Integrated Management of Cottonball

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Cottonball, caused by the fungus *Monilinia oxycocci,* is an economically important disease on many cranberry marshes in Wisconsin. Cottonball has also been described in the Pacific Northwest and southeastern Canada, but losses in these areas have generally been minor. For reasons that are not known, cottonball has increased in importance in Wisconsin over the past 30 years. In recent years, about 20% of bearing acreage has been treated with fungicides specifically for cottonball control. Where cottonball occurs, the incidence of infected fruit is typically 2-10%, but it can exceed 40% if left unchecked. Control of cottonball has depended largely on fungicides. As we gather more information on the interaction of *M. oxycocci* and cranberry in the marsh environment, we are developing safer and more sustainable means of controlling this disease.

Cottonball disease cycle (Fig. 1)

The cottonball fungus, *M. oxycocci,* overwinters in mummified remains of previous seasons' infected fruit, technically known as sclerotia. In the spring, small mushroom-like structures called apothecia grow from some of the sclerotia (mummies). Ascospores are ejected from the apothecia, starting at about budbreak and continuing until just before bloom. Maximal ascospore release occurs over a 10- to 14-day period when the majority of shoots are ½ to 1 ¼ inches long and very susceptible to infection. Infection probably requires water and moderate temperatures, although this has not been determined experimentally. The exact sites on the elongating uprights where the fungus penetrates are not known. Infection results in "tip blight" symptoms: crooked over shoot tips, tan discoloration of leaves, and blasted blossom buds starting about a week before bloom.

Just before bloom, the fungus produces spores (conidia) on infected floral and vegetative uprights. Conidia are carried to flowers by wind, insects, or both. There they germinate on the stigma and grow down the style to the developing ovary, similar to the pattern of pollen germination and growth. As the fruit matures, the fungus fills the seed cavity and eventually grows into the fleshy tissue. By harvest time, sclerotia develop in 25-50% of the infected fruit; berries that do not have sclerotia by harvest time decompose by the following spring.
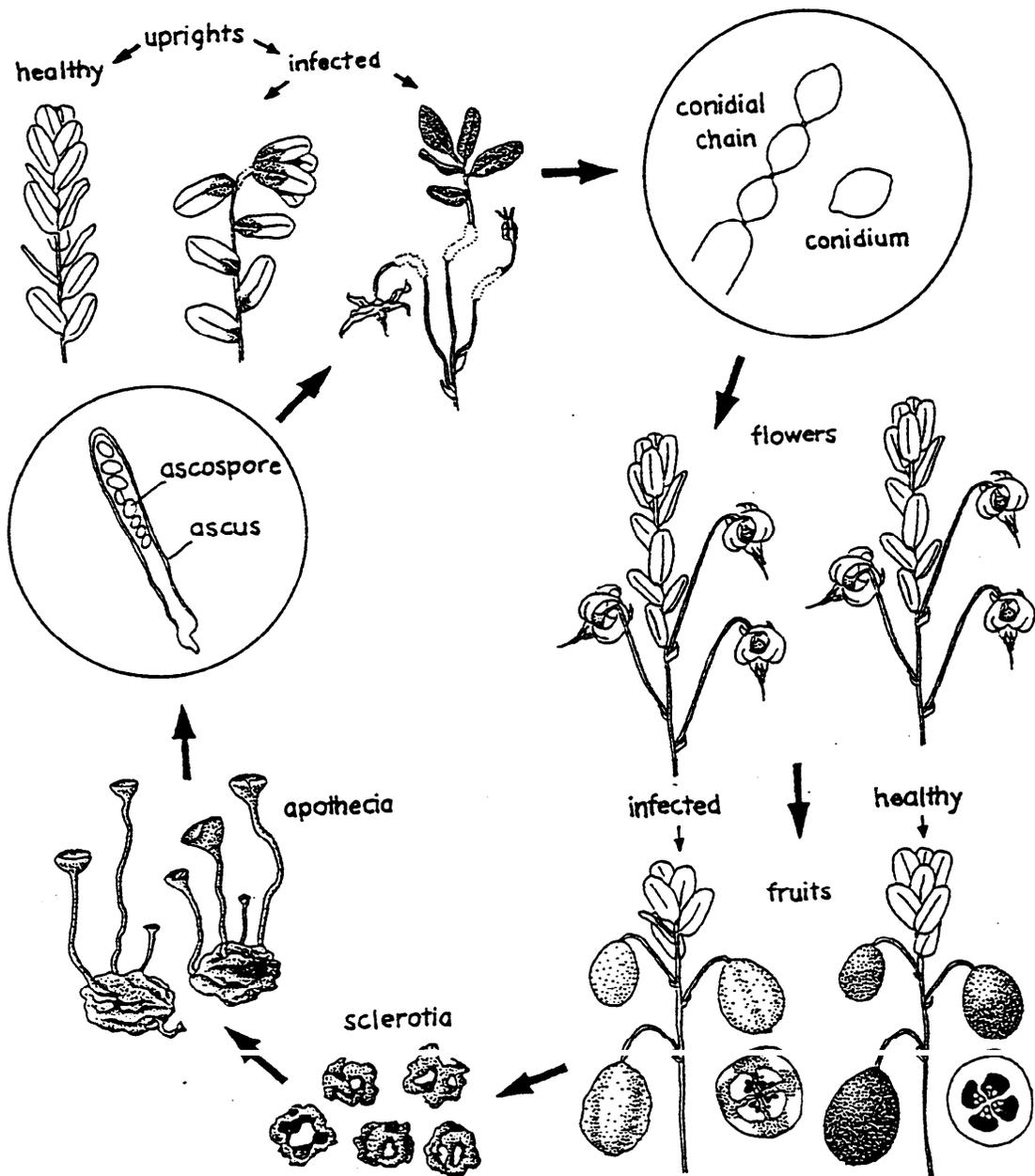
Figure 1. Cottonball disease cycle.

Integrated Management

Sanitation and cultural practices. Most recent research on cottonball has focused on chemical control and efficient use of fungicides. However, there are limited data and circumstantial evidence on the influence of sanitation and cultural practices that guide us in developing an integrated approach to control. For example, cottonball fruit and mummies float, and many are removed during harvest. Some growers have found that re-flooding beds after harvest cleans out not just cottonball mummies but other pests as well. We have noticed that primary infection (shoot infection) is often most severe along ditches, especially where there is dense moss. Perhaps these areas remain wet for prolonged periods and this promotes germination of mummies. Alternatively, vines in these areas may be prone to frost injury. On lowbush blueberry, frost-injured plants are more susceptible to mummy berry, a disease similar to cottonball. Finally, we have observed severe cottonball in areas of beds where newly applied sand remained saturated for several days. Thus, good drainage appears to be important not only for the general health of the cranberry plant but also to prevent cottonball mummies from coming to life.

Chemical control. In the early 1980s Funginex (triforine), a sterol inhibitor (SI) fungicide, was registered on cranberry for control of cottonball. By the mid-1990s, Funginex was no longer being produced, but another SI fungicide, Orbit (propiconazole) became available by Section 18 emergency registration. With both Funginex and Orbit, two sprays during shoot elongation (budbreak) and two sprays during bloom have been permitted. However, most growers who treat for cottonball spray fewer than four times per season. *So which sprays are more important-shoot elongation or bloom?* Answering that question was the *first objective* of our research. A *second objective was to test new fungicides,* especially those that have been deemed "reduced-risk" by the EPA, To delay the development of Orbit-resistant populations of *M. oxycocci, we* need new fungicides with modes of action different from the SIs. A *third research objective was to determine whether fungicide-resistant populations of M. oxycocci had already started to evolve* at sites where SI fungicides (Funginex and Orbit) had been used. The fact that the SI fungicides, which have a single mode of action, have been used frequently, and often exclusively, to control cottonball for the past 16 years is reason enough to be concerned about fungicide resistance in *M.oxycocci.*

Field tests conducted in 1996 and 1997 showed that under low to moderate disease pressure (<15% cottonball berries at harvest), making two sprays during bloom was just as good at reducing cottonball at harvest as making two sprays during shoot elongation *plus* two sprays at bloom (Figs. 2 and 3). In other words, the shoot elongation sprays were a waste of time and fungicide. We also found that some experimental fungicides were as effective as Orbit at controlling cottonball. These will be pursued for future registration. It's encouraging that none of the fungicides tested reduced yield, fruit size, fruit retention, or fruit color.
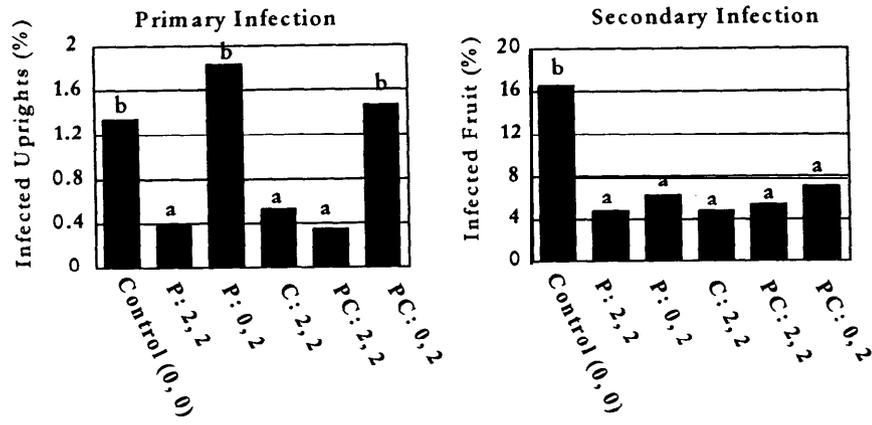
Figure 2. Incidence of primary (shoot) and secondary (fruit) cottonball infection in 1996. Data from two sites were combined. P=propiconazole (Orbit); C=experimental fungicide; PC=mixture of P and C. Numbers after P: or C: are number of shoot elongation (budbreak) sprays, number of bloom sprays. Within a graph, the same letter above bars indicates no statistically significant difference between the treatments.
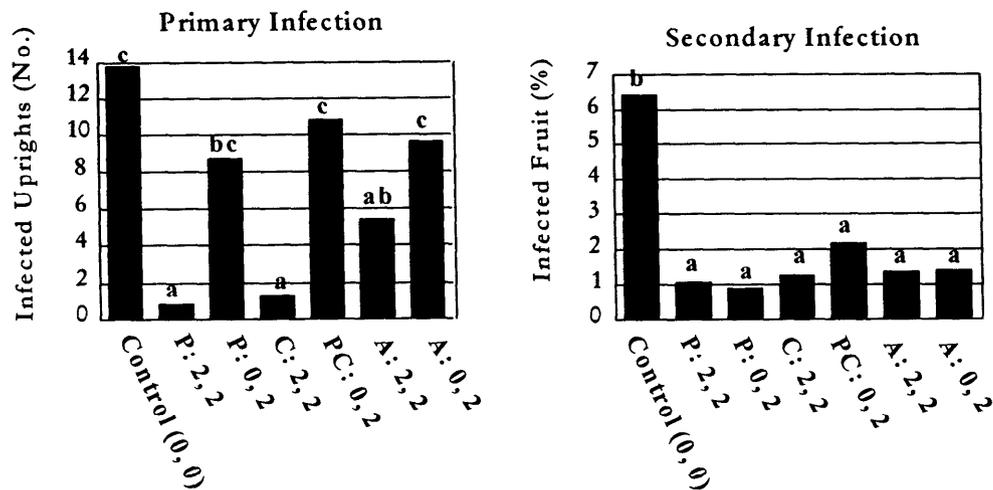
Figure 3. Incidence of primary (shoot) and secondary (fruit) cottonball infection in 1997. P=propiconazole (Orbit); C and A=experimental fungicides PC=mixture of P and C. Numbers after P:, C:, or A are number of shoot elongation (budbreak) sprays, number of bloom sprays. Within a graph, the same letter above bars indicates no statistically significant difference between the treatments.

Fungicide resistance concerns. Despite using fungicides with a single mode of action for several years, there have been no reported suspicions of resistance to Orbit. But if Orbit "failure" is reported in the future, how will we know whether it's because of resistance or some other factor *(e.g.,* too low a rate used or poor spray coverage)? To answer this question in the future, we need to know just how susceptible *M. oxycocci* is to Orbit now, *before* it's been used for several years.

To get a "baseline" fungicide sensitivity standard, and to see whether resistance to Orbit might already be developing, we collected populations of *M. oxycocci* from three sites that differed in fungicide use history. At site 1, fungicides had never been used; at site 2, two to four SI sprays had been applied each year since 1989; and at site 3, two to four SI sprays had been applied each year since the early 1980s along with other fungicides *(e.g.,* copper, mancozeb, captafol, and chlorothalonil). Then, in the laboratory we determined the ED $_{50}$ *(i.e.,* fungicide concentration that reduced fungal growth by 50%) for each member of each population. The frequency distributions for ED $_{50}$ s show that isolates of *M. oxycocci* from a given site vary in sensitivity to Orbit, but the average ED $_{50}$ did not differ significantly among sites (Fig. 4). These data suggest that field populations exposed to the SI fungicides Funginex and Orbit have not become resistant to Orbit. The data also provide a "baseline" sensitivity standard to which we can compare suspected Orbit-resistant populations of *M. oxycocci* in the future.
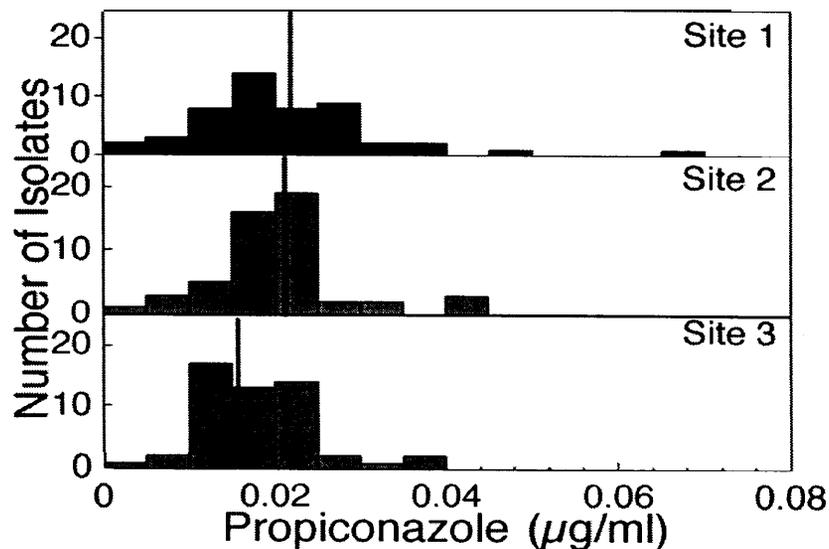
Figure 4.   Frequency distributions of ED $_{50}$ values to propiconazole (Orbit) for populations of *Monilinia oxycocci* from sites with different fungicide use histories (see text for details). Values on the x-axis are ED $_{50}$ fungicide concentrations; values on the y-axis are number of isolates of *M. oxycocci* in each ED $_{50}$ class.   Vertical bars represent the mean ED $_{50}$ for each site.

Susceptibility of popular varieties

Cottonball has been observed on all the popular varieties *(e.g.,* Stevens, Ben Lear, Searles, Pilgrim, McFarlin) in the field, but reports on the relative resistance of these varieties to cottonball have been inconsistent. In the field, susceptibility to cottonball depends on genetic interactions between *M. oxycocci* and the cranberry plant during primary infection of shoots, secondary infection of flowers, the overlap of bloom and spore production on shoots, and environmental factors such as temperature and moisture. But because infection of flowers is the economically important phase of the disease, and we know how to infect flowers under controlled conditions *(e.g.,* the greenhouse), our experiments focused on the susceptibility of the most popular cranberry varieties in Wisconsin--Ben Lear, Pilgrim, Searles, and Stevens--to floral infection. We found that following artificial inoculation in the greenhouse, these varieties did not differ in susceptibility to fruit infection (Fig. 5). In particular, Stevens, which some claim is relatively resistant, was at least as susceptible as the others. We speculate that it enjoys a reputation for resistance in the field only because many Stevens plantings are relatively young and disease pressure has not yet accumulated.
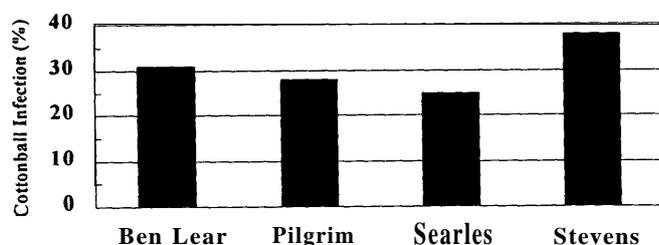
Figure5. Incidence of cottonball secondary (fruit) infection of popular cranberry cultivars in Wisconsin. Approximately 500 flowers of each variety were hand-inoculated in a greenhouse. The differences in percent infection are not statistically significant.

Summary and Recommendations

Experimental data and the observations of growers, crop consultants, and researchers are leading to a better understanding of cottonball. With this information, we are developing sustainable cottonball management programs that integrate sanitation, cultural practices, and fungicide use. The following recommendations should result in disease control that will be safe for humans and the environment and also delay the onset of fungicide resistance in populations of *M. oxycocci.*

- Re-flood beds after harvest to remove cottonball berries and mummies. This will reduce cottonball inoculum and other pests as well.
- Consider all the most popular varieties susceptible to cottonball. Don't expect a bed of Stevens to remain disease-free if planted next to a bed with cottonball.

- Control moss and avoid having areas of saturated sand in the spring when mummies germinate. Mummies germinate through sand, so you can "bury" last year's problem.
- Under "low disease pressure", skip shoot elongation sprays and spray only during bloom. "Low disease pressure" is a subjective term that will vary among growers. If coming into the season, you know you want to treat for cottonball but don't think it's bad enough to justify all four sprays, then consider it "low disease pressure".
- Just before bloom, scout for primary (shoot) infections so you can decide whether or not to spray during bloom. Look especially closely along ditches, wet areas, and where frost may have occurred.
- Two sprays are permitted during bloom. Be certain that the first one goes on at 10-20% bloom. These early flowers are the ones most likely to set fruit and therefore are the most important ones to protect.
- To the extent possible, spray a variety according to ᚕ∙ developmental stage, rather than treating early and late varieties at the same time.
- When using Orbit, do not go below 4 oz per acre. Sterol inhibitor fungicides generally do not perform well if rates are skimmed. Also, for other plant pathogens it's been shown that using lower rates of SIs actually promotes the development of fungicide resistance.