Arctic region during summer. When the summer sunlight weakened, the snow from winter could survive the warm months, building up year after year to form a great glacial cap over Canada and northern Eurasia. When the summer sunlight intensified, it would melt the ice sheets.

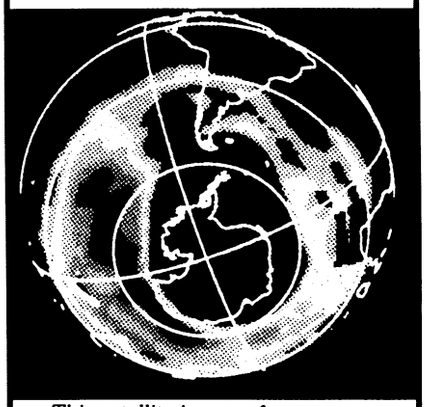
But the Devils Hole record, with its superior chronology, shows that the timing of specific events in the last 500,000 years does not match the predictions of the Milankovitch theory, according to Winograd and his co-workers. Three of the last four ice ages ended when the summer sunlight in the sub-Arctic was relatively weak. What's more, the ice ages lasted different lengths of time, varying from 80,000 to 130,000 years in duration. This suggests that the climate changes follow no regular pacemaker, the researchers contend.

They propose instead that ocean circulation patterns, greenhouse gases in the atmosphere, and the behavior of ice sheets all interact chaotically to cause the irregular glacial cycles. Orbital variations may play a part in this story, but they are by no means principal characters.

James D. Hays, a longtime champion of the Milankovitch theory, calls the Devils Hole data "a beautiful record" but argues that the new data do not sink the idea that orbital changes control the ice ages. Hays, a researcher at Columbia University's Lamont-Doherty Geological Observatory in Palisades, N.Y., points out that although the Devils Hole chronology has more accurate dates than other records, it still faces dating problems. Most important, he says, researchers don't know how many thousands of years rainwater takes to filter through the ground and reach Devils Hole — a factor that could alter the timing of climatic events recorded there.

The Devils Hole record, like the marine ones, includes variations whose frequencies match those of the orbital cycles. This suggests to Hays that these astronomical variables do play an important role. While summer sunlight in the sub-Arctic might not control the growth of ice sheets, a different pattern of sunlight changes may, he says. *— R. Monastersky*

Record size for ozone hole



NASA

This satellite image of ozone concentrations shows a record-breaking region of low levels, depicted in purple hues, sitting over Antarctica. By Sept. 23, this so-called ozone hole had an aerial extent of 8.9 million square miles, almost the size of the entire North American continent. The area of depleted ozone measured about 15 percent larger this year than last year, according to NASA researchers.

Created by chemical pollutants that destroy ozone in the stratosphere, the hole begins to form each year in August and reaches its greatest extent in early October. It then dissipates in November. This image was taken by the Total Ozone Mapping Spectrometer on board the NIMBUS-7 satellite.

The legacy of last year's eruption of Mt. Pinatubo in the Philippines may have worsened this year's ozone depletion. Tiny droplets of volcanic sulfuric acid high in the atmosphere can enhance the destructiveness of the chlorine chemicals that attack ozone. Atmospheric scientists believe such droplets from Pinatubo and a volcanic eruption in southern Chile also added to the severity of last year's ozone hole.

Life-or-death gene sheds light on lymphoma

A gene that normally controls the orderly death of unnecessary cells during the embryonic development of a humble roundworm may offer clues to a better understanding of some common cancers of the human lymph nodes, according to two new reports.

Michael O. Hengartner of the Massachusetts Institute of Technology in Cambridge told a conference of cancer researchers last week that the roundworm gene resembles a so-called proto-oncogene known to cause follicular lymphomas in humans. And in the Oct. 9 SCIENCE, a research team led by Jean-Claude Martinou of the Centre Medical Universitaire in Geneva, Switzerland, reports that injections of the human proto-oncogene, called bcl-2, can prevent the normal death of cultured nerve cells starved of growth factors.

Hengartner discussed his results regarding ced-9, the roundworm equivalent of bcl-2, at the 16th Bristol-Myers Squibb Symposium on Cancer Research, held at the Fox Chase Cancer Center in Philadelphia. He and his colleagues found the ced-9 gene by studying mutant roundworms that develop with extra growths. Hengartner's group determined that these growths consist of cells that would normally die during embryonic development because the worm no longer needs them.

This process of programmed cell death, or apoptosis, occurs in most organisms. Without it, humans would retain the webbed fingers and toes they had as embryos. The same mechanism ensures that old, worn-out cells within the various organs of adults retire and die so that the body can replace them with fresh, lively cells that perform the same function.

"Programmed cell death is a way that multicellular animals have devised to properly get rid of cells that they do not want anymore," Hengartner says. The ced-9 gene regulates the process, he explains, by restraining two "suicide genes" that, when active, kill their own cell. A mutation that damages the ced-9 gene frees the suicide genes to spring into action, leading to cell death, he says.

The similarity between the roundworm gene ced-9 and the human gene bcl-2 may explain how a mutation involving bcl-2 causes human follicular lymphomas, Hengartner suggests. In many such lymphomas, complementary breaks in two different chromosomes allow bcl-2 to switch places with a member of the family of genes responsible for making antibodies. Because the antibody genes function continuously in most white blood cells, says Hengartner, the swap keeps ced-9 permanently turned on, letting some old white cells outlive their usefulness and proliferate as cancer.

"If you inhibit [the] process of cell death, you're going to get a tumor, because the cells are not going to stop dividing when they should," says Frank J. Rauscher III, a cancer researcher with the Wistar Institute in Philadelphia.

Research by Hengartner's team "really illustrates [that] what . . . some may feel are obscure [animal] systems may have absolutely critical relevance and application to what we see in human disease," says Robert L. Comis of the Fox Chase Cancer Center.

Martinou's group has added to Hengartner's cell-death findings by studying rat nerve cells grown in laboratory culture. They report that genetically engineered nerve cells containing extra copies of the bcl-2 gene live two to three times longer than normal nerve cells.

Martinou and his colleagues suggest that their finding might provide insights into degenerative diseases of the nervous system, such as Lou Gehrig's disease and Huntington's disease. Martinou has also found that nerves taken from aborted human fetuses contain active bcl-2 genes. This supports a widely held theory that embryos generate extra nerve fibers — some of which later undergo programmed cell death — to ensure that the nervous system develops the correct nerve connections. *— C. Ezzell*