

ScienceWatch – Darwin in the Dark



I have kept tropical fish since I was a child and early on I became fascinated by an unusual tetra lacking eyes and known as the Mexican blind cave fish (*Astyanax mexicanus*). A very similar fish with normal eyesight is widespread in surface pools so it seems clear that the sighted fish gave rise to the blind ones as they invaded the total darkness of the caves. But how did this happen? How does any organism lose a structure it no longer needs?

A simple explanation would be “use it or lose it”. But disuse in one generation does not result in loss in the next. We could tie back the left leg of every newborn, causing all humankind to hop around on its right leg for many generations. Yet we would still give birth to babies with two normal legs. Non-functional structures are only lost by mutations in the genes responsible for their development and many are retained - consider the nipples borne by men.

Evolution yields changes by starting with random gene mutations that alter traits. According to Darwin, natural selection then selects for those traits that enhance survival and against those that diminish it. We can readily understand how natural selection can act positively on traits that enhance survival of organisms living in darkness, for example, to produce better sensory organs such as longer antennae. But sightless fish living in the dark are no better or worse off than sighted ones. So what could cause these fish to lose their eyes?

This question bedeviled Darwin himself who reluctantly resorted to the disuse idea, “By the time that an animal had reached, after numberless generations, the deepest recesses, disuse will on this view have more or less perfectly obliterated its eyes, and natural selection will often have affected other changes, such as an increase in the length of antennae or palpi, as compensation for blindness”.

The science of genetics was completely unknown to Darwin, but our understanding of genes and mutation provides us with two possible explanations. Knowing as we do that most mutations are harmful, we might guess that eyes are eventually lost because mutations accumulate over time in the genes that make them. In a sighted world these harmful mutations would be selected against, but in the dark they are “neutral”, providing no benefit or harm. But just like the clock that stops ticking after being dropped once too often, a buildup of mutations eventually prevents eye formation.

Alternatively, it is possible that the same gene controls traits that are beneficial in the dark and eye loss. So eye loss is merely a **by-product** of acquiring a trait that enhances survival in the dark. Geneticists know that a mutation in a single gene can have multiple and seemingly unrelated effects. The phenomenon is known as pleiotropy (*pleiotropy*) and is quite common. For example, a single mutation alters the hemoglobin molecule that carries oxygen in the blood and results in sickle cell anemia, a severe blood disease. However, that same mutation also stops the malarial parasite from multiplying in red blood cells, and so confers resistance to malaria.

To learn if mutations in the eye genes had accumulated, a group of biologists performed transplant experiments on cave and surface fish embryos. One team member, Luis Espinasa, at Shenandoah University, Winchester, VA, and his wife Monika have summarized these and other studies that answer the mystery of eye loss in the June 2005 issue of *Natural History* magazine. Normal development of the eye occurs as nervous tissue, destined to become the retina, activates overlying tissue to form a lens. As the lens develops it directs the formation of a complete eye. In cave fish embryos the lens starts to form, but then degenerates and the eye does not form.

The team transplanted an embryonic surface-fish lens into a cave-fish embryo and, conversely, a degenerated lens from a cave-fish embryo into a surface-fish embryo. As expected, the surface fish with the transplanted cave fish lens did not develop eyes, but surprisingly, the cave fish with the transplanted lens from the surface fish developed large normal eyes. These results showed that, even after tens of thousands of years evolving in caves, most of the genes involved in eye formation in the cave-fish remained fully functional. Evidently a buildup of mutations in the eye genes had not occurred.

Further studies showed that cave-fish blindness is caused by a mutation in a master control gene. This gene switches on other genes during embryonic growth and is responsible for normal development of body regions. The mutated gene results in pleiotropic effects; it alters a number of features in the head, not only causing blindness, but also affecting teeth and taste buds. Fish with the mutated gene lack eyes and have more taste buds.

In a separate study of cave-fish cranial bones, Espinasa discovered that these fish also have olfactory pits that are considerably larger than surface fish. Apparently a mutation in the same control gene that stops eye development in the fish also enhances its sense of smell. Natural selection is not acting on cave-fish eyes, instead it is selecting for the fish that can smell better and taste better, and these fish also become blind because the same gene affects all these traits. The light of Darwin's theory of natural selection has been turned on in the cave.

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