Biology

Carol Ezzell reports from San Antonio, Texas, at a meeting of the American Institute of Biological Sciences

Prickly tip-off to ozone decline?

Like Stetson hats and chili peppers, the saguaro cactus symbolizes the U.S. Southwest. A large stand of saguaros near Tucson, Ariz., has even achieved the status of a national monument. But ecologists are now investigating whether these tall, branching cacti, which often live for hundreds of years, might also provide another symbol: a preview of the plant damage to occur during the chlorofluorocarbon-induced degradation of Earth's ozone layer.

An unexplained, accelerating die-off of the Arizona saguaros, preceded by a browning and thickening of the outer flesh (cuticle) and the loss of spines, may result from an increased exposure to ultraviolet-B (UVB) rays as the stratospheric ozone layer thins, says ecologist Kate Lajtha at Boston University. If so, she asserts, "the saguaro might be the first plant species to show damage from increased UVB activity."

Lajtha noticed that the saguaros initially develop the so-called "barking" appearance only on their southern sides, which receive the most sun. She ruled out the possibility that the thickened cuticle represents an attempt to conserve fluids, because healthy and damaged saguaros contain the same proportion of water in relation to the amount of carbon dioxide drawn in. She also ruled out frost damage from winter cold snaps, since frozen cacti get mushy and die quickly. Further, searches for disease-causing organisms and comparisons of environmental lead levels absorbed by each side of the plant turned up no differences, she says.

"It's got to be something in the sunlight," concludes Lajtha, although she concedes, "I'm still skeptical [about the ozone link]."

Lajtha, in collaboration with the U.S. Park Service, now plans to place UVB-shielding glass sleeves around a group of healthy cacti in the Saguaro National Monument. "If UVB is causing the barking, we ought to see a difference [between shielded and unshielded saguaros] within five years," she explains.

Humans blamed for gypsy moth spread

The wave of leaf-eating gypsy moths now surging across the eastern United States would slow to a trickle if it weren't for human activities, according to a computer model of the insect scourge.

Since 1966, the moths' tree-destroying larvae have swept into new areas at an average rate of more than 20 kilometers per year. But without hitching rides on recreational vehicles, moving vans and transported plants, the larvae would spread by only 2.5 km per year, predicts a team of entomologists led by Andrew M. Liebhold at the U.S. Forest Service in Morgantown, W. Va.

The researchers found that the insect's spread is not due to an expansion into more desirable forests. "The higher observed rates of expansion are probably due to human-caused movement of gypsy moths," they conclude.

Gypsy moths were accidentally introduced into the United States in 1868 by a French naturalist living in Medford, Mass. Today, the bristly caterpillars with red and blue spots live as far south as Virginia and as far west as Michigan. If not halted, the moths will spread southward to Florida and westward to Kansas within 50 years, Liebhold's team predicts. One means to stem the tide, he says, could be a fungal infection that naturally kills the moths. The fungus died off soon after its U.S. introduction in 1910, but has recently begun proliferating in the wild in the Northeast (SN: 8/4/90, p.77).

Biomedicine

Recognizing heart attacks in time

Every year, U.S. hospitals admit more than 4 million people with chest pain, but fewer than one-third of them prove to have heart attacks. When patients with chest pain have inconclusive electrocardiograms and no prior history of cardiac problems, physicians face a dilemma. If the pain *is* a heart attack, quick administration of clot-dissolving drugs could help prevent serious injury to heart tissue, perhaps even saving the patient's life. But these drugs also carry serious complications such as bleeding, and physicians hesitate to use them without a definite diagnosis. Yet confirming a heart attack through further testing takes longer than the six-hour "window" available for effective clot-busting therapy.

New research may soon resolve that dilemma. At a meeting of the American Association for Clinical Chemistry, held late last month in Washington, D.C., scientists described two blood tests that could allow physicians to diagnose heart attacks in time to begin treatment with potentially lifesaving drugs. These tests – modified versions of assays already in use – may become available within the next two years, they say.

Both tests detect substances released by damaged hearts. One measures creatine kinase (CK), an enzyme normally used by the heart to pump blood. During a heart attack, the enzyme leaks into the blood and gets converted to a different form of CK – the same form normally used by other muscle tissues. Therein lies the difficulty. Current tests do not distinguish between CK leaked from the heart and CK leaked from other damaged tissue. For an accurate diagnosis, physicians must monitor CK blood levels over a period of eight hours or more.

Alan H.B. Wu, a pathologist at the University of Texas Medical School in Houston, now reports that small quantities of the still-unconverted cardiac enzyme can be detected in the blood as early as two hours after a heart attack. Specific assays for this CK "isoform" already exist, he says, but scientists need to confirm the diagnostic reliability of those tests.

Hemant Vaidya, a biochemist with the Du Pont Co. in Wilmington, Del., points out that physicians in Europe have long used myoglobin – an oxygen-carrying molecule in muscle tissue – as a marker for heart attack. As in the case of CK, the heart and other muscle tissues all release myoglobin when damaged. However, Vaidya says, Finnish researchers have found that carbonic anhydrase – which helps muscle cells metabolize carbon dioxide – is released only by damaged tissues other than the heart. By monitoring the ratio of myoglobin to carbonic anhydrase, doctors could distinguish between a heart attack and other muscle injuries, obtaining an accurate diagnosis within a few hours of the attack, he says.

CD4 counts and AIDS survival

Last fall, preliminary findings hinted that AIDS patients taking the drug zidovudine (AZT) have a better chance of prolonging their survival if their levels of certain immune cells, called CD4, stay above 50 cells per cubic millimeter of blood (SN: 11/4/89, p.298). A retrospective study of 55 people with AIDS or severe AIDS-related complex (ARC) has now confirmed that finding, reports a research group headed by Robert Yarchoan of the National Cancer Institute in Bethesda, Md. Patients "were very unlikely to die from HIV infection as long as their CD4 counts remained at 50 cells/mm^3 or higher," the team writes in the Aug. 1 Annals of Internal Medicine.

Of the 44 patients who died during the four-year study period, 40 had known CD4 levels below the critical limit within the six months prior to death, the researchers say. They emphasize that a CD4 count below 50 should not be viewed as a sign of imminent death; some patients in the study survived more than three years after their counts dropped below the critical level.