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Acrobatic gecko tails

To distract predators, leopard geckos (*Eublepharis macularius*) can shed their tails through a process called autotomy. Though the research community understands how tail loss impacts a lizard's behavior and function, scientists know little about the movement of the detached tail.

Now, researchers have found that the tails can perform acrobatic flips and other complex movements for up to 30 minutes after being severed (*Biol. Lett.* published online 9 September 2009; doi:10.1098/rsbl.2009.0577). Timothy Higham of Clemson University (SC) and Anthony Russell of the University of Calgary (Canada) anesthetized four leopard geckos and implanted electrodes into their tails. After the lizards had recovered from anesthesia, the researchers lightly pinched the base of the tail of each lizard, causing it to detach. They then recorded signals from the electrodes on the tail.

The detached tails began rapidly swinging back and forth, at a rate of four to eight rhythmic movements per second. The tails also jumped, flipped, lunged and performed other complex movements. A neuronal network in the spinal cord generated the rhythmic pattern of motor output, but the source of the stimulus that caused the complex tail movements remains unknown. The authors think further study of the severed gecko tail could help researchers understand spontaneous movements that can occur following human spinal cord injuries.

New chromosome, new species

The formation of new species is typically driven by environmental adaptation. But a recent investigation of neighboring populations of threespine stickleback fish (*Gasterosteus aculeatus*) has now shown, for the first time, that creation of a new sex chromosome can contribute to speciation.

Jun Kitano (Fred Hutchinson Cancer Research Center, Seattle, WA) and colleagues examined a population of sticklebacks in the Japan Sea as well as their ancestral population from the Pacific Ocean. The two populations have been geographically isolated for roughly 1.5–2 million years. They found that in Japan Sea males, the Y chromosome had fused with one copy of an autosome, creating a new Y chromosome. The remaining, unpaired copy of the autosome segregated as a second X chromosome, effectively creating a new sex chromosome system in this population (*Nature* published online 27 September 2009; doi:10.1038/nature08441).

The Japan Sea males had more aggressive mating behavior than Pacific Ocean males, and Pacific Ocean females would not mate with the Japan Sea males. Tests showed that male hybrid fish from crossing the two populations were sterile. The new mating behavior was linked to the new sex chromosome, and it prevented from the two populations from breeding, making the Japan Sea population a new species.

Slowing down immune cells

The human immunodeficiency virus (HIV) uses a variety of tactics to successfully replicate and evade the human immune system. The HIV Nef protein is a prerequisite for such replication; without it, the development of acquired immune deficiency syndrome (AIDS) significantly slows down or even halts. Now, researchers have found that Nef hinders the movement of infected host immune cells (*Cell Host Microbe* **6**, 174–186; 2009).

Oliver Fackler of the University of Heidelberg (Germany) and colleagues investigated the effects of Nef on the migration of germ cells in zebrafish embryos. The majority of germ cells expressing Nef (77%) were nonmotile, whereas most of the germ cells expressing a Nef mutant (75%) moved normally. Fackler and his team completed further tests in a hamster fibroblast cell line. They determined that Nef inactivates cofilin, a protein that helps control cell mobility. To do this, Nef must associate with the cellular kinase Pak2.

These results suggest that by inactivating cofilin, Nef restricts the movement of HIV-infected immune cells. This likely diminishes the immune response to the infection and could also generate microenvironments where the virus can spread to uninfected cells. The authors think that modulating host cell motility might be an effective strategy for improving immune response to HIV infection.