TODO title

TODO

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?

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

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 $A = \{1, \ldots, a_{max}\}$, 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. 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 number of individuals in the population that have allele $a - 1$ (modulo a_{max}) at locus $l - 1$ (modulo L). 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:

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

where z is a baseline fitness, $a_{g,l}$ represents the allelic state of genotype g at locus l, L is the number of adaptive loci, and c is the cost of the cooperative allele. The function 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:

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

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

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:

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

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 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. Mutations occur at each adaptive locus at rate μ_a, and cause a new allelic state to be chosen at random from the set $\{0\} \cup A$. 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. Therefore, the probability that genotype g mutates into genotype g' is given by:

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

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 ($\forall i \in P, l \in \{1,\ldots,L\}$: $a_{\gamma(i),l} = 0$). 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.

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), and 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)
  - 10.1073/pnas.0812644106
- public goods as niche construction
- future QS or other environmental sensing
- Facultative cooperation

- Rodrigues (2012)
- Dumas and Kümmerli (2010)
- Kümmerli and Brown (2010)
- Darch/Diggle
- QS?
- Environmental Sensing? - (Koestler and Waters, 2014, Bernier et al. (2011))

Niche construction and selective feedbacks Niche construction and other social interactions

Public Goods

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 analytically 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.

Primacy/Recency

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

Cooperative Niche Construction

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

protein durability (Brown, 2007; Kümmerli and Brown, 2010), diffusion (Allison, 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). Incorporating host dynamics, co-evolution, and the feedbacks that they produce into models is likely to be equally important for gaining an understanding of how cooperative behaviors evolve in these host-symbiont settings.

Acknowledgments

- TODO: Organizers?
- TODO: Anu

This material is based upon work supported by the National Science Foundation Postdoctoral Research Fellowship in Biology under Grant No. 1309318 (to BDC) and under Cooperative Agreement No. DBI-0939454. Any opinions, findings, and conclusions or recommendations expressed in this material are those of the authors and do not necessarily reflect the views of the National Science Foundation. Computational resources were provided by an award from Google (to BDC and BK).

Figures

Tables

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

References

Allison, S.D. 2005. Cheaters, diffusion and nutrients constrain decomposition by microbial enzymes in spatially structured environments. *Ecology Letters*, **8**: 626–635.

Asfahl, K.L., Walsh, J., Gilbert, K. and Schuster, M. 2015. Non-social adaptation defers a tragedy of the commons in Pseudomonas aeruginosa quorum sensing. *The ISME Journal*, doi: 10.1038/ismej.2014.259.

Bernier, S.P., Ha, D.-G., Khan, W., Merritt, J.H.M. and O'Toole, G.A. 2011. Modulation of Pseudomonas aeruginosa surface-associated group behaviors by individual amino acids through c-di-GMP signaling. *Research in Microbiology*, **162**: 680–688.

Brown, F., Sam P. AND Taddei. 2007. The durability of public goods changes the dynamics and nature of social dilemmas. *PLoS ONE*, **2**: e593. Public Library of Science.

Brown, S.P. and Johnstone, R.A. 2001. Cooperation in the dark: Signalling and collective action in quorum-sensing bacteria. *Proceedings of the Royal Society of London B: Biological Sciences*, **268**: 961–965.

Cosson, P., Zulianello, L., Join-Lambert, O., Faurisson, F., Gebbie, L. and Benghezal, M.*et al.* 2002. Pseudomonas aeruginosa virulence analyzed in a dictyostelium discoideum host system. *Journal of Bacteriology*, **184**: 3027–3033.

Dandekar, A.A., Chugani, S. and Greenberg, E.P. 2012. Bacterial quorum

sensing and metabolic incentives to cooperate. *Science*, **338**: 264–266.

Darch, S.E., West, S.A., Winzer, K. and Diggle, S.P. 2012. Density-dependent fitness benefits in quorum-sensing bacterial populations. *Proceedings of the National Academy of Sciences*, **109**: 8259–8263.

Diggle, S.P., Griffin, A.S., Campbell, G.S. and West, S.A. 2007. Cooperation and conflict in quorum-sensing bacterial populations. *Nature*, **450**: 411–414.

Driscoll, W.W. and Pepper, J.W. 2010. Theory for the evolution of diffusible external goods. *Evolution*, **64**: 2682–2687.

Fletcher, J.A. and Doebeli, M. 2009. A simple and general explanation for the evolution of altruism. *Proceedings of the Royal Society B: Biological Sciences*, **276**: 13–19.

Foster, K., Shaulsky, G., Strassmann, J., Queller, D. and Thompson, C. 2004. Pleiotropy as a mechanism to stabilize cooperation. *Nature*, **431**: 693–696. Nature Publishing Group.

Gardner, A. and West, S.A. 2010. Greenbeards. *Evolution*, **64**: 25–38.

Ghoul, M., West, S.A., Diggle, S.P. and Griffin, A.S. 2014. An experimental test of whether cheating is context dependent. *Journal of Evolutionary Biology*, **27**: 551–556.

Griffin, A.S., West, S.A. and Buckling, A. 2004. Cooperation and competition in pathogenic bacteria. *Nature*, **430**: 1024–1027.

Hafer, N. and Milinski, M. 2015. When parasites disagree: Evidence for parasite-induced sabotage of host manipulation. *Evolution*, doi:

10.1111/evo.12612.

Hagberg, A.A., Schult, D.A. and Swart, P.J. 2008. Exploring network structure, dynamics, and function using NetworkX. In: *Proceedings of the 7th Python in Science Conference (SciPy2008)*, pp. 11–15.

Hamblin, S.R., White, P.A. and Tanaka, M.M. 2014. Viral niche construction alters hosts and ecosystems at multiple scales. *Trends in Ecology & Evolution*, **29**: 594–599.

Hamilton, W.D. 1964. The genetical evolution of social behaviour I & II. *Journal of Theoretical Biology*, **7**: 1–52.

Hamming, R.W. 1950. Error detecting and error correcting codes. *Bell System Technical Journal*, **29**: 147–160.

Hankshaw, S. and Kerr, B. 2015. Thumbs up!: Hitching a ride on the evolutionary superhighway. *Unknown Journal*, **0**: 0–0.

Hardin, G. 1968. The tragedy of the commons. *Science*, **162**: 1243–1248.

Harrison, F. 2007. Microbial ecology of the cystic fibrosis lung. *Microbiology*, **153**: 917–923.

Koestler, B.J. and Waters, C.M. 2014. Bile acids and bicarbonate inversely regulate intracellular cyclic di-GMP in Vibrio cholerae. *Infection and Immunity*, **82**: 3002–3014.

Kuzdzal-Fick, J.J., Fox, S.A., Strassmann, J.E. and Queller, D.C. 2011. High relatedness is necessary and sufficient to maintain multicellularity in Dictyostelium. *Science*, **334**: 1548–1551.

Kümmerli, R. and Brown, S.P. 2010. Molecular and regulatory properties of a public good shape the evolution of cooperation. *Proceedings of the National Academy of Sciences*, **107**: 18921–18926.

Laland, K.N., Odling-Smee, F.J. and Feldman, M.W. 1996. The evolutionary consequences of niche construction: A theoretical investigation using two-locus theory. *Journal of Evolutionary Biology*, **9**: 293–316.

Lehmann, L. 2007. The evolution of trans-generational altruism: Kin selection meets niche construction. *Journal of Evolutionary Biology*, **20**: 181–189. Blackwell Publishing Ltd.

McKinney, W. 2010. Data structures for statistical computing in Python. In: *Proceedings of the 9th Python in Science Conference* (S. van der Walt and J. Millman, eds), pp. 51–56.

Morgan, A.D., Quigley, B.J.Z., Brown, S.P. and Buckling, A. 2012. Selection on non-social traits limits the invasion of social cheats. *Ecology Letters*, **15**: 841–846.

Nadell, C.D., Foster, K.R. and Xavier, J.B. 2010. Emergence of spatial structure in cell groups and the evolution of cooperation. *PLoS Computational Biology*, **6**: e1000716.

Nowak, M.A. 2006. Five rules for the evolution of cooperation. *Science*, **314**: 1560–1563.

R Core Team. 2015. *R: A language and environment for statistical computing*. Vienna, Austria: R Foundation for Statistical Computing.

Ruby, E.G. 1996. Lessons from a cooperative, bacterial-animal association: The Vibrio fischeri–Euprymna scolopes light organ symbiosis. *Annual Review of Microbiology*, **50**: 591–624.

Schwilk, D.W. and Kerr, B. 2002. Genetic niche-hiking: An alternative explanation for the evolution of flammability. *Oikos*, **99**: 431–442.

Sinervo, B., Chaine, A., Clobert, J., Calsbeek, R., Hazard, L. and Lancaster, L.*et al.* 2006. Self-recognition, color signals, and cycles of greenbeard mutualism and altruism. *Proceedings of the National Academy of Sciences*, **103**: 7372–7377.

Us. 2015. Name for data and code.

Van Dyken, J.D. and Wade, M.J. 2012. Origins of altruism diversity II: Runaway coevolution of altruistic strategies via "reciprocal niche construction". *Evolution*, **66**: 2498–2513.

Veelders, M., Brückner, S., Ott, D., Unverzagt, C., Mösch, H.-U. and Essen, L.-O. 2010. Structural basis of flocculin-mediated social behavior in yeast. *Proceedings of the National Academy of Sciences*, **107**: 22511–22516.

Waite, A.J. and Shou, W. 2012. Adaptation to a new environment allows cooperators to purge cheaters stochastically. *Proceedings of the National Academy of Sciences*, **109**: 19079–19086.

West, S.A., Diggle, S.P., Buckling, A., Gardner, A. and Griffin, A.S. 2007a. The social lives of microbes. *Annual Review of Ecology, Evolution, and Systematics*, **38**: 53–77.

West, S.A., Griffin, A.S. and Gardner, A. 2007b. Evolutionary explanations for cooperation. *Current Biology*, **17**: R661–R672.

Zhang, X.-X. and Rainey, P.B. 2013. Exploring the sociobiology of pyoverdin-producing Pseudomonas. *Evolution*, **67**: 3161–3174.