embrace the full range of biodiversity information including geographical, ecological, genetic, and molecular data. A third activity will be to digitize all biodiversity information, now usually embodied specimens in museums continents away from where the samples were collected. "Repatriation of data is a major impetus," says Meredith Lane, vice president for biodiversity at the Academy of Natural Sciences in Philadelphia and a member of the bioinformatics working group. But that process, by which the host country would obtain electronic access to information stored in another country, will require an enormous and sustained effort. "We have 30 million insects on pins, many very small and fragile with tiny hand-written labels. At our current rate of progress, [cataloguing these specimens electronically] would take centuries," says Blackmore.

Of course, all this will take money. And despite the official go-ahead, none has yet materialized for GBIF. The working group has estimated that GBIF will end up coordinating some $40 million a year in ongoing work within member countries, and that GBIF itself can make an important contribution at an annual cost of $8 million a year. But such a budget, paid by member nations, is probably a few years away.

As a first step, science ministers from Australia, Denmark, the United Kingdom, and the United States have signaled their intention to contribute toward the $2 million to $3 million needed to set up a six-person secretariat at a site to be determined. Australia and the United Kingdom are seen as likely bidders for the administrative headquarters, to open next year. Although the United States is unlikely to put in an application, says James Edwards of the National Science Foundation, it is strongly committed to the project. "There is some activity going on now to mobilize data, and there are sporadic efforts to put it on the Internet," he says. "But there's no capacity for the one-stop shopping needed for nations to carry out the CBD and to develop their own biodiversity programs. That's what GBIF will do."

–JUDY REDFEARN

Judy Redfearn writes from Bristol, U.K.

DEVELOPMENTAL BIOLOGY

How to Get a Heart in The Right Place

CHARLOTTESVILLE, VIRGINIA—Like a child learning to put her hand over her heart for the Pledge of Allegiance, a developing embryo needs to know its right from its left. The heart goes on the left and the liver on the right, but how the embryo knows which is which is a long-standing puzzle. At the annual meeting of the Society for Developmental Biology here last month, one promising theory—that twirling "hairs" on embryonic cells set up the left-right distinction—gained strength.

Scientists first proposed a connection between cilia—whiplike protrusions that can propel cells and help keep airways clear—and organ placement nearly 25 years ago. In 1976, Bjorn Afzelius described how human patients with a genetic defect called Kartagener's syndrome have immotile sperm and defective cilia in their airways—and about half have their organs on the wrong side (*Science*, 23 July 1976, p. 317). That connection led to speculation that cilia might somehow help to direct organ placement, but no one knew whether Kartagener's syndrome disables the cilia in the embryo as well.

Turn, turn, turn. Twirling cilia on the node cells of a developing embryo may distinguish its left from its right.

CREDIT: S. POTTER, UNIVERSITY OF CINCINNATI AND J. DUNLAP, UNIVERSITY OF TENNESSEE

The old theory was resurrected 6 months ago, after cell biologist Nobutaka Hirokawa of the University of Tokyo and his colleagues reported that when they knocked out a gene involved in cilia assembly in mice, about half the animals had reversed left-right organ placement, and all lacked cilia on so-called node cells. These cells produce many of the signals that direct the early patterns in a mouse embryo, and the node is the site of some of the first molecular differences between left and right.

When the team made microscopic videos of normal node cells, they found that their cilia rotated counterclockwise, unlike the back-and-forth motion of cilia on sperm or in airways. By tracking fluorescently labeled beads, the scientists determined that the cilia somehow swept the fluid surrounding the cells to the left. That might cause an as yet unknown signal to accumulate, eventually leading to asymmetric organ development. The lack of this lateral cue in the mutant strain could explain the 50% rate of organ reversal.

But other researchers had trouble repeating the technically difficult observations, and many remained unconvinced. One concern was that the mice without cilia on their node cells might have other defects as well, so that something other than the cilia themselves could be the cause of the left-right disturbances. Even Yale University pediatric cardiologist Martina Brueckner, who had been working with a different strain of mutant mice that also suffer a 50% chance of left-right reversal, had her doubts. "It just seemed so weird," she says.

But, at the meeting, she announced that her team has taken a close look at the node cells in their mutant embryos, too. They found that these cells do have cilia, but they stand rigid and straight, instead of twirling. Without that motion, evidently, the signal drifts randomly left or right, which could explain the reversals.

The observation boosts the theory that twirling cilia cause asymmetry, says cell biologist Chris Wright of Vanderbilt University. "Showing that they're rigid is tantalizing," he says. But to really clinch the case, he says, someone needs to show that the cilia in yet another mutant mouse strain called *inv*, in which almost all the animals have reversed organs, twirl backward.

–GRETCHEN VOGEL

SCIENTIFIC MISCONDUCT

EMF Researcher Made Up Data, ORI Says

In a blow to a research area hungry for credible findings, the federal Office of Research Integrity (ORI) reported last month that a biochemist "engaged in scientific misconduct ... by intentionally falsifying and fabricating data and claims" in two studies on how electromagnetic fields (EMFs)—the kind shed by power lines and home appliances—affect living cells. The researcher, Robert P. Liburdy, formerly of the Lawrence Berkeley National Laboratory (LBNL) in California, has agreed to ask the journals to retract the results. "There's a lot of acrimony in the [EMF] debate, and this won't calm things down," says Richard G. Stevens, a cancer epidemiologist at the Pacific Northwest National Laboratory in Hanford, Washington.

Liburdy's findings were among the first to offer a plausible mechanism for a possible link between EMF exposure and cancer or other diseases. In a pair of 1992 papers of which he is the sole author, Liburdy offered evidence that EMFs increase the flow of calcium into lymphocytes, a kind of immune cell produced in the thymus. The papers created a stir, as calcium ions signal cells to turn genes on and off, and play a role in cell division. Because tumor growth is tied to cell proliferation, an alteration in calcium signaling could conceivably lead to cancer. But in an analysis obtained by *Science*, ORI states that "Liburdy's claims that EMF causes cellular effects related to calcium signaling [in three figures in the two journal articles] are

not supported by the primary data."

Responding to an unknown whistleblower's allegations of scientific misconduct by Liburdy, LBNL in January 1995 appointed a panel of four lab scientists to investigate. After reviewing raw data and interviewing Liburdy and other scientists, the panel concluded in a July 1995 report that Liburdy "deliberately created 'artificial' data where no such data existed" in a figure in *FEBS Letters*. In addition, it found, he fabricated data noise for a figure in the *Annals of the New York Academy of Sciences* "in order to mislead the reader." These actions, the panel stated, "fall within the definition of scientific misconduct." When contacted by *Science*, LBNL officials declined to comment, other than to confirm that Liburdy no longer works at the lab.

Because Liburdy had been awarded more than $3.3 million in federal grants for his EMF research, ORI launched a formal review of LBNL's report in fall 1997. ORI approved a request by Liburdy for an interview with ORI staff and two outside experts, which took place in March 1998. At the meeting, Liburdy produced original data he had not shared with LBNL investigators, according to the ORI report. But the data failed to exculpate him: In its analysis, ORI accuses Liburdy of having lied to LBNL and ORI investigators, and it "concurs with [LBNL's] findings of scientific misconduct." "Some of the numbers, essentially, he made up," says John Krueger, an ORI investigator involved in the case.

In a May 1999 agreement signed by Liburdy and ORI acting director Chris Pascal, Liburdy agreed to retract the tainted figures in the two papers and not to receive federal funds for 3 years. He "neither admits nor denies ORI's findings of scientific misconduct," the document states. Liburdy did not respond to requests for an interview.

The misconduct findings are unlikely to shift the playing field in EMF research. Since 1992, 20 to 30 scientific papers have looked at EMF exposures and calcium signaling, without settling the issue, says Christopher Portier, associate director of the environmental toxicology program at the National Institute of Environmental Health Sciences (NIEHS). In a report to Congress released on 15 June, NIEHS director Kenneth Olden states the scientific evidence that EMF exposures "pose any health risk is weak" and that mechanistic and toxicology studies "fail to demonstrate any consistent pattern." The day before the report came out, National Institutes of Health officials had asked NIEHS to determine quickly whether any of Liburdy's research had influenced the report's conclusions, Portier says. The truth was simple, he says: "It had no impact whatsoever."

–DAN VERGANO

Dan Vergano writes for the *Medical Tribune*.

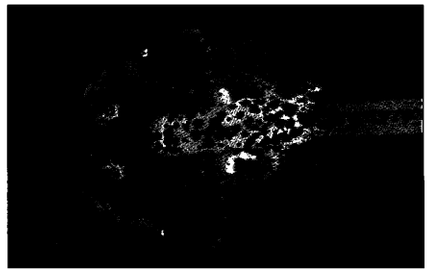
CREDITS: (TOP TO BOTTOM) MARTIN OFFIELD; NICHOLAS HIRSCH

DEVELOPMENTAL BIOLOGY

Frog Is a Prince of a New Model Organism

CHARLOTTESVILLE, VIRGINIA—*Xenopus laevis*, a fist-sized brown frog that is a favorite of developmental biologists, has an embarrassment of genes. For decades, biologists have studied its large, hardy embryos, transplanting bits of tissue to create monster tadpoles with two heads or missing tails—and in the process deciphering some of the key steps that shape a developing vertebrate. But in the age of molecular biology, *X. laevis* has a major drawback: Somewhere in its evolution, the frog's genome doubled, leaving the animals with four copies of most genes instead of the usual two. The extra genes make it nearly impossible for scientists to do the genetic studies that have been so powerful in flies, worms, and mice: interrupting the function of a gene and watching what goes wrong when it is missing.

Now a frog from the damp floor of the West African rainforest, called *Xenopus (Silurana) tropicalis*, may provide the best of both worlds to developmental biologists, who crowded into a recent symposium* here to learn about it. The species is the only close relative of *Xenopus* that has a diploid genome, with just two copies of each gene, like people and most other vertebrates. It is smaller and easier to house than *X. laevis* and also becomes sexually mature in 4 or 5 months instead of 1 to 2 years, so scientists can quickly breed transgenic colonies.



It's easy when they're green. *Xenopus tropicalis* frogs expressing green fluorescent protein in their eyes *(top)* and nervous system *(bottom)* allow researchers to watch the organs develop in live embryos.

Yet the species are close enough that even minor steps in development are the same, and transcription promoters and other genes cloned from *X. laevis* seem to work just fine in *X. tropicalis*. The two species will be "two parts of the same system," predicts cell biologist Marc Kirschner of Harvard Medical School in Boston. "All of this wonderful work and technology in *laevis* has been directly applicable to *tropicalis*," he says.

Kirschner was the first to import *X. tropicalis* to the United States. But most of the work on the new species has been in the laboratories of the symposium organizers: developmental biologist Enrique Amaya of the Wellcome/CRC Institute in Cambridge, U.K., and Robert Grainger of the University of Virginia in Charlottesville.

Already, these labs are toying with the genetics of their new model. At the meeting, developmental biologist Lyle Zimmerman, a postdoctoral fellow in Grainger's lab, described some of the first transgenic *X. tropicalis*. A few years ago, Amaya and Kristen Kroll, now of Harvard Medical School, developed a technique for extracting nuclei from *Xenopus* sperm, treating the DNA so that it efficiently incorporates new genes, and then injecting the transgenic nuclei into eggs. A transgenic frog, with the new genes in all of its cells, then develops. Zimmerman and his colleagues have now used the technique to create frogs that express green fluorescent protein (gfp) in cells destined to become eye, heart, or the nervous system, allowing the scientists to observe the growing organs in live embryos. Although the gfp doesn't disrupt normal gene function, scientists can design DNA inserts that do interrupt key genes, then breed the frogs to produce offspring in which both gene copies are faulty.

Such mutant frogs should prove a powerful tool for developmental biology. The ability to watch gene regulation without killing the embryo "is really unprecedented in a vertebrate," says molecular biologist Barry Knox of the State University of New York, Syracuse. In mice, scientists can do even more sophisticated knock-out experiments, but they cannot observe the embryo as it grows inside its mother. And zebrafish, praised for their see-through embryos, are not as suitable for tissue transplant experiments as the larger frogs.

To lay the groundwork for studying *X. tropicalis*, Grainger, Amaya, and a number of their colleagues hope to launch a major screen for mutant frogs, similar to the systematic screens done in flies and zebrafish. By causing random mutations and then watching their effects, scientists hope to tease out the genes that control various stages of development—and turn their frog into a prince of a model organism.

–GRETCHEN VOGEL

* "Moving into *Xenopus tropicalis*," University of Virginia, 12 June.