

Depleted Ring Around Ozone Hole

Chemical reactions destroy a significant amount of ozone *outside* the main ozone "hole" over Antarctica, according to information gathered in 1987 during a series of high-altitude airplane flights. The newly analyzed data also reveal that ozone destruction in this region starts earlier in the year than previously suspected.

"Now we've shown that the geographic extent of the ozone loss is larger than what we've generally identified as the ozone hole," says Michael H. Proffitt of the National Oceanic and Atmospheric Administration's Aeronomy Laboratory in Boulder, Colo.

In the Nov. 16 NATURE, Proffitt and colleagues David W. Fahey, K.K. Kelly and Adrian F. Tuck describe their analysis of data collected during 12 flights of the NASA ER-2 aircraft from Punta Arenas, Chile. Using a new type of analysis to detect ozone loss, the researchers found evidence of abnormally low ozone concentrations in a ring around the Antarctic ozone hole. In 1987, the ring extended 5° to 10° latitude beyond the ozone hole.

This ring does not cover any populated regions. But ozone destruction in the area could help dilute the ozone layer over more populated portions of the Southern Hemisphere, says Tuck, the project scientist for the airborne experiments (SN: 10/10/87, p.230). Stratospheric ozone protects most life by absorbing harmful ultraviolet radiation from the sun.

The term ozone hole refers to a significant drop in stratospheric ozone concentrations over Antarctica during September and October each year since the late 1970s. At that time of year — spring in the Southern Hemisphere — a belt of stratospheric winds, called the polar vortex, circles the Antarctic region at about 66°S latitude. According to the leading theory of ozone loss, the vortex builds in winter and isolates the polar stratosphere from warmer air over the midlatitudes, allowing temperatures to drop low enough to activate chlorine chemicals that destroy ozone.

Outside the main hole, however, temperatures are much warmer and the concentration of the destructive chlorine chemicals is lower. That's why the suggestion of significant ozone depletion outside the hole comes as a surprise, says Guy P. Brasseur of the National Center for Atmospheric Research in Boulder.

Proffitt and his colleagues used a novel approach to detect and measure this depletion. Over the last four years, scientists have tracked ozone destruction by measuring its concentrations with instruments on satellites, on balloons and on the ground. The method works inside

the Antarctic vortex because ozone levels there drop to extremely low values.

But subtler losses are harder to detect by simply monitoring ozone levels. The concentration swings up and down naturally, so scientists cannot easily determine a "normal" concentration for a particular area, Proffitt says. This problem has plagued researchers trying to detect ozone loss in the Arctic.

Proffitt's group developed an indirect technique to calculate normal ozone levels from the concentration of other, better-behaved chemicals. Their analysis of data collected on flights to various parts of the globe suggests a clear relationship between ozone and certain nitrogen compounds in the atmosphere. The researchers determined that under normal conditions, the ratio between ozone levels and levels of reactive nitrogen exceeds 270:1.

However, flights from Punta Arenas detected a significantly lower ratio in the region near, but still outside, the vortex. Because other chemical measurements suggest the reactive nitrogen concentrations remained normal, the researchers conclude that ozone concentrations

dropped in this region. They calculate that 15 to 30 percent of the ozone at certain altitudes disappeared in a band outside the hole. Inside, the depletions reached 70 to 80 percent at that time, sinking even lower in the next two weeks.

Evidence suggests that destructive chemical reactions caused this drop in ozone levels, but the available information does not indicate whether the chemicals involved were human-made or natural, Proffitt says. He suspects human influence because other measurements have shown a long-term decrease over the last decade in August ozone concentrations for regions between 60°S and 66°S, which is just outside the vortex.

If further work verifies such a significant loss around the hole, scientists will have to determine the mechanism causing the ozone destruction. They must also explain the timing of the loss. The analysis by Proffitt and his colleagues shows that destruction both outside and inside the hole occurred very early in the spring, by mid-August. Other types of measurements did not detect ozone depletions inside the hole until later, Proffitt says.

— R. Monastersky

Prader lacks fader; Angelman misses mom?

At first glance, the two diseases have little in common. People with Prader-Willi syndrome display a lack of muscle tone, an odd facial structure, obesity and a low IQ. People with Angelman syndrome show severe retardation, puppet-like movements and uncontrollable bouts of laughter.

Yet new research suggests these diseases represent two sides of the same genetic coin. Moreover, geneticists examining the syndromes' molecular bases say Prader-Willi now appears to be the first concrete example in humans of a poorly understood genetic phenomenon called genetic imprinting, as well as the best evidence yet linking imprinting to human disease (SN: 5/20/89, p.312).

Most Prader-Willi cases occur when a particular segment of chromosome 15 gets deleted during embryonic development. Oddly, the syndrome results only when that deletion occurs in the chromosome 15 contributed by the father; the maternally contributed chromosome 15 in these patients is normal. But in about 40 percent of Prader-Willi cases, geneticists find no such deletion.

Robert D. Nicholls, Joan H.M. Knoll and Marc Lalonde of the Children's Hospital in Boston and their co-workers performed genetic analyses on six families with Prader-Willi children who lack the char-

acteristic chromosomal deletion. In all cases they found the patients had two maternal chromosome 15 segments and no paternal copy — the result of a rare genetic error during early development. The syndrome provides "the first absolutely clear evidence in humans that it does make a difference which parent a gene comes from," comments geneticist Judith G. Hall of the University of British Columbia in Vancouver. Moreover, preliminary evidence suggests Angelman syndrome results when individuals inherit two *paternal* copies of the same segment on chromosome 15. The researchers reported their findings this week in Baltimore at the annual meeting of the American Society of Human Genetics and in the Nov. 16 NATURE.

Scientists have suspected that genetic imprinting — a difference in gene expression dependent upon which parent contributed that gene — might play a critical role in fetal and adult development, and may account for the unusual inheritance patterns of some diseases. Nicholls, now at the University of Florida College of Medicine in Gainesville, says the new findings should add to scientists' understanding of how genes regulate the expression of other genes — knowledge that may someday aid in the diagnosis and treatment of genetic diseases. — R. Weiss