Highly diverged conifer species share many genetic signals of adaptation to local climate conditions

By Angela M. Hancock

In convergent evolution, different species independently acquire similar traits. Knowing whether divergent species use the same or different genetic solutions to adapt to similar selection pressures can illuminate innate constraints on underlying molecular networks. Most cases of convergence that are understood at the genetic level are relatively simple, involving one or a few genes. On page 1431 of this issue, Yeaman et al. report mapping of locally adaptive genes in lodgepole pine (Pinus contorta) and interior spruce (the species complex that includes Picea engelmannii and Picea glauca) (see the photo). The authors show that many genes implicated in local adaptation are shared among the two species.

Many examples of convergent evolution at the morphological level have been documented, and in some cases the genetic basis for convergence is known. Examples include loss of pigmentation in mice, lizards, and humans via mutations in melanocortin-1 receptor (MC1R) (2), adjustment of reproductive timing in plants via flowering locus C (FLC) and its homologs (3, 4), loss of trichomes in Drosophila via shavenbaby (5), and resistance to toxic cardenalides in insects (6, 7).

However, these well-studied cases tend to involve genetically simple traits, in which one or two genes are credited with the repeated phenotypic changes (8). In these cases, pathway constraints have been invoked to explain cases where different species have adapted via changes in the same genes (9). The idea is that a pathway can only be perturbed in a limited number of ways; thus, the possible genetic modifications that can occur to enable adaptation are also limited. However, many adaptive traits are likely to be genetically complex, involving many small-effect variants (10). In these cases, adaptation could act in more varied ways, making extensive overlap in the genes underlying adaptive responses across diverged species improbable.

Plant species are particularly powerful models for examining local adaptation, because they cannot hide from environmental insults and must face them head-on. Yeaman et al. sequenced the protein-coding genome regions of more than 250 populations from two diverged conifer species, lodgepole pine and interior spruce. For each individual, they collected climatic data from the site of origin as well as phenotypic data, which were measured under simulated natural conditions. They then identified regions of the genome that were correlated with variation in phenotypes or climate to pinpoint loci implicated in local adaptation. In both species, the majority of traits and climatic factors with the strongest signals underlie the ability to deal with cold stress.

Given the observed convergence in cold tolerance adaptation, the authors next explored whether the associated loci were shared or different between the two conifer species. They expected the traits and the adaptive signals examined to involve many genes and overlap of the loci identified in the two species to be limited.

Yet they found extensive overlap, with 47 genes showing signals in both species (see the figure). Moreover, many shared signals were in genes that were duplicated in one or both species. This is consistent with the proposition that duplicated genes may be more likely than nonduplicated genes to be used in adaptive bouts because of reduced constraint on function. The reasoning is...
that when a gene is duplicated, one copy can take responsibility for some or all core functions, leaving the other copy free to evolve new or modified functions (11, 12).

The study by Yeaman et al. provides solid evidence that independent and parallel changes at the molecular level can underlie phenotypic convergence even for traits with complex genetic architectures. But many open questions remain. The genomes interrogated here are large (around 20 billion base pairs) and derived from non-model organisms; the genome assemblies are therefore incomplete, and functional annotation is not available for many loci. Given the interesting finding that duplicated genes show an enrichment of signals of convergent evolution, more complete assemblies will be useful. Improved genome assemblies will allow for finer-scale resolution of the relative adaptive importance of orthologs (genes derived directly from the same ancestral loci) and paralogs (genes derived from ancestral duplications within the genome).

In addition, knowing more about the functional consequences of the signals detected will provide a deeper understanding of the types of changes responsible for the observed adaptive evolution in conifers. For example, it will be interesting to know whether the observed convergent patterns are due to repeated losses of gene function across lineages, or to more subtle changes. Losses of function, which are common in single-gene convergences (2, 8), may occur readily when selection is strong because they have large effects and can be achieved in a variety of ways.

Yeaman et al.’s study is likely to become an important model for future research that examines convergence in local adaptation among species with overlapping ranges. Extending the approach to more diverse species will be important for determining whether the observed patterns are widespread in nature. ■

REFERENCES


NEUROSCIENCE

Cool by neuronal decision

An ion channel expressed in specific brain neurons is required for regulating core body temperature

By Tamas Bartfai

Inside the well-protected and well-ventilated human skull, the temperature of a few hundred thousand neurons can deviate by several degrees centigrade from that of the surrounding 100 billion or so cells. These cells constitute around 15% of the neurons in the brain’s preoptic area (POA) and can change their firing rate dramatically upon a 1° to 3°C change in local temperature (1). Astoundingly, this change in activity can alter the body’s core temperature. Cellular and molecular details of the warm and cold sensitivity of these neurons are not well understood. Although the induction of fever has been well studied, the mechanisms that terminate fever are not yet clear. On page 1393 of this issue, Song et al. (2) report that a transient receptor potential (TRP) cation channel (3, 4) is key to the function of these neurons in thermoregulation, particularly in response to fever.

Human core body temperature is kept within the most narrow range of all the human physiological parameters (compared with blood pressure and blood glucose concentration). Measuring body temperature remains the most widely used, noninvasive method of monitoring the presence of infection or inflammation. The state of having a fever—a coordinated stress response that, through increased thermogenesis and reduced heat loss, is achieved by vasoconstriction—was shown to be regulated by the POA (5, 6). But how does change in the firing rate of temperature-sensitive POA neurons bring about a change in body temperature? A proposed signaling pathway involves the relay of information from POA neurons to brown adipose tissue, via the dorsomedial hypothalamic nucleus, the nucleus raphe pallidus in the brain stem, and postganglionic fibers (see the figure) (7). In this scheme, a fever-inducing substance (pyrogen), such as the hormone prostaglandin E2 (PGE2) or the cytokine interleukin-1 (IL-1), can inhibit the activity of warm-sensitive POA neurons and ultimately change the uncoupling mechanism in the mitochondria of brown adipose tissue, causing heat production (instead of generating energy in the form of adenosine triphosphate).

When transcripts from single, warm-sensitive neurons were eventually characterized, it was clear that the majority release the neurotransmitter γ-aminobutyric acid (GABA), with a smaller population releasing the neurotransmitter glutamate (8). Moreover, it was also established that receptors for various pyrogens—including PGE2, IL-1, and metabolic hormones such as insulin and adiponectin—are present in some warm-sensitive neurons (8). Thus, heat production and concomitant vasoconstriction, which jointly increase core body temperature during fever, could be explained (5–7), but the mechanism(s) responsible for terminating the fever response remained an enigma.

TRP channels expressed in the peripheral nervous system and the skin (TRPV1–8) have been well studied (3, 4). Those expressed in skin keratinocytes are thought to play an important role in nociception, including sensing heat. Although transcripts encoding some TRP channels had been identified in warm-sensitive neurons of the hypothalamus, their functional importance was not understood because the temperature range in which peripheral TRP channels are activated is outside the range that activates POA neurons.

Song et al. show, with in situ hybridization and immunohistochemistry, that many mouse POA neurons express TRPM2, which is a thermosensitive and redox-sensitive channel. The authors observed that the sensitivity of these neurons to warm temperature is affected by TRPM2 sensitizers, including the reactive oxygen species H2O2. They also noted the sensitivity of warm-sensitive POA neurons expressing TRPM2 in vitro to either an intracellular agonist of the adenosine diphosphate receptor or an exogenous inhibitor of TRP channels called 2-aminoethoxydiphenyl borate (2-APB). Moreover, Song et al. show that these TRPM2-expressing neurons

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Editor's Summary

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