Fish Vision

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SPECTRAL TUNING

Evolutionary geneticist Shozo Yokoyama, who studies the evolution of vision in vertebrates, is a pioneer in the field of paleomolecular biology. It's a field that's sexier than it sounds. Not only has he characterized the visual pigments of extant vertebrates but he creates ancestral forms that correspond to transitional nodes in evolution. What's more, these pigments can be tested in the lab to determine the wavelength of light they maximally absorb. Yokoyama then puts all this physical chemistry into ecological and behavioral perspective.

Take, for example, the recent study in which Yokoyama and his colleagues at Emory University report the first violet-sensitive SWS1 opsin found in fish (13 October 2009, *Proceedings of the National Academy of Sciences*). Vertebrates have just five types of visual pigments, or opsins, that mediate vision in different parts of the light spectrum, from ultraviolet (UV) to far red and all the colors in between. Only one of the five opsins, SWS1 (for short wavelength–sensitive type 1), absorbs light at UV wavelengths (around 360 nanometers [nm]). The study reports that the SWS1 of scabbardfish has a single amino acid deletion that shifts its absorption maximum from UV to violet light (423 nm).

Why would scabbardfish shift to seeing blue light? UV vision is relatively common among vertebrates—it is, after all, an ancestral character state (another of Yokoyama's discoveries). There are many advantages to UV vision: It allows organisms to see in poor light, such as that around dawn and dusk; detect UV-reflective cues from other organisms; and find food. Yet UV light doesn't penetrate depths over 20 meters (m), where the light spectrum narrows to about 480 nm. Since scabbardfish live mostly at 25 to 100 m depths, it appears they lost nothing by spectrally adapting to the light available. Lampfish, on the other hand, live at 30 to 1200 m depths but have retained a UV-sensitive form of SWS1. The difference is that lampfish return to the surface to feed on tiny copepods, which are more readily visible in UV.

Visual pigments have proven to be a rewarding system for linking genetic variations with functional adaptations, a connection rarely achieved in evolutionary studies. Yokoyama continues to build on an impressive body of work. His study of rhodopsin pigments and dim-light adaptations in fish (9 September 2008, *PNAS*) exposed how misleading the underlying assumptions of standard evolutionary analyses can be. He demonstrated that functional adaptations in proteins do not always arise from single, advantageous amino acid changes, as the natural selection story goes, but often result after selectively neutral substitutions have had a chance to accumulate. This non-Darwinian type of evolution is random and difficult to track, making statistical analyses of the sequence data unreliable. Evolutionary adaptations become much clearer when they are confirmed at the functional level, as Yokoyama has done.

REGENERATION

When a bright light is shone in adult zebrafish eyes they go blind, but it's only temporary. Within a matter of days, glial cells in the injured fishes' retinas dedifferentiate to form neuronal stem cells, which then regenerate functional photoreceptors. In a study published last June in *PNAS,* University of Michigan scientists working in Pamela Raymond's lab analyzed the transcriptional profile of genes expressed during this process—a total of 953 genes, two of which are also key to regeneration of other tissues.

Zebrafish are a well-characterized experimental model for studying regeneration. In previous studies of zebrafish fins and hearts, the genes necessary for regeneration of amputated tissues have been shown to include a number of both common regeneration molecules and tissue-specific components. In the new study on retinal regeneration, graduate student Zhao Qin and her colleagues identify the same two core players at work in other tissues: *hspd1,* which encodes heat shock protein 60 and is necessary for stem cell formation, and *mps1* (*monopolar spindle 1*), a protein kinase needed for proliferation of progenitor cells. In addition, the temperature-sensitive mutants that have already been developed were useful in characterizing the stages of regeneration at which these genes are required in the retina.

The team improved on past studies of retinal regeneration in a couple of ways. They exposed zebrafish to shorter periods of more intense light, which damaged photoreceptors all at once, and they isolated the glial cells responsible for regeneration from other eye tissues for analysis. The result is a clearer picture of how zebrafish regenerate damaged retinas through the reprogramming of differentiated, nonneuronal cells. By comparing the profile of up-regulated genes in damaged retinas with those published for other regenerating tissues, they concluded that regeneration, and perhaps underlying mechanisms of development, may have much in common.

It was more than 100 years ago, well before the discovery of stem cells, that T. H. Morgan coined the terms that still distinguish two modes of regeneration: *epimorphosis,* which requires cell proliferation at the injured site, and *morphallaxis,* which does not. One wonders if our powerful modern techniques will reveal any similarities between these two superficially different methods of replacing lost tissue, or in creating it in the first place.

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