Mite and fungus: Foe *and* friend?

If you were a plant, being attacked by both spider mites and leaf wilt fungus would spell double trouble, right? Not necessarily, according to a recent study of plants' resistance to pathogens.

Prior exposure to one makes a plant twice as likely to ward off attack by the other, say Richard Karban, Rodney Adamchak and William C. Schnathorst from the University of California at Davis, in a report in the Feb. 6 SCIENCE.

Previous research by other scientists has shown that inoculating cotton seedlings with the fungus causing verticillium wilt protected against later fungal attacks. Other experiments found that a similar resistance could be induced against spider mites if there was prior leaf damage by the insect. But the Davis results are evidence that these very different species can provoke an interspecies, systemic resistance in plants.

The data do not dispel the mysteries of plant "immunity," however. "People have assumed that changes [causing resistance] are defenses on the part of the plant," Karban told SCIENCE NEWS. "However, the change need not be an adaptive effect, but simply a detrimental change that affects the plant and the things that attack it. These plant changes may not be anywhere near as [defined] as [animal immune systems]. I really don't have a clue as to how it works."

The possible mechanisms of plant resistance are many and varied. The plant may produce a chemical that directly affects the plant pathogen. For example, alfalfa under attack by insects produces a chemical that inhibits the insects' digestion (SN: 5/25/85, p.327). Or, suggest Karban and his co-workers, the first species attacking the plant "may deplete the most desirable nutrients or plant parts." Another possibility is that pathogens may activate dormant plant metabolites.

Whatever the mechanism, the commercial potential of inducing "immunity" is already blooming. Karban points out that European growers routinely inoculate tomato plants with viruses to elicit resistance and decrease losses.

In recent years, work on induced resistance in plants to herbivores has become "fashionable," Karban says. But, he adds, it also has become controversial: "Most other scientists are skeptical of these sorts of interactions. At this time, there's not a whole lot of scientific evidence that these interactions exist. . . . But their possible consequences, especially if we can manipulate them, would be fantastic."

— D.D. Edwards

Exposing cancer to a 'light' therapy

An out-of-body blood treatment can control cutaneous T-cell lymphoma (CTCL), a potentially deadly cancer, according to an international group of researchers. Because of the treatment's success with this white blood cell cancer, the scientists are now investigating it for other diseases, including AIDS.

In the therapy, a light-activated drug is triggered in blood cells removed from the patient's body; when the cells are returned to the patient, they appear to act as a type of vaccine. In 27 of 37 people with advanced CTCL, the approach cleared up the redness and scaling caused by cancer cells in the skin, report researchers from several U.S. and European institutions in the Feb. 5 NEW ENGLAND JOURNAL OF MEDICINE. One of the researchers, Richard Edelson of Yale University, had previously described positive results in eight patients (SN: 4/13/85, p.229).

The average survival time for people with advanced CTCL is about 30 months. Since most patients studied haven't been on the drug that long, the researchers aren't ready to call the experimental treatment a cure. But they do claim the therapy, called photopheresis, is the best way to deal with advanced CTCL.

Patients receive a series of treatments, starting each treatment by swallowing an inactive form of the drug psoralen, normally used in treating psoriasis. After the blood cells have absorbed the psoralen, blood is drawn and the white cells, including cancerous ones, are isolated. The rest of the blood goes back into the body; the white cells are exposed to ultraviolet light. The light activates the psoralen, which lethally damages the cells. The cells are then injected into the patient.

The benefits of photopheresis are evidently due not to the immediate damage to the treated cells — only 10 to 15 percent of the white cells are dealt with per therapy session — but to vaccination. The dying cells, when reinfused into the body, set off an immune-system reaction against other cancer cells.

Photopheresis may also be useful in dealing with autoimmune diseases, where white blood cells mistakenly attack the body. The researchers have just begun a trial against pemphigus, a rare autoimmune disease.

Bruce Wintroub, who was involved in the CTCL study, is also part of an effort to determine whether photopheresis has any value in treating AIDS. He and his coworkers at the University of California at San Francisco have found that in the laboratory, photopheresis somehow inactivates the AIDS virus in human white blood cells. Whether the approach will work in people with AIDS remains to be determined.

— J. Silberner

Double (whammy) pneumonia

Having viral pneumonia is a serious matter, but a combined viral-bacterial pneumonia is considered more threatening, with a fatality rate of up to 42 percent. Searching for an explanation of this double-agent danger, researchers in West Germany have discovered that the bacteria may be giving the viral agent a deadly boost in virulence.

Scientists have known that adding the enzyme trypsin can reactivate viruses that have become noninfectious. Now the West German group has found that an enzyme isolated from culture medium used to grow some types of *Staphylococcus aureus* bacteria apparently activates several influenza virus strains. Although the bacteria alone are relatively harmless, staphylococcus infection is often found in influenza pneumonia.

When the viruses are treated *in vitro* with the *S. aureus* enzyme, the infectivity is "enhanced at least 100-fold," according to a report in the Feb. 5 NATURE by scientists at the Institute of Virology at the Justus Liebig University in Giessen and the Institute of Hygiene at the University of Köln. The viruses also are stimulated to undergo multiple growth cycles when the bacterial enzyme is present. This, say the researchers, could explain how the virus spreads in the lungs so rapidly.

Experiments suggest that *S. aureus* can cleave or cut a protein called hemagglutinin on the viral surface, which could contribute to the observed activation of the virus. The hemagglutinins of viral strains whose infectivity was not activated by the bacterial enzyme were not cleaved by the bacterial enzyme.

Co-infections in laboratory mice using strains of influenza virus and *S. aureus* usually meant death within five days, whereas animals infected with only one of the disease agents survived.

— D.D. Edwards

Water veto overridden

On Jan. 20, President Reagan vetoed a $20 billion bill to reauthorize the Clean Water Act (SN: 1/31/87, p.71), calling the legislation too expensive. But last week, Congress passed the bill into law by overriding the veto. The House override came on Feb. 3, and the Senate came a day later.

Though 90 percent of the bill's funding will go to finance sewage treatment, the legislation does provide $400 million for a new program to help states control toxic chemical runoff from nonindustrial sites and $30 million to help clean up the Chesapeake Bay. □