rate (Tolerance to New Stress)	10^{-5}
m	Migration Rate	0.05
d	Population Dilution Factor	0.1
N^2	Number of Metapopulation Sites	625
p_0	Initial Cooperator Proportion	0.5
T	Number of Simulation Cycles	TODO

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