

1 Negative Niche Construction Favors the
2 Evolution of Cooperation

3
4 Brian D. Connelly^{1,3}, Katherine J. Dickinson¹, Sarah P. Hammarlund^{1,2}, and
5 Benjamin Kerr^{1,3}

6 ¹Department of Biology and BEACON Center for the Study of Evolution in
7 Action, University of Washington, Seattle, Washington, 98195, USA

8 ²Current Address: Department of Zoology, University of Oxford, Oxford OX1
9 3PS, United Kingdom

10 ³E-mail: bdcx@uw.edu and kerrb@uw.edu

Abstract

By benefitting others at a cost to themselves, cooperators face an ever present threat from defectors—individuals that avail themselves of the cooperative benefit without contributing. A longstanding challenge to evolutionary biology is to understand the mechanisms that support the many instances of cooperation that nevertheless exist. Hammarlund et al. recently demonstrated that cooperation can persist by hitchhiking along with beneficial non-social adaptations. Importantly, cooperators play an active role in this process. In spatially-structured environments, clustered cooperator populations reach greater densities, which creates more mutational opportunities to gain beneficial non-social adaptations. Cooperation rises in abundance by association with these adaptations. However, once adaptive opportunities have been exhausted, the ride abruptly ends as cooperators are displaced by adapted defectors. Using an agent-based model, we demonstrate that the selective feedback that is created as populations construct their local niches can maintain cooperation indefinitely. This cooperator success depends specifically on negative niche construction, which acts as a perpetual source of adaptive opportunities. As populations adapt, they alter their environment in ways that reveal additional opportunities for adaptation. Despite being independent of niche construction in our model, cooperation feeds this cycle. By reaching larger densities, populations of cooperators are better able to adapt to changes in their constructed niche and successfully respond to the constant threat posed by defectors. We relate these findings to previous studies from the niche construction literature

34 and discuss how this model could be extended to provide a greater under-
35 standing of how cooperation evolves in the complex environments in which it
36 is found.

37 Introduction

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

47 Several factors can prevent this *tragedy of the commons* (Hamilton, 1964;
48 Nowak, 2006; West *et al.*, 2007b). One such factor involves non-random so-
49 cial interaction, in which cooperators benefit more from the cooperative act
50 than defectors. This can occur when cooperators are clustered together in
51 spatially-structured populations (Fletcher and Doebeli, 2009; Nadell *et al.*,
52 2010; Kuzdzal-Fick *et al.*, 2011) or when cooperators use communication
53 (Brown and Johnstone, 2001; Darch *et al.*, 2012) or other cues (Sinervo *et*
54 *al.*, 2006; Gardner and West, 2010; Veelders *et al.*, 2010) to cooperate condi-
55 tionally with kin. Cooperation can also be bolstered by pleiotropic connections
56 to personal benefits (Foster *et al.*, 2004; Dandekar *et al.*, 2012) or through as-
57 sociation with alleles encoding self-benefitting traits (Asfahl *et al.*, 2015). In
58 the latter case, the associated alleles may provide private benefits that are
59 completely independent from the public benefits of cooperation. In asexual

60 populations of cooperators and defectors, this sets the stage for an “adaptive
61 race” in which both types vie for the first highly beneficial adaptation (Waite
62 and Shou, 2012; Morgan *et al.*, 2012). The tragedy of the commons can be
63 deferred if a cooperator, by chance, wins the adaptive race.

64 Hammarlund *et al.* (2015) recently showed that in spatially-structured pop-
65 ulations, the “Hankshaw effect” can give cooperators a substantial leg up on
66 defectors in an adaptive race. This advantage is reminiscent of Sissy Han-
67 kshaw, a fictional character in Tom Robbins’ *Even Cowgirls Get the Blues*,
68 whose oversized thumbs—which were otherwise an impairment—made her a
69 prolific hitchhiker. Similarly, cooperation is costly, but it increases local pop-
70 ulation density. As a result, cooperators are more likely to acquire beneficial
71 mutations. By hitchhiking along with these adaptations, cooperation can rise
72 in abundance. Nevertheless, this advantage is fleeting. As soon as the oppor-
73 tunities for adaptation are exhausted, cooperators are once again at a selective
74 disadvantage against adapted defectors that arise via mutation. However, co-
75 operation can be maintained when frequent environmental changes produce
76 a steady stream of new adaptive opportunities (Hammarlund *et al.*, 2015).
77 Although organisms typically find themselves in dynamic environments, the
78 nature and frequency of these changes might not ensure long-term cooperator
79 survival.

80 Importantly, organisms do more than passively experience changing environ-
81 ments. Through their activities, their interactions with others, and even their
82 deaths, organisms constantly modify their environment. This *niche construc-*
83 *tion* process can produce evolutionary feedback loops in which environmental

84 modification alters selection, which, in turn, alters the distribution of types and
 85 their corresponding influence on the environment (Odling-Smee *et al.*, 2003).
 86 The nature of this feedback can have dramatic evolutionary consequences. One
 87 critical distinction is whether the constructing type is favored in the environ-
 88 ment that it constructs. Under *positive niche construction*, selection favors the
 89 constructor, and evolution stagnates as this type fixes. Whereas under *negative*
 90 *niche construction*, selection favors a type other than the constructor, which
 91 creates an opportunity for novel adaptation. If the resulting adapted type also
 92 engages in negative niche construction, cycles of construction and adaptation
 93 can ensue, such that populations find themselves continually chasing beneficial
 94 mutations as their adaptive landscape perpetually shifts.

95 Here, we show that the selective feedbacks that result from niche construction
 96 can maintain cooperation indefinitely. Further, we find that it is specifically
 97 negative niche construction that is responsible for this result due to the endless
 98 opportunities for adaptation that it produces. These results suggest that by
 99 playing an active role in their own evolution, cooperators can ensure their
 100 survival.

101 Methods

102 Building upon Hammarlund *et al.* (2015), we describe an individual-based
 103 model in which cooperators and defectors evolve and compete in a population
 104 of subpopulations (i.e., a metapopulation). Through mutation, individuals
 105 gain adaptations to their environment, which increase reproductive fitness and

allow those lineages to rise in abundance. More successful lineages spread to neighboring subpopulations by migration.

In the expanded model here, subpopulations additionally modify their local environment. As this process occurs, environmental changes feed back to affect selection. We explore how niche construction affects the evolution of cooperation; specifically, how cooperative behavior can hitchhike along with adaptations to modified environments.

Model Description

Individual Genotypes and Adaptation

Each individual has a haploid genome with $L + 1$ loci, where integers represent different alleles at each locus (see [Table 1](#) for model parameters and their values). An allele at the *cooperation locus* (locus zero) determines whether that individual is a cooperator (allele 1), which carries fitness cost c , or a defector (allele 0). The remaining L loci are *adaptive loci*, and are each occupied by 0 or a value from the set $\{1, 2, \dots, A\}$. Allele 0 represents a lack of adaptation, while a non-zero allele represents one of the A possible adaptations at that locus.

Non-zero alleles signify two types of adaptations, both of which increase fitness. First, adaptations to the external environment confer a fitness benefit δ . This selective value is the same regardless of which non-zero allele is present. We assume $\delta > c$, which allows a minimally adapted cooperator to recoup the cost of cooperation and gain a fitness advantage.

128 Niche Construction and Selective Feedbacks

129 Individual fitness is also affected by aspects of the local environment that are
130 modified by organisms. This constructed “niche” depends on the specific allelic
131 states present in the subpopulation. As allelic states change, the subpopulation
132 alters its environment, creating a unique niche. As described below, the specific
133 alleles at each locus become important.

134 In our model, the feedback that results from niche construction takes the form
135 of density dependent selection, and individuals evolve to better match their
136 constructed niche. We do not represent this niche explicitly, but rather allow
137 the allelic composition of the subpopulation to feed back to affect selection.
138 Specifically, the selective value of non-zero allele a at adaptive locus l —and
139 consequently the fitness of an individual carrying that allele—increases with
140 the number of individuals in the subpopulation that have allele $a - 1$ at locus
141 $l - 1$. For example, if $L = 5$, $A = 6$, and allele 4 has fixed at locus 2, then a
142 genotype with allele 5 at locus 3 is favored. And as allele 5 fixes at locus 3,
143 the niche that this population constructs will favor allele 6 at locus 4 (see [Box](#)
144 [1](#)). As a consequence, genotypes with sequentially increasing allelic states will
145 tend to evolve.

146 We treat both adaptive loci and their non-zero allelic states as “circular”: the
147 selective value of an allele at locus 1 is affected by the allelic composition of
148 the subpopulation at locus L . Similarly, the selective value of allele 1 at any
149 locus increases with the number of individuals carrying allele A at the previous
150 locus. This circularity is represented by the function $\beta(x, X)$, which gives the

integer that is below an arbitrary value x in the set $\{1, 2, \dots, X\}$:

$$\beta(x, X) = \text{mod}_X(x - 2 + X) + 1 \quad (1)$$

Here, $\text{mod}_X(x)$ is the integer remainder when dividing x by X . Using this function, the selective value of allele a at adaptive locus l is increased by ϵ for each individual in the subpopulation that has allele $\beta(a, A)$ at locus $\beta(l, L)$. Thus, ϵ specifies the intensity of selection due to niche construction.

Individual Fitness

Consider a genotype g with allelic state a_l at locus l ; the fitness of an individual with this genotype is defined as:

$$W_g = z - \underbrace{ca_0}_{\text{cost of cooperation}} + \underbrace{\delta \sum_{l=1}^L I(a_l)}_{\text{adaptation to external environment}} + \underbrace{\epsilon \sum_{l=1}^L n(\beta(a_l, A), \beta(l, L))}_{\text{adaptation to constructed environment}} \quad (2)$$

where z is a baseline fitness, $n(a, l)$ is the number of individuals in the subpopulation with allele a at locus l , and $I(a)$ indicates whether a given allele is non-zero:

$$I(a) = \begin{cases} 1 & \text{if } a \in \{1, 2, \dots, A\} \\ 0 & \text{otherwise} \end{cases} \quad (3)$$

Thus, an individual's fitness is determined both by adaptations to the exter-

163 nal environment and by adaptations to its constructed environment. **Box 1**
164 illustrates the process of adaptation to the constructed environment.

165 **Subpopulation Growth and the Benefit of Cooperation**

166 While cooperation is costly, its effects are independent of the external and
167 constructed components of the environment. Cooperation enables a subpop-
168 ulation to reach a greater density. This benefit affects all individuals equally
169 and accumulates linearly with the proportion of cooperators in the subpopula-
170 tion. If p is the proportion of cooperators present at the beginning of a growth
171 cycle, then that subpopulation reaches the following size:

$$S(p) = S_{min} + p(S_{max} - S_{min}) \quad (4)$$

172 During growth, individuals compete through differential reproduction. Each
173 individual's probability of success is proportional to its fitness. The composi-
174 tion of a subpopulation with size P and cooperator proportion p after growth
175 is multinomial with parameters $S(p)$ and $\{\pi_1, \pi_2, \dots, \pi_P\}$, where π_i represents
176 the reproductive fitness of individual i relative to others in its subpopulation
177 (using Equation 2).

178 **Mutation**

179 For simplicity, we apply mutations after subpopulation growth. Mutations
180 occur independently at each locus and cause an allelic state change. At the

181 binary cooperation locus, mutations occur at rate μ_c . These mutations flip
182 the allelic state, causing cooperators to become defectors and vice versa. Mu-
183 tations occur at rate μ_a at each adaptive locus. These mutations replace the
184 existing allele with a value randomly sampled from the set $\{0, 1, \dots, A\}$.

185 **Migration**

186 Populations consist of N^2 patches arranged as an $N \times N$ lattice, where each
187 patch can support a subpopulation. After mutation, individuals emigrate to
188 an adjacent patch with probability m . During each migration event, a single
189 destination patch is randomly chosen from each source patch's Moore neigh-
190 borhood, which is composed of the nearest 8 patches on the lattice. Because
191 the population lattice has boundaries, patches located on the periphery have
192 smaller neighborhoods.

193 **Population Initialization and Simulation**

194 Following Hammarlund *et al.* (2015), we begin simulations with sparse pop-
195 ulations. Subpopulations are first seeded at all patches with size $S(p_0)$ and
196 cooperator proportion p_0 . The population is then thinned. Each individual
197 survives this bottleneck with probability σ . Starting from this initial state,
198 simulations then proceed for T cycles, where each discrete cycle consists of
199 subpopulation growth, mutation, migration, and dilution. Dilution reduces
200 each subpopulation to support growth in the next cycle. Each individual re-
201 mains with probability d , regardless of its genotype.

Simulation Source Code and Software Dependencies

The simulation software and configurations for the experiments reported are available online (Connelly *et al.*, 2015). Simulations used Python 3.4, 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). Reported confidence intervals were estimated by bootstrapping with 1000 re-samples.

Results

Using the model described in the previous section, we perform simulations that follow the evolution of cooperation in a population of subpopulations that are connected by spatially-limited migration. Individuals increase their competitiveness by gaining adaptations. While cooperation does not directly affect the fitness benefits that these adaptations confer, it does have indirect effects on the adaptive process. Specifically, cooperation increases subpopulation density. As a result, larger subpopulations of cooperators experience more mutational opportunities. Cooperation can rise in abundance by hitchhiking along with beneficial mutations, which compensate for the cost of cooperation. Importantly, subpopulations alter their local environments, which feeds back to influence selection. Here, we explore how such niche construction affects the evolution of cooperation.

Cooperation Persists with Niche Construction

Without any opportunity for adaptation ($L = 0$), cooperators are swiftly eliminated (Figure 1A). Despite an initial lift in cooperator abundance due to increased productivity, the cost of cooperation becomes disadvantageous as migration mixes the initially isolated subpopulations. When populations can adapt to the external environment ($L > 0$ and $\delta > 0$), but niche construction is absent ($\epsilon = 0$), cooperators are maintained only transiently (Figure 1B). Here, larger cooperator subpopulations adapt more quickly to their external environment. As previously described by Hammarlund *et al.* (2015), cooperation is subsequently lost once populations become fully adapted. This occurs when isogenic defectors (i.e., defectors with identical adaptive loci) arise via mutation and displace cooperators due to their selective advantage. However, when niche construction feeds back to influence selection ($\epsilon > 0$), cooperation persists in the majority of replicate populations (Figure 1C). We see in Figure 2A that despite some oscillations, cooperation is maintained at high levels in the majority of these populations.

Fitness Increases Alone do not Support Persisting Cooperation

An individual's fitness is affected in this model by adaptations to both the external environment and to the constructed environment. Here, we determine whether cooperation is maintained solely due to the larger selective values that result from the contributions of niche construction. We performed simulations

244 in which these contributions were transferred to supplement the benefits con-
 245 ferred by adaptation to the external, non-constructed environment (replacing
 246 $\epsilon = 0.3$, $\delta = 0.3$ with $\epsilon = 0$, $\delta = 0.6$). In doing so, we conservatively estimate
 247 the selective effects of niche construction. Nevertheless, we find that simply
 248 increasing selective values does not enable cooperators to persist (Figure 2B).
 249 Niche construction, therefore, plays a decisive role here.

250 **Negative Niche Construction is Critical to Cooperator** 251 **Persistence**

252 In our model, an adaptation to the constructed environment initiates a new
 253 instance of niche construction, leading to sequentially increasing allelic states
 254 across the adaptive loci. Under certain conditions, this construction always
 255 makes the constructor sub-optimal for the niche it creates. This negative niche
 256 construction occurs when the number of adaptive alleles (A) does not divide
 257 evenly into the number of adaptive loci (L). In such a case, any sequence of
 258 integers on the circular genome will always contain a break in the sequence;
 259 that is, one locus will have an allele that is not one less than the allele at the
 260 next locus (see Box 1). Given this unavoidable mismatch, any type that has
 261 fixed will always construct a niche that favors selection for a different type.
 262 When negative niche construction is removed (by setting $L = 5$, $A = 5$), coop-
 263 erators are again driven extinct after an initial lift in abundance (Figure 2C).
 264 These results indicate that the type of niche construction matters. Specifically,
 265 negative niche construction is crucial for maintaining cooperation.

Selective Feedbacks Limit Defector Invasion

The adaptation resulting from selective feedbacks can limit invasion by defectors, which arise either through migration from neighboring patches or through mutation at the cooperation locus. This latter challenge is particularly threatening, as these isogenic defectors are equally adapted, yet do not incur the cost of cooperation. As demonstrated in [Figure 3A](#), isogenic defectors rapidly spread when introduced at a single patch in the center of a population of cooperators if mutations do not occur. However, cooperators resist defector invasion in over half of the replicate populations when adaptations can arise via mutation ([Figure 3B](#)). [Figure 4](#) depicts one such instance. In that population, isogenic defectors are seeded at a single patch in an otherwise all-cooperator population. These defectors quickly begin to spread. However, a neighboring cooperator population gains an adaptation, which increases its fitness above that of the defector. This type spreads more quickly, stopping the spread of defectors and eventually driving them to extinction. Because this adaptation occurs in a cooperator population, cooperation is able to hitchhike to safety. Importantly, this new cooperator is favored because of the niche that its ancestral type—and therefore also the defector—constructed. Here, cooperators can find safety in numbers—because their larger subpopulations have more mutational opportunities, they are more likely to gain adaptations that rescue them from invasion. Further, these larger cooperator subpopulations exert greater influence on their niches, which increases selection for an adapted type. This allows that type to appear and to spread more quickly in the population. [Figure 3C](#) shows how quickly an adapted cooperator type can invade a population

290 of defectors.

291 Discussion

292 Despite their negative effects, deleterious traits can rise in abundance through
293 genetic linkage with other traits that are strongly favored by selection (May-
294 nard Smith and Haigh, 1974). In a process termed the “Hankshaw effect”,
295 Hammarlund *et al.* (2015) recently demonstrated that cooperation can ac-
296 tively prolong its existence by increasing its likelihood of hitchhiking with a
297 beneficial trait. In that work and here, subpopulations of cooperators grow to a
298 higher density than those of defectors. These larger cooperator subpopulations
299 therefore experience more mutations and are consequently more likely to gain
300 adaptations. While this process does favor cooperation in the short term, it
301 eventually reaches a dead end: When the opportunities for adaptation are ex-
302 hausted, and cooperators can no longer hitchhike, they face extinction. Here,
303 we have investigated whether niche construction might serve to perpetually
304 generate new adaptive opportunities and thus favor cooperation indefinitely.

305 When niche construction occurs, cooperation can indeed persist (Figures 1C
306 and 2A). In our model, niche construction introduces additional selective ef-
307 fects that influence the evolutionary process, leading to a more pronounced
308 Hankshaw effect. However, these fitness benefits alone do not maintain co-
309 operation (Figure 2B). Niche construction and the selective feedbacks that it
310 produces play a crucial role.

311 We find that it is specifically *negative* niche construction that maintains coop-

312 eration (Figure 2C). As cooperator and defector types gain adaptations, they
 313 alter their environment in ways that favor other types. Thus, negative niche
 314 construction serves as a perpetual source of adaptation. Here we observe an-
 315 other facet of the Hankshaw effect: Because subpopulations of cooperators are
 316 larger, they are better able to respond to the adaptive opportunities that are
 317 created by negative niche construction. By gaining adaptations more quickly,
 318 cooperators resist invasion by defectors (Figure 3B). Even in the presence of
 319 an isogenic defector type, cooperator subpopulations are more likely to pro-
 320 duce the mutant most adapted to the current niche, which can then displace
 321 the slower-adapting defectors. These recurring cycles of defector invasion and
 322 cooperator adaptation underlie the oscillations in cooperator proportion seen
 323 in Figure 2A. When mutations do not confer these adaptations, cooperators
 324 lose the adaptive race and are driven to extinction. This is something that we
 325 see occur stochastically in Figures 2A and 3B.

326 Cooperation as Niche Construction

327 In our model, niche construction and adaptation are independent of cooper-
 328 ation, which allows us to focus on hitchhiking. However, individuals often
 329 cooperate in ways that alter the environment. These cooperative behaviors,
 330 therefore, can be seen as niche construction. For example, bacteria produce a
 331 host of extracellular products that scavenge soluble iron (Griffin *et al.*, 2004),
 332 digest large proteins (Diggle *et al.*, 2007; Darch *et al.*, 2012), and reduce the
 333 risk of predation (Cosson *et al.*, 2002), among many others (West *et al.*, 2007a).

334 As in our model, these forms of cooperation are likely to increase local sub-
335 population density. While many studies have focused on how the environment
336 affects the evolution of these cooperative traits, relatively few have addressed
337 how the environmental changes created by these products feed back to influ-
338 ence evolution.

339 Perhaps most similar to this study, Van Dyken and Wade (2012) demonstrated
340 that when two negative niche constructing, cooperative behaviors co-evolve,
341 selection can increasingly favor these traits, which are otherwise disfavored
342 when alone. In that model, “reciprocal niche construction” occurred when
343 the negative feedback resulting from one strategy positively influenced selec-
344 tion for the other, creating a perpetual cycle that maintained both forms of
345 cooperation. Arguably, this can be seen as an instance of hitchhiking: the
346 currently-maladaptive form of cooperation is maintained by association with
347 the adaptive form.

348 When dispersal is limited, competition among kin can undermine cooperation.
349 To separate kin competition from kin selection, Lehmann (2007) developed
350 a model in which a cooperative, niche-constructing behavior only benefitted
351 future generations. Kin competition was thereby reduced, and cooperation
352 instead benefitted descendants. This work highlights an important aspect of
353 niche construction: Often, the rate of selective feedback from niche construc-
354 tion is different from the rate at which populations grow.

Evolution at Multiple Timescales

In our work, the niche is modeled implicitly by the composition of the subpopulation. Any changes in the subpopulation, therefore, produce immediate effects on the constructed environment and the resulting selective feedbacks. However, timescales in our model could be de-coupled in two ways. First, cooperators modify their niche by enabling their subpopulation to reach larger density (Equation 4). These increased subpopulation sizes play a critical role by effectively increasing the rate of evolution in these subpopulations. Because of the importance of this process, it would be very informative to explore how sensitive our results are to the rate at which cooperators increase subpopulation sizes and the rate at which this benefit decays in the absence of cooperators. Similarly, our results could be substantially affected by alterations in the rate at which the constructed environment changes in response to changes in the subpopulation.

Other studies, while not focused on cooperation, have similarly shown that the timescales at which niche construction feedbacks occur can strongly influence evolutionary outcomes (Laland *et al.*, 1996, 1999). This perspective is likely to be crucial for understanding the evolution of cooperative behaviors like the production of public goods. In these instances, environmental changes are likely to occur on different timescales than growth, which can have profound effects. For example, a multitude of factors, including protein durability (Brown and Taddei, 2007; Kümmerli and Brown, 2010), diffusion (Allison, 2005; Driscoll and Pepper, 2010), and resource availability (Zhang and Rainey, 2013; Ghoul

378 *et al.*, 2014) influence both the rate and the degree to which public goods alter
 379 the environment. While Lehmann (2007) showed that cooperation was favored
 380 when selective feedbacks act over longer timescales, niche construction may in
 381 fact hinder cooperation when selection is more quickly altered. For example,
 382 when public goods accumulate in the environment, cooperators must decrease
 383 production to remain competitive (Kümmerli and Brown, 2010; Dumas and
 384 Kümmerli, 2012). This favors cooperation that occurs facultatively, perhaps
 385 by sensing the abiotic (Bernier *et al.*, 2011; Koestler and Waters, 2014) or bi-
 386 otic environment (Brown and Johnstone, 2001; Darch *et al.*, 2012). To study
 387 how regulatory traits such as these evolve, we could instead represent the niche
 388 explicitly, allowing it to have its own dynamics.

389 **Cooperation and Niche Construction in Host-Symbiont** 390 **Co-Evolution**

391 In many biological systems, the environments modified by organisms are other
 392 organisms. In these instances, the “niche” becomes a biological entity with its
 393 own evolutionary process. A logical extension to our model would be to treat
 394 the environment as an organism. Such a model could be used to explore the
 395 evolution of cooperation in host-symbiont systems, where cooperation among
 396 symbionts affects host fitness. As the host population changes, either in re-
 397 sponse to symbiont cooperation or other factors, so too does selection on their
 398 symbiont populations. In our model, each patch could become hosts with their
 399 own genotypes, and death and reproduction at the host level could be defined

400 in ways that are sensitive to both host and symbiont genotypes. Here, evolu-
401 tionary outcomes depend greatly on the degree of shared interest between the
402 host and symbiont.

403 Of particular importance are cases where the interests of host and symbiont
404 are in conflict. By selecting for new, more resistant host genotypes or by pro-
405 voking a specific immune response, pathogens make their host environment
406 less hospitable and can therefore be seen as potent negative niche construc-
407 tors. The results that we have presented here suggest that such negative niche
408 construction can favor cooperative behavior among these symbiont pathogens.
409 This may be especially relevant when infection is mediated by cooperative be-
410 haviors. For example, the cooperative production of several public goods by
411 *P. aeruginosa* facilitate infection in hosts with cystic fibrosis (Harrison, 2007).
412 Models such as what we have described may permit exploration into how coop-
413 eration and niche construction intersect here and in other medically-relevant
414 instances.

415 More generally, it was recently argued that incorporating the effects of niche
416 construction is critical for improving our understanding of viral evolution
417 (Hamblin *et al.*, 2014) and evolution in co-infecting parasites (Hafer and Milin-
418 ski, 2015). Incorporating host dynamics, co-evolution, and the feedbacks that
419 they produce is likely to be equally important for gaining a greater understand-
420 ing of how cooperative behaviors evolve in these host-symbiont settings.

Acknowledgments

We are grateful to Peter Conlin, Sylvie Estrela, Carrie Glenney, Martha Kornelius, and Luis Zaman for helpful comments on the manuscript, and to Anuraag Pakanati for assistance with simulations. BK thanks Kevin Laland, Marc Feldman, John Odling-Smee, Lucy Odling-Smee, and Doug Irwin for the invitation to participate in the *Frontiers in Niche Construction* meeting at SFI. This material is based upon research supported by the National Science Foundation under Grant DBI-1309318 (Postdoctoral Research Fellowship in Biology to BDC), Cooperative Agreement DBI-0939454 (BEACON STC), and Grant DEB-0952825 (CAREER Award to BK). Computational resources were provided by an award from Google Inc. (to BDC and BK).

Box 1: Description of niche construction in our model

See [Figure B1](#)

(A) Individuals. The genome of each individual consists of a single *cooperation locus* and L *adaptive loci* (here, $L = 5$). At the cooperation locus (labeled 0), this individual has allele 1, making it a cooperator. The adaptive loci (labeled 1 - 5) are arranged as a circular chromosome, where each locus has an integer allele between 0 and A , inclusive. In the description that follows, we focus exclusively on these adaptive loci. Genotypes are given by their allelic states starting with locus 1 . For instance, the genotype shown here is $[2,0,5,2,1]$. Because of their circular structure, allele 2 at the first locus follows allele 1 at the fifth locus.

(B) Niche Construction. Consider a subpopulation fixed for genotype $[1,2,0,0,0]$. This subpopulation constructs environment $E_{[1,2,0,0,0]}$. Every non-zero allele influences selection at the next locus, favoring sequential allelic states. In this constructed environment, allele 3 at locus 3 would be favored. If genotype $[1,2,3,0,0]$ arises via mutation, it is expected to fix. However, genotype $[1,2,3,0,0]$ affects the environment differently. As $[1,2,3,0,0]$ rises in abundance, the constructed environment changes to $E_{[1,2,3,0,0]}$, which favors $[1,2,3,4,0]$.

(C) Niche Construction and Adaptation. The evolutionary transition shown in Part B is indicated in the dashed box. Here, we depict entire sub-

454 populations fixed for a genotype using a single instance of that genotype. Simi-
 455 larly, an arrow represents niche construction and adaptation to the constructed
 456 environment. We start with a case in which there are five alleles ($A = 5$). Sub-
 457 populations begin with the non-adapted genotype $[0,0,0,0,0]$, shown on the far
 458 left. A non-zero allele is introduced via mutation, which represents an adapta-
 459 tion to external aspects of the environment. Here, allele 1 arises and fixes at
 460 locus 1. The remainder of this figure focuses on adaptation to the constructed
 461 aspects of the environment. This genotype has a mismatch (shown by the red
 462 sector), because $E_{[1,0,0,0,0]}$ favors $[1,2,0,0,0]$. Assuming allele 2 arises at the
 463 second locus, it will be selected, creating a match at the first and second loci
 464 (green sector). Now there is a mismatch between the second and third loci
 465 in the resulting environment, which a new round of mutation and selection
 466 corrects, and so on. The green sector grows as the red sector shifts clockwise.
 467 When the population reaches $[1,2,3,4,5]$, it constructs $E_{[1,2,3,4,5]}$. Here, since
 468 allele 1 follows allele 5, there is no longer a mismatch, so no further adaptation
 469 occurs.

470 **(D) Negative Niche Construction.** A different case emerges when the
 471 number of alleles does not evenly divide into the number of loci. Here, we
 472 change the number of alleles to six ($A = 6$). As shown on the far left, we
 473 begin with a subpopulation fixed for genotype $[1,2,3,4,5]$. This genotype has a
 474 mismatch, because the niche constructed by allele 5 favors allele 6 (not 1) at the
 475 next locus (locus 1). A mutant with genotype $[6,2,3,4,5]$ has a fitness advantage
 476 and can fix in $E_{[1,2,3,4,5]}$. However, as this type constructs $E_{[6,2,3,4,5]}$, a new
 477 mismatch appears. In this instance of negative niche construction, adapting

478 to correct one mismatch generates a new mismatch. This system can never
479 escape its mismatches—the red sector just shifts clockwise around the genome
480 perpetually.

481 **Figures**

482 **Figure 1**

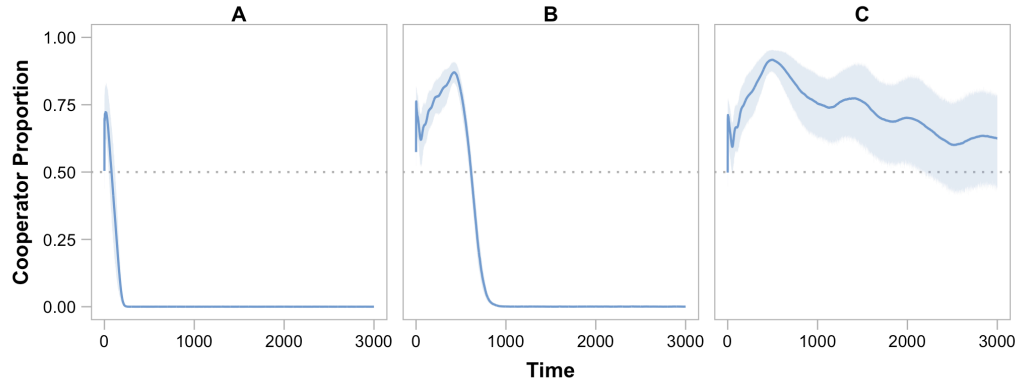


Figure 1: **Adaptation and the Evolution of Cooperation.** The average cooperator proportion among replicate populations for the duration of simulations are shown as curves, and shaded areas indicate 95% confidence intervals. **(A)** Without any opportunity to adapt ($L = 0$), cooperation is quickly lost. **(B)** When adaptation can occur ($L = 5$, $\delta = 0.3$), but niche construction does not affect selection ($\epsilon = 0$), cooperators rise in abundance by hitchhiking along with adaptations to the external environment. Nevertheless, this effect is transient, and cooperators eventually become extinct. **(C)** Niche construction ($\epsilon = 0.00015$) enables cooperation to be maintained indefinitely in the majority of populations. The trajectories of individual populations are shown in Figure 2A.

483 **Figure 2**

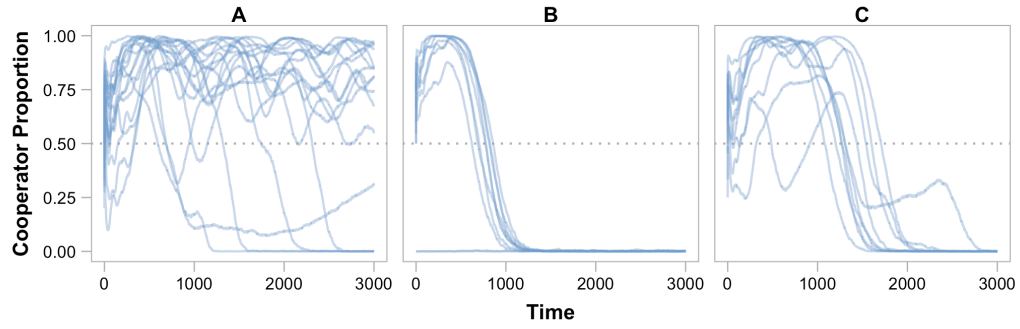


Figure 2: **Niche Construction and the Evolution of Cooperation.** The proportion of cooperators present in each replicate population is shown for the duration of simulations. **(A)** Despite some oscillations, cooperators dominate in 13 of 18 populations when niche construction affects selection. **(B)** When the selective effects of niche construction are transferred to supplement the benefits conferred by adaptation to the external, non-constructed environment, cooperators are driven to extinction by defectors (replacing $\epsilon = 0.3$, $\delta = 0.3$ with $\epsilon = 0$, $\delta = 0.6$). Note that cooperation was not present after initialization in one replicate population. **(C)** Cooperators are also driven to extinction without negative niche construction ($A = 5$).

484 **Figure 3**

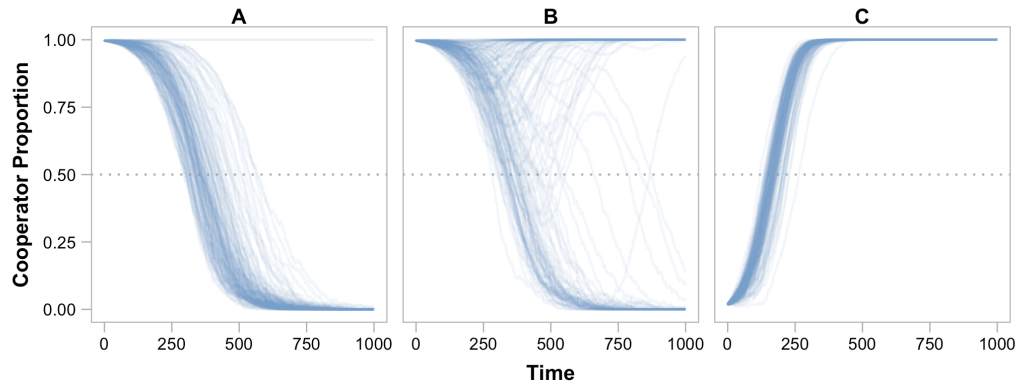


Figure 3: **Niche Construction and Invasion.** The proportion of cooperators present in each replicate population is shown for the duration of simulations ($T = 1000$). In each simulation, a rare type was initiated at a single patch in the center of the population lattice ($N^2 = 121$). Unless otherwise noted, mutations are disabled in these ecological simulations to highlight the dynamics of invasion ($\mu_a = 0, \mu_c = 0$). **(A)** When cooperators and defectors are isogenic (i.e., both types have stress alleles $[1,2,3,4,5]$), rare defectors quickly invade and drive cooperators to extinction due to the cost of cooperation. Defectors were stochastically eliminated in 2 replicate populations. **(B)** However, negative niche construction creates adaptive opportunities that enable cooperators to resist invasion by isogenic defectors. When adaptive mutations occur ($\mu_a = 0.00005$), cooperation remained dominant in 91 of 160 populations. Results from simulations where mutations also occurred at the cooperation locus are shown in Figure S1. **(C)** In fact, a cooperator (stress alleles $[6,2,3,4,5]$, see Box 1) that is adapted to the niche constructed by the defectors can swiftly displace defectors.

485 **Figure 4**

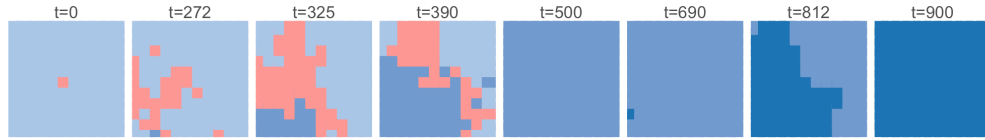


Figure 4: **Cooperator Adaptation Prevents Defector Invasion.** The spatial distribution of dominant types among subpopulations is shown at different time points for one representative simulation in which isogenic defectors arise. To highlight the effects of adaptation, mutations did not occur at the cooperation locus ($\mu_c = 0$). At time $t = 0$ (upper left panel), a single isogenic defector subpopulation (red) is placed within an all-cooperator population (light blue). Because these defectors do not bear the cost of cooperation, they quickly spread ($t = 272$). However, cooperators in one subpopulation gain an adaptation that gives them a fitness advantage over defectors (second panel, medium blue, lower left). At $t = 325$, defectors continue to invade cooperator subpopulations. However, the adapted cooperator type spreads more quickly due to its fitness advantage, invading both defector and ancestral cooperator subpopulations ($t = 390$), until it eventually fixes in the population ($t = 500$). At $t = 690$, a new cooperator type emerges that is favored due to negative niche construction (dark blue). This new type spreads rapidly ($t = 812$) until reaching fixation ($t = 900$). At this point, it becomes susceptible to invasion by the next “adapted” cooperator type, and the cycle continues.

486 **Box 1 Figures**

487 **Figure B1**

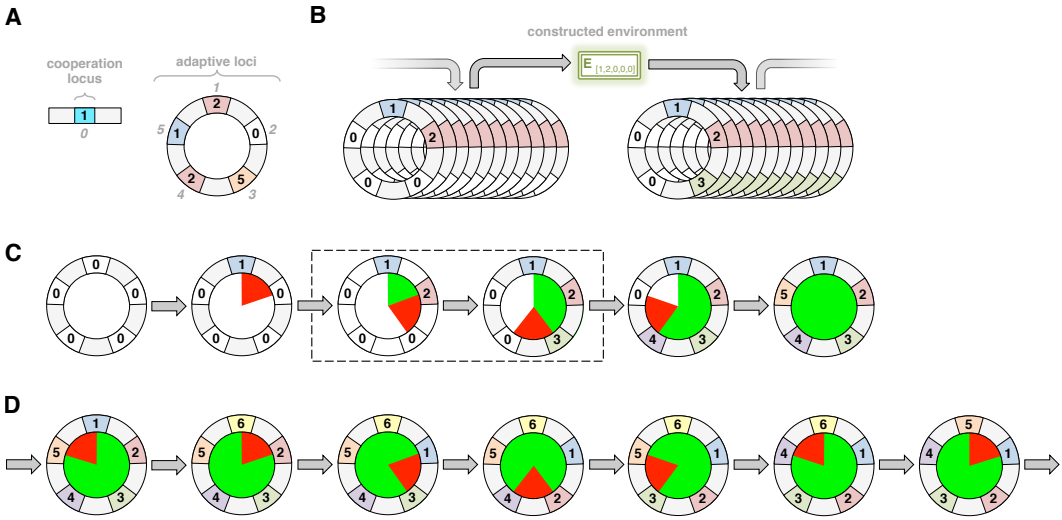


Figure B1: Figure for Box 1

488 **Supplemental Figures**

489 **Figure S1**

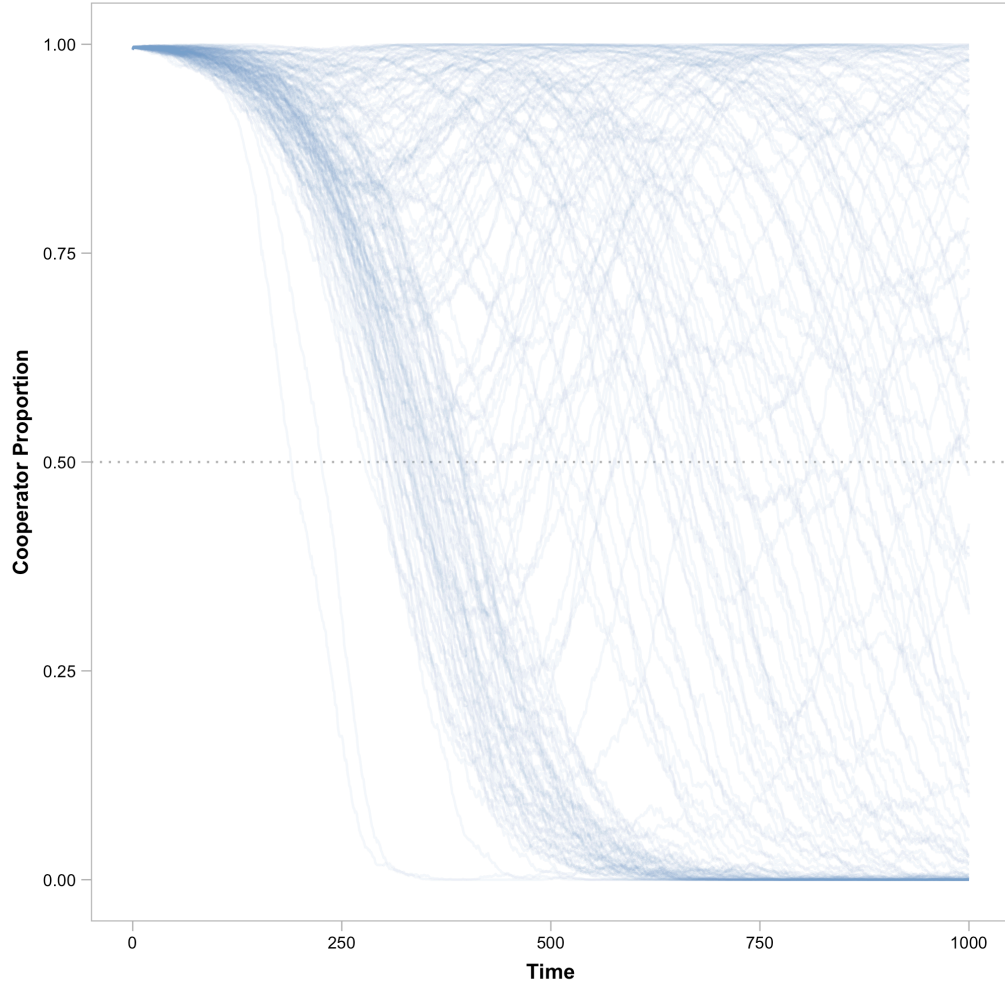


Figure S1: **Defector Invasion with Mutations.** The proportion of co-operators present in each replicate population is shown for the duration of simulations ($T = 1000$). When mutations occur both at the adaptive loci and the cooperation locus ($\mu_a = \mu_c = 0.00005$), cooperation remains dominant in 58 of 160 replicate populations.

490 **Tables**

Table 1: Model parameters and their values

Parameter	Description	Base Value
L	Number of adaptive loci	5
c	Cost of cooperation	0.1
A	Number of alleles	6
δ	Benefit of adaptation to external environment	0.3
ϵ	Benefit of adaptation to constructed environment	0.00015
z	Baseline fitness	1
S_{min}	Minimum subpopulation size	800
S_{max}	Maximum subpopulation size	2000
μ_a	Mutation rate at adaptive loci	10^{-5}
μ_c	Mutation rate at cooperation locus	10^{-5}
N^2	Number of patches	625
m	Migration rate	0.05
p_0	Initial cooperator proportion	0.5
σ	Survival rate at population initialization	10^{-5}
T	Number of simulation cycles	3000
d	Subpopulation dilution factor	0.1

491 **Version 2**

Parameter	Description	Base Value	Values Used
L	Number of adaptive loci	5	0,40
c	Cost of cooperation	0.1	0.1
A	Number of alleles	6	5, 6
δ	Benefit of adaptation to external environment	0.3	0.3
ϵ	Benefit of adaptation to constructed environment	0.00015	TODO
z	Baseline fitness	1	TODO
S_{min}	Minimum subpopulation size	800	TODO
S_{max}	Maximum subpopulation size	2000	TODO
μ_a	Mutation rate at adaptive loci	10^{-5}	10^{-5}
μ_c	Mutation rate at cooperation locus	10^{-5}	10^{-5}
N^2	Number of patches	625	625
m	Migration rate	0.05	TODO
p_0	Initial cooperator proportion	0.5	TODO
σ	Survival rate at population initialization	10^{-5}	TODO
T	Number of simulation cycles	3000	TODO
d	Subpopulation dilution factor	0.1	TODO

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](https://doi.org/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, 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.
- Brown, S.P. and Taddei, F. 2007. The durability of public goods changes the dynamics and nature of social dilemmas. *PLoS ONE*, **2**: e593.
- Connelly, B.D., Dickinson, K.J., Hammarlund, S.P. and Kerr, B. 2015. Model, data, and scripts for negative niche construction favors the evolution of cooperation.
- 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–

514 3033.

515 Dandekar, A.A., Chugani, S. and Greenberg, E.P. 2012. Bacterial quorum
516 sensing and metabolic incentives to cooperate. *Science*, **338**: 264–266.

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

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

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

524 Dumas, Z. and Kümmerli, R. 2012. Cost of cooperation rules selection for
525 cheats in bacterial metapopulations. *Journal of Evolutionary Biology*, **25**:
526 473–484.

527 Fletcher, J.A. and Doebeli, M. 2009. A simple and general explanation for the
528 evolution of altruism. *Proceedings of the Royal Society B: Biological Sciences*,
529 **276**: 13–19.

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

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

533 Ghoul, M., West, S.A., Diggle, S.P. and Griffin, A.S. 2014. An experimental
534 test of whether cheating is context dependent. *Journal of Evolutionary Biology*,
535 **27**: 551–556.

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

538 Hafer, N. and Milinski, M. 2015. When parasites disagree: Evidence for
539 parasite-induced sabotage of host manipulation. *Evolution*, **69**: 611–620.

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

543 Hamblin, S.R., White, P.A. and Tanaka, M.M. 2014. Viral niche construction
544 alters hosts and ecosystems at multiple scales. *Trends in Ecology & Evolution*,
545 **29**: 594–599.

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

548 Hammarlund, S.P., Connelly, B.D., Dickinson, K.J. and Kerr, B. 2015. The
549 evolution of cooperation by the Hankshaw effect. *bioRxiv*, doi: [10.1101/016667](https://doi.org/10.1101/016667).
550 Cold Spring Harbor Labs Journals.

551 Harrison, F. 2007. Microbial ecology of the cystic fibrosis lung. *Microbiology*,
552 **153**: 917–923.

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

556 Kuzdzal-Fick, J.J., Fox, S.A., Strassmann, J.E. and Queller, D.C. 2011. High
557 relatedness is necessary and sufficient to maintain multicellularity in Dic-

558 tyostelium. *Science*, **334**: 1548–1551.

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

562 Laland, K.N., Odling-Smee, F.J. and Feldman, M.W. 1999. Evolutionary con-
563 sequences of niche construction and their implications for ecology. *Proceedings*
564 *of the National Academy of Sciences*, **96**: 10242–10247.

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

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

570 Maynard Smith, J. and Haigh, J. 1974. The hitch-hiking effect of a favourable
571 gene. *Genetics Research*, **23**: 23–35.

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

575 Morgan, A.D., Quigley, B.J.Z., Brown, S.P. and Buckling, A. 2012. Selection
576 on non-social traits limits the invasion of social cheats. *Ecology Letters*, **15**:
577 841–846.

578 Nadell, C.D., Foster, K.R. and Xavier, J.B. 2010. Emergence of spatial struc-
579 ture in cell groups and the evolution of cooperation. *PLoS Computational*

580 *Biology*, **6**: e1000716.

581 Nowak, M.A. 2006. Five rules for the evolution of cooperation. *Science*, **314**:
582 1560–1563.

583 Odling-Smee, F.J., Laland, K.N. and Feldman, M.W. 2003. *Niche construc-*
584 *tion: The neglected process in evolution*. Princeton University Press.

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

587 Sinervo, B., Chaine, A., Clobert, J., Calsbeek, R., Hazard, L. and Lancaster, L.
588 *et al.* 2006. Self-recognition, color signals, and cycles of greenbeard mutualism
589 and altruism. *Proceedings of the National Academy of Sciences*, **103**: 7372–
590 7377.

591 Van Dyken, J.D. and Wade, M.J. 2012. Origins of altruism diversity II: Run-
592 away coevolution of altruistic strategies via “reciprocal niche construction”.
593 *Evolution*, **66**: 2498–2513.

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

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

600 West, S.A., Diggle, S.P., Buckling, A., Gardner, A. and Griffin, A.S. 2007a.
601 The social lives of microbes. *Annual Review of Ecology, Evolution, and Sys-*

602 *tematics*, **38**: 53–77.

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

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