In summary, Drews *et al.* [1] and Matulis *et al.* [2] are the first to describe visual contrast adaptation in *Drosophila*, and narrow the space of models describing how contrast adaptation shapes motion computations. It is interesting to note that *Drosophila* is most active during dawn and dusk, when scene contrast would be expected to change rapidly. Furthermore, increasing the speed of a moving image reduces its effective contrast. Thus, in an organism that undergoes dramatic shifts in self-motion speed, moving between walking and flying during the most contrast-dynamic periods of day, a robust and flexible system of visual contrast adaptation is likely to have evolved. Thus, quite apart from their elegant experiments, the authors of these papers [1,2] have revealed how the fruit fly, long a powerful model for understanding the circuit mechanisms of motion detection, is also an exemplary model to uncover general principles of contrast adaptation. Qualitatively, these papers' perspectives, approaches, and conclusions about the same general biological phenomenon are sometimes similar, sometimes complementary, and sometimes provocatively different, so readers should consider for themselves how and why their conceptual overlap is only partial, and what that suggests about the underlying biology.

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# Evolution: Ancestral Plasticity Promoted Extreme Temperature Adaptation in Thermophilic Bacteria

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**Explaining the origins of adaptive features is a perennial challenge in evolutionary biology. A study on thermophilic cyanobacteria reveals how environmentally induced phenotypic change (plasticity) can pave the way for evolutionary innovation and subsequent adaptation to extreme conditions.**

Where do new complex features — adaptations — come from, especially those that enhance fitness in a given environment? This question lies at the heart of evolutionary biology, and it is generally assumed that new adaptations arise exclusively from genetic changes. Indeed, a variety of adaptive features can be traced to changes in the genome [1,2]. Yet, at least as early as 1896 [3], researchers also proposed that environmentally induced changes to an organism's phenotype (so called 'phenotypic plasticity') might facilitate and promote the emergence of new adaptations. This process has been dubbed 'plasticity-first' or 'plasticity-led evolution'. It occurs when a change in an organism's environment triggers a change in some aspect of its phenotype via phenotypic plasticity. If there is heritable variation in whether or how individuals respond to this change in the environment, then, and over generations, selection can act on this variation and stabilize, refine, and/or extend those adjustments in the phenotype that are best-suited to the altered environmental conditions. Eventually, if a particular phenotype (or phenotypic state) is consistently favored in

the new environment, then selection can lead to the evolutionary loss of plasticity and increased 'canalization' (that is, reduced sensitivity to the environmental change), thereby resulting in the constitutive expression of a trait that was previously inducible by the environment (a process referred to as 'genetic assimilation' [4]). Through this process, ancestral plasticity can 'jump start' the emergence of new adaptive features [5–7].

Despite its long history, plasticity-led evolution was largely ignored for much of the twentieth century because it was viewed as unimportant in evolution [8]. Recent decades, however, have witnessed renewed interest in identifying the role of plasticity-led evolution [5–7]. As part of this renaissance, there have been calls for more studies to evaluate plasticity-led evolution in natural populations of non-model organisms [9–11], and particularly microorganisms, which are underrepresented in such studies [12]. A new study by Miller and colleagues [13] in this issue of *Current Biology* answers this call by uncovering a role for plasticity-led evolution in adaptation of the cyanobacterium *Fischerella thermalis* to extreme temperature conditions.

*Fischerella* sp. are thermophilic, multicellular cyanobacteria found around hot springs and other high-temperature bodies of water worldwide (Figure 1) [14]. When the environment lacks 'combined' nitrogen (that is, nitrogen covalently bonded to other elements), these bacteria facultatively produce specialized cells — heterocysts — that can fix atmospheric nitrogen (that is, convert the nitrogen into a useable form). A requirement for this nitrogen fixation is the formation of a glycolipid layer on the surface of the heterocyst that acts as the primary barrier to gas diffusion. However, many cyanobacteria that form heterocysts harbor temperature-dependent plasticity in the composition of this glycolipid layer, with less permeable glycolipid layer compositions being beneficial at higher temperatures.

Miller and colleagues [13] began by comparing plasticity and growth among *Fischerella* strains that have diverged in thermotolerance. Previous work determined that low tolerance to high temperatures was likely the starting (ancestral) state and high heat tolerance the evolved (derived) state. For most of the strains Miller and colleagues tested, two structural isomers (II and IV) of 1-(O-hexose)-3,29,31-dotriacontanetriol increased at higher temperatures and decreased at lower temperatures. However, two strains independently lost this plasticity and produced these isomers at high levels across all treatment temperatures; that is, these strains became canalized. Notably, even the lowest levels of these isomers in canalized strains (at low temperatures) were greater than the highest levels of these isomers in plastic strains (at high temperatures). Moreover, strains with higher levels of these isomers grew faster at high temperatures, thereby linking isomer levels to performance. In general, these observations are consistent with genetic assimilation having occurred.

To explore the mechanisms and functional consequences of this reduced glycolipid composition plasticity, the authors next compared different strains from the same location as one of their canalized strains. In this population, three strains of the same multi-locus haplotype (MLH3) had canalized glycolipid composition. The authors further demonstrated that not only were these MLH3 strains constitutively expressing an overabundance of high-temperature glycolipid isomers, but doing so had a functional advantage. Namely, this overproduction actually limited diffusion (determined via rate of nitrogen fixation) and enhanced growth at high temperature. But this benefit came with costs: at lower temperatures, these canalized MLH3 strains had poorer nitrogen fixation, and consequently, reduced growth compared to plastic strains. From the opposite perspective, however, plastic strains experienced a cost not experienced by the canalized MLH3 strains at high temperature. Such costs of plasticity are sometimes considered to be crucial for genetic assimilation to occur [15]. Notably, the MLH3 strains predominate in parts of the habitat in which temperature exhibits relatively low variability — these are the perfect conditions for selection to ameliorate costs of plasticity by reducing the expression of plasticity and instead promoting canalization of the selectively favored phenotype(s).

**Figure 1. Plasticity pays off in the heat.**
The multicellular cyanobacterium *Fischerella thermalis* forms filamentous mats in nitrogen-limited, geothermally heated streams, such as White Creek (shown here) in Yellowstone National Park, USA. A paper by Miller and colleagues [13] shows that, during adaptation to high temperatures, specialized nitrogen-fixing cells have evolved multiple times from ancestral phenotypic plasticity. (Photo: David Pfennig.)

Finally, Miller and colleagues uncovered the potential genetic basis for the loss of plasticity in MLH3 strains. They identified 48 unique SNPs that were fixed in the MLH3 strains. Two of these SNPs are located in genes involved in heterocyst development and function, and one of these genes, *hglT*, was previously implicated in production of the glycolipid layer of the heterocyst [16]. The *hglT* SNP is found only in the canalized MLH3 strains and is not present in any other genome of *Fischerella* from around the globe.

Given these findings, the authors lay out a possible evolutionary route to the high-temperature glycolipid innovation present in the specialized nitrogen-fixing cells of *F. thermalis*. First, heterocyst glycolipid-composition plasticity is an ancestral trait: all assayed cyanobacteria genera exhibit changes in composition with temperature. There is also selectable diversity in this plasticity that is uncovered when the cyanobacteria experience high temperatures. Under stable temperatures, selection to maintain plasticity should be weakened (especially if there are costs to plasticity). As predicted, strains canalized for high-temperature isomers predominate in stable thermal environments, where plastic strains incur fitness costs. Thus, exposure to high temperatures likely uncovered cryptic variation in plasticity that allowed some strains to persist in the new environment. Because the environment was stable over generations, and because the plastic responses were sub-optimal, any beneficial mutations that stabilized or refined production of high-temperature isomers were likely favored by selection. In this way, plasticity uncovered variation, facilitated persistence in new conditions, and, in doing so, provided the time for further refinement of a key innovation: specialized nitrogen-fixing cells.

This study makes many contributions to our understanding of how plasticity might impact the evolution of new features. First, it provides an example of plasticity-led evolution in an underrepresented natural system: bacteria. This is crucial because understanding the types of traits and taxa experiencing plasticity-led evolution will help inform the conditions that foster versus impede this evolutionary process. Second, this work helps inform possible mechanisms of plasticity-led evolution. Specifically, this example resembles a 'buying time' model [17], wherein plasticity promotes population persistence in a novel environment (high temperature) by buffering the population from extinction until beneficial mutations or recombinants arise. In this case, plasticity bought time for a new mutation that led to genetic assimilation and fixation of the high-temperature glycolipid isomer phenotype. This mechanism is counter to the original formulation by Waddington [4], in which genetic assimilation came about through selection on standing genetic variation. Whether this process of buying time constitutes plasticity's primary role in evolution remains an open question [18,19]. Finally, Miller and colleagues found that genetic assimilation of the same phenotype has occurred multiple times, independently, and possibly by different means. It will be interesting to see how genetic assimilation occurred in the other canalized strain to determine what degree of parallelism exists at the molecular level.

In sum, Miller and colleagues [13] demonstrate that plasticity can play a leading role in the evolution of innovation in natural populations. Their work also moves the study of plasticity-led evolution forward by uncovering the mechanisms of genetic assimilation and the costs of plasticity, which are two of the remaining frontiers in the field [20].

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