

U.S. BUDGET

Spending Triples on Terrorism R&D

NASA may be best known for sending a man to the moon. Now it wants to show that it's no slouch when it comes to fighting terrorists. The U.S. space agency is one of 11 federal agencies (see pie chart) to share in a record \$1.5 billion that Congress has showered on terrorism-related R&D for 2002 in response to the 11 September and anthrax attacks. The money—nearly triple last year's spending—will be used for everything from new laboratories for studying potential bioweapons to developing hacker-proof computer systems.

The money is a windfall for researchers, who earlier last year were fighting to overturn proposed cuts in many terrorism-related R&D budgets. Even after the attacks, the White House paid scant notice to research in its \$40 billion emergency recovery package that was approved by Congress. But the Senate took up the cause with a vengeance, labeling as urgent more than \$800 million in new terrorism-related R&D projects. "We heard from everyone—university scientists, industry, [federal] researchers—about things they could do to reduce the threat if only they had some money," says an aide to one Senate Democrat.

The final package, mostly inserted late last month into the 2002 appropriations for the Department of Defense, contains \$711 million for R&D. That freshet of funds, when combined with spending approved earlier, will push 2002 spending on terrorism-related R&D up 157% over the \$579 million spent in 2001, according to an analysis by the American Association for the Advancement of Science (publisher of *Science*).

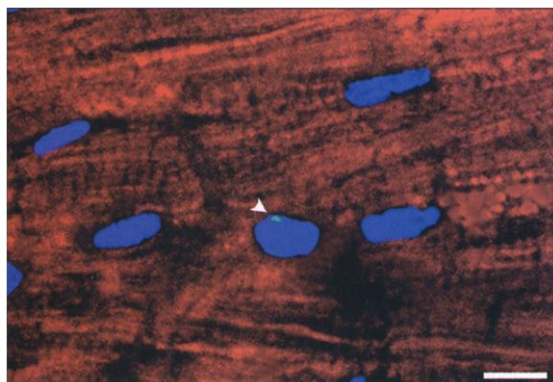
Most of the new money will be used to expand existing programs aimed at preventing terrorist attacks. The military, for example, gets a 50% increase for its multifaceted terrorism research efforts, to \$353 million. The Centers for Disease Control and Prevention, which has played a high-profile role in investigating the anthrax mail attacks, gets \$1 billion overall for security-related expenses, including \$130 million for studying anthrax and other potential bioweapons. That 256% increase "is hopefully just a down payment for research too long neglected," says one science society lobbyist.

benefit in remodeling the heart and possibly improving cardiac function." Several types of cells appeared to regenerate, including cardiac muscle, smooth muscle, and endothelium, Anversa says. Cells containing the Y chromosome appeared "perfectly indistinguishable" from neighboring cells that lacked a Y.

Suspecting that healing the heart might be a job for stem cells, Anversa's group then searched the heart tissue for three molecular markers characteristic of the versatile cells. They found cells bearing these markers both in transplanted and control hearts, suggesting that undamaged hearts harbor populations of such cells. The transplanted hearts contained even higher numbers of the cells, some of which came from the donor and others from the recipient, suggesting that the heart recruits stem cells from other parts of the body to aid in regeneration.

But it is unclear whether these are bona fide heart-specific stem cells and, if so, exactly how they promote regeneration of heart tissue. Nor do the researchers know where the cells originate or how they migrate. Before they can claim to have found stem cells, Anversa says, the team must isolate the cells and demonstrate in vitro that they are self-replicating and capable of differentiating into many types of tissue—work that is now under way.

Discovering how the heart might repair damaged tissue could have enormous, if distant, implications for treating heart dis-



Help from afar? A cell containing a Y chromosome (arrow) has taken up residence in heart tissue from a female donor.

ease. But as yet it's not even clear that the new cells help the transplanted organ, Binkley cautions. "Having recipient cells enter the [heart] could have certain detrimental effects," Binkley says, such as clogging up blood vessels. Only additional studies, he says, can determine if such cells are more balm than bane.

—CAROLINE SEYDEL

Caroline Seydel is a freelance science writer in Los Angeles.

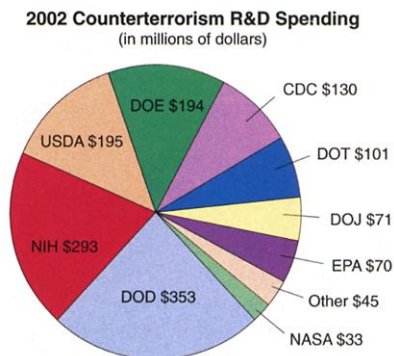
The bioterrorism budget at the National Institutes of Health will soar by nearly 500%, to \$289 million. The total includes \$75 million for a new highly secure laboratory to work with dangerous pathogens. The Department of Agriculture is also slated to get new laboratories; of \$113 million in new funds, \$73 million is set aside for an animal biocontainment facility at the National Animal Disease Laboratory in Ames, Iowa, and improvements at the controversial Plum Island Animal

Disease Center in New York (*Science*, 26 May 2000, p. 1320). The Department of Energy will devote \$78 million of its \$126 million in new funds to help prevent nuclear terrorism.

The country's space agency gets \$33 million for work on two fronts: information systems that terrorists can't penetrate, and imaging systems and other technologies to better detect enemies.

And the Environmental Protection Agency gets \$70 million to, among other things, develop better methods to clean up after any bioweapons are unleashed.

—DAVID MALAKOFF



Spreading the wealth. Eleven U.S. agencies will share a record \$1.5 billion in terrorism-related research funds.

STEM CELL RESEARCH

Rat Brains Respond to Embryonic Stem Cells

In the heated ethical debates over embryonic stem (ES) cells, Parkinson's disease often figures large. Advocates for more research say ES cells offer the best hope for treating or even curing this devastating and deadly disease, which gradually robs patients of their ability to move. The advocates hope that scientists will someday be able to turn ES cells into dopamine-producing cells, replacing those that are lost in the disease. So far, however, evidence that ES cells can make this switch has been limited to experiments in lab culture, not animals.

Now, in a paper published online on 8 January by the *Proceedings of the National Academy of Sciences*, a team of neuroscientists reports that, indeed, mouse ES cells can become dopamine-producing neurons in the brains of rats. These experiments are the first to show that the specific type of neurons missing in Parkinson's disease can develop from an ES cell in an animal's brain and lead to partial recovery.

But the work does not mean doctors will soon be injecting human ES cells into Parkin-

CREDIT: (BOTTOM) F. QUAINI ET AL., *NEURON* 34:8 (2002)

son's patients. "This shows that it can be done, but there are big obstacles to bring this to clinical fruition," says Ole Isacson of Harvard Medical School in Boston, who led the group. Among the caveats: Several rats developed deadly tumors, and the animals' behavioral improvements were limited.

Isacson's approach was surprisingly straightforward: He and his colleagues simply injected untreated ES cells into the rats' brains. In previous experiments, Isacson, postdoc Lars Björklund, and their colleagues had found that undifferentiated ES cells, when injected into animals, seemed eager to become neurons. However, the cells frequently grew out of control and formed teratomas, tumorous growths comprising a mix of cell types.

Isacson and Björklund suspected that the differentiating ES cells were sending conflicting signals to each other that promoted the growth and formation of the teratomas. The team decided to test whether diluting the cells would lessen the chance that the cells would interact with each other, thereby encouraging development along the possible default pathway: making neurons. The team prepared mouse ES cells in a dilute solution and injected about 2000 cells each into the brains of 25 rats. The rats had previously had their dopamine-producing neurons damaged and showed a characteristic tendency to move in circles toward the damaged side of the brain.

Six of the rats showed no evidence that the transplanted cells survived. Five died before behavioral tests were completed and proved to have teratoma-like tumors. But 14 of the rats had surviving mouse cells in their brains 4 months after surgery. All of the surviving grafts contained at least some dopamine-producing neurons. And many of those neurons expressed a protein marker called AHD2, a marker typical of the specific kinds of neurons lost in Parkinson's disease.

"What this work shows is that you can easily get dopamine-producing neurons in the brain," even from undifferentiated ES cells, says developmental neurobiologist Ron McKay of the National Institute of Neurological Disorders and Stroke in Bethesda, Maryland.

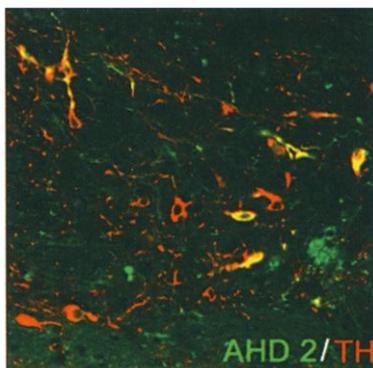
And the new neurons seem to have reduced Parkinson-like symptoms in the animals. The scientists observed a gradual decrease in the abnormal rotations; by 9 weeks following the surgery, the 14 rats with surviving ES cells had improved by an average of 40% over their pretransplant state. Rats that

received sham surgeries showed no improvements. But Anders Björklund of the University of Lund in Sweden cautions that the functional effect is very small. He notes that other transplantation experiments using dopamine-producing neurons from fetal brain tissue (a technique now being tested in humans) regularly produce much more dramatic results.

To Lorenz Studer of the Memorial Sloan-Kettering Cancer Center in New York City, "the results suggest that the fewer cells you put in, the bigger the influence of the environment becomes. If you dilute them sufficiently, you can get them to disregard this tendency to cause tumors."

Studer adds that the relative ease with which dopamine-producing cells developed from the injected ES cells suggests that there might be a way to prompt rare stem cells already in the brain to become dopamine-producing neurons, allowing doctors to avoid the issue of transplanting cells altogether.

—GRETCHEN VOGEL



Pot of gold? Mouse embryonic stem cells injected into rat brains express the AHD2 protein marker (yellow) characteristic of cells lost in Parkinson's disease.

OPTICS

Crystal Stops Light in Its Tracks

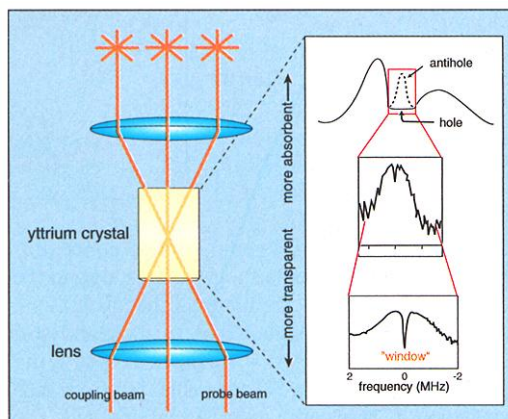
Baby-boomer superheroes, take heart: Despite your aging knees, you can still run faster than light. For years, physicists have been slowing light down to a crawl, and even stopping it in its tracks, by shooting laser beams into cold gases known as Bose-Einstein condensates (BECs). Now, researchers have done the same thing in a much less exotic solid. The advance may

one day lead to memory devices for computers that store information on beams of light.

The work builds on experiments Lene Hau of Harvard University performed in the late 1990s. Hau and colleagues managed to slow light down to a poky 17 meters a second—below the top speed of a bicycle. The group used lasers to poke a spectral "hole" in a BEC of sodium atoms, making it impossible for the condensate's electrons to absorb light of a certain color. Two lasers, known as the probe beam and the coupling beam, zapped the atoms in the condensate, causing their electrons to interfere with each other in ways that made it impossible for them to absorb photons of a certain frequency. As a result, the BEC became transparent to light within a narrow range of frequencies of yellow-orange light. The speed of light in a medium is related to how readily the medium absorbs light of different frequencies; sharp variations in absorption across a narrow range of frequencies dramatically slow a pulse of light. Thus, the tiny spectral hole caused an unprecedented slowing.

Unfortunately, BECs exist only within a few hundred nanokelvin of absolute zero. Solids can exist at warmer temperatures. But in solids, unlike BECs, the laser-induced transparency trick makes too wide a spectral hole to slow light appreciably, says physicist Phil Hemmer, now at Texas A&M University. To narrow the hole, Hemmer and colleagues used two beams from a dye laser to create a large spectral hole in an yttrium crystal. A third beam increased the absorption inside the hole to create a smaller "anti-hole." Happily, the coupling and probe lasers also bleach a narrow anti-antihole of transparency inside the antihole, yielding a range of transparency narrow enough to slow down light to 45 meters per second.

Furthermore, when they shut down the coupling field, the crystal brought the beam to a halt by absorbing and storing the light, and eventually released it when the coupling laser was turned back on—a trick Hau also had performed earlier with BECs (*Science*, 26 January 2001, p. 566). "You preserve phase and amplitude," says team member Alexey Turukhin, a physicist at the Eatontown, New Jersey, branch of laser company JDS Uniphase. As a result, Turukhin says, the light can store information in ways that make it suitable for quantum computing. And although the yttrium crystal must be kept at a chilly 5 kelvin, it is still much easier to handle than a BEC, an important consideration for commercial devices that store light pulses. What's more, Hau notes, light pulses shrink as they slow down, a property that might give scientists an efficient means of compressing information stored on light pulses. —CHARLES SEIFE



Squeeze play. Shrinking the range of frequencies a crystal can transmit slows light to a crawl.