Winter ozone gap detected over the Arctic

Balloon-borne instruments revealed a thin region in the Arctic ozone layer last January — a pattern suggesting the possible birth of a small, transient ozone hole there, according to a new report. Scientists maintain, though, that the Arctic will not suffer the same sweeping ozone loss that afflicts the Antarctic stratosphere each year.

Scores of atmospheric scientists traveled to Norway last winter to assess the threat to Arctic ozone (SN: 2/25/89, p.116). On airplanes flying through dark northern skies, they detected high stratospheric levels of the same chlorine compounds that eat away at ozone in the Antarctic. They described the Arctic atmosphere as primed to destroy ozone and waiting for spring sunlight to combine with chlorine compounds to fuel a catalytic cycle. But they left open the question of how much ozone has been destroyed by the chlorine, which comes largely from human-made chlorofluorocarbons.

Filling in some of the details, a separate team now reports the results of concurrent balloon investigations from Sweden. The balloons carried sensors that measured ozone levels and counted cloud particles. According to the leading theory for ozone destruction, stratospheric cloud particles play a crucial role by activating chlorine compounds to break apart ozone molecules.

The measurements revealed a peculiar gap in the Arctic ozone layer, say David J. Hofmann of the University of Wyoming in Laramie and his colleagues in the July 13 NATURE. On Jan. 23, they observed that the ozone concentration rose relatively steadily with altitude from 10 to 22 kilometers. But the concentration dropped as the balloon ascended beyond about 22 km. It started to rise again after 24 km, resuming a more normal pattern of increase at about 26 km. The particle instrument detected far more cloud particles in the low-ozone region than in adjacent areas.

"This looks very familiar," says Hofmann, who has observed September ozone losses in the Antarctic for several years. The apparent thinning in Arctic ozone between about 22 and 26 km closely resembles a pattern signaling the onset of the Antarctic ozone hole, he says.

Early each September, ozone over Antarctica starts disappearing at about 22 km as the spring sun energizes the destructive chemical reactions. The reactions migrate downward as days get longer and the sunlight reaches farther into the polar vortex — a wind pattern circling the polar regions during winter.

In the Antarctic, the vortex persists into springtime and the destruction process removes a substantial fraction of the ozone from the stratosphere. In 1987, half the Antarctic ozone layer disappeared. In the Arctic, though, the stratosphere is less stable and invading winds can rip apart the vortex several times during winter. With the vortex breached, temperatures remain too high to form stratospheric cloud particles, and the chlorine chemicals get dispersed.

The similarity in ozone profiles from both poles leads Hofmann to suggest the Arctic finding represents the beginning of an ozone hole that never fully developed. However, he says, there's a slim chance the gap is a normal feature of the Arctic stratosphere.

Atmospheric chemist Susan Solomon of the National Oceanic and Atmospheric Administration in Boulder, Colo., agrees the case is not closed. "I think Hofmann's probably right, but it's just not tight enough yet to say," she comments.

If chemicals are thinning Arctic ozone, the vortex instability should keep levels there from dropping as severely as in the Antarctic. Hofmann calculates that last winter's ozone gap represented a 25 percent drop from expected levels at altitudes of 22 to 26 km. This may seem a big drop, but that portion of the stratosphere does not normally hold much ozone. Overall, stratospheric ozone fell short of expected levels by only 3 percent, he says. — *R. Monastersky*

AIDS viral burden far exceeds estimates

People with AIDS carry at least 100 times more of the AIDS virus, HIV, in their white blood cells than previously estimated, researchers reported this week. The new findings may help settle the contentious issue of how such a serious syndrome could be triggered by what seemed so few infectious particles.

Some critics of mainstream AIDS research — most notably Peter H. Duesberg of the University of California, Berkeley — have argued that some factor in addition to HIV must underlie AIDS. Duesberg and others question, for example, how a virus detected in only one of 10,000 targeted white blood cells could so cripple a person's immune system.

The new research indicates that AIDS patients harbor HIV in more than one of every 100 CD4-positive T4 cells — the white blood cells that serve as the major target and reservoir of HIV. In many of these cells, HIV has incorporated itself into the DNA of its host cell, thus resisting detection by standard laboratory techniques. Researchers knew HIV could sequester itself in white blood cells but until now had not shown that the practice was so prevalent.

The new ratio of one infected CD4-positive cell per 100 represents "a very, very high number of infected cells," says Miltiades C. Psallidopoulos of the Georgetown University School of Medicine in Washington, D.C., who performed the work with colleagues from Georgetown, the National Institutes of Health in Bethesda, Md., and Program Resources, Inc., in Frederick, Md. Their data also indicate that AIDS patients carry actively replicating HIV in a concentration 10 times that previously seen.

The researchers accomplished the unprecedented tally by combining two extremely sensitive technologies: a laser-driven flow cytometer capable of separating different types of cells with about 99 percent accuracy, and the polymerase chain reaction, which can detect extremely small quantities of specific DNA sequences such as those from HIV. Their high-tech cell sorter is one of the very few in the United States sealed in a special facility to keep aerosolized, AIDS-infected droplets from escaping. The novel arrangement allowed the researchers to test for HIV DNA in individual cells of known subtypes, providing the best data yet regarding the types and numbers of cells harboring HIV in patients at various stages of AIDS.

In describing the unexpectedly large percentage of infected cells, the researchers also express surprise about two other findings. First, very few of the patients' circulating monocytes — another type of white blood cell — contained HIV, even though scientists have found several monocyte-infecting strains of HIV in laboratory studies of cultured cells. Second, patients' so-called CD4-negative cells contained essentially no HIV, although such cells often contain HIV when grown in culture. These observations suggest that some laboratory studies of AIDS infection may not reflect clinical realities, say research leaders Steven M. Schnittman and Anthony S. Fauci.

"The *in vitro* studies are still very valid," Fauci told SCIENCE NEWS. "But this gives us a caveat that what is going on *in vitro*, although very helpful, may not reflect the very subtle things that are going on *in vivo*." The report appears in the July 21 SCIENCE.

A related study by Psallidopoulos and others, due to appear in an upcoming JOURNAL OF VIROLOGY, shows that as asymptomatic, HIV-infected individuals progress to full-blown AIDS, the percentage of CD4-positive T4 cells infected with HIV increases, as does the ratio of active to latent HIV in those cells. By examining subtle genetic and biochemical differences between individual cells harboring active versus latent HIV, the researchers hope to learn how to maintain viral latency indefinitely in infected individuals. — *R. Weiss*