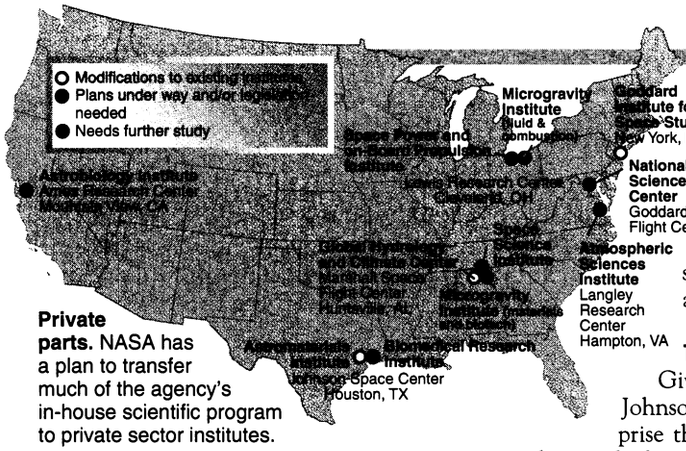

Private parts. NASA has a plan to transfer much of the agency's in-house scientific program to private sector institutes.

SOURCE: NASA

chances when a test subject on Earth fainted during a bungled injection of a drug that accompanied its use. Yet the project was never seriously threatened until NASA's new management ordered the outside review that sparked the protracted struggle between headquarters and the center.

Scientists say that the Houston center's intransigence in removing the experiment from Columbia's manifest even after it was panned by outside reviewers is revealing. "Johnson [Space Center] has a history of independence of operation," says Osborn. "They are not used to having their research reviewed, much less previewed." Other NASA and academic sources, who declined to be identified, agree that the center's attitude has damaged NASA's reputation. "Life scientists at Johnson have been totally isolated and arrogant," says one.

Frank Sulzman, NASA deputy director of life sciences, says removing the experiment from the shuttle "shows our commitment to a fully peer-reviewed program." Although Johnson's Sawin says the delay was the result of a feud between two offices at headquarters, not a refusal to accept outside review, he adds that much of the center's research should not be subject to the normal standards of academic science because its goal is operational, not scientific. "Our internal program has really been set back by this," he says about the push for outside review, adding that it has delayed the launch of some experiments.

NASA's other major center that conducts life sciences research is Ames Research Center in Mountain View, California. Although outside review is common—"Everything we do has been 100% peer reviewed since before 1981," says Ken Souza, Ames associate director for life sciences—some of its projects have come under fire from former employees and outside academics, and an internal panel earlier this year suggested eliminating the science done at the center. Ames is also under attack from the People for the Ethical Treatment of Animals, which charges the center with widespread misconduct in the care of laboratory animals and the use of monkeys in space. The lab's veterinarian resigned earlier this year in protest; an independent panel verified some of the veterinarian's concerns (*Science*, 23 June, p. 1692). The controversy puts an unwelcome spotlight on Ames researchers already nervous about their fate.

Tighter reins

Given the problems at Johnson and Ames, it is no surprise that both are listed in the early round of centers to be converted into science institutes. NASA officials say they are eager to change the perception among some in the academic community that science at its centers is merely an adjunct to multibillion-dollar engineering projects. The new institutes, they argue, would link NASA research more firmly to universities, shrink federal payrolls, and preserve funds for science. The institutes would also reduce the power of the centers, which agency sources say is one of Goldin's overall goals.

In the meantime, however, NASA is reducing the size of headquarters. The NRC panel, which completed most of its work before the institute concept was ripe, warned that this could backfire by passing power back to the centers. That shift, in turn, could weaken NASA's ability to perform quality science. Peer review "has been so strongly centralized because of the suspicions of the community that if the centers do it, they will take advantage of it to capture the research money," says John McElroy, a former senior NASA manager now at the University of Texas, Arlington.

Cordova insists that NASA headquarters will keep a tight rein on peer review until the institutes are in place. She also says she is confident that the institutes and other reforms under way will improve science at an agency with a reputation for scientific isolation and arrogance. Her challenge is to demonstrate to a skeptical research community and a tight-fisted Congress that NASA can deliver on that promise.

–Andrew Lawler

NEUROBIOLOGY

New Clues Found to Huntington's

SAN DIEGO—When researchers cloned the gene that causes Huntington's disease in 1993, its sequence yielded few insights into how the gene's protein product—called huntingtin—may cause this debilitating neurodegenerative disease. But earlier this week at the annual meeting of the Society for Neuroscience, two teams reported results that may help solve this mystery—and perhaps also lead to a better understanding of related diseases, known as the spinal and cerebellar ataxias, that are caused by similar mutations. The researchers have found a protein partner for huntingtin, together with indications that the disease-causing mutations alter the interaction between the two proteins.

It's still unclear how this might cause the neuronal degeneration of Huntington's, but researchers are nevertheless encouraged by the findings. They are "enormously exciting," says Huntington's researcher Nancy Wexler, of Columbia University. "We have two proteins to work with now." *Nature*, which is publishing the work in its 23 November issue, took the unusual step of lifting its embargo 10 days early and published a News and Views piece previewing the work on 9 November.

The proteins involved in Huntington's and the other conditions have a common feature: a stretch of repeated copies of the amino acid glutamine. In the disease-causing mutants these expand in number from less than 35 glutamines in a row to 38 or more. One of the many questions that have puzzled researchers is why huntingtin and the other proteins abruptly begin causing disease when they accumulate 38 to 40 glutamines. That's where the new work comes in.

Frederic Saudou and his colleagues Yvon Trottier and Jean-Louis Mandel, of the University of Strasbourg, France, found a monoclonal antibody that binds to polyglutamine in the disease-causing forms of huntingtin and four of the other proteins. It doesn't bind to versions of the proteins with less than 35 glutamines, suggesting their shape doesn't fit the antibody molecule.

The shape change associated with the mutation may alter the protein's binding to its new partner, a protein called HAP-1 (for huntingtin-associated protein), discovered by Christopher Ross and his colleagues Xiao-Jiang Li and Shi-Hua Li at the Johns Hopkins University School of Medicine. They found that HAP-1 binds to normal huntingtin, but binds even more tightly to the mutant version. That tighter binding may somehow change the way huntingtin and HAP-1 function, causing neuron death, says Ross.

What's more, similar proteins may be at work in the spinal and cerebellar ataxias. Ross's group has found a HAP-1–related protein that doesn't bind huntingtin and is testing to see whether it binds to one of the glutamine-rich proteins that causes the other diseases. If so, they may have found the key players not only in Huntington's, but in related diseases as well.

–Marcia Barinaga