

No sign of ozone loss from launches

Environmental groups have recently voiced concern that exhaust from the space shuttle and other rockets could harm the protective ozone layer. But measurements taken by satellites after shuttle launches do not show any sign of stratospheric ozone destruction, atmospheric scientists report.

Solid-propellant motors used to loft some rockets pose a potential threat to the ozone layer because their exhaust contains chlorine, which in some forms can destroy ozone. Indeed, a scientific flight through a rocket's exhaust plume in the 1970s detected a 40 percent decrease in ozone levels in the plume, reports Steven Aftergood of the Washington, D.C.-based American Federation of Scientists in the Sept. 20 *JOURNAL OF GEOPHYSICAL RESEARCH*. Aftergood suggests that launches may produce local ozone "holes" that persist for several hours.

In the same journal, Richard McPeters of NASA's Goddard Space Flight Center in Greenbelt, Md., and his colleagues respond to Aftergood's comments by examining satellite measurements of ozone in the shuttle launch area. They find no detectable ozone decrease after several launches.

This week, the American Institute of Aeronautics and Astronautics released a report examining the issue of launches and atmospheric problems such as ozone depletion, acid rain and global warming. "There is no pressing need to change the propellants of current launch systems," the report concludes. Any localized ozone loss resulting from launches would dissipate within a few hours and should not significantly affect the Earth's surface because the rockets fly in a slanted trajectory through the atmosphere, says Jerry Grey, the institute's director. Only a vertical trajectory could create a local ozone "hole" through which harmful ultraviolet radiation could penetrate.

According to the report, model calculations suggest that 15 launches a year — a number that exceeds the current launch frequency — could decrease ozone levels over the northern midlatitudes by up to 0.1 percent. While such small changes pose no threat now, they could grow important over time, especially if launch frequency increased substantially, Grey says. The report also calls on scientists to improve on the rudimentary models currently used to assess ozone loss.

A priest's ozone legacy

For 26 years in the late 19th century, Father Francesco Denza kept very regular records that are now helping scientists understand how the industrial revolution increased ozone pollution in the troposphere, the atmosphere's lowest layer.

From January 1868 through December 1893, this theologian and physics teacher took twice-daily readings of ground-level atmospheric ozone at his college in Moncalieri, located at the foot of the Italian Alps. In Denza's time, researchers suspected that ozone might cause epidemic diseases. Scientists now recognize tropospheric ozone — a component of smog — as a greenhouse gas as well as an eye and lung irritant.

A team of Italian scientists has converted Denza's data to a modern scale in order to compare today's ozone levels with those of a preindustrial environment. In the Sept. 20 *JOURNAL OF GEOPHYSICAL RESEARCH*, they report that 19th-century Moncalieri had one-half to one-third the ozone present in a modern rural site. Exhaust from cars, power plants and other sources creates ozone in the lower troposphere.

A quirk of the region's weather enabled the scientists to use Denza's data to gauge ozone changes in the upper troposphere as well. The Alps often cause winds to descend into the Moncalieri region, bringing air from higher levels down to ground level. By identifying such events in Denza's records, the researchers demonstrate that ozone has also accumulated in the upper troposphere.

A dietary shield against lung cancer?

Nonsmokers might reduce their lung cancer risk by cutting cholesterol consumption and by eating more fruits, red and yellow vegetables, and perhaps margarine, suggest three new reports in the Sept. 1 *AMERICAN JOURNAL OF EPIDEMIOLOGY*.

One research group followed 4,538 Finnish men for 20 years. Among the 117 men who developed lung cancer, smoking proved the biggest risk factor. But the data also suggest that nonsmokers eating diets high in antioxidants — mainly carotenoids, vitamin C and vitamin E — lowered their lung cancer risk by at least 60 percent. And these agents appear to act independently, since nonsmokers with diets low in all three faced nearly four times the cancer risk of those whose diets contained the most. Among plant-derived foods, fruits conveyed the biggest benefit, although the study also linked red and yellow vegetables and cereals to lower lung cancer risks.

The 2,300-plus smokers in the group derived no antioxidant benefit, suggesting the micronutrients were not potent enough to counter smoking's influence, say Paul Knekt of the Research Institute for Social Security in Helsinki and his coauthors.

The big surprise? Nonsmokers who ate the most margarine faced only 8 percent of the lung cancer risk seen in those who ate the least margarine. Smokers also derived a benefit from margarine, but theirs was much smaller. Knekt's team speculates that margarine's effect may trace to its vitamin E content or to the possibility that men who chose it over butter took better care of themselves overall.

In another study, Richard B. Shekelle of the University of Texas at Houston and his colleagues reviewed dietary data on 1,878 Chicago men employed by the Western Electric Co. in 1958. During a 24-year follow-up, 57 died of lung cancer. After adjusting for age, smoking and consumption of fat and beta-carotene (a carotenoid), the researchers found that men who had ingested more than 795 milligrams of cholesterol daily ran nearly double the lung cancer risk of those eating less than 605 mg/day. However, this association "was specific to consumption of eggs," the researchers write. Chance, or perhaps a noncholesterol ingredient in eggs, may explain the egg/cancer correlation, they say. But they also note that three other studies have found hints of a cholesterol link to lung cancer.

Marc T. Goodman of the Cancer Research Center of Hawaii in Honolulu, who coauthored one of the three previous studies, has now reanalyzed a portion of his data. While a cholesterol-cancer link remained, there was "no evidence of a dose-response relation between egg consumption and the risk of lung cancer," he writes in a letter published in the same journal. He notes, however, that the Chicago men consumed far more eggs and cholesterol than did the Hawaiians in his own study.

Oxidation diminishes HDLs 'goodness'

Atherosclerosis begins when "foam cells" rich in lipids (mostly cholesterol) accumulate along artery walls. Researchers at the University of California, San Diego, showed that when a buildup of reactive molecules in the body causes low-density lipoproteins (LDLs) to undergo a chemical transformation known as oxidation, macrophages — cells that help LDLs unload their cholesterol — change into foam cells (*SN*: 4/30/88, p.279).

Ordinarily, HDLs — the so-called "good" lipoproteins — can withdraw cholesterol from foam cells and target it for removal from the body. But researchers at Kyoto University in Japan have now demonstrated that oxidized HDLs lose much of their ability to do this. The new data "suggest that oxidative modification of HDL may stimulate development of atherosclerosis" by limiting its removal of cholesterol from foam cells, report Yutaka Nagano and his colleagues in the Aug. 1 *PROCEEDINGS OF THE NATIONAL ACADEMY OF SCIENCES*.