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Eda controls stickleback armor

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Sarah Lovinger
Email: lovdocs2@aol.com

In what could explain earlier findings that just a small number of genetic changes control the widespread evolution of many species of sticklebacks, researchers report in the March 25, 2005, *Science* that mutations in a single gene appear to be responsible for the changes in the armor of these widespread fish.

David Kingsley of Stanford Medical School and the Howard Hughes Medical Institute and colleagues found that reductions in the armor of threespine sticklebacks that have migrated from marine water to freshwater is an example of parallel evolution derived from an allele on a single gene, *Eda*.

"This finding shows that while the morphological change is large, the underlying genetics are simple," Kingsley told *The Scientist*. Marine water fish carry the genetic change at such a low frequency that individual animals do not carry homozygous alleles for the gene, a condition required for the development of low-plated fish, he said. But when these marine fish move to a new freshwater environment, the low-plated phenotype has a selected advantage, and the low-plated fish can appear quickly through natural selection at a higher frequency of the preexisting genetic change.

Eda encodes the signaling molecule ectodermal dysplasin, which controls the development of hair and teeth in human embryos and the bony plate armor of sticklebacks. Sequencing and comparing the complete *Eda* region in marine and freshwater fish revealed that most low-plated populations in the wild shared the same base pair changes.

Sequence amplification from 25 random nuclear genes failed to show evidence for one single origin of low-plated sticklebacks. To confirm that different levels of *Eda* signaling could change plate development in these fish, the researchers injected one-celled embryos from low-plate parents with a full-length mouse *Eda* A1 cDNA. When used in mice carrying a null mutation at the *Eda* locus, this construct has been shown to restore development of teeth and hair. Polymerase chain reaction genotyping of the fish in this cross confirmed that they were all homozygous for the low-plate *Eda* allele. Due to mosaic inheritance of the gene, some transgenic animals developed extra plates, but none of the controls did.

"This is one of the first times to show the actual gene involved in a major evolutionary change," said Catherine Peichel, of the Fred Hutchinson Cancer Center in Seattle, who was not involved in the study but once worked in Kingsley's lab. It is a good example of selective polymorphism, which is hidden in the marine population, she said: "As soon as the fish go into fresh water, there is strong selection for the allele."

In addition to identifying the gene responsible for the change in stickleback plate armor, this study makes another important point about parallel evolution. "Rather than using hundreds of ways to solve a problem, evolution uses a particular mechanism repeatedly to solve a problem," Kingsley said.

"It would be interesting to know how often this [kind of single gene activity] happens in other plants and animals," said John Postlethwait, of the University of Oregon, who was not involved in the study. While Eda is a gene of major effect, but natural selection usually occurs in situations of multiple genes with smaller effects, so it would be interesting to look at this more common situation in natural selection, he said.

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