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Fungicide Resistance Management for Early Blight

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Early blight, caused by *Alternaria solani,* is a very common disease of potato found in most potato-growing areas in Idaho. Although it occurs annually to some degree in most production areas, the timing of its appearance and the rate of disease progress help determine the impact on the potato crop. The disease occurs over a wide range of climatic conditions and depends in large part on the frequency of foliage wetting from rainfall, fog, dew or irrigation, as well as on the nutritional status of foliage and cultivar susceptibility.

Though losses rarely exceed 20 percent, if left uncontrolled, the disease can be very destructive. Disease severity can be especially severe in under-nourished crops due to insufficient nitrogen fertilization. Foliar symptoms of early blight first appear as small, irregular to circular dark-brown spots on the lower (older) leaves. As the disease progresses, these lesions enlarge and several lesions may coalesce to cover the entire leaf. Severely infected leaves eventually wither and die but usually remain attached to the plant. Severe infection of foliage by the early to mid-bulking tuber period can result in smaller tubers, yield loss and lower tuber dry matter content.

Traditionally early blight control has primarily depended on multiple fungicide applications typically applied at approximately 7- to 10-day intervals throughout the growing season. Currently, there is a wide variety of fungicides from which to choose for the control of early blight. However, the quinone outside inhibitor (QoI) fungicides (e.g. kresoxim-methyl, azoxystrobin, pyraclostrobin, trifloxystrobin, famoxadone, and fenamidone) are often favored because they offer broad spectrum protection against a wide range of fungal (e.g. early blight and white mold) and oomycete (e.g. late blight) diseases. Another major benefit of the QoI fungicides is that they have reduced environmental impact and reduced toxicity to mammals and bees as compared with conventional fungicides used to control early blight (e.g. chlorothalonil, mancozeb). However, they do show some toxicity towards aquatic organisms at high concentrations, but since they dissipate in the environment relatively quickly, there is less potential for long-term accumulation of these fungicides in soil and water and hence a lower risk to aquatic organisms.

The QoI fungicides work by disrupting respiration in fungal cells by binding to the so-called Qo site (the outer quinol-oxidation site) of the respiratory enzyme complex cytochrome bc_1. As a result of this narrow mode of action, there is an inherent risk for the development of pathogen resistance. The first QoI fungicides (kresoxim-methyl and azoxystrobin) were released commercially in Europe in the mid 1990's and in the United States in the late 1990's. A Section 18 registration was granted by the Environmental Protection Agency (EPA) that allowed potato growers in Wisconsin to use azoxystrobin (Quadris) for control of early blight mid-season in 1998, and in 1999 the fungicide received full label registration for commercial use on potato. Since 1999, the persistent and intensive use of azoxystrobin and other QoI fungicides alternated with chlorothalonil on commercially grown potato crops in the Midwest and throughout the United States have increased the selection pressure for the development of QoI fungicide resistance in *A. solani*. As a result, the efficacy of these fungicides started to decline after a couple years, and isolates of *A. solani* with reduced sensitivity or complete resistance to QoI fungicides have now been identified. Furthermore, field surveys have shown that these resistant isolates are now widespread throughout the Midwest, eastern U.S. and Canada.

The mode of resistance at the molecular and genetic levels and the existence of mutants resistant to the QoI fungicides have been known for several years in several organisms, including *A. solani*. In *A. solani*, there are two mutations known to cause resistance to the QoI fungicides. The first mutation called F129L causes reduced sensitivity to fungicides, whereas the second called G143A results in complete loss of sensitivity or a full resistance response. Results from field surveys carried out in Wisconsin from 2001 to 2003 showed that the population of *A. solani* isolates present in commercial-growers fields was 20 times more resistant to azoxystrobin than "wild-type" isolates collected prior to the introduction of the QoI fungicides in 1998. Further studies found that isolates with the two mutations were much more common than wild-type isolates of *A. solani*, with only 4 percent of isolates found in 2002 being wild-type.

In 2008, many Idaho growers reported the failure of the traditionally effective QoI strobilurin fungicides (azoxystrobin [Quadris®] and pyraclostrobin [Headline®]) to control early blight. This may also be due to the development of fungicide resistant isolates of *A. solani*. Fungicide resistant isolates of *A. solani* have been reported in Idaho (Jeff Miller, personal communication), but it is not known which type of mutation is responsible for the reduction in sensitivity and/or whether both types of mutation are present in the populations of *A. solani* present in Idaho. However, an alternative hypothesis is that these fungicide resistance problems may be due to misdiagnosis of early blight. Brown leaf spot, caused by *A. alternata* is also a com-

mon disease of potato in Idaho and other potato-growing regions of the U.S., and is often confused with early blight as its symptoms are very similar. The pathogens causing these two diseases are also very closely related. However, unlike the early blight fungi (*A. solani*) where fungicide resistance is known to occur, the brown leaf spot pathogen (*A. alternata*) is inherently more resistant to strobilurins and has never been well controlled by this class of fungicides. Due to the similarity of these two pathogens and their disease symptoms it may be possible that growers are confusing brown leaf spot for early blight and, thus, reporting poor disease control of early blight.

Protectant fungicides recommended for the control of brown leaf spot (e.g. maneb, mancozeb, and chlorothalonil) are also effective against early blight when applied at approximately 7- to 10-day intervals. Due to the fact that the brown leaf spot pathogen, *A. alternate*, is inherently more resistant to the strobilurins than early blight, these fungicides should not be used to control early blight if it is suspected that brown leaf spot is also present in the field. Alternative fungicides which have shown efficacy against both early blight and brown leaf spot contain famoxodone (e.g. Tanos®), difenoconazole (e.g. Revus™ Top), pyrimethanil (e.g. Scala™), fenamidone (e.g. Reason®) and boscalid (e.g. Endura®).

The application of foliar fungicides is not necessary in plants at the vegetative stage, when they are relatively resistant. Accordingly, spraying should commence at the first sign of disease or immediately after bloom. Protectant fungicides should be applied initially at relatively long intervals and subsequently at shorter intervals as the crop ages. Early-season applications of fungicides before secondary inoculum is produced often have minimal or no effect on the spread of the disease, and early blight and brown leaf spot can be adequately controlled by relatively few fungicide applications if the initial application is properly timed. Research has shown that it is possible to effectively control early blight with only three fungicide sprays of an effective fungicide such as boscalid or famoxodone, rotated with a protectant fungicide such as chlorothalonil.

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Did you know?

Although called early blight, most potato varieties are fairly resistant to infection by *A. solani* during the vegetative growth stage and as such the disease rarely shows up early in the season. Early blight begins to show itself about the time potato blossoms appear, which, in Idaho, is usually around the beginning of July.

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