Feuding Over Funds for Cleaner Coal

The bid to establish a $400 million clean-coal technology program was a long, hard-fought political struggle marked by threats of a presidential veto. In the end, an unlikely coalition of utilities, coal companies and environmental groups persuaded Congress that the money would be well spent, and legislation establishing the program was passed last December. Now these groups are complaining that the Department of Energy (DOE), which last week issued its call for proposals to be funded under the new program, has failed to live up to its obligations.

Coal fuels the generation of more than 55 percent of the electricity produced in the United States. Under strong pressure from environmentalists and legislators concerned about acid rain, the coal industry and utilities are stepping up their efforts to burn coal more cleanly. The clean-coal program's success depends on how many research proposals DOE gets from industry. In each case, industry has to supply at least half the funds for a given project.

Getting this program through Congress during a time of tight budgets, and despite the Reagan administration's opposition, was a great achievement, says Sen. Robert C. Byrd (D-W.Va.). "Today, we are at a point which many skeptics thought we would never reach," he said last week in Washington, D.C., at a conference examining the clean-coal program.

The administration now says it strongly supports the program. This shift may have been prompted by a recent report (SN: 1/18/86, p. 37) from special envoys Drew L. Lewis and William G. Davis. That report recommends that the U.S. government spend $2.5 billion over a five-year period to develop new coal-cleaning techniques to reduce sulfur dioxide emissions.

"There is no chance that the administration will propose a $2.5 billion program," says Randal H. Ihara, a Senate staff member. "The clean-coal technology program currently represents the only program to which the administration can point as a basis for further discussions of the acid rain issue with Canada." This subject will probably top the agenda when President Reagan and Canadian Prime Minister Brian Mulroney meet later this month.

However, program supporters are concerned about how DOE has decided to implement the program. Controversy already surrounds at least one clause in DOE's request for proposals. This clause requires industry to pay back the government's share in any project.

"This program was conceived as a partnership between the private sector and the federal government," says Byrd. Turning the government's share into a repayable loan "is not in agreement with the intent of Congress," he says.

"The bureaucrats are trying to steal clean coal," says Carl E. Bagge of the National Coal Association, based in Washington, D.C. "Clean-coal money must be kept separate, clearly visible and free of restrictions that kill incentive."

Environmentalists are also worried about DOE's actions. If DOE selects large, expensive projects, then the program's value in providing cheap and effective air pollution controls will be limited, says John L. McCormick of the Environmental Policy Institute in Washington, D.C.

"The solution to the acid deposition problem must represent a 'win-win' situation for both the Northeast and the Midwest," says McCormick. "That is why we worked so hard to win congressional approval for the clean-coal technology reserve. And that is why the program's ultimate success is so important."

— I. Peterson

Brain pigment and Parkinson's disease

Scientists recently observed that a chemical compound found in synthetic heroin and other illegally manufactured opiates causes brain damage and clinical symptoms that closely match Parkinson's disease (SN: 10/5/85, p. 212). Some researchers believe Parkinson's may be caused by a combination of exposure to the same chemical, known as MPTP, or related compounds in food and other environmental sources and normal brain-cell loss due to aging. But the reason why MPTP destroys only a small, crucial brain area has remained unclear.

In the Feb. 28 SCIENCE, investigators at Johns Hopkins University in Baltimore offer a possible explanation for MPTP's selectivity. After MPTP is converted to the highly toxic substance MPP^+ in the brain, it is pumped into cells that produce the neurotransmitter dopamine and sticks to those that contain the natural pigment neuromelanin, say Robert J. D'Amato, Zoe P. Lipman and Solomon H. Snyder. Most brain areas with the pigment have nerve terminals that channel MPP^+ out of dopamine cells, they explain, but the substantia nigra — the portion of the brain implicated in Parkinson's disease — contains plenty of neuromelanin and few of the protective nerve terminals.

D'Amato and his colleagues came up with this model after first observing that MPP^+ binds to melanin synthesized from dopamine at much higher concentrations than it binds to melanin synthesized from another neurotransmitter, norepinephrine. They then observed that MPP^+ strongly binds to dopamine-rich neuromelanin isolated from a monkey's substantia nigra.

Low doses of MPTP cause extensive cell loss in the substantia nigra of both humans and monkeys, note the researchers. In mice, however, there is no marked loss of neurons, even at higher MPTP doses. There is little or no neuromelanin in the substantia nigra of rodents, they point out, while humans and monkeys have large amounts of the pigment in the substantia nigra.

Although the pigmented locus ceruleus region of the brain also contains neuromelanin, it is largely immune to the cell-destroying effects of low doses of MPTP. The investigators propose that once MPTP is converted to MPP^+ and enters the locus ceruleus, it is accumulated by a dense network of catecholamine (dopamine, norepinephrine and epinephrine) nerve terminals that prevent the substance from entering cell bodies.

"In theory, the key event is that MPP^+ is pumped into dopamine [neurons] by the dopamine uptake system," says Snyder. "But in general, this is not enough. It looks like neuromelanin greatly assists in the killing of brain cells."

The proposed neuromelanin connection, however, has not been endorsed by all MPTP researchers. "The observation [of the Hopkins scientists] is fascinating, but I'm slightly skeptical," says J. William Langston of Stanford University. "I'd label this under the category of an interesting hypothesis that hasn't been thoroughly tested."

Langston and his colleagues recently observed that MPTP does cause cell destruction in the substantia nigra of older mice, a finding that is consistent with the age-related progression of Parkinson's disease. As a result, he says, "I don't believe neuromelanin is part of the key [to MPTP's effects]." Snyder doubts that older mice are as sensitive to MPTP as humans and monkeys are, but at this point, holds Langston, a satisfactory theory for the findings from various laboratories is not possible.

— B. Bower