Plant's genetic 'master switch' subverted

Minutes after a water-starved plant begins wilting, concentrations of a natural hormone soar to 10 times their previous levels. The dramatic rise in the hormone, known as abscisic acid or ABA, triggers a cascade of chemical changes that helps the plant resist drought and disease.

Investigators have also found that ABA influences a plant's early maturation, preventing vulnerable seeds from germinating too rapidly if drought conditions prevail. But until recently, biologists knew little about how the hormone worked on the molecular level.

Researchers have now identified a genetic sequence that appears to act as a "master switch" turning on several genes in response to ABA. The work suggests that by placing copies of the switch near plant genes not normally activated by the hormone, scientists might create plants in which ABA triggers the production of a wide variety of protective proteins. In addition, studies of the switch may provide clues to how other plant hormones function on the molecular level.

Molecular biologist Ralph Quatrano began the work by soaking wheat embryos (the source of wheat germ) in a solution of ABA. He found that the drenched embryos responded by accelerating production of two normal proteins — a storage protein known as triticin and a compound known as Em, which may play a role in drought resistance. Quatrano, then at Oregon State University in Corvallis, documented that ABA had two main effects on the genes encoding these proteins. The hormone increased the transcription of DNA into messenger RNA (mRNA) — a key step in protein production — and it stabilized the normally fragile mRNA produced. ABA's dual role may explain its ability to activate several genes in both mature plants and embryos, Quatrano says.

Working at the University of North Carolina at Chapel Hill with William Marcotte and Mark Guiltinan, Quatrano went on to identify and examine a genetic sequence within the gene encoding the Em protein in wheat embryos. When this sequence, known as the Em promoter, senses high levels of ABA, it activates a chain of commands that switches on the Em gene to produce the Em protein, he says.

Recently, Quatrano and his colleagues isolated a 76-base-pair fragment from the sequence, which has a total of 1,800 base pairs. That fragment contains the active Em switching site common to both wheat and rice plants, he reported this week in Richmond, Va., at the annual meeting of the American Institute of Biological Sciences.

The team also discovered several tantalizing hints that genetic engineering might someday enhance ABA's influence on plant development. Quatrano and his co-workers developed a rapid *in vitro* assay, which showed that the Em promoter, in the presence of ABA, could activate a bacterial gene placed near it — genetic material not normally controlled by the hormone. Moreover, in searching through mRNA derived from wheat embryo, they identified several DNA segments coding for proteins that appear to interact with the 76-base, ABA-sensitive fragment of the Em promoter.

Quatrano suggests that genetic engineers might insert the Em promoter near several plant genes to bring them under the control of naturally occurring or even artificially produced ABA.

Marcotte adds that the group's results complement other researchers' findings that similar master switches influence the response to other hormones — including ethylene, which promotes ripening, and gibberellins, which affect growth. But because ABA can occur in high concentrations in plant embryos and can protect both immature and adult plants from sudden environmental changes, he says, its switch may have special significance for strengthening crops. *— R. Cowen*

Home carpets: Shoeing in toxic pollution

Turkish hosts welcome houseguests with a pair of slippers. While it's a pleasant gesture of hospitality, the practice has a pragmatic purpose: to keep visitors from tracking dirt onto carpets.

Others might do well to adopt this custom, especially if they're parents of crawling youngsters. Two studies, described in Toronto last week at the Fifth International Conference on Indoor Air Quality and Climate, show that home carpeting can become a dramatic reservoir of toxic pollution — much of it apparently tracked in from outdoors.

Seattle consulting engineer John W. Roberts and his colleagues analyzed dust vacuumed from living-room carpets in 40 homes built in the Seattle area before 1950. Typical lead levels in the dust far exceeded levels requiring cleanup at Superfund sites, they report.

However, in five homes where family members habitually removed their shoes at the door, the team found that the geometric mean (logarithmically derived average) concentration of lead was a mere 240 micrograms per square meter ($\mu g/m^2$) of carpet — less than 10 percent of the 2,900 $\mu g/m^2$ mean in homes where shoes were worn. And in six apartments requiring entry through a long, carpeted exterior hallway, the mean living-room carpet level was 440 $\mu g/m^2$.

Lead levels in carpet dust "strongly correlated" with those in the soil just outside, suggesting that soil represents "a major source" of indoor lead contamination, Roberts says. Because high lead levels around a home's foundation tend to reflect the pre-1950 use of heavily leaded paint, older homes carry a higher risk of tracked-in lead problems. Remodeling those homes, however, can pose an acute and even more serious indoor hazard by freeing lead from indoor paint and plumbing. The researchers found mean carpet-lead levels exceeding 12,600 $\mu g/m^2$ in the nine homes remodeled during the year preceding the study.

Nor is lead the only threat. Worrisome pesticide levels have appeared in hundreds of homes studied by David E. Camann of the Southwest Research Institute in San Antonio, Texas. In a new study of one home receiving quarterly professional pest-control treatments — both inside and out — Camann and his colleagues traced residues of many pesticides in the air and carpets primarily to contaminated soil tracked in on shoes. Dust vacuumed from the living-room carpet contained 16 pesticides, including eight of the 10 detected in the room's air. Camann says "about half" the pesticides detected in the 10-day sampling of the home's air — including the once-popular chlordane, heptachlor, dieldrin and DDT — showed rising concentrations as sampling progressed from the carpet to the doormat and doorstep, and finally to the garden. This strongly implicates tracked-in soil as the source, the researchers conclude.

Camann says the lack of a similar carpet-to-soil gradient for the pesticides chlorpyrifos, diazinon, ortho-phenylphenol and propoxur suggests they arose from indoor treatment, with carpets acting as "a significant reservoir."

Roberts, who has made a career of measuring and analyzing house dust, had previously shown that rugs can hold up to 100 times as much of the fine debris as a bare floor. The higher a carpet's pile, he says, the harder it is to pull out that dust, although vacuums with powered carpet beaters can remove up to five times as much dust as suction-only devices.

But outdoor sources can replenish indoor reservoirs for years. In the November 1989 BULLETIN OF ENVIRONMENTAL CONTAMINATION AND TOXICOLOGY, Roberts and Camann reported detecting a toxic wood preservative in the rugs of two of four sampled homes, although the homes' outdoor decks hadn't been treated for three to five years. The team also detected chlordane, dieldrin and DDT, "even though no homeowner could remember using such chemicals" and despite DDT's removal from the consumer market in 1972. *— J. Raloff*