Model Description

Using a computational model, we observed the relationship between adaptation to stress and the production of public good over evolutionary timescales. The simulation software and all configurations for the experiments reported are available at TODO. Simulations were run using Python 2.7.2, NumPy 1.8.1, and NetworkX 1.9. Data analyses were performed using R 3.1.1. Model parameters and their values are listed in [Table X](https://github.com/briandconnelly/niche_hike/wiki/Table%20of%20Parameter%20Values).

# Description of metapopulation, populations, and individuals

Each simulation tracks a single metapopulation comprising sites, arranged as an bounded lattice. Each site could potentially hold a population. Populations consist of individuals, where the genotype of each individual is a binary string of length . Alleles at the first loci (bits) determine the individual’s level of adaptation to the stressful environment. We refer to these loci as “stress loci.” The allele at the th locus determines whether the individual is a producer of a public good (allele ) or a non-producer (allele ). We refer to this locus as the “production locus.”

# Individual fitness

A mutation from to at the th stress locus will improve individual fitness by despite the allelic states of other loci (i.e., there is no epistasis). We assume that are independent and identically distributed (i.i.d.) random variables with . Public good production is costly, reducing individual fitness by . Thus, if the allelic state of the th locus is denoted (with ), then the fitness of an individual is:

where is a baseline fitness (the fitness of an individual with zeros at every locus). If there are no stress loci (), the fitness of a producer and non-producer is and , respectively.

# Overview of basic simulation cycle

Each simulation is run for cycles. Each cycle consists of population growth, mutation, migration, and dilution. We now describe each component of this basic cycle.

## Population Growth

If is the proportion of producers in a population at the beginning of a growth cycle, then that population grows to the following size over the growth cycle:

Therefore, a population with solely non-producers reaches a size of , while a population with only producers reaches a size of (with ). The function gauges the benefit of public good production, as population size increases linearly with the proportion of producers. During population growth, competition between genotypes occurs. There are possible genotypes. Consider an arbitrary genotype (with ). Let be the number of individuals with genotype , and let be the fitness of genotype (see equation [1]). The composition of genotypes after population growth is multinomial with parameters and , where

Thus, is the probability that an individual in the population after growth is genotype (such that ). Population growth occurs at every occupied site in the metapopulation.

## Mutation

For simplicity, we apply mutation after population growth. For each individual, every locus mutates independently. Each stress locus changes allelic state with probability while the production locus changes allelic state with probability . Thus, the probability that genotype mutates into genotype is given by

where and are the Hamming distances between genotypes and at the stress loci and production locus, respectively. The Hamming distance between two bit strings is the number of differing bits.

## Migration

Following mutation, migration of individuals occurs. For each populated site, a random adjacent site in its Moore neighborhood (the 8 nearest sites on the lattice) is chosen. Note, the metapopulation lattice has boundaries, thus sites on the edge of the metapopulation have fewer adjacent sites than those in the interior. Every individual in the focal site moves to the adjacent site with probability .

## Dilution

Following migration, populations were thinned to allow for growth in the next cycle. Each individual, despite its genotype, survived this bottleneck with probability .

## Stress Adaptation

The emergence of an environmental stress subjects populations to an additional bottleneck. Individuals survive this event with probability , which represents the likelihood that a mutation occurs, engendering survival in the new environment. Because individuals are not adapted to this new stress, the allelic state is set to at each stress locus. The fitness effects associated with adaptations at each locus are also regenerated as described in Individual Fitness. Note that this removes the influence of any previous stress. Simulations begin by applying this process to full populations initiated at each site with producer proportion .