

FRANCE

Socialists Reverse Budget Decline

PARIS—When France's new Socialist government unveiled its proposed 1998 budget last week, French scientists were eager to see whether education and research minister Claude Allègre had kept his pledge to give "new hope" to the nation's ailing research effort (*Science*, 13 June, p. 1638). The verdict seems to be a guarded "yes." Although overall the civilian research and development budget, at \$8.8 billion, will remain flat when inflation is taken into account, a reorganization of priorities will give a small but welcome boost to basic research. And young scientists should benefit from a major new recruitment program for universities and public research agencies.

"This is a better budget than we've had in the last few years," says molecular biologist Edward Brody, director of the Center for Molecular Genetics in Gif-sur-Yvette. "It's going to give us a big psychological boost." And developmental biologist Anne-Marie Duprat of Paul Sabatier University in Toulouse calls the budget a "beginning" that "will give some fresh air" to French science.

Researchers have especially welcomed the creation of new research jobs. The budget would add 400 posts to public agencies such as

the basic research organization CNRS and INSERM, its biomedical counterpart, as well as hundreds of positions in the universities. But some scientists expressed disappointment that support for basic lab costs in the public agencies will increase by only 2.5%, not counting a projected inflation rate of 1.4%. "It's good news for creating new positions, but not good news for the lab budgets," says Pierre Chambon, director of the Institute of Genetics and Molecular and Cellular Biology near Strasbourg. University labs, on the other hand, will do significantly better, with a 5.4% increase.

Geophysicist Vincent Courtillot, Allègre's chief adviser, insists that the new budget should not be judged purely in financial terms. "Allègre's view is that money is not the main thing," Courtillot told *Science*. "In both the education and research budgets, the number one priority is creating new jobs."



Kept promise. Science minister Claude Allègre.

Indeed, given France's sluggish economy and the budget slashing required by the European Union for entry into a single European currency, few researchers expected that Allègre would be able to greatly increase the overall science budget. "I think he's doing as well as he can," says Chambon.

Chambon and others argue, however, that if the research budget cannot be expanded further, French science must ultimately undergo dramatic reforms. "If you hire young people but don't increase the lab budget, they can't do research," Chambon says. One solution, he suggests, would be to turn organizations like the CNRS and INSERM into U.S.-style granting agencies and have researchers compete with each other for funds. That would mean a sharp break with France's researcher-for-life tradition. "We need a reform that adjusts the number of active scientists to the realities of the budget," Chambon concludes.

But while such sweeping reforms do not appear to be on Allègre's immediate agenda, most French researchers are taking heart at the modest gains they have made in the new budget. "It's not a major transfusion," says Brody. "But at least it will stop the hemorrhaging."

—Michael Balter

MAD COW DISEASE

New Studies Affirm BSE-Human Link

One of the most worrying consequences of Britain's outbreak of so-called mad cow disease—that humans might become infected by consuming contaminated beef—appears to be confirmed in research published this week in *Nature*.

More than 20 Britons have died over recent months of a variant of Creutzfeldt-Jakob disease (vCJD). The symptoms are similar to those of classic CJD, a fatal brain disease that progresses slowly. But vCJD tends to strike younger people, and it develops much more quickly. A link to mad cow disease—bovine spongiform encephalopathy (BSE)—has been suspected, because both involve dementia, tremors, and what may be an infectious protein called a prion. But the evidence (*Science*, 1 November 1996, p. 721) that eating BSE-contaminated beef might cause vCJD has been inconclusive.

In the new work, Moira Bruce of the Institute of Animal Health in Edinburgh, Scotland, and colleagues injected mice with infectious brain samples from cows with BSE, patients who had vCJD, and patients who had the classic form of CJD. The Edinburgh group had previously shown that different strains of the transmissible encephalopathies have reproducible incubation times and pathology when in-

jected into certain inbred strains of mice. After examining how and where the mouse brains were damaged, along with the symptoms and course of the disease, the researchers concluded that vCJD and BSE in mice are the same, and both are distinct from CJD.

"Epidemiological surveillance continues to indicate that vCJD is a new condition occurring almost exclusively in the U.K.," says Bruce. The new studies "provide compelling evidence of a link between BSE and vCJD." The main distinguishing features of the pathology of vCJD and BSE are the large clumps of so-called prion protein deposited in the brain and the prominent involvement of the cerebellum, she says.

A team from Imperial College School of Medicine at St. Mary's in London, led by John Collinge, adds another piece of evidence to the puzzle in a separate *Nature* paper. Collinge and his colleagues have been looking at the pattern of sugar molecules on prion proteins and the fragment sizes that are produced when prion proteins are digested with a protease enzyme. Previous work had shown that the resulting "fingerprint" of fragments was consistent for different strains of prions when they are transferred to different species. Using both ordinary mice and transgenic mice carrying the human

form of normal prion protein, Collinge's group has produced further evidence that the prions involved in BSE and vCJD appear to be the same, and quite distinct from the sporadic form of CJD. The "inescapable conclusion," says Collinge, is that new vCJD is the human equivalent of BSE. BSE itself is believed to have spread to cattle through feed contaminated with offal from infected animals before the use of offal was banned in 1989.

At least one worrisome possibility has, however, been dispelled. Two farmers who had had BSE in their flocks recently died of CJD, raising concerns that environmental exposure to the disease agent might also be a source of risk. But research on brain material from these patients suggests that they died from classic CJD rather than the variant form linked to BSE.

A key question now is how many people may have been infected by the variant form, but researchers still have no answers. "It may take several years before we can be confident that this is not a period of comparative calm before the storm," says Jeffrey Almond at the University of Reading and John Pattison at University College, London in a commentary in *Nature* accompanying the reports. "Much depends on the average incubation time of vCJD, and at present we cannot calculate it."

—Nigel Williams