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.

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.*, 2007). 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 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. Nevertheless, this advantage is fleeting (Fig. 1A). As soon as the opportunities for adaptation are exhausted, cooperators are once again at a disadvantage against defectors. As shown in Fig. 1B, however, cooperation can be maintained indefinitely when frequent environmental changes produce a stream of non-social adaptive opportunities. Although natural organisms typically find themselves in changing environments, cooperators may not be able to rely on the the environment to provide sufficient adaptive opportunities for their long-term survival.

Materials and Methods

We develop a computational model to observe the evolution of public goods cooperation in a spatially-structured metapopulation of populations. As described below, populations grow to carrying capacity, mutate, and migrate to neighboring patches. During this process, populations adapt to their local environments. The environments are, in turn, modified by the presence of these adapted individuals, allowing each population to construct a unique niche along its evolutionary trajectory. Model parameters and their values are listed in Table 1.

Individuals and Fitness

Each individual has a genotype of length $L + 1$. A binary allele at the first locus determines whether or not the individual is a cooperator, which carries cost c. Note that we refer to all individuals with allelic state 0 at this locus as a "defector", regardless of origin. 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 possible alleles. These alleles represent adaptations to the environment, and the number of loci determines the number of possible adaptations. All non-zero alleles carry fitness benefit δ. 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 i increases with the proportion of the population that has allele $a - 1$ (modulo a_{max}) at locus $i - 1$. The slope of this increase is ϵ (which gauges 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 below, 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 is:

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

where $a_{g,l}$ represents the allelic state of genotype g at locus l, z is a baseline

fitness, L is the number of stress loci, N is the population size at that patch, and c is the cost of the cooperative allele. $I_x(y)$ indicates whether the allelic state y matches allelic state x (1) or not (0). $n(a_{g,l})$ is 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:

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

Population Growth

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:

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

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

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

Thus, π_g is the probability that an individual in the population after growth has genotype g (such that $\sum \pi_g = 1$). G represents the set of all $(a_{max}+1)^L$ genotypes.

Mutation

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

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

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

Migration and Metapopulation Structure

The metapopulation consists of N^2 patches arranged in a $N \times N$ lattice. After 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 ($\forall g \in G, l \in \{2, \ldots, L+1\} : a_{g,l} = 0$). Following initialization, simulations are run for T 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.

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 3.1.2 (R Core Team, 2014).

Results

results...

Discussion

discussion...

- future primacy/recency
- future other types of social interactions
- future QS or other environmental sensing

Acknowledgments

Figures

Tables

Table 1: Model parameters and their value.

Parameter	Description	Base Value
L	Number of Stress Loci	3
a_{max}	Number of alleles	4
δ	Fitness benefit, nonzero alleles	0.5
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 Producer Proportion	0.5
T	Number of Simulation Cycles	TODO

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