25 percent or more of the light-duty vehicles may be powered by diesels by 1990. The improved fuel efficiency of diesel engines is accompanied by a significantly higher rate of particulate emission. Thus, based on current performance, one conservative estimate (*1*) projects 155,000 metric tons of particulates in the United States alone annually by the end of the decade from light-duty diesel vehicles and the total particulate load, including that from heavy-duty diesels, may be more than twice that.

Examination (*2–5*) of the organic extracts of diesel particulate matter indicates significant activity in short-term mutagenicity assays that might be indicative of a *potential* of these materials to induce cancer 20 to 30 years later. Increasingly convincing evidence (*3–8*) for the presence of a class of chemicals called nitroarenes in diesel effluent has been obtained during the last 2 to 3 years. Various groups have reported (*4–5, 7*) that 1-nitropyrene accounts for about 20 percent of the total mutagenicity of diesel effluents. In addition, several investigators have recently detected and reported (*4, 6, 9*) dinitropyrenes as well in diesel particulates. Their contribution to the activity might account for an additional 30 to 80 percent of the total activity. One of these compounds, 1,8-dinitropyrene, is the most potent mutagen reported to date. Thus, it is probable that nitroarenes represent the major class of mutagens in diesel particulates. It can be estimated that the yearly emission in the United States of 1-nitropyrene alone by light-duty diesel engines will be 14,500 kilograms by 1990.

The above observations lead directly to what can be considered the two critical questions:

1) Does inhalation of diesel particulates result in adverse health effects? Specifically, are the results of mutagenicity assays indicative of risks to the environment and humans?

2) Are nitroarenes in general an essential result of the combustion process or do they arise as secondary by-products from the simultaneous presence in the exhaust of polycyclic aromatic hydrocarbons, oxides of nitrogen, and acids? If indeed nitroarenes represent only a small fraction of the mass (approximately 2 percent) yet a major portion of the biological activity of diesel effluents, this provides a unique opportunity for controlling and reducing their concentration. Could optimization of the combustion process or modification of the afterburn result in the control and reduction of nitroarene formation without other adverse consequences?

Since a definitive resolution of the biological consequences of nitroarenes is not likely to be available soon, attempts to minimize their formation in diesel particulates through modification of the combustion process appear to be justified; various afterburn treatments appear promising in this respect. Exposure to mutagenic nitroarenes is not restricted to humans. Because of their widespread distribution, they have the potential of acting on and inducing genetic modifications in the flora and fauna, including the highly inbred and therefore vulnerable food-producing plants, such as corn and wheat.

The widespread distribution, potent mutagenicity, and uncertain biological effects of nitroarenes on higher forms of life indicate there is a need for prompt investigation and caution. This does not appear to be the time to dismiss emissions as a potential health risk and relax the relevant levels of permissible effluents. Nor does it seem wise, as presently contemplated, to curtail federally funded programs to investigate the health effects of diesel emissions. Finally, because it has been found that particulate matter and mutagenic emissions are increased even more drastically in malfunctioning diesel engines, it would appear prudent to alert the public to the importance of a properly adjusted engine and to include appropriate mandatory checks in state vehicular inspection programs.

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Leprosy Vaccine

In Thomas H. Maugh II's excellent article on the leprosy vaccine feasibility studies (Research News, 26 Feb., p. 1083), there is no mention of the crucial role of the National Institute of Allergy and Infectious Diseases (NIAID) of the National Institutes of Health in sponsoring basic and applied research on leprosy. Through both grants and contracts, NIAID funds research on propagating the causative agent in vitro, growing it in armadillos, separating it from infected armadillo tissue, and isolating and characterizing protein and lipid antigens from the purified agent. The NIAID also funds studies on the epidemiology, immunology, and serology of leprosy and coordinates the exchange of materials, such as *Mycobacterium leprae* itself, purified antigens, serum samples from leprosy patients, and polyclonal and monoclonal antibodies. In addition, NIAID administers the U.S.–Japan Cooperative Medical Science Program, wherein scientists from the United States and Japan meet annually to discuss research and progress in several tropical diseases, one of which is leprosy.

According to Darrell Gwinn, Leprosy Program Officer at NIAID, almost $1.5 million is being spent annually on leprosy-related programs. Many of these efforts concern the individual's immunological response to the leprosy bacillus and therefore are directly related to the development of a leprosy vaccine.

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Erratum: In the report "Long-term synaptic potentiation in the superior cervical ganglion" by T. H. Brown and D. A. McAfee (12 March, p. 1411), equation 2 and the following lines should read:

$$I(t) = P\exp(-t/\tau_P) + L\exp(-t/\tau_L)$$

where $P\exp(-t/\tau_P)$ is the early, rapidly decaying component and $L\exp(-t/\tau_L)$ is the slowly decaying, long-term component.