Ear Rots of Corn: Identification, Testing for mycotoxins, and Storage

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IDENTIFICATION

At least four different types of ear rots – Diplodia, Gibberella, Fusarium, and Trichoderma – were observed during the growing season. Ear rots differ from each other in terms of the damage they cause (their symptoms), the toxins they produce, and the specific conditions under which they develop. Most are favored by wet, humid conditions during silk emergence (R1) and just prior to harvest. But they vary in their temperature requirements, with most being restricted my excessively warm conditions. However, it should be noted that even when conditions are not optimum for ear rot development, mycotoxins may accumulate in infected ears. A good first step for determining whether you need to get grain samples tested for mycotoxin is to know which ear rot was most prevalent in your field. Of the four ear rots listed about, Fusarium and Gibberella are the ones of greatest concern in terms of mycotoxin contamination.

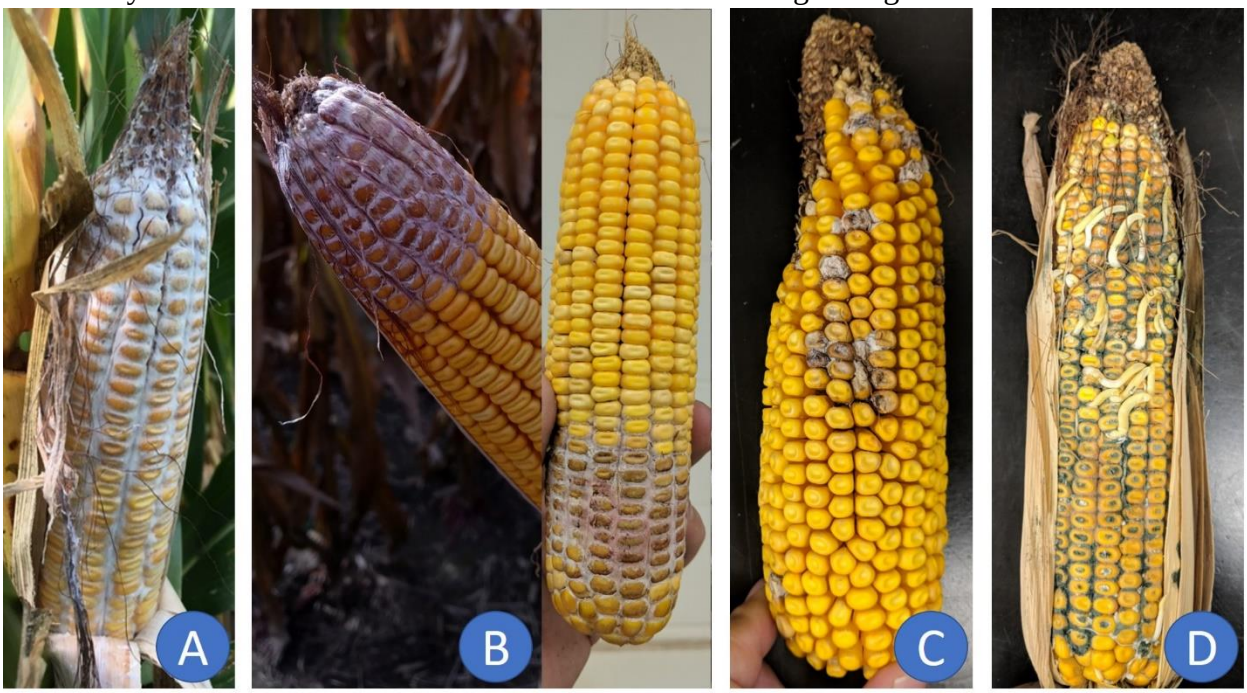
DIPLODIA EAR ROT (Fig. 1A): This is one of the most common ear diseases of corn in Ohio. The most characteristic symptom and the easiest way to tell Diplodia ear rot apart from other ear diseases such as Gibberella and Fusarium ear rots is the presence of white mycelium of the fungus growing over and between kernels, usually starting from the base of the ear. Under highly favorable weather conditions, entire ears may become colonized, turn grayish-brown in color and lightweight (mummified), with kernels, cobs, and ear leaves that are rotted and soft. Rotted kernels may germinate prematurely, particularly if the ears remain upright after physiological maturity. Corn is most susceptible to infection at and up to three weeks after R1. Wet conditions and moderate temperatures during this period favor infection and disease development, and the disease tends to be most severe in no-till or reduce-till fields of corn planted after corn. The greatest impact of this disease is grain yield and quality reduction. Mycotoxins have not been associated with this disease in US, but animals often refuse to consume moldy grain.

GIBBERELLA EAR ROT (Fig. 1B): When natural early-season infections occur via the silk, Gibberella ear rot typically develops as white to pink mold covering the tip to the upper half of the ear. However, infections may also occur at the base of the ear, causing the whitish-pink diseased kernels to develop from the base of the ear upwards. This is particularly true if ears dry down in an upright position and it rains during the weeks leading up to harvest. The Gibberella ear rot fungus may also infect via wounds made by birds or insects, which leads to the mold developing wherever the damage occurs. When severe, Gibberella ear rot is a major concern because the fungus produces several mycotoxins, including deoxynivalenol (***vomitoxin***), that are harmful to livestock. Once the ear is infected by the fungus, these mycotoxins may be present even if no visual symptoms of the disease are detected. Hogs are particularly sensitive to vomitoxin. Therefore, the FDA advisory level for vomitoxin in corn to be fed to hogs is ***5 ppm and this is not to exceed 20% of the diet***.

FUSARIUM EAR ROT (Fig. 1C). Fusarium ear rot is especially common in fields with bird or insect damage to the ears. Affected ears usually have individual diseased kernels scattered over

the ear or in small clusters (associated with insect damage) among healthy-looking kernels. The fungus appears as a whitish mold and infected kernels sometimes develop a brownish discoloration with light-colored streaks (called starburst). Several different Fusarium species are associated with Fusarium ear rot, some of which produce toxins called ***Fumonisins***. Horses are particularly sensitive to Fumonisins. ***Note, vomitoxin and Fumonisins are entirely different types of toxins***.

TRICHODERMA EAR ROT (Fig. 1D): Abundant, thick, greenish mold growing on and between the kernels make Trichoderma ear rot very easy to distinguish from Diplodia, Fusarium, and Gibberella ear rots. However, other greenish ear rots such as Cladosporium, Penicillium and Aspergillus may sometimes be mistaken for Trichoderma ear rot. Like several of the other ear rots, diseased ears are commonly associated with bird, insect, or other types of damage. Another very characteristic feature of Trichoderma ear rots is sprouting (premature germination of the grain on the ear in the field). Although some species of Trichoderma may produce mycotoxins, these toxins are usually not found in Trichoderma-affected ears under our growing conditions.



Diplodia (A), Gibberella (B), Fusarium (C) and Trichoderma ear rots of corn

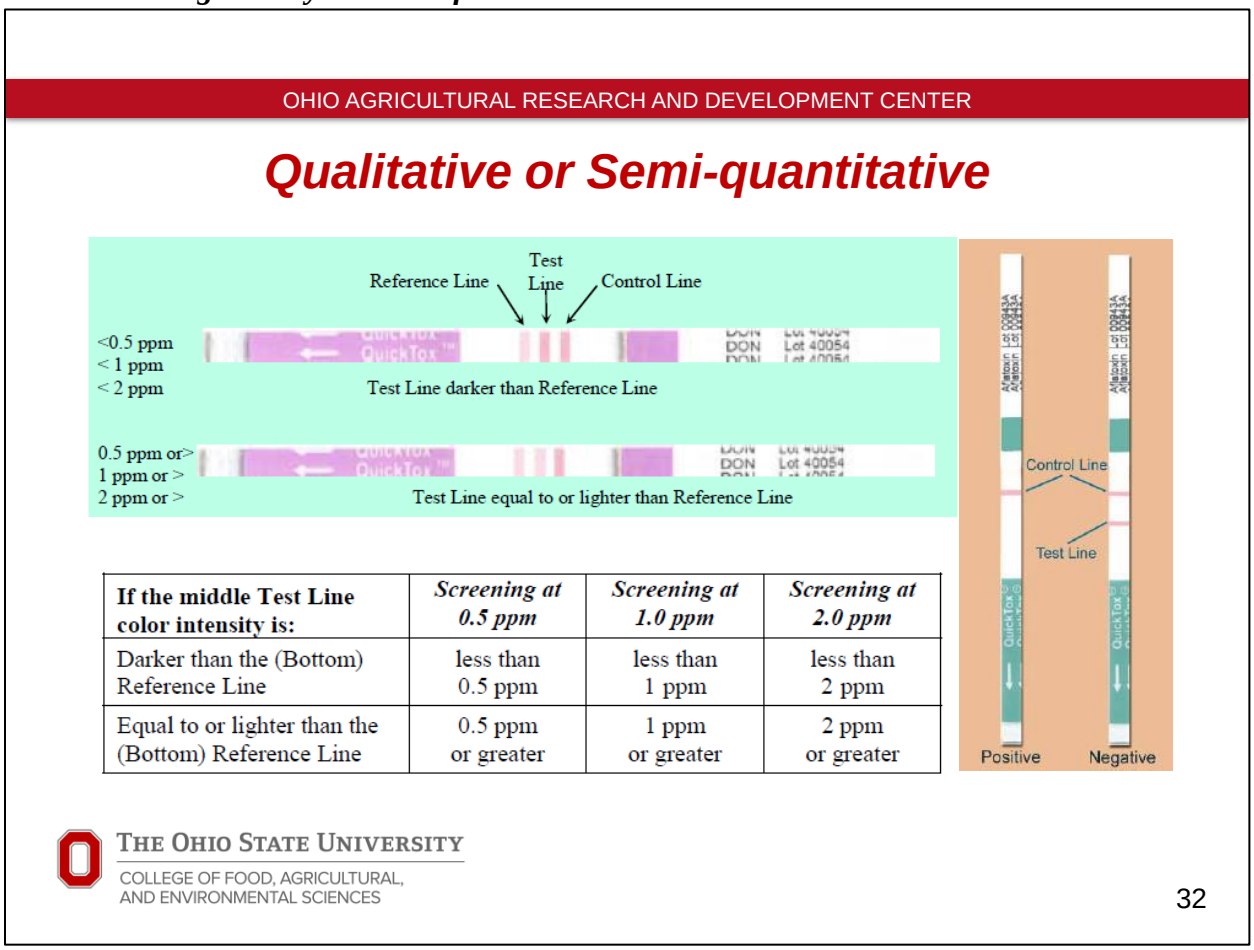
Fig 1. Common ear rots of corn

TESTING FOR MYCOTOXIN

SAMPLING: This is probably the most important step for accurately estimating toxins in grain samples. Since The number of ears infected within a field and number of infected kernels on a given ear are highly variable, moldy grain and vomitoxin levels vary considerably within the grain lot. Poor sampling may result in considerable variation in test results and could result in grain being rejected. To collect a representative grain sample, 5–10 samples should be randomly collected from multiple locations in the bin or truckload. Samples taken only from the central or outer portions of the load will not provide an accurate estimate of toxin contamination. For end-gate

sampling, samples should be drawn from the entire width and depth of the grain stream. For sampling with hand or mechanical probes, multiple samples should be drawn from throughout the bin or truck, along an X-shaped pattern, for example. Once samples are obtained, bulked, and cleaned, the grain should be ground uniformly, in a clean grinder, to resemble flour. Finer particle size increases surface area of the grain and enables efficient extraction of vomitoxin. ***Air (suction) probes are not recommended for sampling grain with ear rot. Moldy and broken kernels are lighter in weight and usually contain high levels of vomitoxin. Air probes are more likely to pull these kernels and overestimate the overall toxin level in the lot.***

TESTING: Mycotoxin tests are either qualitative, semi-quantitative, and quantitative. Qualitative tests provide a yes/no answer for the presence of the toxin in question and are useful for initial screening. Semi-quantitative tests estimate toxin at or above certain levels (>5 ppm) or within a given range, whereas, quantitative tests provide more precise estimates. There is a trade-off between precision, price, and speed. The more quantitative tests tend to be the most precise but are also more expensive and take longer to complete than the qualitative or semi-quantitative tests. Semi-quantitative quick-test kits (see the test stripes in **Fig 2)** are very common and relatively easy to use and inexpensive, but they are often very specific for one particular toxin. ***A test for vomitoxin will generally not work for Fumonisins.***



If the middle Test Line color intensity is:	*Screening at 0.5 ppm*	*Screening at 1.0 ppm*	*Screening at 2.0 ppm*
Darker than the (Bottom) Reference Line	less than 0.5 ppm	less than 1 ppm	less than 2 ppm
Equal to or lighter than the (Bottom) Reference Line	0.5 ppm or greater	1 ppm or greater	2 ppm or greater

Fig 2. An example mycotoxin quick-test strip and interpretation chart.

STORAGE

Poor storage may cause toxin levels to increase. Warm, moist pockets in the grain promote mold development, causing the grain quality to deteriorate and toxin levels to increase. Aeration is important to keep the grain dry and cool. However, it should be noted that while cool temperatures, air circulation, and low moisture levels will minimize fungal growth and toxin production, these will not decrease the level of toxin that was already present in grain going into storage.

- Dry and store harvested grain to below 15% moisture to minimize further mold development and toxin contamination in storage.
- Store dried grain at cool temperatures (36 to 44°F) in clean, dry bins. Moderate to high temperatures are favorable for fungal growth and toxin production.
- Periodically check grain for mold, insects, and temperature.
- If mold is found, send a grain sample for mold identification and analysis to determine if toxins are present and at what level.
- Clean bins and storage units between grain lots to reduce cross-contamination.

The information summarized in this section was taken from factsheet # PLPATH-CER-04 (http://ohioline.osu.edu/factsheet/plpath-cer-04).

Miravis® Ace: A new Fungicide in the Fight against Head Scab and Vomitoxin in Wheat and Barley - Pierce A. Paul and Jorge David Salgado

Miravis® Ace is a new combination fungicide made up of a triazole (propiconazole, the same active ingredient in Tilt) and a Succinate Dehydrogenase Inhibitor (SDHI; Adepidyn®) with promising efficacy against head scab and vomitoxin. Miravis® Ace (abbreviated here as MIR) was compared to Prosaro (abbreviated as PRO) and Caramba (abbreviated as CAR), the industry standards for scab and vomitoxin control, in fungicide efficacy trials (fungicide alone) and integrated management trials (fungicide + genetic resistance).

MIRAVIS® ACE IN EFFICACY TRIALS

Miravis® Ace (13.7 fl. oz. + NIS) was tested alone at 50% early heading (**Fig. 1A**), at 50% early anthesis (flowering; **Fig. 1B**), or at 4-6 days after flowering or in combination with Prosaro (6.5 fl oz. + NIS) (**MIR_PRO**, **Fig. 2**), Caramba (13.5 fl oz + NIS) (**MIR_CAR**, **Fig. 2**) or Folicur (4 fl oz + NIS) (**MIR_FOL, Fig. 2**).

Early heading (A) and Early Flowering (B)

Fig 1. A - note the head only partially out of the flag leaf, **B** - note the anthers extruded in the center of the head

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Miravis Ace Compared to and combined with Prosaro and Caramba

Combination	Description
PRO_A	Prosaro (6.5 fl oz) at 50% early anthesis (flowering)
CAR_A	Caramba (13.5 fl oz) at 50% early anthesis
MIR_A	Miravis Ace (13.7 fl oz) at 50% early anthesis
MIR_H	Miravis Ace at heading (Feekes 10.3 - 10.5)
PRO_H	Prosaro at heading (Feekes 10.3 - 10.5)
MIR_L	Miravis Ace at 4-6 days after 50% early anthesis
MIR_PRO	Miravis Ace at 50% early anthesis followed by Prosaro 4-6 days after
MIR_CAR	Miravis Ace at 50% early anthesis followed by Caramba 4-6 days after
MIR_FOL	Miravis Ace at 50% early anthesis followed by Folicur 4-6 days after
CK	Non-treated check

All treatments were applied with non-ionic surfactant at 0.125 v/v, volume 15-20 GPA
Susceptible cultivar used in all trials and all plots were inoculated

Fig 2. Treatment list and description

- Miravis® Ace was just as effective as Caramba and Prosaro when applied at flowering (**Fig. 3A** and **Fig. 4A**).
- When applied at early anthesis, early heading, or at 4-6 days after flowering, Miravis® Ace showed comparable effective against heads scab (**Fig. 3A and B**). However, the heading application was considerably less effective than flowering and late applications in terms of vomitoxin reduction (**Fig. 4A and B**).
- Two-treatments programs, with an application of Miravis® Ace at anthesis followed by an application of Caramba, Prosaro, or Folicur 4-6 days later, led to the greatest scab and vomitoxin reduction (**Fig. 3C and 4C**).

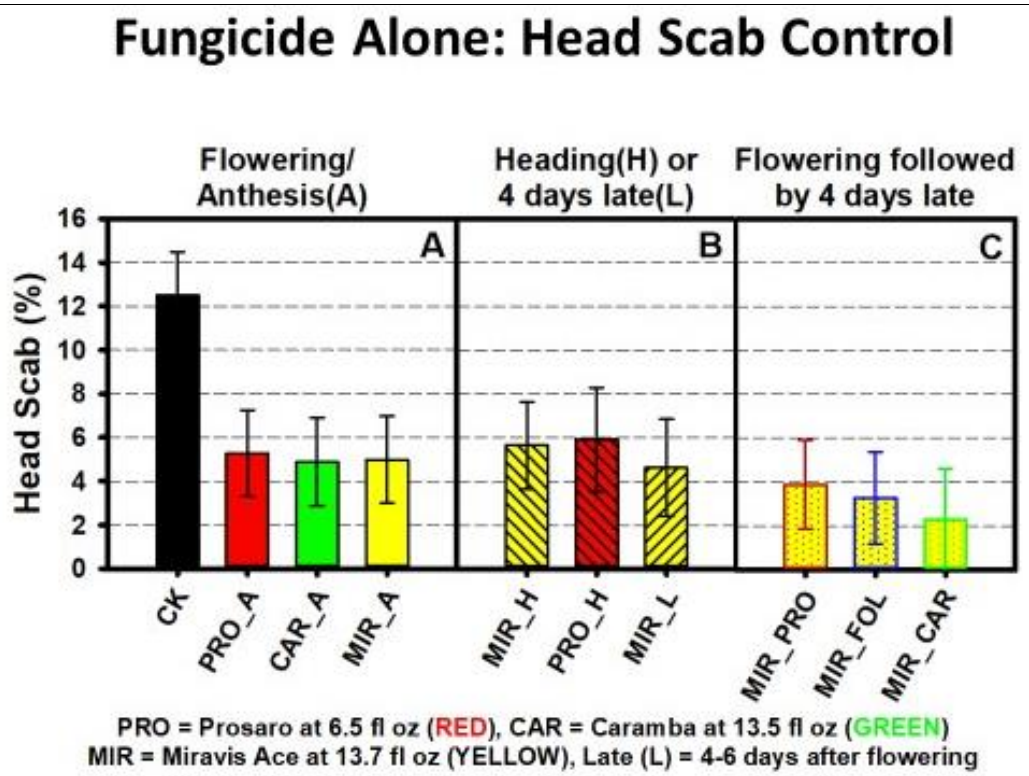

Fig 3. Efficacy against head scab when applied at flowering/anthesis (**A**), at heading or after flowering (**B**) or combination treatments (**C**, two-treatment programs).

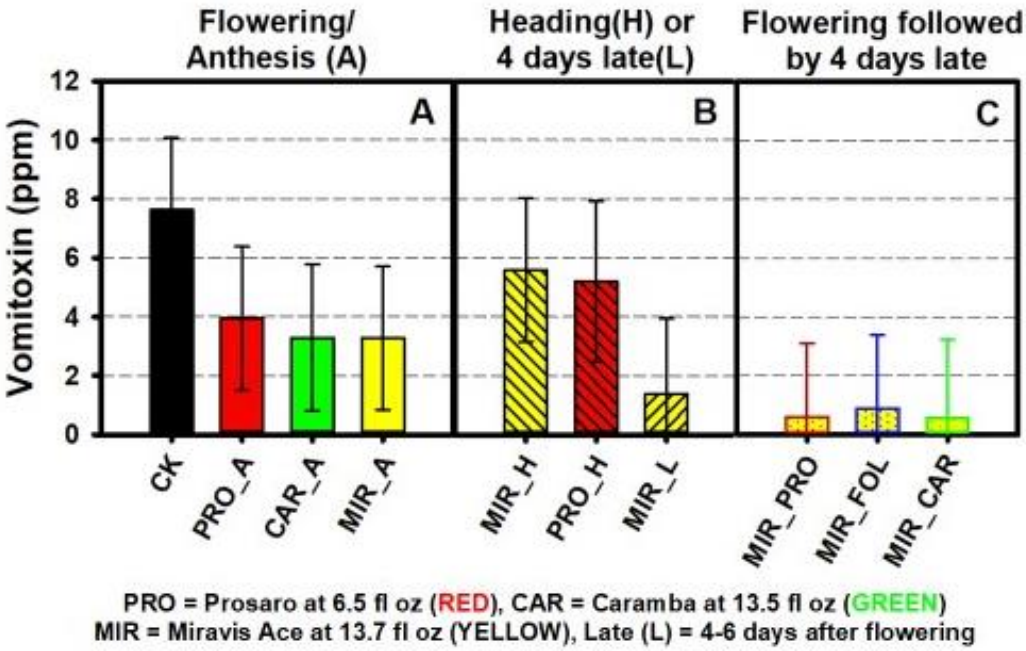

Fig 4. Efficacy against head scab when applied at flowering/anthesis (**A**), at heading or after flowering (**B**) or combination treatments (**C;** two-treatment programs).

MIRAVIS® ACE IN INTEGRATED MANAGEMENT TRIALS

The efficacy of Miravis® Ace (13.7 fl. oz. + NIS) when applied at 50% early heading or at 50% early anthesis (flowering) was evaluated on susceptible (S), moderately susceptible (MS) and moderately resistant (MR) cultivar (**Fig. 5**).

Miravis Ace + Genetic Resistance: Integrated management

Combination	Description
MR_I	Moderately resistant, with Prosaro (6.5 fl oz) at 50% early anthesis
MR_II	Moderately resistant, with Miravis Ace (13.7 fl oz) at 50% early anthesis
MR_III	Moderately resistant, with Miravis Ace (13.7 fl oz) at heading
MR_CK	Moderately resistant, non-treated
MS_I	Moderately susceptible, with Prosaro (6.5 fl oz) at 50% early anthesis
MS_II	Moderately susceptible, with Miravis (13.7 fl oz) Ace at 50% early anthesis
MS_III	Moderately susceptible, with Miravis Ace (13.7 fl oz) at heading
MS_CK	Moderately susceptible, nontreated
S_I	Susceptible, with Prosaro (6.5 fl oz) at 50% early anthesis
S_II	Susceptible, with Miravis Ace (13.7 fl oz) at 50% early anthesis
S_III	Susceptible, treated with Miravis Ace (13.7 fl oz) at heading
S_CK	Susceptible, nontreated

All treatments were applied with non-ionic surfactant at 0.125 v/v, volume 15-20 GPA
All plots were inoculated

Fig. 5. Fungicide treatment x cultivar resistance combinations evaluated for efficacy against head scab and vomitoxin.

- All fungicide treatment x cultivar resistance combinations reduced head scab and vomitoxin over the non-treated susceptible check (**Fig. 6** and **7**).
- When applied at heading or at anthesis, Miravis® Ace show comparable efficacy to Prosaro, particularly against scab.
- Compared to the non-treated susceptible check (S_CK), average scab and vomitoxin levels were lowest when the fungicide treatments were applied to moderately susceptible or moderately resistant cultivars than to a susceptible cultivar (**Fig. 6** and **7**).
- The lowest average levels of vomitoxin occurred when a moderately resistant cultivar was treated with a Prosaro or Miravis® Ace at flowering (**Fig. 7C**).
- Planting a more resistant cultivar without applying a fungicide (MR_CK or MS_CK) resulted in similar levels of vomitoxin to the susceptible-treated (S_I, S_II, or S_III; **Fig. 7**).

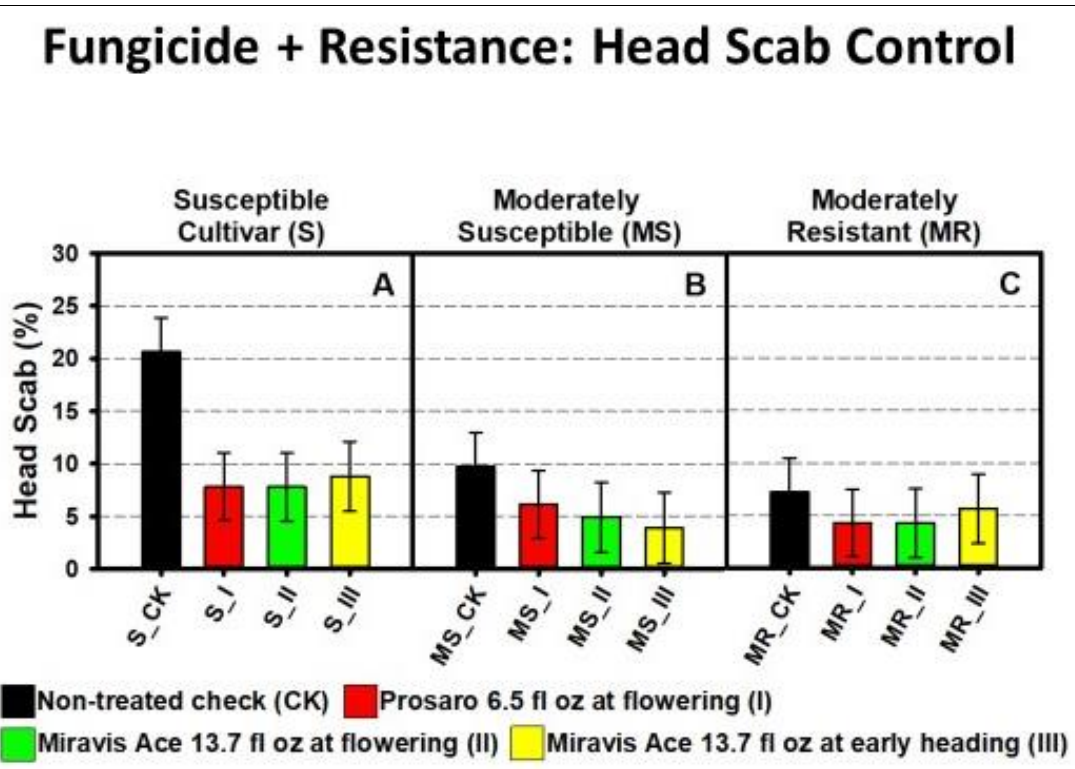

Fig 6. Effect of fungicide x genetic resistance combinations on head scab.

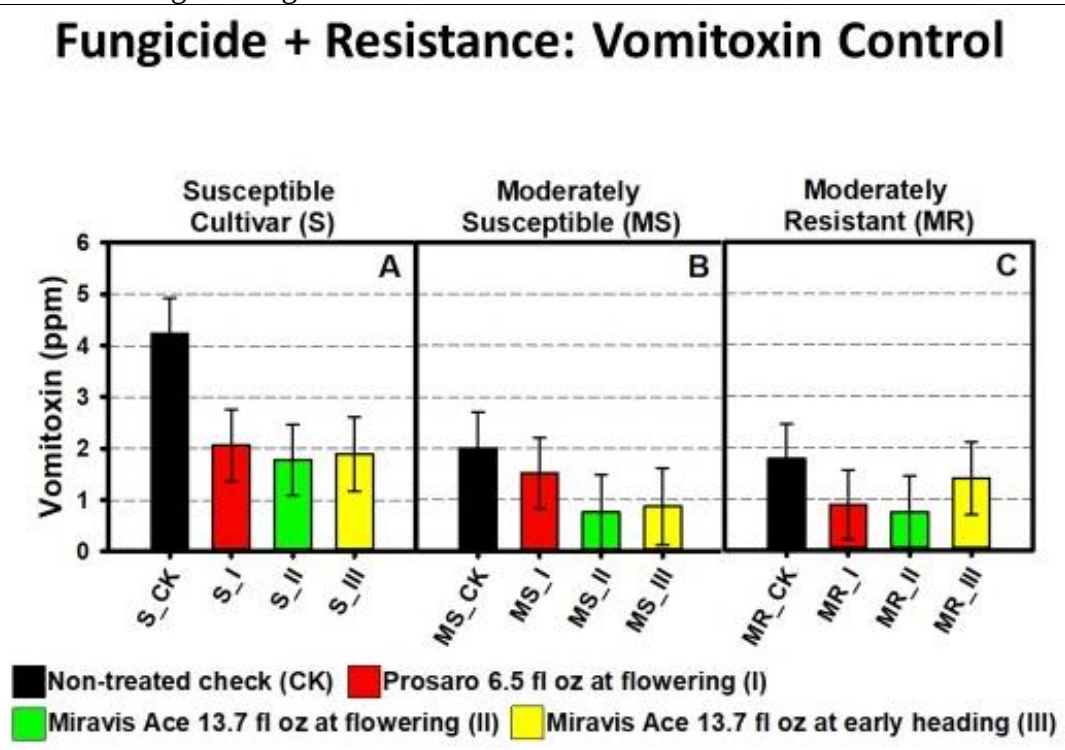

Fig 7. Effect of fungicide x genetic resistance combinations on vomitoxin.