SCIENCE NEWS of the week

New Process KOs All NO_x

Pollution control engineers have had a difficult time and only limited success in battling nitrogen oxides, or NO_x. Produced by combustion, these gases play a major role in the formation of both smog ozone and acid rain. But a scientist has invented a chemical process that is "capable of completely removing NO_x from the products of combustion," according to a newly published paper by researchers at Sandia National Laboratories in Livermore, Calif.

The anti-NO_x discovery, by Sandia's Robert A. Perry, is an offshoot of his research into the fundamental chemistry of hydrocarbon combustion. The new finding involves mixing combustion gases with isocyanic acid (HNCO), a gas formed when the nontoxic cyanuric acid, or $(HNCO)_3$, is heated. In a series of rapid chemical reactions that have not yet been fully characterized, the HNCO will mix with nitric oxide (NO), for example, forming elemental nitrogen gas, carbon monoxide, carbon dioxide and water vapor. In a laboratory test with the exhaust from a small diesel engine, the HNCO treatment was capable of removing more than 99 percent of the nitrogen oxides present in the untreated exhaust, according to a report by Perry and Dennis L. Siebers in the Dec. 18 NATURE.

This process "will work, as far as we know, for any process that uses hydrocarbons for combustion," Perry says. And unlike the limited (and far less efficient) anti-NO_x systems now available, he says, this one can work in the presence or absence of oxygen. It also does not appear to be affected by the presence of sulfur contaminants in the fuel that is burned, whereas anti-NO_x systems employing catalysts frequently are.

Still, there are several important engineering questions to be answered, Perry says, such as whether the process will operate fast enough to fully eliminate the NO_x produced in a large exhaust stream, whether devices that employ it can be made small enough to fit on a car and whether systems incorporating these devices can be produced inexpensively enough to encourage widespread use.

Perry expects to resolve many of these questions within a year. He says a commercial prototype device — probably initially designed for diesel engines — might be available within five years.

Ordinarily, the Department of Energy (DOE) would own patent rights to any technology invented at one of its labs. But hoping to speed the commercial development of this process, DOE officials have formally announced an unusual decision in which Perry will be allowed to keep the rights to his invention. As soon as he can obtain the money to start his own firm, Perry says, he will leave Sandia to develop devices that employ this process, which he calls RAPRENO$_x$, for Rapid Reduction of NO_x.

Harry H. Hovey Jr., director of New York State's division of air resources, characterizes the process as "exciting." "If such a device is workable and economically feasible — two big ifs — it would help in the acid deposition problem in the East," he says, "because NO_x is inherent to that process." Probably just as important, he says, is NO_x's role in smog formation and the ozone problem plaguing all major U.S. metropolitan areas (SN: 6/28/86, p.405). "Without the NO_x," Hovey told SCIENCE NEWS, "there's a very good likelihood we could meet the ozone standard."

Linas P. Gobis, an environmental engineer with the Motor Vehicle Manufacturers Association in Detroit, says that because "NO_x is one of the hardest emissions to lower," this process could also prove very important to the diesel industry.

— J. Raloff

AIDS studies suggest new directions, therapies

The variability of the AIDS virus — from its genes to its effect on people — sometimes seems matched only by the diversity of approaches that can be taken toward its study. Three new reports suggest a variety of actions for the virus or for the body's reaction to it; each of the three could lead to new therapeutic tacks.

The reports detail a newfound importance for immune system suppressor cells in controlling human immunodeficiency virus (HIV), a suggestion that the virus sparks an autoimmune attack, and the discovery of a small protein that blocks the virus's binding site on its target cells. How the three sets of findings mesh with one another remains to be seen.

Jay A. Levy and his colleagues at the University of California at San Francisco came up with what is perhaps the most paradoxical finding of the three — that *suppressor* immune cells, rather than effector cells that initiate and direct immunity, are key to fighting off HIV infection. Boosting suppressor cells could prevent or help counter infection, they suggest.

Levy and his colleagues studied the blood of three healthy homosexual men who had antibodies to HIV but from whom no HIV could be cultured. When the researchers grew the men's white blood cells in the laboratory and removed the suppressor cells, called CD8 cells, they found biochemical signs of the virus's presence. When they added the suppressor cells back to the culture, signs of the virus disappeared. They also found that a person's own suppressor cells protected his cells better than other people's, and that the more CD8 cells, the stronger the effect. They report on the results in the Dec. 19 SCIENCE.

That suppressor cells could hold in check an immunosuppressive virus is not as counterintuitive as it sounds. Further experiments indicated that the CD8 cells may be releasing a factor that squelches replication of the virus, Levy and his colleagues report. The discovery also suggests an explanation for why some people infected by the virus don't develop the syndrome: It could be that their suppressor cells are more active.

Anthony Fauci, director of the National Institute of Allergy and Infectious Diseases, commented in a prepared statement, "It's an interesting study of potential importance in understanding the mechanism of how the body defends itself against replication of HIV. We look forward to further studies confirming this observation and delineating the precise mechanism of the suppressive effect which was noted."

Levy says he'd like to try growing CD8 cells from AIDS patients' blood samples and giving them back to the patients in higher doses, after he first determines the desired levels of suppressor cells. Too many suppressor cells could have a damaging effect. "We've got to be very careful before we go to clinical trials," he says. If all goes well, such trials could begin within the next six months, he estimates.

Meanwhile, other San Francisco researchers have come up with a theory for how the virus wreaks its havoc. According to John L. Ziegler of the Veterans Administration Medical Center and Daniel P. Stites, who was also an author of the CD8 study, HIV may cause the body to attack itself. While the idea of AIDS as an autoimmune disease has been proposed before, these researchers suggest a precise mechanism for how the virus could induce the attack.

The problem, they say in the December CLINICAL IMMUNOLOGY AND IMMUNOPATHOLOGY, could result from a similarity between the "hook" the virus uses to grab its target — immune effector cells called CD4 or T4 cells — and an unrelated protein on other white blood cell types that tells the immune system that these cells are "self" and shouldn't be rejected.

According to Ziegler and Stites's hy-