

One hallmark of the Anthropocene period is a rapid change in the ever-new ways our bipedal species affect the wild cohabitants on Earth. Emerging evidence on many fronts suggest that human-induced environmental change can lead to marked evolution on decadal or even faster timescales. One eye-opening study from 2002 simulated intensive fishing and reported a twofold difference in body weight after just four generations of selectively harvesting either small or large individuals (1). However, merely documenting that rapid evolution takes place falls short of deciphering how such evolution occurs at a mechanistic level—a prerequisite for predicting evolution in new cases. On page XXXX of this issue, Therkildsen *et al.* (2) show that genomic changes that took place during the original 2002 experiment (1) partly evolved to align with variation along a natural gradient in the wild, but that strong selection also quickly erodes genetic variance.

Behind the many documented facets of contemporary global change—climate, species invasions, land-use modifications—lie untold stories of new and strong selection pressures (3). “A new selection pressure” is used as a gentler way of saying that individuals die sooner or reproduce less. Other individuals that better tackle the new environment will pass on their heritable traits, and so the population will evolve and maybe persist. This is how the ancestors of existing species have outlived every environmental challenge until now. But with the Anthropocene rate of change, a key question is whether evolution is fast enough to keep up?

A first approach to understanding evolutionary change is phenotype-centric and starts from knowledge about how organisms function in their environment (for example, physiology, behavior, and ecology). The key challenge here is to understand how phenotypes with certain heritable traits survive or reproduce better than alternative phenotypes. The Atlantic silverside, the species used in the experiment, occurs along a latitudinal gradient where clever experiments have demonstrated advantages of fast growth found toward north because of a short productive season, while southern fish grow more slowly because summers are longer and winters milder (4).

Rather ignorantly, the phenotype-centric approach assumes that when phenotypic traits are selected, corresponding changes at the molecular level will follow suit (5). This is easiest to grasp for continuous traits, that is, phenotypes that display a range of presentations such as body height or growth rate, as these are often influenced by the joint effects of perhaps hundreds of genes. If there is, say, a selective advantage of fast growth, then individuals having more of the gene variants (alleles) for fast growth also produce more offspring, and alleles for fast growth will accumulate in numbers (see the figure). The breath-taking diversity of life tells us that genomic constraints are less important if there is sufficient time, but with the current rate of anthropogenic change, time for evolution is limited.

When evolution approaches maximum rates, or when only a few genes in specific pathways are involved (6), it begins to matter that genes i) may have multiple effects in the complex biochemical network of a cell, and ii) are physical molecules—tiny stretches of nucleotides positioned somewhere on a vast DNA string. A next approach is needed, one that is DNA-centric and views evolution bottom-up, from genes to organisms. The focus is now on distinct alleles and their effects and interactions, which can make evolutionary trajectories deviate from those predicted by phenotype-centric methods. Under strong selection, certain alleles might confer significant benefits to their bearers and, over few generations, spread through the population like a brushfire (a so-called selective sweep). Another gene that happens to reside in a nearby chromosomal location can hitchhike to fame, and its effects on the organism can influence the evolutionary outcome, often substantially.

What happened in the silversides experiment (1) is particularly interesting, then, because selection was strong and evolution fast. From two populations, large fish had been removed and after four generations their phenotypes were markedly smaller. The opposite happened when small fish were harvested, and where harvesting was random there was no change in body size. Therkildsen *et al.* (2) pulled the experimental fish from 2002 (1) out of the freezer, sequenced their genomes, and searched for changes in genetic markers. In all of the experimental populations, thousands of genetic markers spread over nearly every chromosome changed in frequency and contributed to the evolved size differences. These results support the phenotype-centric view of polygenic evolution and are not surprising for a trait such as growth, which involves multiple underlying processes in biochemistry, behavior, metabolism, etc.

However, there was also strong evidence that attributes of the physical DNA-molecule altered evolutionary outcomes. In experimental tank Down2, one individual had a chromosome variant typical of slow-growing fish from the southern range of this species' distribution in the wild; this chromosomal variant swept through the Down2 experimental population to near total dominance. All alleles on this large part of chromosome became common, which is well explained by the DNA-centric view of evolution but would come as a surprise to anyone relying on phenotype-centric methods.

Importantly, Therkildsen *et al.* demonstrate how maintaining genetic diversity may be crucial to conserving species diversity. Rapid evolution of growth was made possible in part by pre-existing growth differences along the species' natural range. Alleles that became common in the fast-growing lab populations were shared with northern, fast-growing fish in the wild, and populations evolving slow growth accumulated southern-type alleles. Without the broad array of alternative alleles available in the starting populations, evolution would not have had the raw material to work from. If new genetic material instead had to arise from random mutations, the rate of evolution would have been orders of magnitude slower.

It is worrisome how quickly alleles disappeared during the short experiment. Alleles that strongly increased in frequency in one population did not necessarily change in parallel populations subjected to the same treatment. Thus, several genomic configurations led to similar phenotypes (7), and different alleles were lost in different populations. Such chance events are typical for small populations, known as the bottleneck effect. But, genetic variance eroded more quickly in the selected lines because it was not only few, but also similar, individuals that survived and bred. This suggests that one should extend the concern for small populations also to somewhat larger population size if they recently have been or currently are under strong selection.