



# Vegetable Crop Update

*A newsletter for commercial potato and vegetable growers prepared by the University of Wisconsin-Madison vegetable research and extension specialists*

**No. 21 – September 5, 2022**

## ***In This Issue:***

- Metabolic herbicide resistance
- Potato disease risk and management recommendations for early blight and late blight
- Cucurbit downy mildew
- Tar spot on sweet corn

## ***Calendar of Events:***

**November 29-December 1, 2022** – Midwest Food Producers Assoc. Processing Crops Conference, Kalahari Convention Center  
**January 29-31, 2023** – Wisconsin Fresh Fruit and Vegetable Growers Conference, Kalahari Resort, Wisconsin Dells, WI  
**February 7-9, 2023** – UW-Madison Div. of Extension & WPVGA Grower Education Conference & Industry Show, Stevens Point, WI

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**Is metabolic herbicide resistance the straw that will break weed management's back?** In a long-term tillage research project in Kansas, a Palmer amaranth population was identified that was resistant to six herbicide sites of action in individual plants. While that's challenging enough, here's the scariest part: in some cases the plants had evolved resistance to herbicides that had never been sprayed in the field (Shyam et al. 2021).

Similarly, in Illinois a waterhemp population was recently identified that's resistant to dicamba, yet the field had never been treated with dicamba or 2,4-D. The population was also resistant to five other herbicide sites of action, which may have been the source of resistance to the sixth herbicide site of action that includes dicamba (see <https://aces.illinois.edu/news/first-dicamba-resistant-waterhemp-reported-illinois> for an informative summary of this work).

Weeds that have become resistant to herbicides they've never been sprayed with may sound like something out of a CSI type show. The phenomenon is not new but is becoming more common. In fact, one of the potential causes – metabolic resistance – isn't even limited to plants. So how could this happen?

In a broad sense, herbicide resistant weeds can be divided in two groups: those with target site resistance, and those with non-target site resistance. In target site resistant weeds, the specific enzyme that the herbicide targets is either mutated so that the herbicide can't bind to it (think of pieces of a puzzle not fitting together) or the target enzyme is overproduced to the point that the herbicide can't effectively bind to all the sites.

Non-target site resistance can happen in a few ways: in resistant weeds the herbicide may not be absorbed or translocated (moved within the plant) as well, the herbicide may get sequestered in plant parts away from the target site, or the herbicide may get metabolized by the plant. The remainder of this article will focus on metabolic herbicide resistance because it likely has the greatest implications for production agriculture.

Herbicide metabolism involves the breakdown of the active ingredient into metabolites that are less mobile and less toxic to the plant, and then the "dumping" of the metabolites into plant parts where they are sequestered and not active. Enzymes cause the breakdown, and two of the most involved include cytochrome P450 monooxygenase (P450) and glutathione S-transferase (GST). P450s are among the most common enzymes in living organisms and have the ability to metabolize 11 of the 26 herbicide modes of action. GSTs are also common in living organisms and are responsible for some grass tolerance to herbicides and some observed cases

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of insecticide resistance (Rigon et al. 2020). Crop safety with many herbicides is based on metabolism by these broad enzymatic families.

Herbicide metabolism has been researched and observed over the past few decades with challenging grass weeds such as rigid ryegrass in Australian wheat production (Yu and Powles 2014). More recently, however, metabolic resistance has been reported among broadleaf weeds and close to home. For example, metabolic resistance to the herbicide S-metolachlor was reported in two waterhemp populations in Illinois (Strom et al. 2020). In this case the resistant waterhemp metabolized 90% of the S-metolachlor in less than 3.2 hours.

Metabolic resistance has sometimes been referred to as “creeping resistance” because of the way that it evolves in populations, where plants that can survive low herbicide doses by metabolizing some of the active ingredient produce seed, and subsequent generations are selected that can metabolize more and more herbicide until they are no longer useful for control. For example, waterhemp control with dicamba in the Illinois population noted above decreased from 80% to 65% over just a few years, and dicamba wasn’t even sprayed during that time.

So why is metabolic resistance so concerning compared to target site resistance that’s been addressed for years? Target site resistance is very specific to an herbicide active ingredient, the individual target site that it binds to, and a mutation that changes those puzzle pieces. In contrast, in metabolic resistance the enzymatic activity that breaks down the herbicides and other toxins is not specific. Once high metabolic activity is selected for, the plant can breakdown a broad range of herbicides across modes of action, potentially including active ingredients that have never been sprayed on that population before, and even herbicides that have yet to be discovered. For example, in the Kansas study mentioned above, the authors concluded that “these results suggest predominance of metabolic resistance possibly mediated by cytochrome P450 and GST enzyme activity that may have predisposed the KCTR Palmer amaranth population to evolve resistance to multiple herbicides” (Shyam et al. 2021). In practical terms, metabolic resistance adds tremendous unpredictability to weed management decision making and outcomes.

These metabolic enzymatic activities are also not specific to plants and herbicides, which makes for complex resistance scenarios. For example, Clements et al. (2018) reported that some of the fungicides commonly used for potato disease control can upregulate GST enzyme production in Colorado potato beetles, and that increase in enzymatic activity can negatively affect insecticide performance.

Additionally, not only is metabolic resistance more challenging to research than target site resistance, it’s also harder to observe in the field. For years growers and scouts have been told to keep an eye out for living target plants that normally would have been killed and that are among other dead weeds, and that stark contrast of living versus dead was often the smoking gun of resistance. In metabolic resistance, the selection pressure creeps along where target weeds may be injured but eventually recover enough to produce a few viable seeds, and the high metabolism selection cycle continues on until multiple herbicides are ineffective.

The increase in likely cases of metabolic resistance observations in recent years speaks to the dire need to develop practical and economical alternatives to herbicides – it’s simply not just about rotating herbicides anymore. In the short term, much effort is currently being directed to intervening in the seed production and dispersal step of the resistance selection cycle with mechanical tools like combine weed seed cleaners and collectors. Research is also underway to gain a better understanding of the complex metabolic interactions among pesticides and pests, and how that affects practical management decisions. In the longer term, alternative technologies like weed sensors and highly efficient robotic weeders need to be developed and available for adoption in reasonable and affordable ways.

For the details:

Clements J, Schoville S, Clements A, Armezian D, Davis T, Sanchez-Sedillo B, Bradfield C, Groves RL (2018) Agricultural fungicides inadvertently influence the fitness of Colorado potato beetles, *Leptinotarsa decemlineata*, and their susceptibility to insecticides. *Nature* 8:13282 (doi: 10.1038/s41598-018-31663-4)

Rigon CAG, Gaines TA, Kupper A, Dayan FE (2020) Metabolism-based herbicide resistance, the major threat among the non-target site resistance mechanisms. *Outlooks on Pest Mgt.* 31:164-168 (doi: 10.1564/v31\_aug\_04)

Strom SA, Hager AG, Seiter NJ, Davis AS, Riechers DE (2020) Metabolic resistance to S-metolachlor in two waterhemp (*Amaranthus tuberculatus*) populations from Illinois, USA. *Pest Mgt. Sci.* 76:3139-3148 (doi: 10.1002/ps.5868)

Shyam C, Borgato E, Peterson D, Dille JA, Jugulam M (2021) Predominance of metabolic resistance in a six-way-resistant Palmer Amaranth (*Amaranthus palmeri*) population. *Frontiers in Plant Sci.* 11:614618 (doi: 10.3389/fpls.2020.614618)

Yu Q, Powles S (2014) Metabolism-based herbicide resistance and cross-resistance in crop weeds: A threat to herbicide sustainability and global crop production. *Plant Phys.* 166:1106-1118

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**Current P-Day (Early Blight) and Disease Severity Value (Late Blight) Accumulations.** Thanks to Ben Bradford, UW-Madison Entomology; Stephen Jordan, UW-Madison Plant Pathology; and our grower collaborator weather station hosts for supporting this disease management effort in 2022. A Potato Physiological Day or P-Day value of  $\geq 300$  indicates the threshold for early blight risk and triggers preventative fungicide application. A Disease Severity Value or DSV of  $\geq 18$  indicates the threshold for late blight risk and triggers preventative fungicide application. Red text in table indicates threshold has been met or surpassed. Weather data used in these calculations will come from weather stations that are placed in potato fields in each of the four locations, as available. Data from an alternative modeling source: <https://agweather.cals.wisc.edu/vdifn> will be used to supplement as needed. Data are available for each weather station at: <https://vegpath.plantpath.wisc.edu/dsv/>.

Location	Planting Date		50% Emergence Date	Disease Severity Values (DSVs) 9/4/2022	Potato Physiological Days (P-Days) 9/4/2022
Grand Marsh	Early	Apr 5	May 10	72	875
	Mid	Apr 20	May 15	72	834
	Late	May 12	May 25	72	776
Hancock	Early	Apr 7	May 12	44	860
	Mid	Apr 22	May 17	44	840
	Late	May 14	May 26	44	781
Plover	Early	Apr 7	May 15	114	815
	Mid	Apr 24	May 20	114	781
	Late	May 18	May 27	113	746
Antigo	Early	May 1	Jun 3	49	693
	Mid	May 15	June 15	45	619
	Late	June 10	June 24	45	534

In addition to the potato field weather stations, we have the UW Vegetable Disease and Insect Forecasting Network tool to explore P-Days and DSVs across the state (<https://agweather.cals.wisc.edu/vdifn>). This tool utilizes NOAA weather data (stations are not situated within potato fields). In using this tool, be sure to enter your model selections and parameters, then hit the blue submit button at the bottom of the parameter boxes.

Accumulations of P-Days were high (~61 in most locations) over the past week. Potatoes should continue to be on a preventative fungicide program with effective disease management selections to limit early blight in long-season potatoes.

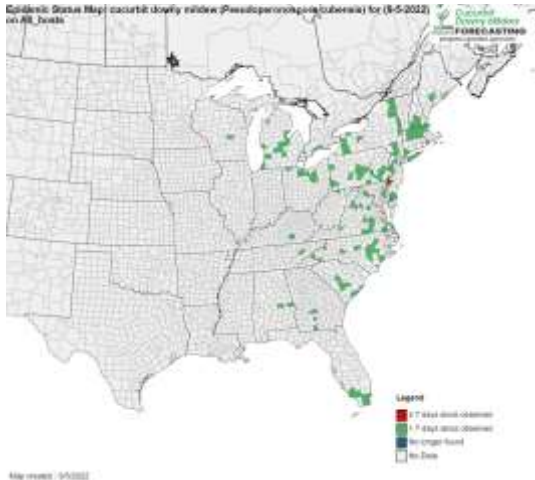
Our UW Hancock Agricultural Research Station foliar early blight management trial on Russet Burbank potato has reached the point where our non-treated controls are completely senesced (pic below on the left). Some of the 'best' foliar fungicide programs are maintaining canopy at roughly 50% coverage (pic below on the right). This particular treatment includes Bravo WeatherStik and Miravis Prime in a 10-week foliar fungicide program. We have one final foliar application in this trial which will be harvest in early October.



All monitored Wisconsin locations accumulated 0 DSVs this past week indicating low to no risk week for promoting late blight in potato plantings in Grand Marsh, Hancock, Plover, and Antigo. All plantings have now reached/exceeded the threshold for receiving a preventative application of fungicide for the management of late blight. A fungicide list for potato late blight in Wisconsin was provided in last week's newsletter and is available here: <https://vegpath.plantpath.wisc.edu/2022/07/03/update-10-july-3-2022/>

To my knowledge, there have been no reports of late blight in Wisconsin on potato or tomato so far this season. According to [usablight.org](https://usablight.org) there were also no diagnoses of late blight in the US in the past week. Previous diagnoses in the US this season included those in NC, FL, CA, TN, and Ontario Canada. These have been primarily on tomato, with only the FL report on potato in early spring.

**Cucurbit Downy Mildew:** During this past week, cucurbit downy mildew was confirmed on summer squash and cucumber in New Jersey. Previously this growing season, the disease was confirmed in AL, CT, DE, FL, GA, KY, MA, MD, ME, MI, NC, NH, NJ, NY, OH, PA, SC, VA, and WI. The first report in WI had come from Waushara County (UW Hancock ARS) on Aug 15. Red counties, on the figure below, indicate recent reports (less than 1 week old) of cucurbit downy mildew.



<https://cdm.ipmpipe.org/>

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HIGH Risk for cucurbits in southern and western GA, eastern and northern AL, the NC mountains, east-central and eastern TN, central and eastern KY, extreme southwest VA, WV except the northeast, northeast OH, western PA, southern ON, western NY. Moderate Risk for southeast MI, northwest OH, central and eastern NY, central and southern VT and NH, southwest ME, MA / CT / RI, Long Island, northern NJ, central and eastern PA, northeast WV, western VA, western NC except the mountains, western and southern SC, eastern GA, and southern FL. Low Risk to cucurbits in eastern SC, southeast and south-central NC, southwest MI, and northern IN. Minimal Risk elsewhere.

As a reminder, the pathogen is now known to have two ‘strains’ for clade types. The type (Clade 2) which infects cucumber, can also infect melon. Due to fungicide resistance within the downy mildew pathogen population, especially in Clade 2, selection of fungicides is important. Management of cucurbit downy mildew requires preventative fungicide applications as commercial cultivars are generally susceptible to current strains (Clades) of the pathogen. Management information can be sourced here:

<https://vegpath.plantpath.wisc.edu/2022/07/03/update-10-july-3-2022/>

**Tar spot of sweet corn** has been diagnosed in Waushara County in our Midwest Food Products Association-partnered sweet corn variety trials at the end of August. Dr. Damon Smith, UW Field Crops Extension Pathologist had previously diagnosed this emerging disease on field corn in early July in Wisconsin <https://ipcm.wisc.edu/blog/2022/07/we-found-tar-spot-of-corn-in-2022-now-what/>. This is a relatively new foliar fungal disease (caused by *Phyllacora maydis*) of corn crops with potentially devastating effects. Damon has been developing fungicide programs and disease forecasting approaches for management of this disease over the past

few years in field corn and has recently collaborated with me and the MWFPA and other industry partners to address the disease in sweet corn.

**A bit more about tar spot of corn:** Growers and processors have indicated recent and growing concern with tar spot in sweet corn (*Zea mays*) grown on low organic matter soils in central Wisconsin. The disease has been reported primarily in the Midwestern US and the southeastern US in recent years. In Latin America, where tar spot is more common, fisheye lesions are associated with another fungus, *Monographella maydis*, that forms a disease complex with *P. maydis* known as the tar spot complex. *M. maydis* has not been detected in the U.S., to date. *Phyllachora maydis* is the causal fungus of tar spot of corn and can cause severe yield loss on susceptible hybrids when conditions are favorable for disease (high humidity and prolonged leaf wetness). Tar spot appears as small, raised, black spots scattered across the upper and lower leaf surfaces (Picture below). These spots are ascomata (fungal fruiting structures). Microscopically, hundreds of sausage-shaped asci (spore cases) filled with spores are visible. When severe, ascomatium can even appear on husks and leaf sheaths. Tan to brown lesions with dark borders surrounding the ascomatium can also develop. These are known as ‘fisheye’ lesions.

The pathogen overwinters in infested corn residue. Residue management, rotation, and avoiding susceptible hybrids may reduce tar spot development and severity. Some fungicides may also reduce tar spot, although little data exists regarding application timing for efficacy and economic response in sweet corn. The Corn ipmPIPE helps track the appearance of tar spot (<https://corn.ipmPIPE.org/tarspot/>). Today’s map from this tool is provided below. Additional information is aggregated and provided online by the Crop Protection Network (<https://cropprotectionnetwork.org/series/tar-spot/publications/tar-spot-preface-and-introduction>).

Late in the growing season, common and southern rust pustules can be mistaken for tar spot ascomata as these rusts switch from producing orange to red spores (urediniospores) to black spores (teliospores). However, rust spores burst through the epidermis and the spores can be scraped away from the pustules. Tar spots cannot be scraped off of the leaf tissue.

