

HYDROLOGY

USGS Braces for Severe Budget Cuts

When the Department of the Interior (DOI) unveiled its budget request last month, the news was gloomy for the U.S. Geological Survey (USGS): The low-profile agency is slated for a 7.9% cut in fiscal year 2002, which starts on 1 October. It's a victim of DOI's scramble to reduce its overall budget by 3.7% while boosting big-ticket items such as repairs to Bureau of Indian Affairs schools promised during the election campaign. Hardest hit within the USGS is the water resources division, which provides a wealth of

gy Program. All its research would be shut down. Since 1982, this program has studied how contaminants move and break down over years in heavily instrumented aquifers and throughout watersheds. "This is expensive [research], and it requires a lot of high-level scientific expertise," says Bruce Rittman, a civil engineer at Northwestern University in Evanston, Illinois, who chaired a National Research Council review on in situ bioremediation. "The USGS really has been the most thorough and successful at putting these programs together." Rittman adds that the USGS has examined only a fraction of contamination settings so far.

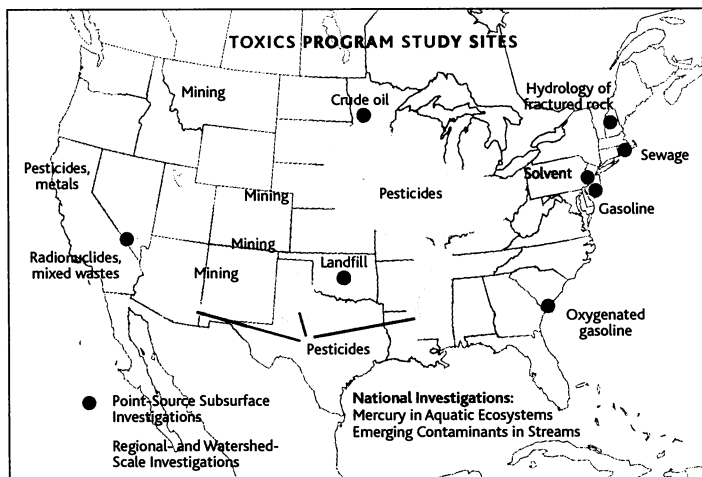
Another hard-hit program is the National Water Quality Assessment (NAWQA). Its

funding would drop by 30% to \$45 million. The program has been running for 10 years as a long-term evaluation of different kinds of watersheds and aquifers. "It's the only program we have that really begins to assess the status and look at trends in the nation's water quality," says George Hallberg, a hydrogeologist with The Cadmus Group in Waltham, Massachusetts, who chairs a National Academy of Sciences review com-

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—ERIK STOKSTAD



High and dry? All of the USGS's toxic-hydrology research would be shut down under the proposed budget, unless co-funders can be found.

data that underlie research and regulation.

The proposed cuts have shocked state water-quality agencies, civil engineers, and other groups that use USGS research. "We're extremely concerned about this," says Erik Olson of the Natural Resources Defense Council, an environmental organization based in New York City. "It would make it almost impossible for the federal government to have a meaningful understanding of water quality in the United States."

DOI says it doesn't intend to eliminate or scale back these programs; it just wants users to help pay for the data. The trouble is that cost-sharing arrangements haven't been set up yet, and USGS officials warn that cutting the budget in the meantime will shut down research, eliminate at least 506 jobs, and create logistical problems that will ultimately raise costs. Congress began picking over the budget request in detail this week; USGS scientists are hoping that several well-placed supporters in the appropriation subcommittees will come to the rescue, but the outlook is uncertain.

Facing the biggest cut—a 71% drop to \$4 million—is the Toxic Substances Hydrolo-

gical Program.

The cuts could force the USGS to prune NAWQA from 42 sites to 24, which would mean that some environmental settings would not be studied at all. "You reach a point of diminishing returns," Hallberg says. "You just can't keep reducing the size and call it a national program." The program had originally intended to include 60 sites, but those plans were scaled back in previous budgets.

DOI hopes to soften the cuts by having USGS share costs with users of its data. Observers point to several potential problems with that plan. One is that states and regulatory agencies would bring their own research agendas to the table. USGS scientists fear that might balkanize what is supposed to be a program with national standards. Moreover, cost-sharing projects often last only a few years—not long enough to spot trends in water quality.

Some observers also worry that Environmental Protection Agency (EPA) funding of USGS research might taint the results. "The risk is that users may view the information as less credible because it comes from an agency that has a political rather than a sci-

UNIVERSITIES

Princeton Picks Biologist Tilghman as President

Princeton University named Shirley Tilghman its president on 5 May, making her the first woman to hold that post and the first prominent genome leader to head a major university. Tilghman will take the helm in June, succeeding Harold Shapiro, who announced last fall that he was ready to step down after 13 years.

Tilghman, 54, is known for her research on "imprinting"—the subtle chemistry by which mammalian cells suppress genes from one parent while allowing other genes to be expressed. But she's also valued as a clear-headed policy adviser and teacher. "Shirley is capable of sorting through complex issues and coming up with the ideal solution—just what you want in a university president," says Francis Collins, director of the U.S. National Human Genome Research



Breeze in the ivy. Biologist Shirley Tilghman will be Princeton's first woman president.

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Institute, who has asked her to serve on many panels. Collins adds: "She will be a great champion for science and for women in science." Princeton graduate and genome researcher Eric Lander of the Massachusetts Institute of Technology in Cambridge describes Tilghman as a "great scientist, a true humanist, and a wonderful person," as well as "a spectacular choice for Princeton."

Tilghman was on the search committee until 6 weeks ago, according to the chair of the trustees' executive committee and head of the search committee, Robert H. Rawson Jr. She left a meeting early to teach, and in her absence, the other members decided they wanted her to become a candidate. She agreed and withdrew from the search committee. The remaining members chose her unanimously.

Tilghman—like Shapiro—was born in Canada. She joined Princeton's faculty in 1986, became a Howard Hughes Medical Institute investigator in 1988, and was elected to the National Academy of Sciences and the Institute of Medicine less than a decade later. She received Princeton's top teaching award in 1996. Since its founding in 1998, she has run Princeton's Lewis-Sigler Institute for Integrative Genomics; no successor has yet been named.

—ELIOT MARSHALL

THE 1918 PANDEMIC

Killer Flu With a Human-Pig Pedigree?

LONDON—Scientists have come up with a new explanation for what made the Spanish flu the biggest killer of the 20th century. The deadly influenza did not jump from birds into humans, they argue, but rather was the unfortunate result of an unprecedented recombination of pig and human flu genes. "This is a great step away from the existing theories of the origin of 1918 flu," says virologist Mark Gibbs of the Australian National University in Canberra, whose team presented its findings at a symposium here on 25 April at the Royal Society.

Reassuringly, such genetic recombination appears to be an exceedingly rare event, suggesting that the odds of a reprise of the Spanish flu pandemic—which killed 40 million people around the world in 1918 and 1919—are vanishingly low. Some experts, however, argue that this proposed explanation for the Spanish flu's virulence is flawed: Recombination among flu strains, they assert, does not happen at all.

Four years ago in *Science*, a team led by virologist Jeffery Taubenberger of the Armed Forces Institute of Pathology in Washington, D.C., published the initial RNA sequences of the 1918 flu strain after isolating it from the preserved tissue of victims (21 March 1997, p. 1793). Poring over these

data, Gibbs's team homed in on the gene for hemagglutinin (HA), a viral protein used to gain entry into target cells. Novel HA configurations are harder for the immune system to recognize, making the protein a key determinant of a strain's virulence.

The researchers used standard software to compare the sequence of the 1918 HA



Spanish flu victims. New work suggests that the 1918 strain derived its lethal powers from an unprecedented genetic recombination.

gene to those from 30 closely related influenza strains from birds, pigs, and humans. "Within a few hours we had a preliminary signal," says Gibbs. Pursuing this further, they discovered that the 1918 version appeared to be a chimera of sorts: One end bore a marked resemblance to human flu sequences, the middle was strikingly similar to pig, while the other end again was human. The "simplest scenario," says Gibbs, is that the HA gene (which, researchers concur, originated in bird flu strains) slipped into mammals sometime before 1918 and diverged into two lineages, human and pig.

Then in a bad twist of fate, these HA genes recombined to form the version that made the Spanish flu so much harder for the immune system to recognize—and therefore more virulent. "It looks for all the world like the signature of recombination, and I can't see how to explain it otherwise," says Eddie Holmes, who studies viral evolution at the University of Oxford. "We are talking about a rare event, but evolution is all about rare events."

The idea has met with skepticism from some top flu researchers, however. Taubenberger, for one, argues that the Gibbs group has misinterpreted the 1918 sequence. He believes that an influenza virus with an avian HA gene had started circulating in humans just before the start of the pandemic. In the fall of 1918, this virus infected humans and swine simultaneously and split into two lineages, and disparity in the rate of evolution between the two strains since then has con-

fused the picture, says Taubenberger. "Human flu viruses are subject to huge immunological pressure and are forced to mutate rapidly," as humans are so long-lived and develop immunity to many strains, he says, while pig strains mutate slowly because their hosts don't live long enough to develop such broad immunity. Thus, Taubenberger argues, the HA gene in the 1918 human samples resembled that of the avian ancestor of both pig and human strains. But by the 1930s—the date of the earliest strains that can be directly compared—the human gene had changed dramatically, while the pig gene had changed little. That, he says, would explain why sections of the 1918 human virus look similar to those of later pig viruses.

Taubenberger does not believe that flu strains—like other negative strand RNA viruses—are capable of recombination, in which new genes are patched together from sections of other genes. Not that flu strains are limited to evolution through mutation: A kind of whole-gene

swapping between strains, called reassortment, is thought to have spawned the virulent avian flu strains that jumped into mammals in 1957 and 1968.

Gibbs acknowledges that it will be an uphill battle to convince some of his colleagues that influenza strains are capable of recombining. "One of the paradigms," he says, "is that flu goes in for reassortment but not for recombination." But, he argues, recombination provides the best explanation for the genetic data.

—JOHN PICKRELL

U.S. SCIENCE POLICY

Former Advisers Fret Over OSTP Vacancy

CAMBRIDGE, MASSACHUSETTS—Although billed as a celebration, the largest gathering of U.S. presidential science advisers had more the air of a wake. Meeting here last week, the former government officials were all too aware that a new Administration is busy making critical decisions without benefit of the kind of scientific advice that has guided most presidents in the past half-century. "Nobody is celebrating the future here," says Neal Lane, former science adviser to President Bill Clinton and now a professor at Houston's Rice University.

Lane was one of eight former science advisers who gathered on 1 May at the Massachusetts Institute of Technology (MIT) to celebrate the 25th anniversary of the Office of Science and Technology Policy (OSTP).