

Herbicide resistance no longer a black box for scientists

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Pat Tranel, pictured along with millions of waterhemp seeds, is getting closer to determining the genetic architecture of herbicide resistance in the problematic weed species. Credit: Fred Zwicky

When agricultural weeds evolve resistance to herbicides, they do it in one of two ways. In target-site resistance, a tiny mutation in the plant's genetic code means the chemical no longer fits in the protein it's designed to attack. In non-target-site resistance, the plant deploys a whole slew of enzymes that detoxify the chemical before it can cause

harm.

Target-site resistance is easy for scientists. They know what the [target protein](#) is, which means they can look directly at the [genetic code](#) to figure out the mutation responsible. But for non-target-site resistance, it's a guessing game. Researchers can sometimes tell what class of enzymes detoxifies the chemical, but they know next to nothing about what [genes](#) code for those enzymes. In other words, non-target-site resistance is a black box.

A University of Illinois study is the first to open that box in a new way, identifying gene regions responsible for non-target-site [herbicide](#) resistance in waterhemp.

"We used a genetic mapping approach with the [reference genome](#) for waterhemp, a species that can cause yield losses upwards of 70% in corn and is resistant to seven herbicide modes of action," says Pat Tranel, professor and associate head in the Department of Crop Sciences at the University of Illinois and co-author on the study. "We were able to narrow it down to two regions of the genome, or about 60 genes."

Being able to pinpoint the genes for non-target-site resistance could enable tools for early detection and herbicide management.

"We eventually want to develop an assay farmers can use to tell if the waterhemp in their field is resistant to a given chemical, either to confirm why a previous application did not work or before they spray to see if they're going to waste money," says Brent Murphy, doctoral researcher and lead author on the study. "These genomic assays exist for target-site resistance because we know the region of the genome where those mutations are, but for non-target-site resistance, we have had no idea where to look until now."

"Now we know the genes responsible are somewhere in these two small regions of the genome. So we've come to an intermediary step to eventually developing an assay that growers can use to determine whether or not they should be spraying a certain chemistry."

The researchers specifically looked for genes that allow waterhemp to evade HPPD-inhibiting herbicides such as tembotrione, a chemical commonly applied in seed corn and other production systems.

To find the genomic regions responsible, they mated waterhemp plants that showed resistance or sensitivity to HPPD inhibitors. Then they exposed the grandchildren of those parent plants to HPPD-inhibiting herbicides to see how they fared. Because the complete waterhemp genome is now available, they were able to look for commonalities among plants that survived the HPPD-inhibitor application.

"You basically ask the question, for the [resistant plants](#), what part of their genome do they share in common? And that gets you to what part of the genome is controlling the trait of interest. Using this approach, known as genetic mapping, we identified two regions of the genome that seemed to be associated with resistance," Tranel says.

Murphy was able to determine which plants had each of the two regions, and which had both. This allowed him to rank the importance of the [gene regions](#).

"A lot of times, we know a trait is controlled by two genes. But does that mean both genes are equally important, or one gene is 90% responsible, and the other gene is 10%? That's part of what we're looking at in the genetic architecture of a trait: the number of genes, where they are, and the relative importance of these different genes," Tranel says. "Here, we saw a nice stepwise effect. If you had one of the regions, you were kind of resistant. If you had the other one, you were kind of resistant. If you

have them both? You're pretty resistant. Essentially like the resistant parent."

While the researchers still don't know which of the 60-ish genes are essential to HPPD-inhibitor resistance – they have follow-up studies in mind to narrow the search even farther – they know none of the genes encode [p450 enzymes](#). These have been implicated in multiple studies as key players in non-target-site resistance.

"While a p450 enzyme might still be involved, our mapping study indicates the change causing resistance is in a gene regulating the p450, rather than in the p450 gene itself," Tranel explains.

HPPD-inhibitors are commonly used in seed corn and other maize production systems, but, interestingly, they hadn't been used in the field where the researchers collected the waterhemp for the study.

"There wasn't a previous field-use history of this chemistry. So, it was really interesting to see that our population was resistant to it. How did this develop? Most of the time you expect resistance to develop as a result of some form of selection pressure. But here, we don't have an obvious one," Murphy says.

Tranel thinks non-target-site resistance to one class of herbicides might confer cross-resistance to other classes. The population in the study was resistant to 2,4-D, a herbicide from another class that might have triggered resistance to HPPD-inhibitors.

"Why do we have this plant that's resistant to multiple herbicides? Are there some genomic changes in common to facilitate that resistance? It's really important to understand this, as we try to give farmers advice about what can they do to mitigate non-target-site resistance, because it's still a bit of a black box," Tranel says. "With target site resistance, we

can tell them to use herbicides with different modes of action. But in non-target-site resistance, different herbicides could be metabolized by, for example, different p450s that are regulated in the same way. That's why we need to unravel this further to come up with better, more informed strategies to mitigate non-target-site resistance."

Tranel expects that, as more weed genomes become available, genetic mapping will become a mainstay for investigating non-target-site resistance.

"Finally, we are getting the tools we need to really get to the bottom of metabolic herbicide resistance, which is the greatest threat to contemporary weed management," he says.

The article, "Genetic architecture underlying HPPD-inhibitor resistance in a Nebraska *Amaranthus tuberculatus* population," is published in *Pest Management Science*.

More information: Brent P Murphy et al, Genetic architecture underlying HPPD -inhibitor resistance in a Nebraska *Amaranthus tuberculatus* population, *Pest Management Science* (2021). [DOI: 10.1002/ps.6560](https://doi.org/10.1002/ps.6560)

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