

Coalition For Animals & Animal Research

CFAAR Arizona Newsletter

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To join the Arizona CFAAR, please fill out the membership form on the back page. Donations publish our newsletter and educational materials. A year's subscription is included with your contribution.

CFAAR: Who We Are

CFAAR is a nonprofit educational organization which formed in response to activists who were attempting to discredit animal research and animal researchers in 1988. Several local CFAAR chapters have since sprung up across the country. These groups share the following objectives:

- 1) To **organize** students, faculty, and staff at institutions where animal research is performed so effective letter writing campaigns can be initiated quickly.
- 2) To **educate** the public, in general, and the campus, in particular, about the true nature of animal research and animal researchers.
- 3) To **support** responsible and humane use of animals in biomedical research.

The first of these objectives will be the primary function of the group. As legislation is introduced that affects animal research, we need to respond so our representatives know exactly how we, the people, want them to vote. Accordingly, through our newsletter, we will help inform you about legislation and other "happenings" concerning attacks on animal research. Our goal is to make it as easy as possible to contact your Washington, D.C. representatives.

The key to the effectiveness of this organization is you! We need your willingness to write an occasional letter, perhaps talk with a school group and, of course, give a few dollars to cover the cost of printing the newsletter and educational materials.

**HELP SUPPORT CFAAR
SO WE CAN SUPPORT YOU**

"Thank you Research" Growing

Researchers feeling beleaguered by animal rights activists are receiving support from a most likely source - the patients and others who have or will benefit from their work. Soon even more researchers will be hearing from grateful patients.

For the last two years, a very informal effort to connect patients with researchers has been ongoing. Spearheaded by a few grateful parents of children who literally owe their lives to medical research, other parents all over the country have been signing simple thank you cards and sending them, with photos of their children, to those to whom they owe so much - biomedical researchers.

Nora Rumpf began the project after her son Lee's life was saved by extracorporeal membrane oxygenation (ECMO). Born with severely damaged lungs, Lee's chances of surviving with conventional therapy were less than 20% when he was placed on ECMO, a lung bypass procedure which has saved the lives of more than 5000 newborns since 1976. Animal research was essential to ECMO's development, continued refinement, future advancement, and the training of hospital staff who treat children while they are on the ECMO machine.

The effect on researchers who have received the thank you cards has been gratifying - and exactly as intended. Ms. Rumpf has received dozens of letters from researchers. Universally surprised and gratified, the researchers have expressed their reactions in the following ways:

After receiving hundreds of pieces of hate mail, it is really heartening to hear from people who appreciate what we are trying to do.

Your card was so encouraging - it has touched many people's hearts because it really is the "first" real thank you for a difficult job. One of our caretakers - a middle aged man who has cleaned cages for 12 years - had tears in his eyes.

When people like you go out of your way on our behalf, it makes a world of difference.

Now the effort is expanding. Ms. Rumpf and Kathy Braga, another ECMO mom, have established a new organization in Washington, DC, called Thank You Research. Through them, even more thank you cards will be sent to researchers from patients and their

families. Working first with the American Diabetes Association, Thank You Research will be collecting cards and pictures from among 14 million Americans suffering from the debilitating and life-threatening disease.

“Our hope is that, some day, one of our cards will hang over every lab bench in American,” said Ms. Rumpf. “Whenever researchers are confronted by the faces of animal rights demonstrators, we hope they will remember our faces and know that those for whom they labor do know the value and honor their work.

(CFAAR Berkeley News, Winter/Spring 93)

Animals in Diabetes Research

If you know 20 people, chances are one of them is among the 11 million Americans suffering from diabetes. In 1989, an additional 600,000 will develop the disease. Because diabetes is so prevalent, almost everyone is familiar with the disease. Or are they?

Most people are really unaware of just how serious a disease diabetes is. For instance, did you know diabetes is the third leading cause of death by disease? Or that it is the leading cause of new blindness for people under 65? Or that a person with diabetes has twice the chance of a heart attack or stroke? Or that kidney disease is 17 times more common among diabetics?

And the dollar cost of diabetes is enormous, \$20.4 billion in 1987. It accounted for more than 11.5 million days of hospital care costing \$6.9 billion. Another \$1.7 billion was spent for doctor visits and medications, and \$942 million for nursing home care. \$10.8 billion was attributed to lost productivity.

What is diabetes? Diabetes is a disease where the body fails to produce or effectively use insulin. Diabetes in children, if not treated, can kill in a short time; in older patients, it may go undetected for years. Even with treatment, complications (such as blindness, kidney failure, loss of limbs, heart disease, or stroke) almost inevitably occurs.

What kinds of diabetes are there? Diabetes occurs in two forms – Insulin-Dependant Diabetes Mellitus (IDDM) and Non-Insulin-Dependant Diabetes Mellitus (NIDDM). Insulin-dependant diabetics require regular injections of insulin to keep their blood sugar levels within acceptable limits. IDDM is usually diagnosed at an early age, and is often referred to as juvenile onset diabetes. IDDM's major characteristic is a loss of the beta cells that secrete insulin from the islet of Langerhans located in the pancreas. IDDM appears most often in people with a genetic tendency towards diabetes and who then acquire a viral infection. The infection sets in motion a chain of events resulting

in the death of all beta cells. Once the cells die, these people become insulin-dependent.

Nearly **one million** IDDM patients live in this country, but if insulin were not available, they would not be diabetics, they would be dead.

NIDDM is the most prevalent form of diabetes, with approximately **ten million** NIDDM patients in America, many undiagnosed. A non-insulin-dependant diabetic is able to maintain proper blood sugar levels through special diets. Many non-insulin-dependant diabetics are able to live their entire lives with dietary control alone – others eventually become insulin dependant.

Are humans the only animals to get diabetes? In addition to humans, diabetes also naturally occurs in rats, cats, dogs, (Michigan State University has a colony of diabetic dogs) and several primate species (the Oregon Regional Primate Research Centers has several insulin-dependant Macaca nigra monkeys). For this reason, these animals are used to study diabetes, and the new treatments developed through this research benefit those animals as well as man.

What has animal research done to help diabetics? The major turning point for treatment of diabetes came in 1921 when Dr. Frederick Banting and Charles Best developed a means of extracting insulin from the pancreases of dogs (pound dogs, by the way). In a short time, insulin from cows and pigs was being produced in large quantities and saving the lives of thousands of diabetics.

Since then, many important developments have come about through animal research. Information about dietary management of diabetes has increased due to controlled animal experiments. Research and safety testing on dogs made possible external insulin pumps, now becoming available for diabetics having difficulty regulating their blood sugar levels through conventional injections. The first human trials of internal insulin pumps (PIMS), are underway at Johns Hopkins University, following several years of carefully evaluation and refinement in dogs.

What hope does animal research hold for the future? Perhaps the most exciting phase of diabetes research is that which is currently underway into curing the disease. Researchers are pursuing two promising courses to reach this goal – islet cell transplantation and gene therapy – and animals are vital to both of these pursuits.

What about transplantation? In recent years, surgeons have successfully performed several transplants of entire pancreas gland. However, all organ transplants carry with them the drawback of organ rejection. For most people with diabetes, the side effects of the required anti-rejection drugs are worse than the symptoms and complications of diabetes

itself. However, if a diabetic has kidney failure and the only hope of survival is a kidney transplant, a simultaneous pancreas transplant is often considered. iiFAR has a member in California who was an insulin dependent diabetic since childhood and suffered kidney failure. She had a combination kidney/pancreas transplant, and is now completely cured of her diabetes.

The most promising of transplant techniques is that of transplanting only the insulin producing islet cells. This has proven successful in inbred mice, and is now being perfected in random source dogs (pound dogs). Islet cell transplantation is not a major surgery, and the problem of rejection is not nearly as great as with an entire organ.

What is gene therapy? The really high-tech approach to curing diabetes is through gene therapy. Scientists have been able to isolate the human gene that produces insulin. When this gene is injected into a single-cell mouse embryo and the embryo is re-implanted into the mother mouse, the resulting offspring produces human insulin and the insulin production is regulated in the same way as in humans. This type of gene therapy is called germ-line, and is the easiest to accomplish.

Gene therapy research for diabetes has now progressed to the more difficult technique, known as somatic cell therapy. Genes put into somatic cells only affect the animal – or person – treated. By contrast, genes put into germ-line cells (such as embryos) would be inherited by the offspring. For ethical reasons, most scientists believe germ-line gene therapy is inappropriate for humans.

Mice are currently being used in somatic cell gene therapy. Researchers have been successful in injecting human insulin-producing genes into mice with diabetes, and the human gene causes insulin production (too much, in fact). Before this process can progress upwards to dogs then humans, more work with rodents is needed to understand the complex ways in which the insulin gene is normally regulated.

Animals are important to diabetes research! Animal research is critical to the understanding of diabetes. If research on diabetes is stopped because laboratory animals are not available, the additional cost in human suffering and the financial burden to society would be enormous. Both will be dramatically reduced when diabetes can be prevented and/or cured, hopefully not-too-distant future.

(iiFARsighted Report, 11/88, Vol 2, No 4)

Arthritis Sufferer Hopes Studies Bring Him Relief

Each day for Kenneth G. Brown is like licking honey off a cactus. About 19 years ago, he lost

some time from his career as a house painter in Michigan when he went through a battle with flu that left him flat on his back.

The “flu” never ended. It turned into advanced rheumatoid arthritis. Unable to work, Brown moved to Tucson in 1972 with his wife and two children, where, he says, “I did nothing. I stayed at home under my wife's feet for 10 years. She had to work.”

Today, at age 46, Brown lives with hope – hope that research to help him “will come through real soon.” He takes 20 medications daily. They cost \$200 a month. One of them is a prednisone derivative to maintain cortisone levels in his body. “One of the problems with rheumatoid arthritis,” Brown explains, “is that the gland that makes cortisone in your body shuts down.” But use of prednisone, he knows, eventually leads to kidney and liver damage.

He's been in a wheelchair for about eight years. He has artificial knees. His ankles are torn up. A chronic staphylococcus infection between the bone and the knee prostheses won't heal, which prohibits his being able to have additional surgery. Yet Brown is busier than most healthy people, says his wife, Sharon L. Brown. Thanks to a rehabilitation program at The Arthritis Foundation that led to training in electronics, Brown has worked as a master control engineer at television station KVOA for nine years.

In addition, says his wife, Brown takes classes at Pima Community College; has served as president of a local ham radio club and gets involved in public-service ham radio activities such as walk-a-thons, circuses and rodeos. He bowled on a handicapped league and builds remote control airplanes and sailboats with his son. “You can't just sit around and be sick,” says Brown. “The thing to do is focus on what you can do and go on. It's easy to say; hard to do. When I find myself trying to do something I could do a month ago and can longer do, I find myself getting depressed.

Few people connect medical research using animals with human health unless they meet up with a person such as Brown, whose future may depend on that research. There is no cure for rheumatoid arthritis, which is estimated to afflict about 7 million Americans. It's the leading cause of disability in the United States; more than 1 million people are totally disabled from it – about one out of every seven.

Drugs help reduce some pain and symptoms of the disease; rest and exercise also can help. Arthritis is only partly understood. Most of what is known about is has been unraveled using animal models.

Eric P. Gall, director of the Arizona Arthritis Center, says rabbits have proven to be effective models of the disease for researchers to study. One model was developed by injecting egg albumin into the animals'

joints; this led to responses that resembled human rheumatoid arthritis, Gall says. The rabbits helped them realize that rheumatoid arthritis continues to affect victims' joints even after the viral infection that triggers it goes away, Gall says. "The material that stimulates the disease is stored in a part of the joint where you wouldn't expect to see it. Rather than in the lining of the joint, it's stored in fibrous material like the ligaments surrounding the joint of the meniscus in the knee."

This material seems to cause something to go haywire in cells of the protective fluid surrounding the joints. As this fluid volume increases, the joints swell. They hurt, and they get stiff. Eventually, the joints, as well as muscles and other nearby tissues, are destroyed. The final tissue reaction is that the joint fuses.

In another rheumatoid arthritis study, also involving rabbits, a new class of immunomodulator drugs is being studied for its effect on the crippling disease, Gall says. David Yocum, research director for the Arizona Arthritis Center, developed a different rabbit model to study these drugs, which work by modifying a specific function of cells rather than by killing them.

Yocum developed a model using the same bacteria that causes Strep throat, Gall says. When injected into the abdomens of genetically susceptible rabbits, the bacteria created symptoms of rheumatoid arthritis. Yocum used cyclosporin-A, a drug commonly known for its use with transplant patients to combat organ rejection. The drug seems to help control joint pain and swelling by subduing T-helper cells, a type of immune-system cell. "It's very effective immunomodulator drug," says Gall. "From studies in animals and then humans, he found he could predict in which patients the drugs would work, so you don't waste time and money and the possible toxicity of drugs in patients for whom cyclosporin won't work."

(Lo Que Pasa, March 1990)

Holding Human Health Hostage by Michael E. DeBakey, M.D.

As a patient-advocate, both in and out of the operating room, I feel a responsibility to protest the rights of patients to medical advances resulting from animal research. Had the use of pound animals in research been prohibited when I began my career, it would have prevented me from developing a number of lifesaving procedures in my research laboratory. Instead of restoring thousands of patients to a normal life and a return to productive work, my colleagues and I would have been helpless to offer many of our patients any real hope at all. "If scientists abandon cat and dog experiments for other models that are not as well

understood, many potential medical breakthroughs may be severely crippled or halted."

Even with today's technology, I could not have developed on a computer the roller pump that made open-heart surgery possible or the artificial artery that restored to health previously doomed patients with aneurysms. Nor could we have attempted the first successful coronary artery bypass or implanted the first temporary mechanical heart which we saved a patient's life two decades ago. Would animal-rights activists have objected to the first kidney, heart or liver transplant? Would they forgo the protection humanity enjoys today against polio, tetanus, diphtheria, and whooping cough or the treatment for strep throat, ear infection, bronchitis, and pneumonia – all the products of animal research? Would they have denied the 11 million diabetics the right to life insulin has given them – or victims of cancer the help they have received from radiation and chemotherapy? It was in monkeys the deadly AIDS virus was isolated, and that isolation is the initial step in the ultimate development of a vaccine. Would the animal-rights activists halt that research and allow an epidemic to rage unopposed? The truth is there are no satisfactory insentient models at present for certain types of biomedical research and testing. A computer is not a living system and would not have produced the dramatic medical advances of the past few decades.

Only about 1% of abandoned dogs are released for research. If pounds are such a meager source of research animals, you may ask, why am I concerned about losing that source? My reasons are well founded, I believe: not only are pound animals of particular value in research on heart and kidney disease, brain injury, stroke, blindness, and deafness, but a ban on their use could have grave and far-reaching consequences for human and animal health. In addition, such a ban would impose an extra burden on taxpayers and could price many important research projects out of existence. Each dog and cat bred specifically for research costs hundreds of dollars more than a pound animal. Legislation prohibiting the use of these animals makes no accommodation for this substantial rise in cost. For many of our most productive researchers, the additional expense would shut down their laboratories. Critical work on inducing tolerance in organ grafts, for example, and on minimizing damage to cardiac muscles after heart attacks has been halted in some research labs because of the soaring cost of dogs.

Moreover, eliminating the use of pound animals in research would, paradoxically, cause even more animals to die. According to the American Humane Society, 7 million dogs are abandoned to pounds or shelters each year, 5 million of which are killed– 600 "trusting pets" killed hourly. Yet some would have you believe killing animals in a pound is more virtuous than

having them help advance medical knowledge and ultimately benefit human and animal health. I don't like to see life taken from any species unnecessarily, and that would happen if pound animals are prohibited from research. Every year we would have to breed an additional 138,000 dogs and 50,000 cats for research to replace the pound animals, which would then be put to death anyway because no one wants them. With the current overpopulation of dogs and cats, the logic of such a policy escapes me.

It was humane concerns that led me into medicine. I strongly disapprove of cruelty to animals as well as humans. Medical scientists are not engaged in cockfighting, bullfighting, or any other "sport" imposing stress or violence on animals. Rather, they are searching for ways to relieve suffering and preserve life. Unquestionably, every precaution should be taken, and enforced, to ensure laboratory animals are treated humanely. Responsible scientists observe humane guidelines, not only because their search for new medical knowledge is motivated by compassion for the suffering, but because they know improper treatment adversely affects the quality of their research. Scientists are also obligated to use insentient models when these are satisfactory, but again, no responsible scientists would incur the substantial expense and devote the considerable space required for housing and caring for animals when other equally satisfactory models were available.

If scientists abandon cat and dog experiments for other models that are not as suitable or as well understood, many potential medical breakthroughs may be severely crippled or halted. Grave diseases such as AIDS, cancer, heart disease, muscular dystrophy, Alzheimer's disease, and other serious conditions will, however, continue to plague our families, friends, and fellow citizens.

Remember, too, pets have also profited from animal research. It is doubtful animals could be treated today for heart or kidney disease, leukemia, or other serious disorders, if animal research had been prohibited previously. If an animal is seriously ill or injured, would the animal rights activist deny it a form of treatment potentially beneficial but never used before—and therefore experimental? Until one is faced with a life-threatening condition of a loved one—human or animal—it is difficult to answer that question truthfully.

We have aggressive advocates of the rights of trees, sharks, bats, whales, seals, and other mammals, but what about the rights of ailing humans? Shrill attacks against speciesism are difficult to defend when one observes pit bulldogs mauling and killing children, wolves killing deer, cats consuming rats and birds consuming worms. And even vegetarians destroy living plants for consumption. Self-preservation is a primary instinct of all members of the animal kingdom, and patients with that instinct deserve our compassion, protection, and assistance as much as other species.

There is a difference, moreover, between animal welfare and antiscience. Infiltrating laboratories surreptitiously posing as volunteer workers, destroying research records, vandalizing research facilities, bombing and threatening scientists are all irrational methods of persuasion. At one research institution, damages amounted to more than a half million dollars when computers were destroyed, blood was poured on files, and liberationist slogans were painted on laboratory walls. Research on infant blindness was halted for eight months while claims of animal abuse were investigated, only to be found baseless. Such harassment, demoralization and interference, divert funds from productive research to security and discourage bright young people from entering research. Once the manpower chain is broken, it will not be easily restored. And where will we then turn for answers to devastating human diseases? Guerilla tactics, lurid pictures, and sensational headlines may inflame emotions, but they do not lead to rational judgement. More important, should we condone harassment, terrorism, and violence masquerading as concern for animal rights?

As a physician, my greatest concern is, of course, for the suffering human beings who will be denied effective treatment because we took action that seems superficially humane but may ultimately render us powerless against certain diseases. What do I tell dying patients who are waiting for the medical advances these threatened investigations may produce—there is no hope because we have been prevented from acquiring the new knowledge needed to correct their conditions? As a human being and physician, I cannot conceive of telling parents their sick child is doomed because we cannot use all the tools at our disposal. Surely those who object to animals in research laboratories must be equally distressed at seeing sick children hooked up to tubes. How will these parents feel about a society that legislates the rights of animals above those of humans?

Through research, we have made remarkable advances in medicine, but we still do not have all the answers. If the animal-rights activists could witness the heartbreaking suffering of patients and families I encounter daily, I doubt they would deliberately pose a direct threat to human and animal health by demanding we abandon some of our most fruitful methods of medical investigation. The American public must decide: Shall we tell hundreds of thousands of victims of heart attacks, cancer, AIDS, and numerous other dreaded diseases that the right of abandoned animals to die in a pound supersede the patient's rights to relief from suffering and premature death? In making that decision, let us use not anger and hatred, but reason and good will.

(liFARsighted Report, Vol 1, No 2, 8/87)

Vaccine Shown To Block HIV Infection In Chimps

An experimental vaccine has proved in a laboratory test on chimps that it can immunize against AIDS-infected blood cells, a common pathway for spread of the disease among intravenous-drug users. Chimps inoculated with the vaccine remained free of the human immuno deficiency virus, or HIV, for up to a year after they were injected with contaminated blood, said Patricia Fultz of the University of Alabama in Birmingham. "This proves we're on the right track," she said.

The study, to be published today in the journal *Science*, tested how effective an AIDS vaccine would be against a virus that is within blood cells. The vaccine in an earlier study was tested against free-floating virus-virus particles in the plasma of the blood—but not within the blood cells.

HIV, which causes AIDS, is known to cause infection in both forms. "Many people felt this was a stronger challenge for the vaccine because infected cells have the potential of producing much more virus," said Fultz, who was the lead author of the study. The research involved three chimpanzees inoculated with a vaccine and then injected with HIV-infected blood cells. Fultz said the vaccine mimics the molecules found on the outside of the AIDS virus and on the outside of cells that are infected with the virus.

After inoculation, all three chimps were injected with doses of blood cells from a chimp infected with HIV. Researchers then tested the three chimps periodically to see whether they developed an HIV infection. One chimp died of heart failure, not associated with AIDS, after seven months, Fultz said. Tissue samples taken from this chimp were cultured for six weeks, and there was no evidence of viral infection, she said. The two other chimps were found to be immune to HIV for a full 12 months. "This tells us that the vaccine candidates with which we are working have the potential to protect against both forms of the virus and that we don't need to revise our vaccine at this point or try additional types," Fultz said. Although the new test has the promise of an AIDS vaccine, Fultz said much more work needs to be done before it can be tested in humans.

(The Arizona Republic, 6/19/92)

Monkey-Human Viral Hybrid is New Weapon in AIDS Fight by Jon Cohen

On the eve of the Eighth International Conference on AIDS in Amsterdam, researchers at Boston's Dana-Farber Cancer Institute have announced that they have made a combination human-monkey AIDS virus, which they believe will be a powerful new weapon in the battle against the pandemic. Called "SHIV" by the group, the weapon is a hybrid of the

human AIDS virus, HIV-1, and its simian cousin, SIV. In the June issue of the *Journal of Acquired Immune Deficiency Syndrome (JAIDS)*, the team reports that a genetically engineered SHIV has persistently infected four cynomolgus monkeys, a relatively plentiful species.

Dana-Farber's Joseph Sodroski - whose graduate student, John Li, engineered the SHIV - adds that if these monkeys end up getting sick from SHIV, the model may help solve the mystery of how the AIDS virus savages the immune system. "I'm ecstatic," says Sodroski's colleague William Haseltine, who is both a coauthor of the SHIV paper and an editor of *JAIDS*. "What this does is break a bottleneck in vaccine testing." But an Amsterdam, Haseltine's enthusiasm could be tempered a bit by a competing group of Japanese researchers - who claim they developed a similar combination virus first but had trouble publishing their results while the Americans hustled to print.

The bottleneck Haseltine refers to is the fact that the only animal model so far available for testing HIV-1 vaccines has been the chimpanzee, a rare and expensive species that can be infected with HIV-1 but does not seem to develop AIDS. The chimp model, in fact, has so many limitations that most AIDS vaccine work has been done in monkeys using the monkey virus, SIV. Though SIV handily infects several monkey species and causes disease, as a model for human infection it has the handicap of having significant genetic and functional differences from HIV-1.

So does SHIV, but the Dana-Farber group argues that it has enough in common with HIV-1 to be, as they write, "a valuable model for study of the efficacy of anti-HIV-1 vaccines." And in that view they have supporters, one of whom is Alan Schultz of the National Institute of Allergy and Infectious Diseases: "It's so sweet, it's so fantastic," says Schultz, acting director of the vaccine branch at the Division of AIDS. Schultz is particularly elated because real-life tests of AIDS vaccines are right around the corner and SHIV could be the route to revealing which vaccines have the best chance of working - a route researchers desperately need. "There's virtually no way right now to evaluate the efficacy of HIV-1 vaccines in an animal model," laments Schultz. Masanori Hayami, head of the Japanese group developing chimerics at Kyoto University's Institute for Virus Research, agrees the SHIV data "looks good". But his group came up with the original idea for chimeric SIV-HIV-concedes - and has made several variations. Hayami's group even came up with a hybrid much like SHIV, which they call NM-3n, that they have isolated from a cynomolgus monkey that was infected 37 weeks ago. But the Americans have published their animal data and the Japanese haven't, and that has produced some ill feelings.

Hayami contends his group was on the verge of publishing their animal data after 4 years of work and that the Dana-Farber group "hurried their publication" in *JAIDS* because they "know what we are doing." He

adds: "It is our surprise (that) Dr. Sodroski's paper was received (by JAIDS) May 1, accepted on May 7 and published in the June issue. Usually we must wait for the answer for 2 to 3 months and it takes a half a year for publication." The Japanese paper is now in press at the Journal of General Virology.

Sodroski acknowledges that "to be in a competitive position" his lab turned on the afterburners. "Were we interested in rapid publication?" he asks. "Sure we were." But both Sodroski and Haseltine emphasize that their paper was peer reviewed and they believe their chimeric is better. "We have a great deal of respect for Dr. Hayami," adds Haseltine. Attendees at the Amsterdam meeting will have a chance to hear the American and Japanese teams describe their chimerics and, perhaps, divvy up credit.

They will also hear more about the fact that SHIV is not the only recent development that could help break the AIDS vaccine testing bottleneck. Only weeks ago, a University of Washington research team working with pigtail macaque model for testing HIV-1 vaccines (Science, 19 June, p. 1630 and 3 July, p. 103). But some researchers doubt that the macaque model will pan out. And in any case, some researchers think introducing SHIV into readily available species of monkeys could be more practical than attempting to infect the pigtail with HIV-1 since the latter, though not endangered, are still in short supply.

SHIV is essentially the monkey virus SIV dressed in the outer, or envelope, proteins of the human AIDS virus. It contains two critical genes from HIV-1 - tat and rev - that help to regulate viral replication. The value of this "chimeric" virus lies in the fact that envelope proteins are the key components of many experimental HIV-1 vaccines. HIV-1 cannot infect cynomolgus monkeys, but a vaccine made from HIV-1 envelope proteins should stimulate their immune systems. If the vaccine works, then when the monkeys are later "challenged" with chimeric virus, they should be able to resist infection.

Of course, it will be a while before anyone can say whether SHIV has proven out. Norman Letvin of Harvard's New England Regional Primate Research Center, who infected monkeys with SHIV and continues to evaluate them, says the animals have had the virus in their blood cells for more than 3 months now. And what if both the SHIV model and the pigtail macaque work hold up? A debate between partisans of each school likely will follow, because, to anticipate only one argument, since the current chimeric model is based on HIV-1 envelope proteins only, it cannot be used to test vaccines that rely on HIV-1's inner, or core, proteins. Indeed, this point has already led Washington's William Morton to assert that the pigtail system holds more promise. "You want to try to come as close as you can to human disease and pathogenesis," says Morton. "If you can use the whole HIV-1 in the monkey model, that's the best of all possible worlds." Then again,

partisans on both sides would much prefer to join in such a debate than to find either or both of these approaches falling by the wayside.

(Science, Vol. 257, July 24, 1992)

New Neurons Produced in Mouse Brains

Researchers at the University of Calgary have found that, contrary to scientific belief, cells from the striatum region of adult mouse brains are capable of producing new neurons in culture. Scientists say if the procedure can be duplicated in humans, it would raise the possibility that someday certain forms of brain damage and disease could be treated by inducing the brain to heal itself.

Samuel Weiss, assisted by graduate student Brent Reynolds, placed cells from the brains of adult mice in a solution containing epidermal growth factor, a substance produced by the body that stimulates cell division. Many of the cells died, those that survived began to multiply and then to differentiate into neurons & glia, cells which provide nutritional support to nerve cells.

The finding is astonishing because it contradicts a long-held dogma of neuroscience stating that mammalian brains cannot produce new nerve cells after the embryonic stage. Although exceptions to this rule have surfaced in the past, there was never any evidence of neuron production in the striatum, the region of the brain affected by oxygen deprivation as well as degenerative diseases such as Huntington's disease. "It challenged everything I had read, everything I had learned when I was a student," Weiss told the journal Science. "I couldn't imagine how I would try to convince people this was real."

Although there is no guarantee that production of new nerves would take place in adult brain tissue, the finding does indicate that progenitor cells, the cells which in the embryonic brain reproduce and differentiate into neurons and glia, are indeed present in the brain after it has reached maturity. Weiss and Reynolds concluded that although these cells are present in the adult brain, they must somehow be dormant.

The challenge now is to discover why, if those cells do exist in the adult brain, they do not produce new nerve cells on their own. If researchers can somehow enable the brain to produce new neurons, either by inserting cultured cells back into the brain tissue or enabling progenitor cells to reproduce and differentiate, new approaches to treatment of brain injury and diseases such as Alzheimer's and Parkinson's may result.

(FBR Newsletter, Vol. 4, No. 3)