# How Fungi Infect and How Fungicides Work

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In Wisconsin, approximately 25 diseases have been reported on cranberry, and the majority of these are caused by fungi. The most important fungal diseases in Wisconsin are: cottonball (*Monilinia oxycocci*); the fruit rot complex (several fungi, but most notable in recent years, *Colletotrichum* and *Phomopsis*); and upright dieback (*Phomopsis vaccinii*). *Phytophthora* spp. that infect roots and runners, are superficially similar to fungi, but genetically very different. Cranberry stem gall, which is a serious problem in some locations in some years, is most likely caused by bacteria that produce a plant growth hormone.

Fungi (singular form: fungus) are a unique kingdom of life. Despite attempts to lump them with plants, fungi are actually more closely related to animals than plants. Fungi lack chlorophyll, and therefore cannot make their own food. They also lack vascular tissue, and therefore cannot effectively move water throughout their "bodies." Therefore, fungi need to live in close contact with a source of nutrients and water. Since plants don't have true immune systems to thwart off such freeloaders, it's not surprising that some fungi are parasites (=pathogens) of plants.

The fungal infection process consists of a series of steps. Taken together these steps make up the disease cycle for a pathogen. Fungicides act by disrupting one or more of these steps.

- *Inoculation*—the fungal spore lands on a plant surface.
- *Adhesion*—the fungal spore exudes a glue so that it sticks to the plant even if there is rain.
- *Germination*—the fungal spore takes up water and a germ tube emerges.
- *Penetration*—the germ tube enters the plant, either by poking directly through the epidermis or by going through stomata or wounds.
- *Plant-pathogen recognition*—chemical or molecular signals are exchanged between the fungus and the plant so that the fungus knows it is on a suitable host.
- *Infection*—the fungus invades the plant by growing into or between cells, and the fungus produces spores that are released at the plant surface.

The fungicides registered on cranberry vary in the steps of the infection process that they disrupt. To some extent, this is determined by where the fungicide ends up after it is sprayed onto the plant. **Contact fungicides** remain on the surface of the plant, and move only when rain or irrigation water redistributes them. For this reason, a little rain after a fungicide application is not a bad thing. Fungicides are formulated to adhere to the plant, and they are rainfast except under extreme circumstances. Contact fungicides inhibit spore germination but do not work if a plant is already infected. This type of activity is referred to as **protectant**. Examples of contact fungicides with protectant activity are chlorothalonil, mancozeb, and copper. **Systemic fungicides** move through a

plant's vascular tissue. Examples of systemic fungicides are Aliette and phosphorous acid products, which are taken up by leaves and transported to runners and roots to inhibit *Phytophthora*. **Locally systemic fungicides** are taken up by leaves or flowers and move a short distance within the leaf or flower. For example, they can move from one surface of a leaf to the other or from the base of a flower to the stigma (where the cottonball pathogen infects). Uptake is better through soft tissues, such as growing shoot tips and young fruit rather than hard tissues such as mature leaves and fruit. Examples of locally systemic fungicides are propiconazole and azoxystrobin. Locally systemic fungicides do their best work by inhibiting growth of fungi after infection, and are said to have **post-infection activity.**

| **Brand name** | **Active ingredient** | **Protectant** | **Post-infection** |
|---|---|---|---|
| Bravo | chlorothalonil | X | |
| Dithane, Pennozeb | mancozeb | X | |
| Champ, Kocide, etc. | copper | X | |
| Orbit | propiconazole | X | X |
| Abound | azoxystrobin | X | X |
| Ridomil | mefanoxim | X | X |
| Aliette | fosetyl aluminum | | X |
| Phostrol, Prophyt, etc. | phosphorous acid | | X |

For a fungicide to work, it must reach its target. That is, it either has to contact the fungus at the surface of the plant or inside the plant. Contact fungicides must be present at the infection court, the site on the plant where the fungus penetrates. This means that coverage with a contact fungicide has to be nearly perfect for the fungicide to work. Coverage can be improved by using higher spray volumes and by applying under calm conditions. Systemic and locally systemic fungicides are a little more forgiving of incomplete coverage, but obviously, they won't reach their intended targets if applied when there is a breeze. Some infection courts are easier to protect than others. *Phomopsis vaccinii* (upright dieback pathogen) and *Monilinia oxycocci* (cottonball pathogen) infect through elongating shoots. These "roughneck" shoots are a relatively large infection court and have a lot of nooks and crannies for spores to hide in and escape contact fungicides. Consequently, controlling primary cottonball infections and upright dieback is not always successful, even when the most effective fungicides are used. On the other hand, the infection court for secondary cottonball infections is the floral stigma, a relatively small, soft target that probably takes up the systemic fungicide propiconazole very well. In this way, control of secondary cottonball infections is usually successful if an effective fungicide is used during bloom.

Sometimes fungicides are used, but disease still develops. What goes wrong? The issue of getting the infection court covered is discussed above. This can be improved by increasing spray volume and spraying during calm conditions. Increasing spray volume will also reduce the risk of phytotoxicity from chlorothalonil, because the concentration of the product is reduced. Fungicide timing must be right for the fungicide to reach the susceptible infection court on the plant. For primary cottonball and upright dieback, this means spraying during shoot elongation. Research has shown that the best time for

cottonball sprays are when more than half of the shoots show about ½ inch of new growth. To prevent secondary cottonball infections, sprays must go on during bloom. Fruit rot sprays are most effective if applied at late bloom and early fruit set. Plant growth stages vary among varieties and across a cranberry marsh. Although it's not always practical, disease control will be better if problem beds are sprayed according to their own growth stage. More details on spray timing and disease cycles are available in other extension publications. Lastly, it's important to use a fungicide that is effective against the disease you are trying to control. A fungicide that is cheap to buy is expensive to use if it doesn't work! Research done here in Wisconsin and in other states has provided us the following information about which fungicides are best for which purpose.

| | **chloro-thalonil** | **mancozeb** | **copper** | **propi-conazole** | **azoxy-strobin** | **mefanoxim** | **fosetyl-Al** |
|---|---|---|---|---|---|---|---|
| **Cottonball** | F | P-F | P | E | F-G | ? | ? |
| **Upright dieback** | G-E | NA | ? | ? | NA | ? | ? |
| **Fruit rot** | E | F-G | P-F | P-F | F-G | ? | ? |
| **Phytophthora** | ? | ? | ? | ? | ? | G | F-G |

E=excellent; G=good; F=fair; P=poor; NA=not allowed based on timing of permitted sprays; ?=unknown. Phosphorous acid has not been tested on the strains of *Phytophthora* present in Wisconsin, but in other disease systems its performance has been similar to that of fosetyl aluminum.