

FUNGICIDE RESISTANCE MANAGEMENT

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Resistance to systemic fungicides has been recognized as a major challenge for crop protection since the early 1970s. It became an issue of concern to southeastern peach producers when resistance to benomyl arose in *Monilinia fructicola*, the causal organism of blossom blight and brown rot. Since then, several other groups of systemic and semi-systemic fungicides belonging to the dicarboximide, demethylation inhibitor (DMI), anilinopyrimidine (AP), and strobilurin type action and resistance (STAR) fungicide families have been introduced and made available in stone fruits in the southeastern United States and elsewhere.

- **Benzimidazole (MBC) fungicides:** Members of this family are benomyl (Benlate) and thiophanate methyl (Topsin-M). Control failure due to resistance development was found in brown rot (*M. fructicola*) and scab (*Cladosporium carpophilum*) within a few years of their introductions in the mid-1960s. Benomyl and thiophanate methyl, although different chemicals, both break down upon wetting into methyl benzimidazole carbamate (MBC). It is actually MBC that provides most of the fungicidal activity of these two products. Research also demonstrated that these resistant pathogen strains were resistant to MBC rather than either specific product.
- **Demethylation Inhibitor (DMI) fungicides:** Members of this family are tebuconazole (Elite), propiconazole (Orbit), myclobutanil (Nova), and fenbuconazole (Indar). The DMIs were introduced in the early 1980s and represent an important class of agricultural fungicides. They make a major contribution to world agriculture via their broad spectrum of disease control. Although DMIs are structurally diverse and may have active groups containing a pyrimidine, pyridine, imidazole, or a triazole, they all share a common mode of action that targets a discrete aspect of sterol biosynthesis. This highly specific mode of action suggests a considerable risk of resistance development. Field experience and research results, however, have indicated that DMI resistance evolved more slowly than for the benzimidazoles. Studies on *M. fructicola* isolates from South Carolina indicated a shift of pathogen populations from commercial orchards toward reduced sensitivity, but it was suggested that the shift is not yet strong enough to cause control failure.
- **Dicarboximide fungicides:** Compounds in this class of chemistry include vinclozolin (Ronilan) and iprodione (Rovral). Dicarboximide fungicides were introduced in the mid-1970s, and within a decade resistant isolates of *Botrytis cinerea* (gray mold) were common in several crops. *B. cinerea* remains the only resistant pathogen of real concern. In a 1996 survey, resistance to dicarboximides in other fungal species, for example *M. fructicola* and *Alternaria* spp., was sporadic and did not cause practical problems.
- **Anilinopyrimidine (APs) fungicides:** Of this family only one member, cyprodinil (Vanguard), is currently registered. The APs are a novel group of fungicides and are active against a broad range of fungi. The mode of action includes inhibition of methionine biosynthesis and secretion of hydrolytic enzymes. Although field monitoring has shown no sensitivity shifts in either gray mold or apple scab (*Venturia inaequalis*) under practical conditions, evidence from field and lab trials indicates a substantial resistance risk. APs are volatile in high temperatures, thus restricted in use for the cooler springtime.
- **Strobilurin Type Action and Resistance (STAR) fungicides:** Only one member, azoxystrobin (Abound), is currently registered in peach; other products are expected. The STAR fungicides were introduced in the late 1990s and also represent a novel group of fungicides highly active against a broad range of fungi. They inhibit electron transport in the mitochondrial membranes of fungi. Although it has been known for some time that a single point mutation within the cytochrome *b* gene could confer high levels of resistance, it was generally believed to be unlikely that this point mutation would occur in a majority of the mitochondria within a fungal cell. Thus, it was expected that resistance would develop slowly. However, control failures due to resistance development caused by exactly this point mutation have been reported worldwide in six powdery mildew species on cereals, cucurbits, and bananas. Most recently, resistance development in apple scab was reported.

For updates on resistance to fungicides, consult the web page of the Fungicide Resistance Action Committee (FRAC), <http://www.frac.info/>. All fungicides that have run into trouble with pathogen resistance have one thing in common: they are single-site inhibitors, which means they interfere in a very specific way with a single process of fungus development. Often the systemic fungicides are the ones that will run into resistance trouble. There are several reasons for this:

- A systemic fungicide is taken into a plant, resulting in more uniform distribution through the plant, providing, at least in theory, the maximum uniform coverage. Coverage is certainly much more uniform than randomly placed dried droplets of a fungicide on the outer plant surface. The greater the portion of the target pathogen population exposed to a fungicide, the greater the risk of selection for resistance. As coverage becomes more uniform, more of the pathogen population comes in contact with the fungicide. Because a systemic fungicide provides the maximum in uniform application, then it also provides the maximum contact with a pathogen population. In any case, the effect of the improved coverage factor for systemics is almost certainly trivial compared with the effects of repeated fungicide applications during a season on selection for pathogen resistance.
- Fungi are related to plants. As a consequence, systemic fungicides with modes of action that are toxic to multiple vital life processes have been of no use, because of toxicity to both fungi and higher plants. In fact, most usable systemic fungicides developed to date have a rather specific mode of action. It is this specific mode of action, rather than the systemic property, that leads to resistance selection.

WHERE DOES RESISTANCE COME FROM?

It is believed that pathogen populations contain naturally occurring resistant types in very low numbers. There is normally no adaptive advantage to this trait in the absence of the particular fungicide, so this portion of the population remains very minor. However, when a fungus population is exposed to a particular fungicide, the resistant strains have a great advantage and are left with no competition, thus they build up. Eventually they may become dominant in at least a significant part of the population and control failures are observed. The more times per season an at-risk fungicide is used, the more rapid the shift to resistance will be.

WHY DON'T ALL FUNGICIDES EVENTUALLY RUN INTO RESISTANCE PROBLEMS?

Many fungicides have a variety of antifungal actions that interfere with several unrelated fungus life processes. These materials are very unlikely to run into widespread resistance problems. Those fungicides most likely to have problems with fungal resistance have a very specific site of activity.

HOW CAN FUNGICIDE RESISTANCE PROBLEMS BE AVOIDED?

Basically, there are two different approaches to avoid rapid resistance development:

- Use at-risk fungicides in tank-mix combination with other fungicides with different modes of action.
- Alternate applications of at-risk fungicides with other fungicides with different modes of action.

These two ideas are very reasonable, and in one form or another are used as the major means of fungicide resistance management. Several mathematical models have been developed to test which of these two approaches would be better. The best approach, combinations or alternating, depends on the specific situation. The results of testing these models can be briefly summarized:

- The more often an at-risk fungicide is used in any way, the more quickly it selects out the resistant strains.
- Neither fungicide combinations nor alternating prevent the selection of resistance. The best either approach can do is slow the process.
- Combinations usually can be expected to slow the process more than alternating. However, combinations are more expensive and may mask the selection of resistance. This can be a major concern to peach producers in the Southeast.

In most cases, cross-resistance exists between members of a fungicide family. Thus, when any one product fails because of resistance, the other product in that same family would no longer work either. As a consequence, when switching to a new fungicide for resistance management purposes, one needs to switch to a compound from a different fungicide family (i.e., different mode of action).

WHAT APPROACH TO FUNGICIDE RESISTANCE MANAGEMENT SHOULD BE FOLLOWED?

The best approach to fungicide resistance management is to use a program consisting of both combinations and alternating of fungicides with different modes of action. An at-risk fungicide should be used only at key times in the season when absolutely necessary. Other products should be used at other times. When used, an at-risk fungicide should be used in combination with another product of a differing mode of action. The combination helps slow the rate of resistance selection and provides some control of resistant strains already present. Use of a combination is critical if some documented resistance has already occurred. In the total absence of locally documented resistance, combinations are less critical, as long as an alternating program to limit use of the at-risk fungicide is followed. The fungicide recommendations of the [Southeastern Peach, Nectarine and Plum Pest Management and Culture Guide](#) are written to emphasize this sort of program. Any and all fungicide programs should be supplemented, wherever possible, by cultural and sanitation practices that reduce disease carryover or otherwise assist disease control.

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