

Budget tips from science adviser's lips

With President Clinton preparing the public for the prospect of new taxes and a streamlining of both government and its spending, researchers have become understandably nervous. Will the new administration favor applied research over basic? Will such big efforts as the human genome project or the Superconducting Super Collider (SSC) proceed at the expense of smaller programs? Where will any "peace dividend" go?

Presidential science adviser John H. Gibbons tackled such questions at a press briefing last week. And the bottom line, he warned, is that "everyone's going to have to take a hit."

That includes Space Station Freedom, a perhaps \$30 billion project the space agency envisioned paying \$16.9 billion toward. Uncle Sam so far has spent \$8 billion — \$2.1 billion this fiscal year alone. Completing the program as originally planned would require federal spending "that simply is not realistic" in light of growing budget deficits, Gibbons said. So the space community is being asked to design a less costly mission that still achieves most of the old research objectives. A solution "hasn't been written yet," Gibbons said Feb. 15. Three days later, however, NASA acknowledged it would target the space station's \$2.3 billion budget for next year on a new, more "streamlined, cost-effective design."

The SSC can't be scaled back and still perform as expected. So here, Gibbons said, the administration is asking "how urgent is it to proceed at the highest speed?" To complete the massive accelerator by 1999 — and hopefully hold to its projected \$8.2 billion price tag — Congress would have to allocate \$710 million for the project next year. Instead, the White House will ask only \$640 million, Department of Energy (DOE) officials said last week. The President also will look to "further internationalize this effort" and its financing, Gibbons said — perhaps by drawing in the Japanese "a little more closely."

In the Cold War's warming, Clinton will also re-tailor military objectives. For instance, "it would be absolutely silly to continue to produce nuclear-attack submarines and space-based intercepts for multiple warheads," Gibbons said, since the threat they were to counter has all but disappeared. But as the Defense Department redefines research needs, he said, "I would fight" shifting money from research into procurement programs.

Overall, Gibbons said, the President will look to shift back to a historic balance in federal research and development (R&D) spending of roughly 50 percent for defense programs, 50 percent for civilian. Currently, that ratio is closer to 60:40.

The President will also recommend some investments, Gibbons said. High on the list will be initiatives to spur "green" (pollution-preventing) technologies, such as those that conserve energy or other resources. The science adviser pointed to the National Institute of Standards and Technology's advanced technology program (ATP) as another investment target. And the Commerce Department confirmed ATP's favored status in a briefing prior to the President's Feb. 17 speech to a joint session of Congress. Clinton will seek to more than double ATP's budget in the current fiscal year. If Congress agrees, that \$103 million increase — to \$171 million — would hike ATP to 34 percent of NIST's entire federal appropriation.

Though Gibbons conceded there would be a strong push for research that supports improving U.S. industrial productivity and job formation, he added that redirecting basic research to this end "may be a misplaced focus." Recent productivity losses appear to stem less from problems in basic research, he said, than to a "disconnect" between it and those who translate research findings into economic activity. He indicated the new administration would seek to establish a "reconnect," not only by making federally funded information more readily available to entrepreneurs, but perhaps also by translating and disseminating important research from foreign-language journals.

A genetic basis for adult leukemia

Two reports now add weight to the suggestion that mutations affecting a protein that regulates the production of interferon, an immune-system messenger that also affects cell growth, may lead to leukemia in adults.

That protein, one of two interferon regulatory factors, keeps cell division in check, report Hisashi Harada, Motoo Kitagawa, and their colleagues at Osaka University in Japan. They monitored the amount of each factor produced by normal mouse cells during growth and division. The level of the second factor remained fairly constant. But the amount of the first factor — which started out lower than the second factor — increased, peaking in cells that stopped dividing, say the scientists.

They also discovered that mutant mouse cells that produce way too much of the second factor multiply much like malignant tumor cells. By adding the gene for the first factor to these mutants, however, the scientists were able to restore normal growth. Thus it appears that the two factors work to balance each other and that a deficit in the first factor could lead to a tumor, the researchers conclude in the Feb. 12 *SCIENCE*.

Even if just one of the two genes coding for that first factor is missing or mutated, cells fail to produce the protein in the right amounts, adds Cheryl L. Willman, a cell biologist at the University of New Mexico School of Medicine in Albuquerque. After conducting genetic analyses of 13 patients with leukemia or preleukemia syndromes, she and her colleagues concluded that the gene for this regulatory protein lies on the stretch of chromosome 5 found faulty in many people with leukemia. They also describe their results in the Feb. 12 *SCIENCE*.

Shark gut goop no snake oil

Scientists have discovered a new class of antibiotic — a type of steroid — in the stomach of the dogfish shark. Called squalamine, this steroid resembles plant compounds now used to kill intestinal parasites. In laboratory tests, it works against fungi, protozoa, and both gram-negative and gram-positive bacteria, says Karen S. Moore of Children's Hospital of Philadelphia.

This antibiotic destroys bacteria as effectively as ampicillin, a broad-spectrum antibiotic, Moore and her colleagues report in the Feb. 15 *PROCEEDINGS OF THE NATIONAL ACADEMY OF SCIENCES*. In squalamine, a bile salt links with a steroid to work better than either of those compounds alone, say the researchers, who have now made the chemical in the lab.

Cancer-cell vaccine halts tumor spread

Scientists have shown in mice that genetically altered tumor cells can stimulate the immune system to halt the spread of an otherwise lethal cancer, says cell biologist Michael Feldman of the Weizmann Institute of Science in Rehovot, Israel.

Working with U.S. and Japanese researchers, Feldman and his colleagues treated mice with rapidly spreading tumors on their feet. After removing the tumors, the scientists inserted the gene coding for a type of interferon into the tumor cells. Interferon revs up white-blood-cell activity and causes the tumor cell to express proteins on its surface that make it a more visible target for the white blood cells. The scientists then injected the genetically altered tumor cells into half the mice weekly for five weeks. New tumors developed in untreated mice, but the vaccinated mice remained healthy, the researchers report in the Feb. 15 *JOURNAL OF IMMUNOLOGY*.

"The almost complete prevention of metastasis under conditions where the animal would almost certainly die makes it very attractive," says Feldman. "This is a model that people would like to apply to humans."

Researchers have already used genetically altered cancer cells to bolster the immune system's fight against advanced melanoma, the most deadly skin cancer (SN: 10/19/91, p.253).