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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.

Introduction

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

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

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-

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

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). At the first locus, a binary allele 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. The remaining L loci are *stress loci*, and are each occupied by a 0 or an integer from the set $A = \{1, \ldots, a_{max}\}$, 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. 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 stress 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 stress locus is affected by the last). The slope of this increase is ϵ, which specifies the intensity of niche construction. As a consequence of this form of frequency dependence, genotypes with sequentially increasing allelic states will tend to evolve. Because mutations are random, as described later, each population will evolve sequences that start with different allelic states. These different sequences represent the unique niches constructed by populations. Under this model, the fitness of an individual with genotype g in population P is:

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

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

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

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

Cooperators produce a public good that is equally accessible to all members of the population. This public good increases the carrying capacity at that patch, allowing the population to reach greater density. This benefit increases 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 and $S(p)$ and $\{\pi_1, \pi_2, \ldots, \pi_{|P|}\}$, where:

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

Here, $\gamma(i)$ is the genotype of individual i, and $W_{\gamma(i)}$ is its fitness (see Equation 1). π_i is proportional to $W_{\gamma(i)}$ (and $\sum_{i \in P} \pi_i = 1$).

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 stress locus at rate μ_s, 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:

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

where $H_c(g,\ g')$ and $H_s(g,\ g')$ are the Hamming distances between genotypes g and g' at the cooperation locus and stress 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 the onset of an environmental stress. First, populations are seeded at all patches with cooperator proportion p_0 and grown to density $S(p_0)$. Stress 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 stress, the allelic state of each individual's genotype is set to 0 at each stress locus ($\forall i \in P, l \in \{2, \ldots, L+1\} : a_{\gamma(i),l} = 0$). 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. The individuals that remain are chosen by binomial sampling, where each individual persists with probability d, regardless of allelic state.

Source Code and Software Environment

The simulation software and configurations for the experiments reported are available online (Us, 2015). Simulations used Python 3.4.0, NumPy 1.9.1,

Pandas 0.15.2 (McKinney, 2010), NetworkX 1.9.1 (Hagberg *et al.*, 2008). Data analyses were performed with R 3.1.3 (R Core Team, 2015).

Results

results…

Discussion

- summary of results
- similarities/differences from previous work
    - Schwilk and Kerr (2002)
- future primacy/recency
    - Laland et al. (1996)
    - Lehmann (2007)
- public goods as niche construction
- Host symbiont - many instances of cooperation occur among pathogens.
- future QS or other environmental sensing
- Facultative cooperation
    - Rodrigues (2012)
    - Dumas and Kümmerli (2010)
    - Kümmerli and Brown (2010)

206 – Darch/Diggle

207 – QS?

208 – Environmental Sensing?

209 Niche construction and selective feedbacks

210 Niche construction and other social interactions # Acknowledgments

211 • PRFB

212 • BEACON

213 • Google

214 • Organizers?

Figures

Tables

Table 1: Model parameters and their value

Parameter	Description	Base Value
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}
N^2	Number of Metapopulation Sites	625
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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