New Phylum Found Residing on Lobsters

Even the most sophisticated lobster lovers probably give little thought to the creatures' lips. If they only knew what they were missing.

On the mouths of Norwegian lobsters lives a tiny invertebrate that fits into none of the animal kingdom's 35 or so broad taxonomic groups called phyla, claim Peter Funch and Reinhardt Møbjerg Kristensen of the University of Copenhagen. They have named the creature *Symbion pandora* and have assigned it to an entirely new phylum, which they call Cycliophora.

Their discovery of "what appears to be a new phylum of metazoans has to be the zoological highlight of the decade," asserts Simon Conway Morris of the University of Cambridge in England in a commentary accompanying the report in the Dec. 14 NATURE.

"I think that there will be a lot of response, both positive and negative" to the report, acknowledges Funch. Scientists often argue over phylum designations.

For example, Funch and Kristensen state that the new creature most nearly resembles the phyla Ectoprocta and Entoprocta. Yet scientists fail to agree that Ectoprocta and Entoprocta are closely related, Morris notes.

Tom Fenchel of the Marine Biological Laboratory in Elsinore, Denmark, first observed the creature in the 1960s. Three decades later, using a state-of-the-art electron microscope that can peer deep into cell parts, Funch and Kristensen described the creature's unique body structure and behavior. They find that it reproduces both sexually and asexually and performs some odd stunts in the process. It also has several types of larvae, only some of which feed.

While attached to a lobster, a feeding-stage *S. pandora* uses tiny hairs, called cilia, around its mouth to capture food intended for the lobster. Periodically, its entire feeding apparatus, including the stomach, deteriorates. But *S. pandora* remains stuck to the lobster and grows new feeding structures.

After several cycles, so-called pandora larvae develop inside the feeding-stage animal. Before they emerge, each pandora produces a feeding-stage larva inside itself. When a pandora larva emerges, it settles on the lobster and soon dies. Its feeding-stage larva remains attached to the lobster.

Before the lobster molts, something—perhaps the hormones that tell it to molt—triggers *S. pandora* to produce either a female or a dwarf male, which has only a nervous system, reproductive organs, and cilia for swimming. Then sexual reproduction begins.

Funch

A feeding-stage S. pandora *with a pandora larva in its brooding chamber.*

The dwarf male seeks out an *S. pandora* that is carrying a female and fertilizes her eggs—exactly when remains unclear, Funch says. The fertilized female quickly dies. Yet another type of larva, a chordoid, emerges from the eggs, swims to another lobster, and dies, leaving a few bud cells that develop into a feeding-stage larva.

When their lobster hosts finally molt, all the *S. pandora* disperse and find a new lobster or return to their original one after it finishes molting. — *T. Adler*

San Andreas looms larger in L.A.'s future

When the San Andreas fault eventually unleashes the Big One, it will batter Los Angeles with waves of seismic energy far greater than seismologists had ever imagined, according to a ground-breaking computer simulation—the largest yet attempted for a San Andreas quake.

"We're looking at numbers that are two to three times what had been predicted previously," says seismologist Ralph J. Archuleta of the University of California, Santa Barbara. Archuleta worked with colleague Kim B. Olsen and with Joseph R. Matarese of the Massachusetts Institute of Technology to simulate the effects of a magnitude 7.75 tremor originating north and east of Los Angeles.

Seismologists put the chances of such a quake at one in four by the year 2024.

"When I look at the calculated ground motions, they are just breathtakingly large," says seismologist Paul G. Somerville of Woodward-Clyde, a consulting firm in Pasadena, Calif.

Angeleños shouldn't rush to pack their bags, however. Somerville and other researchers warn against placing too much faith in the computer simulation. Despite its massive size, they say, it suffers from limitations and uncertainties.

Even Archuleta hesitates to interpret the results. "Is this bad news? I don't know. I don't know if this will be significant or not," admits the seismologist, who discussed the work this week in San Francisco at a meeting of the American Geophysical Union. Archuleta and his coworkers also published their results in the Dec. 8 SCIENCE.

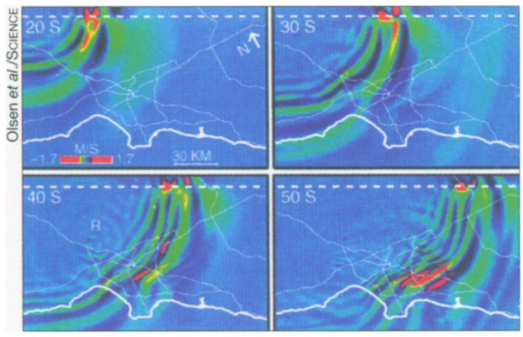
In the simulation, the quake ruptured a 170-kilometer-long stretch of the San Andreas fault from a region near the town of Gorman to the San Bernardino area east of Los Angeles. The virtual vibrations rippled through a three-dimensional representation that included realistic details of the local geology.

Other research groups have conducted simulations with the same algorithm, but Archuleta's team is the first to attempt computations for so big a quake. The simulation took 23 hours on a supercomputer with 512 parallel processors.

"It's a calculation that many have dreamed about for years," says Thomas H. Heaton of the California Institute of Technology in Pasadena.

Despite all this computing power, Archuleta's group had to limit the simulation to keep it manageable. The team looked only at vibrations with periods greater than 2.5 seconds. The rolling motion from these long-period waves threatens tall buildings and long bridges but does not usually harm buildings of a few stories or less, which constitute the majority of structures in Los Angeles.

Olsen *et al.*/SCIENCE



Snapshots of the seismic waves emanating from the San Andreas fault (dotted line). White lines are Los Angeles freeways; red shows strongest shaking.

Not surprisingly, the simulated vibrations were strongest in regions close to the San Andreas fault, including heavily populated San Bernardino. Unexpectedly, however, the computer exercise also showed the ground moving powerfully in parts of Los Angeles some 60 kilometers from the fault.

The waves are amplified as they pass beneath Los Angeles, explains Archuleta, because the city sits on a vast, sediment-filled basin. The soft sediments slow the waves as they enter, and the basin structure traps them—both factors that boost the size of the waves. The ground in some areas of Los Angeles moved at 1.4 meters per second, several times the rate expected.

If they struck an area with tall buildings, these exceptional waves could cause tremendous damage. But the simulation lacks sufficient resolution to show engineers whether the waves would hit the few sections of Los Angeles with high-rises. "It's going to take a fair amount of additional work to tell what this means for existing structures," says Heaton.

The study does not address short-period vibrations, the rapid jerking that tears small buildings apart. Some seismologists fear that the short-period waves from a San Andreas temblor would also exceed expectations.

Heaton disagrees, arguing that a giant quake outside Los Angeles is unlikely to shake the city with short-period waves much more destructive than those of last year's magnitude 6.7 Northridge shock. If big San Andreas jolts did create monster short-period waves, then the last superquake in 1857 should have devastated the city. Most of its 5,000 residents survived the tremor, however.

— R. Monastersky

U.N. to oversee methyl bromide phaseout

In Vienna last week, representatives of some 110 countries voted to strengthen the Montreal Protocol, a United Nations treaty to protect Earth's stratospheric ozone layer. On Dec. 8, industrial nations agreed to phase out their use of methyl bromide by 2010. The bromine released by this short-lived, gaseous pesticide is 50 times more destructive to ozone than chlorine is.

Previously, industrialized nations—which account for about 80 percent of methyl bromide use—had agreed only to freeze that use at 1991 levels.

"Phasing out methyl bromide offered the biggest percentage savings of future ozone loss" of any of three major treaty changes considered, explains Daniel L. Albritton of the National Oceanic and Atmospheric Administration in Boulder, Colo.

New analyses also indicate that further regulation of hydrochlorofluorocarbons (HCFCs) would offer the smallest ozone rewards, Albritton, a cochair of the U.N. scientific assessment on stratospheric ozone, told the delegates. After much debate, however, the delegates voted to accelerate by about 10 years—to 2020—the HCFC phaseout by most industrialized countries and to extend the phaseout to developing nations.

Last year, as scheduled, production of firefighting chemicals called halons came to an end. The Vienna meeting did not take action on Albritton's third proposal: prohibiting release of existing halons from unused fire extinguishers.

The United States had pushed for a 2001 phaseout of methyl bromide to coincide with its slated ban under the Clean Air Act. Environmental groups responded to the treaty change with a statement chastising the United States and other leaders for backing "an extremely weak compromise that will extend ozone depletion for several decades through continued use of methyl bromide."

The 2010 timetable "is ridiculous" and "far too long . . . for something that we are sure is contributing to ozone destruction," argues atmospheric physicist Rumen Bojkov, an adviser on global environmental issues to the secretary general of the World Meteorological Organization in Geneva. He noted, however, that most major users of methyl bromide categorically rejected earlier deadlines.

The 2010 deadline now threatens to derail the U.S. phaseout. A bill introduced by Rep. Dan Miller (R–Fla.) would delay the 2001 deadline until there is a worldwide phaseout or until cost-effective substitutes exist.

— J. Raloff

DNA manipulation goes large-scale

Geneticists have scored another victory on the playing field of the mouse genome. In what they call chromosome engineering, researchers have succeeded in deleting, inverting, and rearranging not single genes but large, selected blocks of mouse DNA.

In genetic engineering, investigators routinely pop extra genes into mice and knock out specific genes to create mice that develop without those genes' proteins. Now, with their new skills, researchers from Baylor College of Medicine and Texas A&M University, both in Houston, have robbed a mouse chromosome of 10 percent of its DNA.

"That's a mighty big chunk of DNA," marvels Kenneth Paigen, director of Jackson Laboratory in Bar Harbor, Maine, which collects mutant mice.

Chromosome engineering will speed the search for new genes, especially those that normally prevent uncontrolled proliferation of cells, says Allan Bradley, a Howard Hughes Medical Institute researcher at Baylor. The technique may also help create rodent examples of many human difficulties, since chromosomal rearrangements often cause failed pregnancies, familial diseases, and cancers.

"We're going to have quite a few different types of applications using this technology," says Mario Capecchi of the University of Utah in Salt Lake City.

Bradley and his colleagues, who describe their experiments in the Dec. 14 NATURE, use an unusual enzyme produced by a virus that infects bacteria. The enzyme, Cre recombinase, or simply Cre, recognizes short viral DNA sequences called loxP sites.

When Cre encounters two loxP sites that scientists have inserted into a mouse chromosome, the enzyme cuts out the intervening DNA (SN: 7/9/94, p.20). Depending on the orientation of the loxP sites, the enzyme then either inverts the DNA fragment and places it back in the chromosome or discards the snipped DNA, says Bradley.

Since every cell contains two copies of most chromosomes, the loxP insertions sometimes land on different copies of a particular chromosome. In those cases, Cre uses the loxP sites to define a region of one chromosome that it will cut off and attach to the other chromosome. "It puts one piece of chromosome onto another," says Capecchi.

To create mice with manipulated chromosomes, Bradley's group alters the DNA of stem cells and injects these immature cells into early-stage embryos. If the altered stem cells develop into the mouse's reproductive cells, all cells in the embryo's offspring will have the modified chromosomes.

Deleting large chunks of one chromosome does not generally kill the offspring, says Bradley, because the second copy usually contains a spare of each missing gene. The deletion does make it easier to search for tumor-suppressing genes, for example, since both copies of such genes must be deactivated before cancer results.

Bradley's group removed about one-tenth of one copy of chromosome 11 from a mouse strain. The deleted region resembles the part of human chromosome 17 that contains genes implicated in suppressing breast cancer. By mutating genes in the same region of the unaltered copy of chromosome 11 and observing whether cancer develops, investigators hope to identify the tumor-suppressor genes.

This search technique can also help identify genes whose functions are normally masked by dominant counterparts on the other copy of the chromosome. Chromosome engineering promises "to revolutionize mouse genetics," asserts Bradley.

— J. Travis