Earth Science

From a meeting in San Francisco of the American Geophysical Union

A dusty way to break the ice age spell

Motes of floating dust may seem insubstantial, but they have the power to change the world. Computer simulations of Earth's climate suggest that atmospheric dust may have triggered repeated warm spells during the last ice age and could even have ended that glacial period more than 10,000 years ago.

From measurements of ancient particles trapped in glaciers, scientists know that the atmosphere was far dustier during the ice age than it is now. According to traditional climate theory, this dust helped maintain cool conditions by blocking sunlight. The computer simulations, which model both atmosphere and oceans, suggest a far more complex effect of dust, says Jonathan Overpeck of the National Oceanic and Atmospheric Administration in Boulder, Colo.

"The paradigm that glacial dust causes cooling is just wrong," he told attendees.

During the ice age, a large fraction of northern land was covered with bright snow, which reflected most of the sunlight that hit it. Dust is darker than snow, so extra dust in the atmosphere absorbed solar radiation that would otherwise have been reflected into space. Simulations with the added dust showed warmer temperatures over the continents than those without the dust. The scientists also reported their results in the Dec. 5, 1996 NATURE.

The amount of dust in the sky varied markedly during the ice age. When the land-based ice sheets grew large, they stole water from the oceans and caused global sea levels to drop. Exposed ocean sediments were then picked up by the wind, adding dust to the sky. Overpeck and his colleagues suggest that sudden surges in atmospheric dustiness could have triggered abrupt warmings during the ice age and eventually pushed the planet out of its glacial conditions. — *R.M.*

The tropics throw their weight around

Despite its frosty name, the ice age was not a period of unrelenting cold. Global temperatures in the glacial epoch swung up and down radically from one millennium to the next. During the warmest of these spans, vast armadas of icebergs sailed across the northern Atlantic Ocean for reasons that scientists have not yet discovered.

Most researchers have searched for answers in the far north, the site of the iceberg flotillas, which are called Heinrich events. But the explanation for Heinrich events may actually lie much farther south, propose Andrew McIntyre and Barbara Molfino of the Lamont-Doherty Earth Observatory in Palisades, N.Y.

McIntyre and Molfino came to this conclusion after studying seafloor sediments extracted from drill holes in the equatorial Atlantic. They examined fossilized remnants of the marine alga *Florisphaera profunda*, which grows near the ocean surface but below most other algae. Because *F. profunda* flourishes whenever westward-moving equatorial winds slacken, the scientists used the alga as a record of past winds.

The sediment records revealed that all Heinrich events during the last 45,000 years occurred during times of extremely weak equatorial winds. When winds were strong or normal, warm water in the Caribbean and Gulf of Mexico was largely contained in those regions. When the winds died, the warm water flooded into the Atlantic, where it was carried north by the Gulf Stream. Once it reached the far north, this water could have caused the great discharges of ice into the Atlantic, the researchers proposed at the meeting and in the Dec. 13, 1996 SCIENCE.

What controls the equatorial winds? Because the winds appear to follow specific cycles over thousands of years, McIntyre and Molfino hypothesize that slow changes in Earth's orbit drive the wind variations and therefore deserve ultimate blame for the Heinrich events. — *R.M.*

Nutrition

Alcohol's electric effects

Many New Year's revelers enjoyed their libations a little too much again this year, some staggering to bed in a drunken stupor. Though people have long recognized that too much alcohol impairs brain functioning, no one has quite understood how intoxication occurs.

Indeed, observes biochemist Richard W. Gross of Washington University School of Medicine in St. Louis, many test-tube studies have shown that ethanol does not appear to affect brain cells until its concentration becomes so high that it would kill a person. Gross suggests that the flaw in such studies was their use of ethanol—plain alcohol.

When an individual drinks ethanol, the body immediately begins breaking it down. Working with cells grown in the lab, Gross and Rose A. Gubitosi-Klug, also of Washington University School of Medicine, provide evidence that certain ethanol breakdown products, known as fatty acid ethyl esters, are likely to be the direct agents of intoxication.

In the Dec. 20, 1996 JOURNAL OF BIOLOGICAL CHEMISTRY, the pair showed that these ethyl esters speed up the movement of potassium ions, which are positively charged, from brain cells through channels in their outer membranes. This flow of ions increases the negative electric potential inside the cells, impairing the action of the voltage-dependent calcium channels.

The cells rely on calcium for responding to messages from other cells. When ethyl esters depress calcium concentrations, Gross says, communications between these cells can become uncoordinated. "And when their timing is off, you're going to have slurred speech" and other symptoms of drunkenness, including a breakdown in the inhibitory pathways that would normally curb inappropriate speech and behavior, he says.

"Our report is the first to show . . . these profound changes in the electrical functions of a [brain cell] at concentrations of alcohol which are present after people drink," Gross told SCIENCE NEWS. "If you could prevent those changes from occurring," he says, "then presumably you could interrupt or even prevent the phenomenon of dependence" in alcoholics. "That," he reflects, "is our dream." — *J.R.*

Neural tube defects: B-yond folate

Last March, the Food and Drug Administration announced it would soon require U.S. food manufacturers to fortify many of their products with folate. This B vitamin can cut the risk of neural tube abnormalities—a class of potentially debilitating and life-threatening birth defects (SN: 3/30/96, p. 198).

Some 30 percent of such defects, however, appear unrelated to folate. These additional cases might be prevented by supplementation during pregnancy with inositol, another B vitamin, actually a "quasi-vitamin," according to a report by Nicholas D.E. Greene and Andrew J. Copp of the University of London's Institute of Child Health. It appears in the January NATURE MEDICINE.

Curly-tail mice are extremely susceptible to developing folate-resistant neural tube defects. Working with laboratory-grown cells and embryos from these mice, the scientists traced the vulnerability to the strain's abnormally low activity of a gene known as *RAR-beta*. It appears that a mutation in another gene dampens *RAR-beta*'s activity, Copp says. By treating female mice midway through pregnancy with large doses of inositol, he and Greene pumped *RAR-beta*'s activity back up—preventing the development of neural tube defects.

If this mouse represents a good model of folate-resistant neural tube defects in humans, then only persons carrying the faulty gene would be at risk. Copp says the animal data indicate that "the average diet would not provide enough [inositol] to overcome the defect," necessitating supplementation with large amounts of the nutrient. — *J.R.*

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