nanced by "debt for nature" swaps that divert funds from debt payment to science. Additional money could come from profit-sharing agreements with, say, pharmaceutical companies. Some of the profits from new natural products would be reinvested in conserving natural habitat. "This is a treasure hunt in which part of the profit is put back into saving the treasure itself," Eisner says.

Metcalf thinks a way to prevent the dissipation of entomology's "core" skills would be the establishment of entomology institutes, within a university framework but free from the pressures of teaching. These, he says, would be better vehicles than university departments for conserving knowledge of specific groups of organisms. Such institutes would be "anchored at one end in agriculture and at the other in medical entomology" and would draw on many specialities in between. "But we ought to hurry," Metcalf says, "If we wait 10 years to do these things, we won't be any better off in 100 years than we are now."

Gwadz favors a program of training fellowships for medical entomologists that would make it possible for them to maintain an affiliation with a U.S. university while spending considerable time in the developing world. Gwadz has proposed such a program, funded by the U.S. Agency for International Development and administered by the National Institutes of Health. But, he says, AID wasn't interested.

Wilson takes a broader, cross-disciplinary point of view. "We need homeostatic devices," he says, "refugia, floors below which disciplines are not allowed to fall. This activity might be a function of the National Research Council conducting discipline surveys with a view to maintaining subsistence-level support for disciplines even when they appear to be wholly out of fashion. Then, when there is a scientific breakthrough or a sudden societal need, it will be possible to start up again quickly."

The picture is not entirely bleak. Eisner, for example, says that after many years of crying in the wilderness he now sees increased awareness of the problem on the part of private institutions—and even to some extent in the government—and more willingness to look for ways to finance projects related to biological surveys and chemical prospecting.

Nonetheless, the world of entomology faces the real danger of losing hard-won fundamental knowledge in the excitement of learning and applying remarkable new techniques. And the problems of food, disease, and species extinction mean that the time for resolving these issues is short.

■ CONSTANCE HOLDEN

Neuroscientists Track Nerve Development

Last week more than 12,000 researchers descended on Phoenix, Arizona, for the annual meeting of the Society for Neuroscience—a striking testament to the growth and vitality of their chosen field of endeavor. Some of the symposia provided new insights into the fundamental problems of nerve cell regeneration and development.

A Piece of the Nerve Regeneration Puzzle

Peripheral nerves, such as those in the arms and legs, will grow back after an injury. But severed spinal cord nerves will not. The $64,000 question is why not. Martin Schwab and his colleagues at the University of Zurich, Switzerland, may have found part of the answer. The new information may aid the search for ways to spark nerve regeneration in people who have suffered spinal cord injuries.

Work with rats suggests, the Zurich researchers say, that spinal neurons are unable to regenerate because their growth is inhibited by two related proteins present in the fatty myelin sheath surrounding them. In contrast, myelin from the animals' peripheral nerves lacks both the proteins and the growth-inhibitory activity. The activity is also lacking, Schwab told *Science*, in the myelin of lower vertebrates such as fish, whose spinal neurons regenerate following injury.

Schwab presented recent results indicating that partial spinal nerve regeneration can take place if the inhibitory proteins are either absent or inactivated. The researchers found that some spinal neurons would grow back in rats in which the proteins had been neutralized by antibodies or eliminated by killing the myelin-producing cells.

But this news does not mean the problem of nerve regeneration has been solved. The number of neurons that grew back was small, probably because the inhibitory proteins are only one of several impediments to

Animal Activism 101

"We're here to confront one of the most important issues our society is dealing with." So said David Hubel, president of the Society for Neuroscience, when he opened one of the best attended sessions at last week's neuroscience meeting in Phoenix.

Nearly 1000 neuroscientists had packed the lecture hall to get some practical advice on how to combat attacks by animal activists. They heard from two of their colleagues, Rick Van Sluyters of the University of California, Berkeley, and Stephen Lisberger of UC San Francisco, who know only too well how vulnerable neuroscientists are to such attacks. Their message: Prepare your case before trouble arises and enlist the support of your colleagues and your institution.

The pair had learned those lessons the hard way. In 1983, Lisberger's research on eye movement in monkeys became the target of a prolonged and personal campaign. Animal activists broke into his lab, staged a mock funeral in front of UCSF for one of his monkeys, and bombarded him with letters, phone calls, and death threats for more than a year. Van Sluyters came under attack last year when activists held a press conference denouncing his research on vision in cats as "the most cruel and worthless animal research of the year," in an unsuccessful effort to stop state funding of a new animal facility on the Berkeley campus.

Lisberger and Van Sluyters emphasized that researchers who come under similar attack should never speak in their own defense. "You absolutely cannot defend yourself," Van Sluyters said. "It is by definition self-serving and there is no way you can be credible in that situation." What helped in their cases was a swift showing of public support by their institutions and colleagues. But no defense will work, they warned, if either the institution or the researcher is guilty of not giving humane treatment to the animals used in research.

So all animal researchers, Van Sluyters said, should work to see that their institutions have sound animal care and use programs. And they should be sure their own research protocols are up to date, approved by the appropriate committees, and followed to the

neuronal regrowth. Nerve cells may also fail to regenerate because of poorly understood inherent properties, or because they lack growth factors or the molecules that help them cling to a surface as they grow.

How to Build a Synapse

Once a growing axon reaches its destination it has to form a link, called a synapse, with its target cell. Researchers are now beginning to clone the genes for the proteins that help form the most accessible type of synapse, the one between nerve and muscle cells. If they can understand how this synapse forms, researchers hope, they may be able to learn how less accessible synapses, such as those in the brain, form.

Early on, work by Uel J. McMahan, of Stanford University had suggested that specific molecules located in the extracellular sheath surrounding muscle cells might direct the formation of nerve-muscle connections. Now Joshua Sanes and his colleagues at Washington University in St. Louis have cloned the rat gene for one of those proteins, which they call s-laminin (pronounced "slaminin"), for synaptic laminin, because it is closely related to laminin, a ubiquitous extracellular protein on which neurons readily grow. The evidence is still preliminary, Sanes says, but s-laminin may be the signal that tells regenerating motor neurons that they have arrived at the right spot to stop and form a synapse with a muscle cell

The muscle side of the neuromuscular junction contains clusters of receptors for acetylcholine, the chemical nerve cells release to trigger muscle contraction. Researchers have also recently cloned the genes for two neuronal proteins that may play a role in forming those clusters. Gerald Fischbach and colleagues at Washington University School of Medicine obtained the gene for a protein they call ARIA (for acetylcholine receptor–inducing activity), which was identified originally by its ability to turn up acetylcholine receptor production by muscle cells. And McMahan, Richard Scheller, and their co-workers at Stanford cloned the gene for agrin, which causes acetylcholine receptors to aggregate when it is applied to muscle cells in culture.

Both agrin and ARIA are produced in motor neurons in very young embryos, suggesting the proteins could play a role in development as well as in nerve regeneration. But s-laminin, ARIA, and agrin may have additional functions beyond those postulated for them in the neuromuscular junction as they, or at least some close relatives, are present in other tissues as well.

And the cloning of ARIA has revealed an interesting twist—it is very similar to the cellular prion protein, part of which is the infectious agent in neurological diseases such as scrapie. What that resemblance might mean is still a mystery. But with the three clones in hand, the researchers can now set out to clarify just where the proteins and their relatives are made, as well as what they might be doing there.

A Temporary Scaffolding Helps Build Brains

The nerve cells of the embryonic brain are faced with a formidable problem. As they develop, they send out long projections that must find their exact destinations within the complex layers of the brain. Researchers at Stanford University have found the first evidence that the mammalian brain may solve this problem by constructing a temporary scaffolding of cells that act as roadways to guide neurons to their destinations.

Several months ago, Carla Shatz, Susan McConnell, and Anirvan Ghosh described neurons in the developing cat brain that send the first projections from the cortex to other brain areas. The neurons, called "subplate cells," for the area of the cortex where they originate, make their journey when the brain is young and still relatively simple. Like scaffolding, they are temporary and later die. The Stanford group proposed that the neurons may serve a "pioneering" function, as some neurons have been shown to do in insects and fish, blazing trails through the nervous system for other neurons to follow.

At the neuroscience meeting, McConnell and Ghosh reported that they now have evidence that the subplate cells do indeed serve such a function. When Ghosh killed the subplate neurons in a particular region of the cortex early in development, neurons that normally grow to that region from deeper in the brain seemed to lose their way, growing past the point where they would normally turn to enter the cortex.

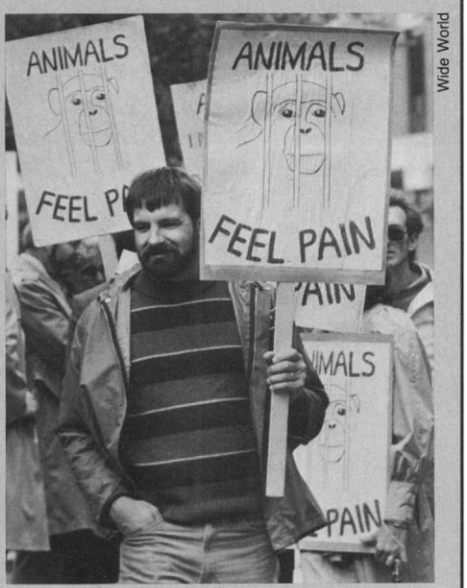
And do the subplate cells also help neurons growing out of the cortex find their way to other parts of the brain? It's too early to tell for sure, but Ghosh presented what McConnell called a "tantalizing hint" that this is true. The researchers killed the subplate cells and then looked for signs that cortical neurons were able to grow permanent connections to one of their normal distant destinations in the brain. But in the absence of the subplate cells, the cortical neurons were apparently unable to find their way. ■ MARCIA BARINAGA

letter, Lisberger advised. "Is there anything you are doing in the lab that you could not defend to your colleagues?" he asked. "If you are in compliance with both the letter and the spirit of the law, the scientific community will come to your defense. If you are doing anything wrong, I can't predict what will happen."

But just following the rules is not enough. "Your institution must have a designated spokesperson who knows about biomedical research," said Van Sluyters. "Prepare a brief summary [for that person] that says what you do and why it is important," he suggested. That statement, Lisberger added, will be the springboard for a timely press release, should one be needed.

"There are times when there is no course that will be smooth," said Lisberger. Though he was "kept afloat" by the knowledge that he had done nothing wrong, he said the personal threats were emotionally grueling, and he still jumps when his doorbell rings after dark. He advised animal researchers not to list their phone numbers or at least not their addresses.

The two warned their audience not to assume that these things only happen to other people. "What happened to me will probably never happen to all of you, but it will happen to some of you," said Van Sluyters. "Have [everything] ironed out ahead of time, so it doesn't have to be done over the telephone, at 11 at night, when the details of the attack are sketchy and you are panicked." ■ MARCIA BARINAGA



Wide World

Picketers protest *animal use in research.*