Antarctic ozone hole returns with a bang

'Tis the season for ozone depletion over the Antarctic, and this year's ozone hole is developing at record speed. Ozone has rapidly dwindled in the Antarctic stratosphere despite weather conditions resembling those that checked the growth of an ozone hole in 1988.

'Tis also the season for satellite setbacks, and NASA's main ozone-sensing instrument is threatening to die of old age before the agency can launch a replacement — a situation that would drastically hamper efforts to monitor ozone loss over the entire globe.

The stratospheric ozone layer protects life on Earth by blocking out much of the sun's damaging ultraviolet radiation before it reaches the planet's surface. Every September since the late 1970s, the Antarctic has experienced a temporary but dramatic loss of ozone. The ozone hole develops because extremely cold conditions in the polar stratosphere help chlorine and bromine pollutants to destroy ozone through chemical processes.

Balloon measurements made above Antarctica's McMurdo Station indicate that stratospheric ozone levels this year have dropped as swiftly as they did in 1987 and 1989 — two years when ozone reached its lowest recorded level (SN: 10/14/89, p.246), says David J. Hofmann of the National Oceanic and Atmospheric Administration in Boulder, Colo.

In early September, the rate of ozone loss exceeded even that of 1987 and 1989, according to measurements by the Total Ozone Mapping Spectrometer (TOMS) aboard NASA's Nimbus 7 satellite. "It's clear that the ozone is somewhat lower this year than it was last year at the same time," says Arlin J. Krueger of the NASA Goddard Space Flight Center in Greenbelt, Md. TOMS data show that the minimum ozone level over Antarctica plummeted from 200 Dobson units in mid-August to 133 Dobson units in mid-September.

Ozone losses normally continue through the early part of October. Scientists say they cannot predict whether this year's ozone layer will thin to the same extent it did in 1987 and 1989, when levels reached a minimum value of around 103 Dobson units.

The amount of ozone lost so far this year already exceeds the total lost in 1988, a year when several large weather patterns formed in the Southern Hemisphere and disturbed the Antarctic vortex — a ring of winds circling the pole that seal off the Antarctic stratosphere. In 1988, the weakened vortex let in winds from the midlatitudes, which kept the stratosphere relatively warm and inhibited the development of a strong ozone hole.

Similar but weaker weather patterns developed this year, disrupting the Antarctic vortex to some degree in late August and early September. Because dramatic ozone losses occurred during the same period, scientists now wonder whether weak atmospheric disturbances might sometimes enhance the growth of ozone holes.

At the same time, many researchers express concern that the aging TOMS instrument may be approaching death's door. Launched in 1978 with a planned lifetime of two years, the monitoring device is plagued by a rotating wheel that occasionally drifts out of sync with other parts. The problem has affected TOMS on and off since 1984, but this summer "was by far the most severe episode," says Krueger.

Data collected during these episodes remain usable, but eventually the drift will worsen and TOMS will send only static back to Earth. NASA plans to launch the old design model for TOMS on a Soviet satellite in 1991 as a stopgap until a new instrument goes up in 1993. Researchers want to check the calibration of the current TOMS against another model in order to resolve questions about how much the global ozone layer has thinned since the late 1970s, but if the present instrument fails before a replacement arrives, the calibration opportunity will be lost. — *R. Monastersky*

Extending cell life yields clues to growth

In Tom Robbins' novel, *Jitterbug Perfume,* characters search for the essence of life in an ancient perfume bottle. Muses the novel's narrator: "To physically overcome death — is that not the goal? — we must think unthinkable thoughts and ask unanswerable questions."

In the laboratory, researchers on a similar quest employ a different sort of vessel — a petri dish packed with cultured cells — to identify chemical factors that might either extend life or hasten its demise. And though the scientists may pose questions that have no immediate answers, the ultimate goal, notes molecular biologist Thomas Maciag, seems clear: to painstakingly amass clues about the regulation of cell survival and proliferation that might help elucidate the causes of cancer, atherosclerosis and other illnesses in which cell growth runs amok.

Using cultured endothelial cells derived from a human umbilical vein, Maciag and his colleagues at the American Red Cross in Rockville, Md., say they have now succeeded in prolonging *in vitro* cell survival, defined as the number of times a cell population doubles during its lifetime. Though endothelial cells in the petri dish normally stop dividing after 20 to 60 doublings, those treated with a new chemical agent retained their youthful appearance and lasted long enough to undergo as many as 140 population doublings, the researchers report in the Sept. 28 SCIENCE.

Maciag and his co-workers based this study on their previous work demonstrating that cultures of endothelial cells show signs of aging, including an inability to divide — changes perhaps similar to those that cells undergo in the aging body. They also relied on reports from other researchers that the cellular protein interleukin-1 alpha (IL-1 alpha) appears to inhibit growth of these cells *in vitro.* In their current work, Maciag's team detected elevated levels of IL-1 alpha and its messenger RNA — the genetic material that normally directs production of the protein — in cell cultures that had doubled their population many times, but not in younger cultures.

Injecting the older cultures with daily doses of a compound that blocked the messenger RNA from producing IL-1 alpha, the team prevented the onset of cellular senescence and extended the lifespan of the cells. Stopping the injections allowed the aging process to resume unimpeded, the scientists note.

Because continued exposure to the compound that halted production of IL-1 alpha cannot stave off cell aging and cell death indefinitely, Maciag suggests that the protein may be the first of a repertoire of chemicals that slow endothelial cell growth. "IL-1 alpha may be the 'switch' of choice in the repertoire, but other factors may [sequentially] kick in to inhibit growth," he notes.

Attempts to uncover this secret arsenal, he says, may help cancer biologists and cardiovascular researchers understand why some cells in the body — such as breast-cancer cells and those that form plaque beneath the endothelial lining of blood-vessel walls — proliferate out of control, while others do not. He adds that factors governing proliferation of endothelial cells *in vitro* are likely to differ from those regulating other cells, such as fibroblasts, which also undergo senescence but appear to recognize IL-1 alpha as a growth promoter. Maciag notes that similarities in structure between IL-1 alpha and fibroblast growth factor suggest a common evolutionary origin.

Besides its influence on growth, IL-1 alpha acts to differentiate endothelial cells, he observes, prompting those cells to mature into capillary-shaped cells that line blood vessels and the lymphatic system. Maciag speculates that IL-1 alpha might clamp down on cellular growth in order to stimulate maturation, since a limited amount of energy may prevent both processes from proceeding simultaneously. — *R. Cowen*