Northern ozone suffered heavy winter loss

Earth's ozone layer weathered a tough winter this year. Concentrations of the protective gas were well below normal over the United States and much of the Northern Hemisphere, apparently brought down by natural weather patterns and chemical pollution. Ozone amounts set record lows above some Arctic regions, but the northern depletions were not severe enough to qualify as an ozone hole.

In years past, atmospheric scientists had discerned the seeds of Arctic ozone loss in the form of destructive chlorine molecules amassing within northern skies. Such pollutants would take a substantial bite out of the polar ozone, researchers claimed, if the stratosphere stayed cold in late winter. Those predictions came true as temperatures set record lows this March, enabling pollutants to devour ozone in the far north.

The National Oceanic and Atmospheric Administration last week released satellite measurements of wintertime ozone collected by its orbiting SBUV/2 instrument. The greatest depletions occurred over Siberia, where ozone concentrations dropped 35 percent below the values observed in 1979, before substantial ozone destruction had begun.

Sensors on NASA's Upper Atmosphere Research Satellite also confirmed that chlorine chemicals munched away ozone in the Arctic. The satellite instruments detected high concentrations of the destructive molecule chlorine monoxide in the Arctic vortex — a region of cold air enclosed by swirling winds that circle the pole. "There's no question there has been significant loss in the Arctic this winter and ozone values are low," says Joe W. Waters of NASA's Jet Propulsion Laboratory in Pasadena, Calif.

Ozone in Earth's stratosphere (12 to 50 kilometers above the surface) protects life by blocking out harmful ultraviolet radiation from the sun. Chlorine and bromine pollution began eating away the beneficial layer in the late 1970s. Each September, the chemicals destroy most of the ozone above Antarctica, creating an ozone hole. They have also gnawed slowly at ozone in the midlatitudes, lowering its concentration by about 2 to 4 percent per decade.

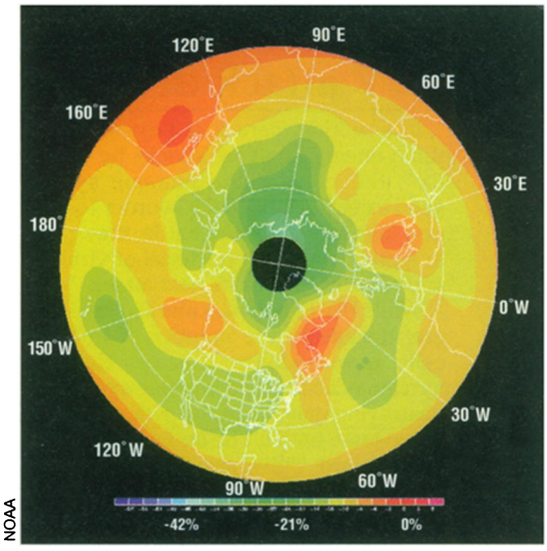
The extremely cold Arctic temperatures this year contributed to ozone depletion by creating icy cloud particles in the stratosphere. Such particles provide surfaces for the chemical reactions that transform inactive chlorine into chlorine monoxide. In contrast to what happens in Antarctica, however, the destructive process in the Arctic does not remove enough ozone to generate an ozone hole.

According to the SBUV/2 data, low ozone concentrations also persisted above the northern midlatitudes, home to much of the world's population. March values above the United States fell 15 to 20 percent below the amounts observed during the same month in 1979. Ground-based observations, gathered by the World Meteorological Organization (WMO), recorded a 10 percent deficiency over Europe for the winter season, says WMO scientist Rumen D. Bojkov in Geneva.

The midlatitude ozone declines present a difficult problem for scientists. Although evidence indicates that chemical pollutants deserve most of the blame for the Arctic loss, meteorological factors could have played a major role in the midlatitude changes. Wind shifts above the tropics in late 1994 may have reduced atmospheric circulation in the stratosphere, cutting the flow of ozone-rich air into the midlatitudes.

International agreements to eliminate many ozone-destroying pollutants will help the ozone layer recover in the next century. But scientists expect that the problem will worsen over the next decade, as chlorine accumulates and greenhouse warming in the lower atmosphere causes cooling in the stratosphere.

— *R. Monastersky*



Comparison of March ozone values in 1979 and 1995. Red indicates no change. Green shows where 1995 amounts fall 20 percent below 1979 values.

Did evolution really anticipate dioxin?

Most, if not all, mammalian cells possess a clump of proteins that will bind to dioxin, polychlorinated biphenyls (PCBs), and many other toxic pollutants. Once this pairing occurs, the complex travels as a unit to the cell's nucleus, where it can wreak havoc with DNA's normal, genetically prescribed activities.

For more than a decade, toxicologists have wrestled with the question of why animals evolved a receptor for pollutants emitted largely since World War II.

A study now appears to confirm what these scientists had come to suspect: The "dioxin receptor" harbors a split personality. While toxicologists probed the dark side of this aryl hydrocarbon (Ah) receptor — its ability to unleash dioxin's toxicity — a benign alter ego has quietly hidden away. And while the precise nature of its beneficial persona still remains concealed, the new data suggest that this receptor performs several indispensable functions.

To probe the receptor's role, Frank J. Gonzalez of the National Cancer Institute in Bethesda, Md., and his colleagues developed a line of mice in which they genetically knocked out, or eliminated, the Ah receptor. In the May 5 SCIENCE, they note that these mice exhibit several potentially lethal defects.

For instance, they begin life with only 10 to 20 percent as many infection-fighting T and B cells circulating in their blood as mice who carry the Ah receptor. Not surprisingly, roughly half these receptor-free newborns die within 2 weeks of birth. In survivors, numbers of these immune cells eventually build to near normal levels — only to plummet permanently following puberty.

The knockout mice also develop liver fibrosis — pockets of dead tissue, Gonzalez notes. The affected area covers 10 percent of the newborns' livers and grows with time. Such damage usually signals toxic chemical exposures.

Gonzalez concludes that the receptor appears to offer protection from still-unidentified toxicants. During fetal development, mom's receptors may take over for those her offspring lack. Certainly, he says, this scenario would explain why the animals' vitality declines immediately after birth.

The new knockout data "confirm that this [receptor] has an important role in development," says dioxin toxicologist Linda Birnbaum of the Environmental Protection Agency in Research Triangle Park, N.C. She says this study also goes a long way toward establishing that some "natural partner" of the Ah receptor exists in the body — one that either becomes dislodged or blocked by dioxin and its toxic cronies.

Chris Bradfield of Northwestern University Medical School in Chicago is also working on an Ah receptor knockout. One justification for developing these mice has been to test the receptor's role in any toxic effects linked to dioxin and PCBs. But that won't be possible in such sick mice. So before such studies can begin, Bradfield says, researchers may have to "tweak this model a little more" to "rescue these mice" — perhaps by reinserting the receptor back into certain tissues.

— *J. Raloff*