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Evolution: Adapting to a Warming World

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To be able to cope with climate change, species need to evolve. Demonstrating such evolution in wild populations is notoriously difficult. Replication of a 21-year-old experiment demonstrates that a long-distance migratory songbird has genetically adapted to climate change.

Our world’s climate is rapidly changing, and species need to adapt to this warming world to persist. One key impact of climate change is on the seasonal timing — or phenology — of annually recurring events, such as migration, diapause or reproduction. There is ample evidence that phenology is shifting in response to climate change: marmots change their hibernation timing [1], birds lay their eggs earlier in spring [2] and the flowering dates of plants are advancing [3]. These shifts are to a large extent the result of phenotypic plasticity [4]: a bird will lay its eggs at an earlier time in spring in a warm compared to a cold spring. But this plasticity alone will not be sufficient to keep up with climate change and thus species need to adapt via genetic changes [5].

It is notoriously difficult to demonstrate that shifts in phenology in wild populations are (partly) due to genetic changes [6]. Distinguishing plasticity from genetic change by simply carrying out statistical analysis of long-term phenotypic data will not work but there are a few approaches that may be used to infer genetic change [7,8]. One of them is to analyse long-term data on

populations with a known pedigree. Using quantitative genetics, the genetic component underlying the trait (the so-called ‘breeding value’) can be calculated and regressed against year [9–11]. However, the long-term data on pedigreed populations needed for this are only available for very few species. Genomic analysis can also be used, either through the calculation of genomic breeding values or by analysing selective sweeps in the genome, and then associating phenotypes with genes near the genomic locations of these sweeps [12]. The latter approach has the advantage that no long-term data are needed but does rely on knowledge on which genes are involved in shaping the phenotype, which is often lacking for species that are not genomic model species. A recent study by Barbara Helm and colleagues [13] in *Current Biology* uses a much rarer but potentially powerful method to infer genetic change.

Helm and colleagues [13] carefully repeated an experiment in 2002, which was originally carried out in 1981, 21 years earlier, and assessed the change in phenology. This has been done for insects [14,15] but not for vertebrates.

The new study thus provides a unique example of a repeated experiment to study adaptive evolution using a vertebrate species: a long-distance migratory songbird, the pied flycatcher (*Ficedula hypoleuca*; Figure 1). Pied flycatchers winter in Africa and need to time migration to the European breeding grounds such that they arrive on time to take advantage of profitable spring conditions to raise their offspring. For this, the birds rely on an endogenous circannual timer, simply because there are no environmental signals in Africa that indicate when conditions at the breeding grounds are beneficial. This lack of informative cues for departure makes adaptation through phenotypic plasticity difficult, although once the journey has started flycatchers may adjust their migration speed depending on the conditions *en route*. To substantially shift flycatchers’ arrival and breeding phenology, genetic changes are needed.

Helm and colleagues [13] made use of the fact that endogenous circannual timing programs can be studied using captive birds. In captivity, migratory songbirds also go through their normal annual-cycle events, such as fattening,





Figure 1. Ready to breed. Pair of pied flycatchers (*Ficedula hypoleuca*) preparing to breed. The female carries nesting materials. Photo with kind permission from Ralph Martin (www.visual-nature.de).

moulting, gonadal activation and even ‘migration’, which can be recorded from the so-called ‘migratory restlessness’ of the birds, which, rather than sleeping, remain active at night. Helm and colleagues [13] took in nestlings from a long-term study population, the same population as was used 21 years earlier in the original study [16], which demonstrated that annual cycle in flycatchers was controlled by an endogenous circannual rhythmicity. The nestlings were hand-raised and their annual-cycles studied under natural photoperiods. From the timing of annual-cycle stages, timing indexes were calculated. Autumn timing index had shifted to a later date, winter timing index had not significantly shifted, but spring timing index had shifted clearly to an earlier date (by 9.3 days), and this shift was stronger in males than in females. Over the same 21-year period the local wild population, monitored by citizen scientists, advanced spring egg-laying by 11.2 days. Because the experiment was a meticulous repetition of the original one (see below), these differences cannot be due to plastic responses to different conditions and thus the flycatchers had changed genetically.

One of the limitations of repeated experiments is that there is often just

a single replicate after many years, although some insect studies have multiple repetitions [14,15]. Especially with a single repetition, the experiment needs to be replicated exactly to minimize spurious findings due to phenotypic plasticity, for instance. In the study of Helm and colleagues [13], the original set-up was very carefully repeated, and that was possible because the late Eberhard Gwinner, who conducted the original and repeated experiments, had retained the original set-up. Also, when new equipment was used, for example to measure activity, it was calibrated against the original equipment, in this case micro-switches against inkwriters, which were still available; sometimes it pays off to keep the old stuff around. Another key problem is to obtain the ‘same’ birds to be used in the repeat experiment. This was tricky, given that the wild population had shifted its phenology over the 21-year period. Birds can be collected at the same calendar date or at the same time relative to the mean population phenology. A strong point of the new study is that, although the new cohort was collected on the same calendar date, and thus from relatively later-laying parents, the birds shifted to an earlier date, opposite to what would be

expected based on relative sampling date. A final point that makes these results convincing is that it was predicted from literature that spring phenology would advance, while there was no prediction for autumn and winter phenology, and that the shift in phenology for spring events would be stronger in males. These predictions were confirmed by the findings.

Helm and colleagues [13] show that there have been genetic changes in a long-distance migratory songbird. There thus seems to be scope for contemporary evolution, which will contribute to the adaptation of species to climate change. But a word of caution: it is highly unlikely that the rate of this genetic change is sufficient to keep up with the speed of climate change. The phenotypic shifts that we see in the wild, which are a combination of phenotypic plasticity and genetic change, are often not sufficient to keep up with how much species should be shifting, *i.e.* the time of year where fitness peaks. The resulting mistiming [17] may lead to population declines, as has been shown for the pied flycatcher [18]. Only substantially reducing the rate of climate change to historical rates of warming, perhaps even with as much as a factor of 100 [19], will allow species to keep up via genetic change, as they have always done on our ever-changing planet.

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Tissue Repair: Guarding against Friendly Fire

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Following tissue injury, cells produce reactive molecules that fight off invading pathogens, but these factors might also damage the host tissue. A new study has characterized a network of defense pathways that synergize to protect cells from collateral damage and drive repair.

The ability of the body to repair itself following injury has been of central importance throughout evolution. From lower organisms to humans, sophisticated repair mechanisms work quickly to close the wound and restore tissue function, although to varying degrees depending on the species, its health condition and the location of the wound. In humans, defects in the body's tissue repair response can lead to an inability to close wounds in a timely manner, often resulting in infection. A frequent consequence is the development of chronic, non-healing wounds, which cause severe morbidity and can reduce life expectancy. Following injury, inflammatory cells travel to the wound site and produce large

amounts of highly reactive compounds called reactive oxygen species (ROS), which serve to damage and destroy invading pathogens. Despite the antimicrobial benefits, high amounts of ROS also cause damage to host cells, potentially leading to healing defects and the development of skin ulcers. It is therefore essential that host tissues have powerful mechanisms to protect cells from ROS-mediated damage. A new study published in this issue of *Current Biology* has thoroughly dissected these cytoprotective mechanisms: here, Weavers *et al.* [1] identify a collaborative relationship between different cell defense pathways that function to provide cells with a robust ability to withstand the harsh

conditions in wounded tissue, while working collectively to drive efficient repair.

Using *Drosophila*, which allows for easy genetic manipulation and is well-suited for live-tissue imaging, Weavers *et al.* [1] show that ROS levels increase substantially following injury, and originate primarily from the inflammatory cells that are recruited to the wound site. This explosion of ROS creates a destructive environment for nearby tissues and slows down the healing process: the authors found that depletion of immune cells (and the accompanying ROS) accelerated wound healing (Figure 1A) [1]. Certainly, this detrimental effect is outweighed by the importance of

