tracking temperature, incinerator operators can minimize dioxin production. For the Pittsfield plant, carbon monoxide levels had to be below 100 parts per million and the temperature between 1,500°F and 1,800°F.

"Minimizing conditions will be different for different plants," says NYSERDA's Joseph R. Visalli, project manager for the incinerator test. "You have to know the incinerator, and you have to do some testing."

This research and other studies also hint that burning garbage at temperatures higher than a certain level may be counterproductive. "There's been this feeling that the higher the temperature, the better off you are," says Visalli. Higher combustion temperatures may, in fact, lead to greater carbon monoxide production because of incomplete combustion and to the vaporization of larger quantities of heavy metals, which become part of the fly ash and act as catalysts for the production of dioxins.

Chemist Francis W. Karasek of the University of Waterloo in Waterloo, Ontario, reports in the Aug. 14 SCIENCE that fly ash plays a major role in the production of dioxins. "We found that [fly ash] is indeed a very strong catalyst," says Karasek, "which causes dioxins to form from almost anything." That process seems to occur most readily after the fly ash has cooled to about 300°C, often in a pollution control device such as an electrostatic precipitator. The chief culprits are the heavy metals present in the fly ash particles.

Karasek suggests that one way to solve the dioxin emissions problem is to add a substance that "poisons" the catalyst, preventing it from contributing to the formation of dioxins. "We've been doing quite a bit of experimental work in which we are able to introduce compounds that render the fly ash completely inactive," he says. "It's possible to completely block the formation of dioxins."

Controlling combustion is still important, says Visalli. "Whatever pollution control you put on, you still want to minimize the amount [of pollutants] that goes into it," he says. "The less that's produced and the more that's destroyed, the less you have to worry about it in the ash."

Another alternative is to eliminate metals from garbage before it is incinerated. "We need to know more about where some of these chemicals are coming from in the waste," says Visalli. Pigments in printing inks, for example, may contribute heavy metals. The Pittsfield researchers also found traces of dioxins (but not furans) in the garbage even before incineration. "There are a whole host of things that should be removed from refuse and recycled," Visalli says.

Much remains to be learned. Still unclear is the role that metals play in the formation of compounds other than dioxins. More full-scale tests of incinerators should be done to confirm the Pittsfield results. And researchers need a better understanding of how to take samples and analyze them for dioxins so that the results are reliable and accurate.

Nevertheless, says Floyd Hasselriis of ASME's dioxin committee, the Pittsfield study "is the first clear road map [for incinerator operating conditions] that we've had. We really understand it pretty well now."

"To me," says Visalli, "the dioxin controversy, if it's done anything positive, has inspired research into learning how to better combust [garbage] and better control emissions. I think we're achieving that." — *I. Peterson*

Gene therapy takes aim at liver, lungs

Two studies released last week describe progress in an experimental technique that may someday replace organ or tissue transplants as a means of correcting certain metabolic disorders. In both cases, researchers are using recombinant DNA technology to correct for a class of diseases in which one or more defective genes result in an inability to produce particular proteins in the body.

Savio L. C. Woo of the Baylor College of Medicine in Houston reports that he and his colleagues successfully infected liver cells with recombinant retroviruses, and that these viruses directed the liver cells to produce a new protein. The research points to the possibility of stimulating genetically defective liver cells to produce normal proteins by using custom-crafted viruses as genetic delivery vehicles — a process known as somatic gene therapy.

"The liver may be the preferred target for somatic gene therapy of many inborn errors of metabolism that are currently indications for liver transplant," the researchers write in the August PROCEEDINGS OF THE NATIONAL ACADEMY OF SCIENCES (Vol.84, No.15).

The research, however, confirms the importance of incorporating the proper genetic "switch," or promoter, into a genetically engineered carrier virus in order to get expression of an inserted gene. Working with liver cells cultured from mice, the researchers experimented with three different viral promoters. They found that only one of them — the herpes TK promoter — was capable of being "turned on" in liver cells. "That's in contrast to skin cells," says one of the researchers, Fred D. Ledley, "where all three promoters work just fine."

The research is aimed at developing a treatment for one of the most common inborn errors of metabolism — phenylketonuria, or PKU (SN: 2/8/86, p.84), in which liver cells fail to produce the protein phenylalanine hydroxylase. Each year in the United States about 1 in 12,000 infants is born with the deficiency, which carries potential for toxicity and mental retardation.

"These kids can be kept on a [phenylalanine-free] diet, but that's palliation, not a cure," Ledley told SCIENCE NEWS. In addition, he says, "There are really dozens of liver disorders — many of which are lethal — that we just can't treat." Although liver transplants are becoming increasingly successful, Ledley notes that "the key advantage of gene therapy over organ transplants is that you don't need to find a donor."

In related research, scientists at the National Heart, Lung, and Blood Institute (NHLBI) in Bethesda, Md., transplanted gene-altered cells into mice, then tracked the long-term production of a human protein by those cells. The cells had been induced via retroviral gene transfer to produce alpha 1-antitrypsin, a protein that protects lung tissue from naturally occurring but potentially damaging enzymes. Inherited deficiencies of alpha 1-antitrypsin today account for 20,000 to 40,000 cases of emphysema in the United States.

Robert I. Garver Jr. and his colleagues report in the Aug. 14 SCIENCE that alpha 1-antitrypsin diffused into the blood and lung tissue of mice for four weeks after genetically engineered alpha 1-antitrypsin-producing cells were injected into the rodents' abdominal cavities. The research suggests that physicians may someday treat genetic deficiencies of certain circulating proteins by implanting "colonies" of specially engineered protein-secreting cells.

These findings differ from those of Woo and others at Baylor, who found that the addition of PKU-correcting protein to the general circulation was insufficient to correct that genetic deficiency. The difference, according to Ronald G. Crystal of the NHLBI team, may be that phenylalanine hydroxylase — the PKU protein — needs to interact with cofactors inside liver cells, while alpha 1-antitrypsin works in the extracellular space. "I think the approach we're using can be useful for conditions in which the deficient protein is an extracellular protein, such as alpha 1-antitrypsin, growth hormone, or complement [an immune system protein]."

In addition, Crystal says, his team's technique may prove more useful than the current practice — also still experimental — of using bone marrow cells to manufacture missing proteins. Instead of using marrow cells, which are genetically variable and can respond to gene transfers in unpredictable ways, the team uses monoclonal fibroblast cells that are genetically uniform and that express inserted genes more efficiently. — *R. Weiss*