ber of an entangled pair together with her message photon. This measurement does not tell her the state of her message photon, but it does entangle her two photons in such a way that they end up with opposite states. As a result of her measurement, Bob's photon becomes instantly primed so that when Alice tells Bob what to do to his photon, he will find that it is identical to the original message photon.

Although the process destroys the original state of the message photon, that state lives on in Bob's photon, the second entangled photon, irrespective of his distance from Alice. There is no transport of actual photons from Alice to Bob—only a flow of quantum information. "It is more like faxing than teleportation," says Sudbery.

Although the setup sounds simple, the team's apparatus is a bewildering tangle of lasers, mirrors, and optical instruments, and the work "required a great deal of experimental finesse," says Bennett. One key was the development over the past 2 years of pure and bright sources of entangled photons, explains Zeilinger. Another, he says, was learning how to take measurements like Alice's, which register information about the joint properties of two photons that lose their individual identities in the process.

A separate group, headed by Francesco De Martini at Italy's National Institute of Nuclear Physics in Rome, has performed a similar experiment, which the researchers will report in *Physical Review Letters*. The Rome group's experiment departs from Bennett's original vision but is optically simpler than the Innsbruck work. It relies on a single pair of entangled photons, with one twin forced to play the role of Alice's message photon in addition to its teleporting role.

"I think one of the main uses to which teleportation will be put is moving data around inside a quantum computer," says Bennett. In quantum computation, "there's the very serious problem that quantum information is very delicate. If it leaks out of the computer at all in the course of the calculation, the result will be spoiled." Teleportation can help beat this kind of problem. "This is a way of sending quantum information reliably through a noisy channel," he adds.

Sadly for *Star Trek* fans, however, quantum teleportation cannot be scaled up to move Captain Kirk from place to place. It is more akin to teleporting "states" of Captain Kirk around the universe. One could imagine the captain's amorous mood being teleported to a far-off clone, a prospect sure to strike fear into the hearts of all female aliens. One consolation: The original amorous state of the captain would be destroyed in the process.

–Andrew Watson

Andrew Watson is a science writer in Norwich, U.K.

DEVELOPMENTAL BIOLOGY

Possible New Roles for *HOX* Genes

MADRID, SPAIN—The best discoveries in the trendy field of "evo-devo" shed light on two processes simultaneously: how genes shape the bodies of today's organisms during embryonic development, and how those same genes may have guided the organisms' evolution (*Science*, 4 July, p. 34). New results from Peter Holland's team at the University of Reading in the United Kingdom seem to have achieved both objectives.

At a workshop on development and evolution held here last month by the Madrid-based Juan March Foundation, Holland reported that he and his colleagues found a putative second cluster of *HOX*-like genes in *Amphioxus*, a fishlike marine invertebrate that is seen as a crucial evolutionary link to vertebrates. A great deal of evidence has shown that the *HOX* genes—so-called because they carry a DNA sequence known as the homeobox—play important roles in laying down the head-to-tail patterns of embryos of organisms ranging from worms to flies to humans.

In addition to providing a better understanding of the genes controlling *Amphioxus* development, the Holland team's result is intriguing because it may also help explain a key development in the rise of complex organisms like vertebrates during evolution: the creation of multiple "germ layers"—the primitive embryonic tissues that give rise to all of a creature's tissues and organs. *HOX* genes have long been known to be active in ectoderm, the outermost germ layer, but Holland found that the new "sister cluster" of *HOX*-like genes is expressed in the innermost layer, the endoderm. The results suggest, he said, that the appearance of the new cluster—presumably resulting from duplication of a primordial *HOX* gene cluster—is related to the creation of multiple germ layers in early evolution.

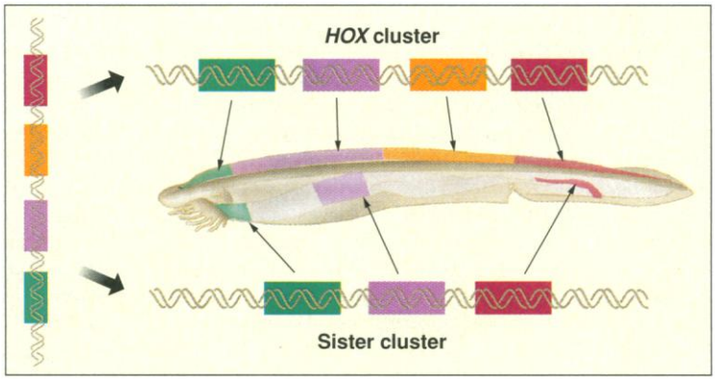


ILLUSTRATION: K. SUTLIFF

Division of labor. The descendants of a proto-*HOX* gene cluster (left) may help shape the *Amphioxus* ectoderm (above) and the gut (below).

Although other researchers want more evidence before accepting that suggestion, it helped make Holland's presentation the most talked-about at the workshop. "These were the newest and best results at the meeting," says Andre Adoutte, an evolutionary biologist specializing in molecular phylogeny at the University of South Paris.

Holland made this discovery while studying a set of *HOX* genes that has long puzzled developmental biologists. Almost all *HOX* genes are arranged in clusters of roughly nine genes each. The expression patterns of these genes along the head-to-tail axis of the embryo typically follow their arrangement in the clusters, with those at one end tending to be expressed more anteriorly, while those at the other end are active farther back. But researchers have also identified three or four types of *HOX*-like genes that don't seem to fit this neat pattern.

While they carry a typical homeobox sequence, for example, they have never been shown to be part of a cluster. That presented a conundrum: If these "dispersed" or "orphan" *HOX* genes weren't in clusters, they could not have arisen the way true *HOX* genes supposedly did—by duplication of successive genes along a single chromosome. But their close similarity to true *HOX* genes makes it unlikely they arose by chance.

At the meeting, Holland described new data from his laboratory showing that three of these orphan *HOX* genes are in fact clustered in *Amphioxus* the way true *HOX* genes are. Using probes made from vertebrate *HOX* genes, he found that two dispersed *Amphioxus* genes were located in adjacent regions of a single chromosome. He then used the technique of "chromosome walking" to track down a third orphan *HOX* gene located close to the other two. Finding the genes close together means, Holland said,

that they couldn't have just "hopped out" of the single original *HOX* cluster one by one. Instead, he concluded, "the two gene clusters originated suddenly, by duplication of a primordial cluster."

What's more, the team found that the genes' order of appearance on the chromosome corresponds to the head-to-tail order of their activity in the body. However, they differ from other *HOX* genes in one key respect: They are expressed only in the endoderm, the innermost of the three embryonic germ layers, which gives rise to the gut and other organs of the viscera. Other *HOX* genes—including the first *HOX* gene cluster of *Amphioxus* identified by the Holland team in 1992—are active only in the embryonic ectoderm, the outermost germ layer, which produces tissues including the skin, nervous system, and sense organs. "What we've got here are two gene clusters, each obeying spatial colinearity but in different ... layers," Holland said.

To back his contention that the genes of the new cluster were key to the development of the inner germ layer, Holland used data from other laboratories to show that analogous genes in mice and frogs are expressed in the gut. One of the *Amphioxus HOX*-like genes, for instance, is analogous to the *IPF* gene in mammals which, gene knockout experiments show, is crucial for development of the pancreas in mice. Putting this all together, Holland proposed that doubling of the primordial *HOX* cluster occurred before an ancestor of *Amphioxus* acquired an endoderm and was, in fact, instrumental in its creation.

Other meeting participants view this suggestion cautiously. Michael Akam of the University Museum of Zoology in Cambridge, U.K., says that while Holland's data linking the three genes "look ironclad," his hypothesis that one *HOX* cluster took over the patterning of the inner body layer and the other the outer one is "almost too clean a model to be probable." Diethard Tautz of the University of Munich in Germany agrees. *Amphioxus*, he says, "is only one data point. You need to have more than one organism to form a complete picture."

The additional data points may come soon, as the search for *HOX* genes and their relatives in primitive organisms such as acorn worms is heating up. The implications are so important, says participant Axel Meyer, an evolutionary biologist at the University of Konstanz in Germany, that the race to find these genes "is a gold rush." But the "gold" in this case will be a clear idea of how evolution really happened.

–Steven Dickman

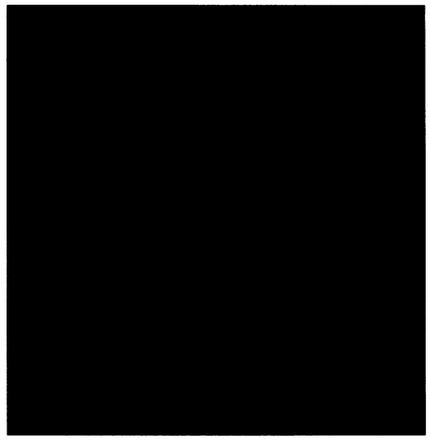
Steven Dickman is a writer in Cambridge, Massachusetts.

CELL BIOLOGY

Pinning Down Cell Division

The events in the cell just before it divides are some of the most dramatic in biology. The chromosomes condense, the nuclear membrane disappears, and the cell starts to build its mitotic spindle—a set of fibers that will eventually pull the chromosomes to the opposite poles of the dividing cell. How the cell choreographs these complex changes is unclear, but on page 1957, molecular biologist Kun Ping Lu of Beth Israel Deaconess Medical Center in Boston and Harvard University and his colleagues report evidence for a new mechanism that may play a key role.

Cell biologists have long known that the cell's progress toward division is controlled by a group of kinases, enzymes that add phosphate groups to a variety of cell proteins. For the most part, though, they've had few clues to what those phosphate additions actually do. That's where the Lu team's work comes in. It suggests that the phosphates serve as a sort of tag for attracting an enzyme called Pin1, which may cause the phosphorylated proteins to change their shapes. Researchers don't yet know exactly what this accomplishes, although they point to several possibilities, such as turning off an active enzyme, directing a protein to a new place in the cell, or targeting a protein for degradation. Whatever the precise result, however, the work provides "a new function for phosphorylation," says molecular biologist Tony Hunter of the Salk Institute in La Jolla, California.



LU ET AL.

Twister. The Pin1 protein may help regulate mitosis by binding to phosphorylated proteins. The bright green region at left is the phosphate binding site.

Lu and Hunter first discovered Pin1 2 years ago as a protein that interacts with and inhibits another critical cell regulator, called NIMA, which helps turn on mitosis. Pin1 itself is an isomerase enzyme that changes the configuration of the peptide bond preceding proline, an amino acid that is an important determinant of protein structure because it can put kinks into a protein chain. Previous studies also showed that Pin1 is crucial for both yeast and human cells to divide properly. Without it, for example, cells can't complete mitosis. But its precise role in the cell remained a mystery.

Researchers got a clue earlier this year, however, when Joseph Noel of the Salk Institute solved Pin1's three-dimensional structure. It showed that the enzyme has a pocket for binding phosphate next to the site where it binds its proline target, says molecular biologist Lewis Cantley of Harvard, a coauthor on the *Science* paper. That suggested Pin1 might bind phosphorylated proteins.

To confirm that hunch, the team searched through a library of protein fragments for peptides that bind to the enzyme. Sure enough, says Cantley, Pin1 preferentially picked out peptides that have a phosphate attached to an amino acid adjacent to a proline. With some sequences, in fact, the phosphorylated version bound thousands of times better than the unphosphorylated peptide.

Other unpublished work suggests that Pin1 might help orchestrate cell division by interacting with other proteins involved in mitosis. When members of Lu's team went "fishing" through the contents of ruptured cells for proteins that bind to Pin1, they landed at least a dozen that are also targeted by an antibody, called MPM-2, that binds to proteins involved in mitosis in a wide range of cells. These proteins, too, contain a proline and an adjacent phosphate.

Taken together, say Lu and his colleagues, the experiments suggest that Pin1 helps regulate a two-step process that governs cell division. Adding phosphates to proteins involved in mitosis creates binding sites for Pin1, which can then latch onto them and twist the peptide bond next to the prolines it contacts. That might, in turn, change the shape of the whole protein, perhaps altering its ability to interact with still other proteins, its location in the cell, or its life-span.

Whatever the binding does, Cantley suggests that it might enable Pin1 to serve as a sort of checkpoint on the way to cell division. He notes that while cells lacking the protein can't divide—indeed, they die instead—manipulations that increase Pin1 production delay the onset of mitosis. Based on that, he proposes that by binding to phosphorylated proteins, Pin1 may slow down the activity of any proteins that are getting