Orbiting sensors study threat to ozone layer

Despite glitches that delayed the delicate operation, crew members aboard the space shuttle Discovery last week deployed a satellite designed to vastly improve scientific understanding of Earth's protective ozone layer and the chemical pollution that degrades it. The successful release of the instrument-packed satellite kicked off NASA's "Mission to Planet Earth," a multi-decade effort to examine how human activities affect the global environment.

The largest U.S. satellite ever built for studying the Earth, the Upper Atmosphere Research Satellite (UARS) will probe the physical and chemical processes that dominate the stratosphere and mesosphere – the two middle layers of atmosphere, extending from an altitude of about 15 kilometers up to 80 kilometers.

"We're trying to understand the processes so that we don't have any more surprises. The ozone hole, when it was discovered in 1985, was a surprise. People hadn't anticipated things like that," says UARS chief scientist Carl A. Reber of NASA's Goddard Space Flight Center in Greenbelt, Md.

The $710 million spacecraft, designed to operate for at least three years, carries 10 sensors. Four of the instruments measure atmospheric temperatures and concentrations of important chemicals. Two sensors record the speed and direction of stratospheric winds, which, among other things, set the stage each September for the development of the Antarctic ozone hole. Another four measure the energy deposited by the sun in the upper and middle atmosphere. This energy creates the ozone layer and powers the chemical reactions that allow chlorine pollution to destroy ozone.

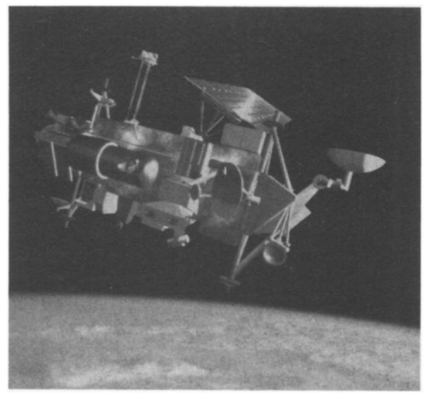
The UARS sensors are not designed for long-term monitoring of ozone concentrations; rather, they will help scientists study the processes that control ozone levels. With instruments that can "see" between 80°N and 80°S, UARS will provide essentially global measurements of many factors previously studied only with aircraft and balloons, which collect information from limited regions of the atmosphere.

Perhaps most important, one instrument will provide the first nearly worldwide measurements of chlorine monoxide, the chemical that plays the principal role in creating the Antarctic ozone hole. Scientists have also detected extremely high levels of chlorine monoxide in the Arctic and elevated values over the United States, but they do not know the extent of these elevated levels.

The new satellite "is sort of a godsend," says Michael J. Prather, an atmospheric scientist at NASA's Goddard Institute for Space Studies in New York City. Prather leads an effort to determine whether fleets of high-speed, Concorde-like planes could damage the ozone layer. He and his collaborators hope to answer this and other questions about ozone loss by using computer models to simulate chemical reactions in the atmosphere. Measurements made by UARS will provide the data needed to test these models, he says.

NASA delayed the satellite's release from the shuttle when a check revealed a wiring mistake – detectable only in space – that caused its signals to have the wrong polarity. In two hours, engineers on the ground made simple changes to reverse the polarity, says project manager Charles E. Trevathan of the Goddard Space Flight Center.

But several hours later, UARS' primary transponder failed to tune in to a communications satellite that transmits information between the ground and UARS. Ground control switched to an identical, backup transponder on UARS, which successfully locked on to the communications signal. Soon afterward, the main transponder began working, says Trevathan. Engineers now seek to understand the problem. – *R. Monastersky*



NASA

Largest Earth explorer: UARS measures 10.3 meters long and weighs 6,800 kilograms.

Finally, proof of 'good' cholesterol's perks

By now, most people know that high blood levels of the so-called "good" cholesterol – high-density lipoprotein particles, or HDLs – help lower the risk of developing atherosclerosis. Epidemiologists established this medical truism through scores of studies showing that people with elevated HDL levels are less likely to suffer heart attacks caused by narrowed arteries. But researchers have never known for sure whether HDLs actually prevent the fatty arterial deposits characteristic of atherosclerosis, or whether the link is simply a clinically useful coincidence.

Now, a research team at Lawrence Berkeley Laboratory in Berkeley, Calif., has demonstrated that mice with an inherited genetic susceptibility to atherosclerosis fail to develop the initial stage of the disease if they are genetically engineered to carry a human gene for HDL. Other researchers say the finding suggests a new strategy for treating people with high levels of "bad" – low-density lipoprotein (LDL) – cholesterol.

LDLs ferry cholesterol to blood vessel walls, depositing it as fatty streaks. HDLs clear LDL cholesterol from the bloodstream by transporting it to the liver for breakdown and excretion (SN: 9/9/89, p.171). Until now, however, scientists lacked proof that HDLs remove enough LDL cholesterol to prevent formation of the fatty streaks, which can eventually harden into plaques on vessel walls.

The new evidence that HDLs inhibit the earliest phase of atherosclerosis "supports what people have observed through epidemiology," says study leader Edward M. Rubin. Beverly Paigen, who studies atherosclerosis in mice at the Jackson Laboratory in Bar Harbor, Maine, says the work "provides really direct evidence that increasing the level of HDLs ... will give protection against heart disease."

Rubin and his colleagues inserted the human gene for HDLs' primary protein into mice that ordinarily develop telltale fatty streaks in their arteries when fed a high-fat diet. HDL particles contain equal parts of protein and cholesterol; the protein component consists mainly of apolipoprotein A-1 (apo A-1).

The team reports in the Sept. 19 NATURE that the transgenic mice produced nearly three times more apo A-1 than control mice. The high apo A-1 levels prevented fatty arterial streaks even in engineered mice fed high-fat "cookies" made of mouse chow and large amounts of dairy butter. Fatty streaks did develop in engineered mice receiving ultra-high-fat cookies containing cocoa butter, but these deposits covered only one-eighth as much area as those of control mice on the same diet.

The apo A-1 increase, which in turn elevated total levels of HDLs, "had a very profound effect" in preventing fatty streaks, Rubin concludes. But he cautions that the streaks are only the precursors of life-threatening atherosclerotic plaques. Researchers need to conduct longer studies to show that apo A-1 prevents plaque formation, he says.

In the meantime, Paigen predicts the new work will spur researchers to develop drugs that can raise levels of HDLs. Current drug treatments that lower the good cholesterol along with the bad "may not be the optimal kind of therapy," she says. "We really want something that will lower LDL cholesterol and leave the HDL where it is or raise it." – *C. Ezzell*