napus, known as oilseed rape or canola. Farmers grow this mustard for its edible oil. The crop species hybridizes readily with wild mustards, including one of its ancestors, *Brassica rapa.*

To track any wandering of chloroplast genes, the researchers checked 47 cropweed hybrids found near commercial fields. All hybrids showed the weed, or maternal, chloroplast DNA. That convinced Wilkinson that pollen wafting from fields does not carry chloroplast DNA.

In practical terms, errant crop pollen doesn't have that many places to go, Wilkinson notes. He and Scott found that of more than 140 patches of wild *B. rapa* in farmland, only 2 grew near oilseed rape fields.

In such patches, just 0.4 to 1.5 percent of the plants have mixed parentage, he and Scott reported last year. All in all, there will probably be "no or negligible" escape of chloroplast genes through crop pollen, he predicts.

The researchers also considered the other escape avenue: the female flower parts. If crop seeds spill near wild plants, the resulting plants may be pollinated by weed species to create hybrids carrying the modified chloroplast. In another generation or two, the wayward genes could get into highly fertile wild plants.

However, when a crop plant gets loose, "it doesn't last very long," Wilkinson says. He and Scott monitored 18 patches harboring crop plants that had gone wild. Fifteen of the patches disappeared or failed to set seed during the 3-year study.

Hybridization is "inevitable but will occur only extremely rarely," Wilkinson says. "It all comes down to what the transgene actually is." A transplanted gene that gives a plant whopping advantages in the wild might spread even through a tiny keyhole escape avenue.

"You have to look at each plant on a plant-by-plant basis," agrees Dean Chamberlain of the University of North Carolina (UNC) in Greensboro. In a commentary in the same issue of NATURE BIOTECHNOLOGY, he and UNC's C. Neal Stewart Jr. note that chloroplast genes are so difficult to work with that, to date, only tobacco has been transformed in this way.

Just wait, responds Daniell. He expects several researchers soon to announce transfers of chloroplast genes.

The chloroplast strategy is still no panacea, warns Joseph E. Cummins of the University of Western Ontario in London, Ontario. Chloroplasts are passed on through pollen in many conifers and through both parental lines in alfalfa. Also, Cummins points out that chloroplast DNA can leak into mitochondria, a cell structure that does show up in oilseed rape pollen.

Wilkinson speculates that both fans and foes of genetically modified crops will quote the new paper as supporting evidence. "To us, it's just data," he sighs. —*S. Milius*

Stopping leaks may boost cancer drugs

Almost every medicine produces side effects. The crucial issue is whether a drug has a therapeutic window, a dose range that allays a patient's illness without causing greater problems.

In a finding that may widen the therapeutic windows of two experimental cancer medicines, researchers have uncovered the molecular explanation for a side effect—leaky blood vessels—that both therapies cause. Known as vascular leak syndrome, the condition occurs when fluid from the bloodstream escapes into surrounding tissues.

"You sort of become a water balloon," says Ellen S. Vitetta of the University of Texas Southwestern Medical Center at Dallas. While a body can often slowly expel this excess water, fluid buildup in organs such as lungs can turn deadly.

Vitetta and her colleagues encountered vascular leak syndrome when they began testing immunotoxins in cancer patients. These artificial proteins consist of a plant or bacterial toxin attached to antibodies that home in on cancer cells.

The immunotoxins have lived up to their billing as cancer killers, but they also trigger changes in cells lining blood vessels. The cells become rounder than normal, leaving gaps through which fluid could seep out. The problem limits the amount of immunotoxins people can receive as a treatment.

"This has stalled the field a great deal," says immunotoxin investigator Daniel A. Vallera of the University of Minnesota Cancer Center in Minneapolis.

"You don't have a wide therapeutic window, because you hit this toxicity," agrees Christopher A. Pennell, also of the University of Minnesota Cancer Center.

Like the immunotoxins, interleukin-2, a protein that stimulates the immune system's cells, causes vascular leaks at high doses. The side effect has frequently thwarted its use in people with cancer and, more recently, AIDS.

Speculating that immunotoxins and interleukin-2 generate leaky blood vessels in the same way, Vitetta's team compared the proteins. "You line up the [amino acid] sequences and ask if there's a consensus sequence. Lo and behold, out came this motif," says Vitetta. All the molecules share a particular combination of three amino acids, her group reports in the March 30 PROCEEDINGS OF THE NATIONAL ACADEMY OF SCIENCES.

The researchers also made protein fragments containing this motif but no other parts of interleukin-2 or the immunotoxins. Injected into animals, those segments caused leaky blood vessels. "You don't need the rest of the molecules," says Vitetta. "You just need this tiny, little piece."

The scientists are now trying to eliminate this dangerous motif by mutating the genes that encode the immunotoxins. They expect that the modified immunotoxins will retain their cancer-killing prowess but leave blood vessels alone.

Making interleukin-2 safer may prove more difficult since the motif falls in a region crucial to the protein's therapeutic function. Investigators could instead try to block the proteins on blood vessels that the immunotoxins and interleukin-2 bind, Vitetta notes.

"She's putting together a really nice story," says Pennell. —*J. Travis*

Big dust, little harm

Dust storms are blowing away the argument that eroded soil and other relatively large, airborne particles are as hazardous to health as the far smaller particles generated by combustion.

Over the past decade, a host of studies has linked the outdoor buildup of combustion particles to a rise in hospital admissions and death rates for respiratory illness (SN: 4/6/91, p. 212) and heart disease (SN: 7/1/95, p. 5). Such data convinced the Environmental Protection Agency to create new limits (SN: 7/5/97, p. 6)—not yet in effect—for particles that measure 2.5 micrometers (μm) in diameter or smaller (termed PM-2.5). Federal rules already limit a broader class of particles, those with diameters of up to 10 μm (PM-10).

Representatives of combustion-intensive industries say that errors in measuring large particles have made the relative health impacts of large and small particles hard to distinguish. Thus, they have argued against rules focusing on PM-2.5, notes Joel Schwartz of the Harvard School of Public Health in Boston.

"It's a big fight," he explains, and it has threatened to derail implementation of the PM-2.5 limits. Hoping to settle the controversy, Schwartz teamed up with researchers from two universities in Washington State to study Spokane death rates during 17 major dust storms over 6 years. The average PM-10 concentration on storm days was 263 micrograms per cubic meter ($\mu g/m^3$) of air—well above the current EPA limit. On the stormless days, PM-10 averaged only 42 $\mu g/m^3$. During dust storms, PM-10 consists primarily of particles larger than 2.5 μm in diameter, but the amount of fine particles present changes relatively little with weather.

Nonaccidental death rates were slightly lower during the dust storms than on calm days of the same date during the study, the researchers report in the May ENVIRONMENTAL HEALTH PERSPECTIVES. These data, they contend, argue against the industry position and confirm results of other studies "that toxicity of coarse particles is substantially less than that of fine particles." —*J. Raloff*