

Fossils Push Back Origin of Land Animals

Paleontologists have discovered fossilized fragments of the oldest known land-adapted creatures: centipedes and tiny, spider-like arachnids dating back about 414 million years. The finds are forcing scientists to revise their thoughts about animal colonization of the continents, one of the most important steps in evolutionary history.

The fossils come from the city of Ludlow in Shropshire, England, where they were embedded within rocks from Earth's Silurian period, report Andrew J. Jeram of the Ulster Museum in Belfast, Paul A. Selden of the University of Manchester and Dianne Edwards of the University of Wales, who announced their discovery in the Nov. 3 SCIENCE. Prior to the Ludlow finds, the oldest known land animals dated to the early Devonian period, about 398 million years ago.

"This represents a very substantial step back in time," says William A. Shear, who studies the early evolution of land animals at Hampden-Sydney (Va.) College. "What this tells us is that we can look much farther back in the fossil record and expect to find more communities like this. We'll probably have to look much, much farther back to find the actual transitional forms [from which these land creatures evolved]."

Jeram and his colleagues say the age of the fossils suggests that the earliest land animals — ancient arthropods — emerged from the ocean soon after plants began spreading over the continents. Until now, paleontologists conjectured that animals lagged far behind plants in their adaptation to terrestrial life.

The researchers uncovered the fossils by dissolving Silurian rocks in hydrofluoric acid, which leaves behind exoskeleton fragments. Then they examined the animal parts under a microscope, attempting to decipher how the fragments fit together.

"It's a bit like doing a jigsaw puzzle — or maybe a dozen jigsaw puzzles that have

been thrown together — without knowing what the picture looks like," Selden told SCIENCE NEWS.

The acid treatment unveiled pieces of legs, back plates and trunks from centipedes of unknown size and the body of a spider-like animal called a trigonotarbid arachnid. The trigonotarbid fossil measured 1.3 millimeters long, suggesting an animal about the size of a common flea. The most complex land plants of that era grew only a few millimeters tall and would have looked like an outdoor carpet covering the landscape, Shear says.

Because both trigonotarbids and centipedes were predatory animals, the researchers reason that early terrestrial communities must have included other arthropods that served as prey. The Ludlow remains did not offer clear evidence of such creatures, but Selden suggests the prey animals were small arthropods that munched on tiny, easily digestible bits of decayed plant material. This contrasts with the modern world, where animals at the lower end of the food chain subsist on live vegetation.

— R. Monastersky

Genetic trickery probes tropical parasites

When it comes to genetic sleight-of-hand, the one-celled trypanosomatids surely rank as masters. Some of these pathogenic protozoans sneak past their host's immune surveillance by cloaking themselves in an ever-changing wardrobe of surface proteins. Others escape detection by hiding in the very immune cells that would normally attack them. In each case, the covert maneuvers spring from a complex and cryptic chain of genetic commands.

Scientists have now devised a genetic ruse of their own for deciphering those commands. By slipping a foreign gene between DNA sequences identical to those in the parasites, and then inserting the engineered material into the organisms, they have tricked two types of trypanosomatids into substituting the foreign material for the coding sequence of a naturally occurring gene.

"The basic idea is that we can figure out the role of a gene by deleting it and studying the biology of the [altered] organism," says parasitologist Angela Cruz, who coauthored one of two reports on this work in the Nov. 8 NATURE.

Investigators say the gene-replacement technique could eventually reveal the function of a slew of trypanosomatid genes. Moreover, they suggest that the ability to decipher and disrupt genetic functions might lead to more effective drugs to treat the many tropical diseases caused by these parasites, and might enable researchers to create weakened strains that could serve as vaccines against some of the parasites.

Working separately, two research teams used a gene-insertion technique that mimics a natural mechanism for replacing damaged genes. Although this technique is becoming standard in studies of yeast and mice, it had never been tried with trypanosomatids. For their engineered material, both groups chose a bacterial gene for resistance

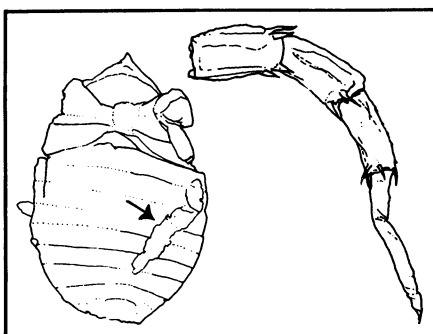
against the antibiotic neomycin so that they could confirm insertions by exposing the parasites to the drug.

At Harvard Medical School in Boston, Cruz and Stephen M. Beverley hid the gene for neomycin resistance between certain genetic sequences taken from the trypanosomatid *Leishmania major*. By themselves, these sequences cannot code for proteins, but the researchers used them as homing devices to pinpoint the site of a particular *L. major* gene that codes for an enzyme involved in building nucleotides. After inserting the genetic sandwich into the parasites, the researchers found that 45 percent of their *L. major* colonies substituted the neomycin "marker" gene for the critical enzyme-encoding gene.

Piet Borst and his colleagues at the Netherlands Cancer Institute in Amsterdam report similar success in using the neomycin gene to replace a targeted gene in *Trypanosoma brucei*, which causes African sleeping sickness.

The new work opens up the long-range prospect of deleting trypanosomatid genes that confer virulence and using the weakened organisms as the basis for vaccines, asserts Mario Capecchi, a molecular geneticist at the University of Utah in Salt Lake City, in a commentary accompanying the reports. Borst told SCIENCE NEWS that while such an approach might work with *Leishmania* species, he doubts it could yield a vaccine for *Trypanosoma* parasites, in part because the variety of strains and the changeability of their surface proteins would likely enable them to evade vaccine-triggered antibodies. Borst adds, however, that new drugs for sleeping sickness and related diseases might ultimately emerge from studies identifying the genes that regulate the unique feeding habits of *Trypanosoma* parasites and that allow these organisms to change their protein cloaks.

— R. Cowen



Left: Fossilized body of a spider-like arachnid, with arrow indicating poorly preserved leg. Right: Leg of a Silurian centipede.