Here Comes the Sun-Climate Connection

A study of millennia-old pine trees growing on the upper reaches of the Sierra Nevada hints that flickerings in the sun's brightness altered Earth's climate in the past — a finding that suggests scientists must consider solar changes when forecasting global warming.

Solar physicists and geoscientists long have wondered whether hot and cold spells in Earth's history could have resulted from changes in the sun. As concern over global warming has surged in recent years, the solar issue has attracted renewed interest, particularly because some skeptics of warming have suggested that the sun will ameliorate the predicted temperature increase from greenhouse gas pollution.

But clear evidence of a sun-climate connection has remained elusive.

The new analysis of Sierra Nevada pines adds weight to the suggestion that the sun plays a noticeable role in changing Earth's climate. "I think this is some of the best proof we have to date that the sun is actually forcing climate," says Louis A. Scuderi of Boston University.

Scuderi studied the annual growth rings of foxtail pine trees living at the 3,600-meter-high timberline on the harsh, windswept slopes of Cirque Peak. In these dry heights, foxtail pines can survive more than 1,500 years. By comparing the living trees with dead, fallen snags, Scuderi constructed a 2,000-year-long record of tree-ring width, which he reports in the March 5 SCIENCE.

Because tree growth at such altitudes depends largely on temperatures during the growing season, Scuderi could translate the growth data into a temperature record for the site. To test whether the sun caused part of those temperature swings, Scuderi matched the data with a record of atmospheric carbon-14 concentrations, also determined from tree rings. Carbon-14 forms when solar radiation enters Earth's atmosphere, producing energetic neutrons that collide with nitrogen in the air. The carbon-14 concentration thus provides an indirect measure of the sun's strength.

Comparing the temperature and carbon-14 records, Scuderi found that they exhibit similar cycles, particularly one with a period of 125 years. He also discovered that both records show major changes at the same times, around the years 1050 A.D. and 1650 A.D. Such similarities lead Scuderi to suggest that periodic solar variations induced the Cirque Peak temperature cycles.

In the 15 years of satellite observations, the sun's brightness has changed only by 0.1 percent — too little to alter Earth's climate substantially. But Scuderi's study and others suggest that longer term solar fluctuations have had the strength to change the climate.

Minze Stuiver, a geochemist at the University of Washington in Seattle, says Scuderi's study presents a good case for a sun-climate link: "It's one of the better studies." But Stuiver cautions that a simple correlation between two factors does not necessarily indicate that one caused the other. Proponents of the sun's influence on climate have yet to propose a mechanism whereby such minor changes in solar brightness can cause major swings in climate on Earth.

If the sun does wax and wane enough to warm and cool the Earth, then solar variations may have caused part of the global temperature increase since the late 1800s. In the past, climate experts have presumed that carbon dioxide pollution caused much of that warming. But if the sun played a substantial role, then carbon dioxide may affect the climate less than researchers have presumed, which might reduce the expected global warming, Scuderi suggests.

Don't look to the sun to eliminate global warming, however. Two studies in the Nov. 26, 1992 NATURE conclude that the warming power of greenhouse gases outweighs the climatic influence of the sun. "No matter what the sun does, the greenhouse effect still dominates in the future," says Tom M.L. Wigley of the University Corporation for Atmospheric Research in Boulder, Colo., who coauthored one of the papers. *— R. Monastersky*

Free-radical scavenger gene tied to ALS

Successful pinpointing of the defective gene involved in one inherited form of Lou Gehrig's disease (ALS) has shed light on a possible cause — and potential treatment — of nerve-cell damage in this degenerative disorder.

In ALS (amyotrophic lateral sclerosis), nerve cells in the spinal cord and brain that control muscles disintegrate, causing paralysis and eventual death. By studying the 10 percent of people who inherit a genetic defect that causes one type of ALS, researchers have been homing in on how the disease develops.

In about half the people who inherit ALS, that defect lies on chromosome 21. There, a mutation occurs in the gene that codes for an enzyme called superoxide dismutase, a team of 31 scientists reports in the March 4 NATURE.

"It's the most significant finding to date in ALS research," says Lynn M. Klein, a vice president at the ALS Association in Woodland Hills, Calif. "We are really closing in on this disease."

Led by Robert H. Brown Jr. at Massachusetts General Hospital in Boston and Teepu Siddique of Northwestern University Medical School in Chicago, researchers from the United States, Canada, Belgium, and Australia examined chromosome 21 in dozens of families with the familial form of ALS. In 13 families, they discovered 11 different mutations in the gene for superoxide dismutase. Each mutation led to the switching of an amino acid in a key part of the enzyme, says Brown.

"It's an enormously important discovery," comments Carl M. Leventhal, a neurologist at the National Institute of Neurological Disorders and Stroke in Bethesda, Md. For the first time, scientists have found a specific gene involved in a degenerative nerve disease of older adults, he adds.

In the body, this enzyme helps destroy highly reactive oxygen molecules called free radicals, says James O. McNamara, a neuroscientist at the Duke University Medical Center in Durham, N.C. In a separate report in the March 4 NATURE, he and Duke biochemist Irwin Fridovich propose that if the enzyme is missing or not working properly, then free radicals typically generated during nerve-cell activity may build up and slowly destroy neurons. Thus, the finding suggests that therapies to get rid of free radicals may slow the progression of this disease, he adds.

However, scientists do not know how these mutations affect the enzyme or its activity. Preventing the disease may be more complicated than simply getting rid of free radicals, Siddique cautions. "We haven't slain Goliath," he says. "But we feel like a David who has been shown the slingshot."

The results clarify what needs further study. The human body contains other kinds of superoxide dismutase that may play a role in other types of inherited ALS, in nonfamilial — or sporadic — ALS, and other degenerative nerve disorders. "One suspects that free-radical toxicity will be implicated," Brown says.

Even as scientists investigate how the enzyme can lead to ALS, Brown expresses hope that companies will start developing and testing drugs that get rid of free radicals to see whether these therapies work against this form of ALS. *— E. Pennisi*