

## FITNESS OF FUNGICIDE RESISTANT STRAINS OF THE PINK ROT PATHOGEN AND USE OF PHOSPHITE BASED FUNGICIDES IN MANAGING PINK ROT OF POTATO

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Pink rot is caused by the soil-borne pathogen, *Phytophthora erythroseptica*. The “fungus” is not a true fungus, but considered a water mold (Oomycete). As a result, some fungicides that interfere with the biochemical processes of true fungi are ineffective in managing water mold “fungi.” However, *P. erythroseptica* looks like a fungus, grows like a fungus, and in many other ways behaves like a fungus, so we can talk about it as a “fungus.” Growers in eastern Idaho experienced significant losses due to pink rot in 2003. The pathogen infected plant stems, stolons and tubers in early August, resulting in early plant death and severe tuber rot. Some fields were abandoned due to the extent of pink rot decay. Extended periods of warm weather may have been responsible directly by making the environment more favorable for disease, or indirectly by causing growers to apply excess irrigation water in an attempt to cool the potato crop.

The pink rot pathogen overwinters in potato fields via oospores. Oospores are thick-walled structures resistant to environmental conditions typically considered adverse to the pathogen. Primary pink rot infection in storage can lead to secondary infection by soft-rotting bacteria. This in turn can lead to extensive tuber break-down in storage. Several disease management practices are required to minimize pink rot damage. These include water management, establishing a good skin set prior to harvest, harvesting tubers below 68°F, and using fungicides that have mefenoxam as the active ingredient (Ridomil Gold products, Ultra Flourish, and Flouronil). Use of these fungicides has been questioned recently, however, with the detection of pathogen strains resistant to mefenoxam in Idaho in 1998. Many of the fields in eastern Idaho with problems in 2003 had received multiple applications of Ridomil Gold.

A survey of tubers from the Magic Valley and eastern Idaho seems to indicate that the frequency of fungicide-resistant isolates is increasing (Table 1). The survey is not complete, and does not include all counties. As infected tubers are submitted to the University of Idaho diagnostic laboratories, tests are done to determine how sensitive the pink rot pathogen is to mefenoxam. With the widespread use of Ridomil it is not surprising that fungicide resistance is increasing. Almost all of the fungicide-resistant isolates have come from eastern Idaho. Only one resistant isolate has come from the Magic Valley.

Table 1. Occurrence of mefenoxam resistance in the pink rot pathogen in southern Idaho. The percentage of isolates is given in parenthesis.

Fungicide response	2001	2002	2003
Resistant	47 (66)	118 (77)	171 (80)
Sensitive	24 (34)	35 (23)	43 (20)
Total	71	153	214

It is not uncommon for fungi to develop some kind of fitness cost when fungicide resistance develops. This means that in the absence of the fungicide, the fungicide-sensitive fungi survive

and reproduce more efficiently than the fungicide-resistant fungi. Studies have been conducted at the University of Idaho to compare the fitness of mefenoxam-resistant and mefenoxam-sensitive isolate of the pink rot pathogen. A total of 30 isolates (15 mefenoxam-resistant and 15 mefenoxam-sensitive) were selected for tests and grown on artificial growth media at five different temperatures. The rate of fungal growth was measured, along with the number of oospores (sexual reproductive bodies) produced per volume of growth medium. These tests were repeated on five separate occasions to ensure reliability of results.

Table 2. Growth rate and oospore production of mefenoxam-resistant and mefenoxam-sensitive isolate of the pink rot pathogen.

Fitness parameter Fungicide sensitivity class	Temperature (F)				
	50	60	70	77	86
Oospore production (oospores/mm <sup>3</sup> )					
Sensitive	5 a	17 a	49 a	13 a	0
Resistant	278 b	302 b	287 b	239 b	0
Growth rate (mm/day)					
Sensitive	2.4 a	4.0 a	5.9 a	6.1 a	1.1
Resistant	7.5 b	10.2 b	14.0 b	15.1 b	3.6

Values with different lowercase letters are significantly different from each other ( $P > 0.05$ ).

From 50 to 77 F, mefenoxam-resistant isolates of *P. erythroseptica* were able to produce significantly more oospores than mefenoxam-sensitive isolates (Table 2). Additionally, mefenoxam-resistant isolates grew faster (higher growth rate per day). Fast growth of mefenoxam-resistant isolates has been so consistent, that at the diagnostic lab at the University of Idaho we can now estimate the mefenoxam sensitivity of an isolate based on how fast it grows out of infected tissue onto artificial growth media. At high temperatures (86 F), both classes of isolates grow and produce oospores similarly. These results indicate that mefenoxam-resistant strains of the pink rot pathogen may be more fit than mefenoxam-sensitive isolates. However, further research is needed to determine how long the different isolates can survive in the soil.

Phosphite based products (salts of phosphorous acid) offer some hope in managing pink rot caused by mefenoxam-resistant isolates. Two phosphite-based products that have been tested at the University of Idaho include Phostrol (Nufarm Americas) and Fosphite (J. H. Biotech). Phosphite based products are directly harmful to the growth of the water mold fungi, but are also known to stimulate natural plant defenses. Phosphite is systemic, moving both up and down in the plant. This allows for root and tuber protection from foliar applications. Recent research conducted by the University of Idaho and Washington State University has shown that when phosphite products are applied to the foliage, tubers are protected from late blight and pink rot.

A trial performed at the Aberdeen Research and Extension Center in 2003 showed that Phostrol gave some protection to tubers against infection by the pink rot pathogen (Table 3). Tubers were challenged with a mefenoxam-sensitive isolate two weeks after harvest. Where no fungicides were applied, 84% of all inoculated tubers developed pink rot. All treatments except the Ridomil Gold in-furrow application significantly reduced the amount of pink rot. The disease control provided by Phostrol was not as good as the control provided by Ridomil, but there was a significant reduction compared to the untreated control. Applying the foliar pre-pack Ridomil

Gold three times did not offer any better protection than applying only twice. Combining Ridomil Gold and Phostrol did not give any better protection than Ridomil Gold alone.

Table 3. Control of pink rot by Ridomil and Phostrol at the Aberdeen R & E Center (cv. Russet Norkotah) as determined by a laboratory test.

Treatment	Pink rot incidence
Untreated control	84 a
Ridomil Gold EC in-furrow at planting (6.2 fl oz/acre)	70 ab
Ridomil Gold EC at hilling (6.2 fl oz/acre)	66 b
Ridomil Gold Bravo foliar (2× at 2 lb/acre each time)	44 c
Ridomil Gold Bravo foliar (3× at 2 lb/acre each time)	44 c
Phostrol foliar (3× at 10 pt/acre each time)	66 b
Ridomil Gold Bravo foliar 2× (2 lb), Phostrol 3× (10 pt)	48 c

In another trial conducted at Bonners Ferry, Fosphite was tested for its ability to control late blight compared to standard fungicide programs. Healthy tubers from the trial were inoculated with a mefenoxam-resistant isolate of the pink rot pathogen after the tubers had been in storage for two months. Tubers from Fosphite treatments had significantly less pink rot than tubers from treatments not receiving Fosphite (Table 4). Because this test was done after several weeks in storage, the level of infection was not high. However, foliar-applied Fosphite still offered some protection to the tubers.

Table 4. Effect of Fosphite on pink rot incidence (mefenoxam-resistant isolate) in a laboratory test.

Treatment (# of apps, rate per acre)	Pink rot incidence
Untreated control	24 a
Fosphite (3×, 10 pt each time)	7 b
Fosphite (as above), Quadris (1×, 6.2 fl oz), Dithane (2×, 2 lb)	4 b
Quadris (as above), Dithane (6×, 2 lb)	27 a

Various post-harvest tuber treatments have been used in an attempt to reduce tuber rots (including pink rot) in storage. Since phosphite fungicides showed some promise as foliar applications, it was decided to try them as post-harvest applications. Oxidate and Zoxamide (the active ingredient in the Gavel fungicide) were also included in the test. Washed tubers were dipped in a suspension of *P. erythroseptica* zoospores and then treated 0, 1, 2, 4, and 6 hours later. Oxidate, Zoxamide, and Phostrol were all effective at reducing disease incidence when applied immediately (0 hour) after inoculation (Table 5). When tubers were treated one hour after inoculation, only Zoxamide and Phostrol were effective. After two hours, Phostrol and Zoxamide were effective, with Phostrol performing the better of the two. Phostrol was the only treatment to reduce pink rot incidence when tubers were treated 4 or 6 hours after inoculation.

Table 5. Effect of post-inoculation interval on post-harvest treatment for incidence of pink rot (cv. Russet Burbank).

Treatment (lb a.i./ton tubers)	Post inoculation interval (hours)				
	0	1	2	4	6
Untreated control	87 a	92 a	88 a	90 b	100 a

Oxidate (1:50 dilution)*	43 b	85 ab	87 ab	98 a	95 a
Zoxamide (0.124)	3 c	70 b	72 b	92 b	97 a
Phostrol (0.669 lb)	0 c	3 d	3 c	22 c	29 b

\*5,400 ppm rate.

Values in the same column with different lowercase letters are significantly different from each other.

In summary, pink rot continues to be problematic in eastern Idaho. Increasing mefenoxam resistance in the pink rot pathogen underscores the need for new disease management options. Foliar and post-harvest applications of phosphite based fungicides are effective in reducing pink rot incidence. Foliar phosphite applications do not provide the same level of protection that mefenoxam may provide with mefenoxam-sensitive populations, but phosphite is effective against mefenoxam-resistant pink rot isolates. When treating tubers post-harvest, Oxidate was somewhat effective when applied immediately after inoculation. Phostrol provided protection even when applied as much as six hours after inoculation.