Fungicide Resistance in Turfgrass - An Introduction

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Author's note: This is the first in a three-part series looking at fungicide resistance in turfgrass.

Like many important discoveries in history, the first fungicide was discovered completely by accident. Bordeaux mixture, discovered in 1885 and applied to grapes in France, was originally meant to discourage passersby from picking the fruit. But it also was remarkably effective in controlling powdery mildew, and so the first fungicide was discovered and a fundamental shift in the way agricultural diseases are controlled had begun (De Waard, 1993). More effective fungicides were developed in the years to follow, and by the turn of the 20th century many non-selective and inorganic fungicides were being applied to agricultural and horticultural crops (Eckert 1988).

Nearly all fungicides developed before 1970 were multi-site inhibitors that protected the surface of the plant from pathogen infection, what would be known as contact fungicides today (Sisler, 1988). These fungicides were cheap and effective, and were the primary means of disease control for many growers. But beginning in the 1960's and influenced by the release of Rachel Carson's Silent Spring, public concerns over possible environmental contamination and mammalian toxicity led to the development of fungicides that were more selective in targeting the pathogen (De Waard, 1993). The selective nature of these fungicides left them more vulnerable to the development and proliferation of organisms that had developed resistance (Eckert 1988).

It is widely accepted amongst those who study fungicide resistance that the application of fungicides do not actually cause the fungi to become resistant to the fungicide. Instead, applications of fungicides control normal or "wild-type" isolates of fungi but cannot control those isolates that have undergone random mutations that render the fungicide ineffective (Couch 1995). With repeated applications of the same fungicide, the mutated isolates proliferate in the population and soon come to dominate the population (Figure 1). It is when these mutated isolates make up a significant proportion of the overall fungal population that we see a loss of disease control and observe "field" or "practical" fungicide resistance.

While much has been made about the loss of disease control due to fungicide resistance in the past 20 years, reports of resistance to fungicides have been around for over 40 years. The first reports of fungicide resistance were to the cadmium and mercury-based fungicides in the late 1960's (Cole *et al.*, 1968; Massie *et al.*, 1968). In the 1970's, shortly after their introduction into the market, dollar spot resistance was reported to the benzimidazole class of fungicides that includes active ingredients such as thiophanate-methyl and benomyl (Warren *et al.*, 1974; Warren et al., 1977). Widespread reports of decreased fungal sensitivities to the demethylation-inhibitor (DMI) class of fungicides has been documented with many pathogens since the early 1980's (Detweiler *et al.*, 1983; Leroux *et al.*, 1988; Koller *et al.*, 1991; Golembiewski *et al.*, 1995; Peever and Milgroom, 1994; Franke., 1998). Most recently, reports of resistance to the strobilurin class of fungicides has been

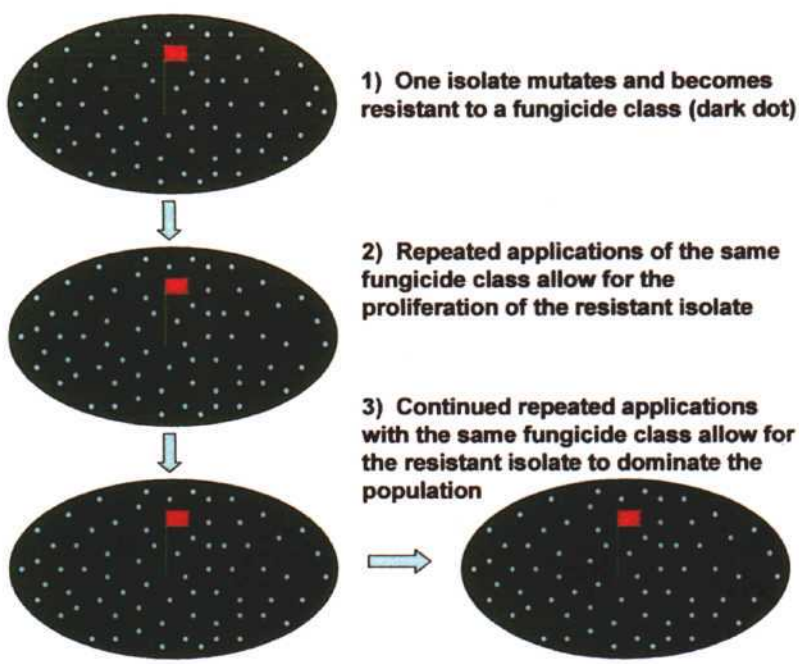

Figure 1: Under repeated applications of the same fungicide class, a single resistant isolate (dark dot) can proliferate and dominate the population.

observed in turfgrass and other crops as well (Wong and Wilcox, 2002). The speed and severity that resistance has developed to each of these fungicide classes has varied, and to understand why one must know the basics about how each of these classes inhibit fungal growth.

The benzimidazole class of fungicides inhibits fungal growth by interfering with microtubule assembly in the fungal cell, which in turn disrupts the development of the spindle fibers (Ishii, 1992). Thinking back to high school biology class, the spindle fibers are the structures that pull the chromosomes apart during cell division. So in a simple sense, benzimidazole fungicides inhibit cell division. The site where the fungicide binds to and inhibits cell division is controlled by one gene. If by chance there is a single mutation at that gene, the binding site will be altered and not allow for the binding of the benzimidazole fungicide. This fungal isolate has now obtained resistance to benzimidazole fungicides, and in the continued presence of benzimidazole fungicide applications will quickly proliferate and dominate the fungal population. Once the resistant isolate becomes the dominant isolate in the population, a sharp reduction in control in the field is observed. This drastic and rapid selection of resistant organisms in the population is known as disruptive or qualitative selection (Figure 2), and often leads to two distinctly different subgroups with very different fungicide sensitivities within the overall population (Koller and Scheinpflug, 1987).

The DMI group of fungicides is actually part of a larger class of fungicides known as the sterol biosynthesis inhibitors (SBI). While it is unknown exactly how other groups within the SBI class inhibit fungal growth, the DMI fungicides bind to a site in the fungal cell's sterol biosynthesis pathway (Koller, 1988). Sterols play an important role in many different cell functions,

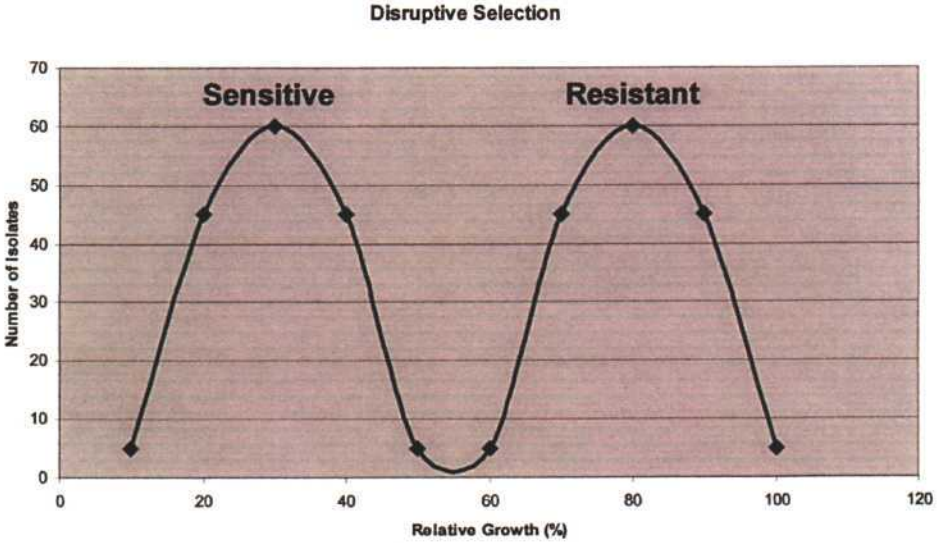

Figure 2: Repeated applications of the same fungicide class that act in a disruptive way can rapidly divide the fungal population into two subgroups with very different sensitivities to fungicides. Disease control failures in the field can develop rapidly. The benzimidazole class of fungicides acts in this manner.

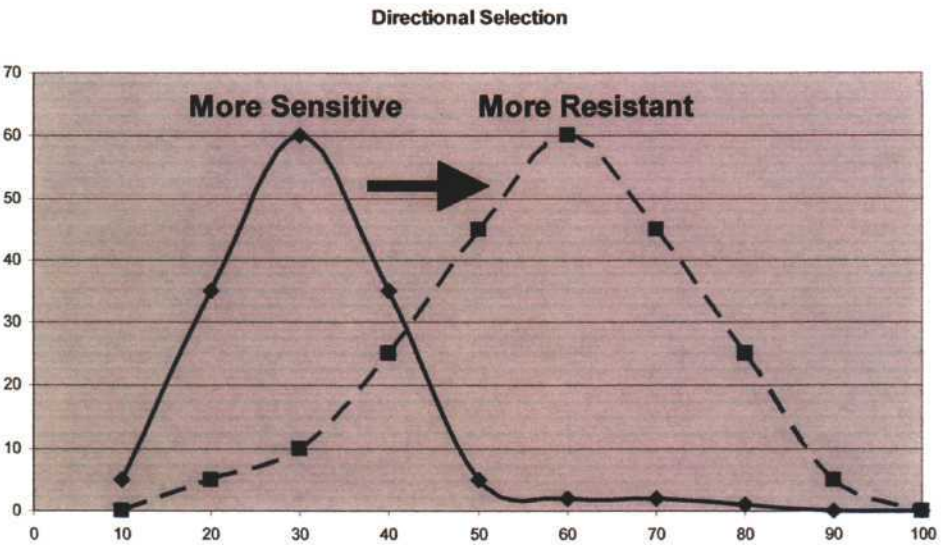

Figure 3: Repeated applications of the same fungicide class that act in a directional way can gradually shift the overall sensitivity of a fungal population towards being more resistant to the fungicide. Disease control failures develop slowly, even under repeated applications of the same fungicide class. The DMI class of fungicides acts in this manner.

including maintenance of the cell membrane and synthesis of hormones (Koller, 1992). In contrast to the benzimidazoles, several genes regulate the site that DMI fungicides bind to. This results in a gradual, step-wise process of worsening resistance with each subsequent mutation in the sterol biosynthesis pathway. With repeated applications of DMI fungicides, there is a gradual decrease in control of the target organism. This is observed in the field as shorter lengths of fungicide efficacy and disease "breakthrough" in higher pressure areas. This gradual resistance buildup in the fungal population is known as directional or qualitative selection (Figure 3), and often results in the delayed onset of observed fungicide resistance in the field for many years (Koller and Scheinpflug, 1987).

Strobilurin fungicides are also

part of a larger class known as the QOI fungicides. They work to inhibit fungal growth by inhibiting mitochondrial respiration in the fungal cell (Heany *et al.*, 2001). While the exact mechanism for development of resistance to the strobilurins is unclear, widespread field resistance in pathogens such as *Blumeria graminis, Venturia inaequalis*, and more recently *Colletotrichum cereale* in turfgrass has been documented (Wong and Wilcox, 2002; Wong, 2003). This rapid resistance development soon after the introduction of strobilurins to the market suggests that resistance develops in a manner similar to the benzimidazoles rather than the DMI's, but more research is needed to clarify this point.

Fewer new fungicide chemistries are being produced due to the onerous cost, and increased governmental regulation of older, effective fungicides such as chlorothalonil have left turfgrass managers with diminished disease control options. Many strategies for managing fungicide resistance have been touted for years, but little scientific data exists to actually support these strategies. Continued scientific research needs to go into the population dynamics of fungicide resistance to better understand how the resistant isolates relate to the sensitive ones in the turfgrass environment. This will allow for more effective fungicide management strategies, prolonging the effectiveness of our current fungicides years into the future.

References

Cole, H., Taylor, B., & Duich, J. (1968). Evidence of differing tolerances to fungicides among isolates of *Sclerotinia homoeocarpa*. *Phytopathology*, 58(5), 683-686.

Couch, H. B. (1995). Diseases of Turfgrasses, 3rd ed. Krieger Publishing Co., Malabar, FL.

Detweiler, A. R., Vargas, J. M., & Danneberger, T. K. (1983). Resistance of *Sclerotinia homoeocarpa* to iprodione and benomyl. *Plant Disease*, 67(6), 627-630.

De Waard, M. A., Georgopoulos, S. G., Hollomon, D. W., Ishii, H., Leroux, P., Ragsdale, N. N. et al. (1993). Chemical control of plant-diseases: problems and prospects. *Annual Review of Phytopathology*, 31, 403-421.

Eckert, J. W. (1988). Historical development of fungicide resistance in plant pathogens. Pages 1-3 in: Fungicide Resistance in North America. C. J. Delp, ed. The American Phytopathological

Society, St. Paul, MN.
Franke, M. D., Brenneman, T. B., & Stevenson, K. L. (1998). Stem rot of peanut: relationship between *in vitro* fungicide sensitivity and field efficacy of fungicides. *Peanut Science*, 25, 76-80.
Golembiewski, R. C., Vargas, J. M., Jones, A. L., & Detweiler, A. R. (1995). Detection of demethylation inhibitor (DMI) resistance in *Sclerotinia homoeocarpa* populations. *Plant Disease*, 79(5), 491-493.
Heany, S. P., Hall, A. A., Davies, S. A., Olaya, G. (2001). Resistance to fungicides in the QOI-STAR cross resistance group: Current perspectives. Pages 755-762 in: Brighton Crop Protection Conference - Pests and Diseases - 2000. Vol 2. Major Print, Nottingham, UK.
Ishii, H. (1992). Target sites of tubulin-binding fungicides. Pages 43-52 in: Target Sites of Fungicide Action. W. Koller, ed. CRC Press, Boca Raton, FL.
Koller, W. (1988). Sterol demethylation inhibitors: mechanisms of action and resistance. Pages 77-88 in: Fungicide Resistance in North America. C. J. Delp, ed. The American Phytopathological Society, St. Paul, MN.
Koller, W. (1992). Antifungal agents with target sites in sterol functions and biosynthesis. Pages 119-206 in: Target Sites of Fungicide Action. W. Koller, ed. CRC Press, Boca Raton, FL.
Koller, W., Parker, D. M., & Reynolds, K. L. (1991). Base-line sensitivities of *Venturia inaequalis* to sterol demethylation inhibitors. *Plant Disease*, 75(7), 726-728.
Koller, W., & Scheinpflug, H. (1987). Fungal resistance to sterol biosynthesis inhibitors - a new challenge. *Plant Disease*, 71(12), 1066-1074.
Leroux, P., Gredt, M., & Boeda, P. (1988). Resistance to inhibitors of sterol biosynthesis in field isolates or laboratory strains of the eyespot pathogen *Pseudocercosporella herpotrichoides*. *Pesticide Science*, 23(2), 119-129.
Massie, L. B., Cole, H., & Duich, J. (1968). Pathogen variation in relation to disease severity and control of *Sclerotinia* dollar spot of turfgrass by fungicides. *Phytopathology* 58(12), 1616-1619.
Peever, T. L., & Milgroom, M. G. (1994). Lack of correlation between fitness and resistance to sterol biosynthesis-inhibiting fungicides in *Pyrenophora teres*. *Phytopathology*, 84(5), 515-519.
Sisler, H. (1988). Fungicidal action and fungal resistance mechanisms. Pages 6-8 in: Fungicide Resistance in North America. C. J. Delp, ed. The American Phytopathological Society, St. Paul, MN.
Warren, C. G., Sanders, P., & Cole, H. (1974). Sclerotinia homoeocarpa tolerance to benzimidazole configuration fungicides. *Phytopathology*, 64(8), 1139-1142.
Warren, C. G., Sanders, P. L., Cole, H., & Duich, J. M. (1977). Relative fitness of benzimidazole-tolerant and cadmium-tolerant populations of *Sclerotinia homoeocarpa* in absence and presence of fungicides. *Phytopathology*, 67(5), 704-708.
Wong, F. P. (2003). Baseline sensitivity distribution of Colletotrichum graminicola (turfgrass anthracnose) to thiophanate-methyl and detection of resistance in California populations. *Phytopathology*, 93(6), S91
Wong, F. P., Wilcox, W. F. (2002). Sensitivity to azoxystrobin among isolates of *Uncinula necator*: Baseline distribution and relationship to myclobutanil sensitivity. *Plant Disease*, 86(4), 394-404.