Speciation's roots?

About four years ago, Janne Lempe, then a graduate student at the Max Planck Institute for Developmental Biology in Tübingen, was hard at work on a series of experiments on the genetic regulation of flowering time in Arabidopsis thaliana. She crossed two strains, chosen for their difference in flowering time, and set off to the greenhouse to leave the seeds to grow.

Researchers generally raise Arabidopsis at 23° C, but with the lab’s main greenhouse full, Lempe plunked her experiment into the 16° C greenhouse next door. A few weeks on, her seedlings had produced stunted, stumpy, deformed versions of the hearty white-flowered weeds she was expecting. Her plants were not only unusually small, but their leaves were also covered in necrotic brown spots, and they’d failed to flower.

ARTICLE EXTRAS

Slideshow: Crossing Arabidopsis Strains

Lempe’s two starting strains, UK1 and UK3, were perfectly normal. So why would their offspring be sick? Right away, says Detlef Weigel, an evolutionary geneticist who supervised Lempe’s doctoral research, “I thought this might have some bearing on speciation.” Perhaps genes from one strain were incompatible with those in the other, creating in the offspring a gene-flow barrier, which is the first step to the evolution of a new species. Researchers have mapped genes in Drosophila that keep two species apart, but such studies can’t resolve whether genetic incompatibilities drove speciation, or instead accumulated after the species diverged. Weigel wondered whether Lempe’s sick hybrids might shine light on the process from the other side, by showing how such incompatibilities could arise in a single species.

Lempe was surprised to find that her plants recovered, and even flowered, when she
transferred them from the cooler greenhouse to the warmer one. (It's unclear why, but it's not unusual for growth effects to be temperature sensitive.) So she took the seeds and established a homozygous strain from the sick crosses. The results suggested that the interaction of two genes, an allele from each parent strain, had caused the necrosis in the plants.

When Weigel showed pictures of stunted UK1/UK3 crosses to Jeff Dangl, a plant immunologist at the University of North Carolina, Chapel Hill, Dangl pegged the problem at a single glance. "I said, 'that's R-genes,'" he recalls. R-genes, one of the cornerstones of plant immunity, are activated in response to specific pathogens; he had seen similarly necrotic specimens in which these genes were overexpressed. Lempe's microarray studies had shown that most of the differentially expressed genes in the crosses were involved in pathogen resistance and immunity. The duo plotted a collaboration.

Dangl's instinct proved correct. Kirsten Bomblies, a postdoc who joined Weigel's lab a few months on, mapped one of the incompatible alleles to an R-gene ( PLoS Biol, 5:1962-72, 2007). The second allele is still mysterious, but Bomblies suspects that it's an R-gene, since several genes on the affected locus are R-genes; unpublished work on other necrotic crosses has also pointed to immune genes. Moreover, the hybrid plants are more resistant than normal to pathogens, suggesting that a revved up immune system - the plant world's version of autoimmunity - was causing the necrotic phenotype.

Indeed, the literature on crop breeding is replete with descriptions of two normal strains producing a stunted hybrid. In a paper published in 1929, a researcher suggested that the sickness looked like an immune response, proposing that plants must therefore have circulating antibodies. His idea was ridiculed, and it was another 30 years before breeders began again noting a possible link between hybrid necrosis and a pathogen response, Bomblies says. The key now, she notes, is to track the effect in the wild and in other species. So far she has performed 500 or so hybrid crosses from Arabidopsis strains collected throughout Europe; about 2% of those crosses are necrotic. "As far as I know," she says wryly, "it's the biggest intercrossing scheme of natural strains in Arabidopsis."

Weigel is quick to point out that the researchers may not be witnessing a speciation event. "We cannot predict that these things are going to turn into new species; in fact, it's very unlikely," he says, adding that there must be many cases in which the process starts but is never completed. Still, he insists, "it's important as a model."

"I think that's fair," says John Willis, an evolutionary geneticist at Duke University who works on Mimulus, commonly called monkey-flower. What's interesting, he notes, is that the effect seems to have evolved "as a byproduct of adaptive natural selection acting on these genes for completely other purposes." His group has found sterile hybrids of Mimulus strains, and he and Bomblies recently discussed plans to study the genetics.

"A lot of people have very strong feelings on how this is going to play out," says Weigel. "I just say, 'I don't know, we'll see.'"