ALTERNATIVE METHODS FOR CONTROL OF POTATO DISEASES

by
Dennis C. Gross [1]

Early dying of potatoes is usually caused by the interactions of one or more soilborne pathogens which can be classified as either wilt-inducing or stress-inducing pathogens. The fungus, Verticillium dahliae, and the bacterium Erwinia carotovora, are the two pathogens responsible for causing the wilt symptoms (5,7), but other soilborne pathogens now appear to increase disease severity by stressing plants to make them more susceptible to one or both of the wilt pathogens. This report will briefly describe these pathogens and how they are able to survive from one season to the next. Our research towards developing a broad-spectrum biocontrol method for controlling one or more soilborne pathogens involved in early dying is also discussed.

Vascular Wilt Pathogens

Verticillium dahliae is generally considered the most important pathogen involved in potato early dying (6,7). Although this fungus is sometimes the sole cause of early dying, severe cases of early dying usually involve an interaction with Erwinia carotovora or one of the stress-inducing pathogens. The fungus resides in the soil as microsclerotia. In response to root exudates the microsclerotia germinate to form hyphae which can directly penetrate nearby roots early in the season. The fungus invades the root cortex, gaining entry into the xylem vessels by which it can systemically spread throughout the plant. It is during this systemic invasion of the xylem that the wilt and chlorotic symptoms begin to occur. As plants die the fungus produces the thick-walled, dark microsclerotia which serve as the resting structures of the fungus.

Erwinia carotovora has long been known to cause tuber soft rots and blackleg of potato. Only recently has there been strong evidence that this bacterium can cause the typical wilt symptoms associated with potato early dying (5,7). Greenhouse inoculations of potato plants with E. carotovora alone usually result in little or no symptom development. In contrast, coinoculations with V. dahliae or pathogens which stress the plants stimulate the bacterium to invade the vascular system to cause or accentuate wilt symptoms. In contrast to V. dahliae, the bacterium cannot live in soil for long periods of time; E. carotovora maintains itself by living on the roots of alternate crop plants or weeds.

---

[1] Associate Professor, Department of Plant Pathology, Washington State University, Pullman, Wa.

This Presentation is part of the Proceedings of the 1989 Washington State Potato Conference & Trade Fair.

A common source of the bacterium is contaminated potato seed pieces, although recent evidence shows the bacterium to occur in water reservoirs used as sources of irrigation such as the Columbia river (1). During midseason, the bacterium typically colonizes root and foliar potato surfaces to high populations, sometimes exceeding one million cells per gram of tissue (3). Large increases in bacterial populations correspond with the occurrence of higher temperatures and the development of a dense canopy.

Potato Stress Pathogens

Several soilborne microorganisms can contribute to potato early dying by stressing potato plants to make them more susceptible to invasion by one or both of the wilt pathogens. Evidence from the Columbia Basin and elsewhere (4) shows that root-lesion nematodes, such as Pratylenchus penetrans, increase susceptibility of potatoes to systemic infection by the wilt pathogens. Moreover, the effect of root-lesion nematodes is most pronounced when plants are infected with both wilt pathogens.

Other soilborne pathogens capable of inducing stress are the fungi, Pythium and Fusarium sambucinum. Pythium typically attacks roots and fleshy tissues to cause root rots and tuber soft rots. The fungus lives in the soil and is readily found in nearly all potato fields surveyed in the Columbia Basin. Although Fusarium has not yet been shown to affect the severity of early dying, in a 1987 field trial at Othello we demonstrated that the fungus can interact with E. carotovora to cause a significant decrease in total tuber yield.

Control of Potato Early Dying

Although there is no single method for controlling early dying, practices which incorporate sound management practices can lessen disease severity. Avoidance of water or nutrient stresses to potato plants by close control of irrigation rates and levels of nutrient availability can reduce the severity of disease. For example, poor distribution of available nitrogen within the rhizosphere has been shown (2,7) to increase the severity of early dying in Idaho. Nevertheless, most early dying control programs are aimed at reducing the inoculum potential of soilborne pathogens, especially V. dahliae, by chemical fumigants such as metham-sodium and chloropicrin (2). Although fumigant treatments are usually aimed at killing the microsclerotia of V. dahliae residing in the soil, they are also beneficial in reducing populations of nematodes and the resting spores of Pythium and F. sambucinum. However, soil fumigation has no appreciable effect on E. carotovora since the bacterium is introduced into potato fields at planting on seed pieces or later by the use of contaminated irrigation water (1).

Future Prospects for Using Biological Control

The use of root-inhabiting bacteria to suppress soilborne pathogens and promote plant growth is a potentially useful means of controlling potato early dying and reduce dependence on or the need for chemicals.

Because early dying is usually caused by the interactions of more than one pathogen, we have been attempting to develop a method of biological control effective against a broad spectrum of potato pathogens. From these efforts we have selected bacteria, identified as strains of Pseudomonas fluorescens, which are especially promising based on: (i) their ability to grow to high populations on potato roots; (ii) persist on developing root systems throughout the growing season; (iii) produce antibiotics inhibitory to several potato pathogens; and (iv) yield significant levels of disease control in greenhouse and field trials.

Our Pythium tests have been conducted at Plymouth, Wa. in a sandy soil conducive to this fungus. In 1986, we obtained our most exciting results due to favorable conditions for the development of seed piece decay as a result of infection by Pythium, which naturally occurred in the soil. Both emergence and plant height were substantially increased by Pseudomonas strains W4F1080 and R4a-80; at harvest a significant (P=0.05) increase of 18% in marketable yield was measured.

The effects of pseudomonad strains on the interactions between E. carotovora subsp. atroseptica and F. sambucinum have been tested at Othello, Wa. Treatments consisted of a given pseudomonad strain applied to seed pieces together with 10^6 cfu/ml E. c. atroseptica, 2.5 x 10^6 conidia/ml F. sambucinum, or combinations of the two pathogens. In June, we observed the occurrence of blackleg which was most serious in those blocks treated with both pathogens, occurring at a relatively high incidence of 12% (Table 1). Plants treated with F. sambucinum alone did not show blackleg while plants treated with E. c. atroseptica alone showed about a 1% incidence of this disease. But of most importance was the observation that strains W4F1080 and W4F1043 suppressed blackleg 32-45% as compared to the check. This indicated that the pseudomonads decreased blackleg not through control of E. c. atroseptica but through suppression of F. sambucinum, which interacted with the bacterial pathogen to cause disease. Both total and U.S. No. 1 yields were significantly reduced in the E. c. atroseptica treatments; statistical analysis showed a significant (F=2.5) interaction between E c. atroseptica and F. sambucinum to decrease total yield.

Table 1. Effect of interactions among E. c. atroseptica (Eca), F. sambucinum (Fs) and P. fluorescens on blackleg at Othello, Wa. 1987.

	Blackleg (%)[a]			
Treatment	−Eca −Fs	+Eca −Fs	−Eca +Fs	+Eca +Fs
Check	0	1.4	0	11.9
R4a-80	0	0.9	0	9.8
W4F1043	0	0.6	0.6	8.1
W4F1080	0	1.8	0	6.5

[a]LSD (P=0.05) = 3.6

Despite the promising results described above, the biological control system will require further improvements before it can be used commercially. The most serious limitation is that existing biocontrol systems are not as effective as chemical treatments aimed at reducing the inoculum potential of a target pathogen. Although biocontrol does not necessarily need to equal that of chemical control to prove useful, it must give predictable results if it is to gain acceptance by growers. Another problem is that because potato early dying is a disease complex composed of more than one pathogen, an effective biocontrol agent will need to be broad in its ability to control soilborne pathogens. Although these may seem to be insurmountable problems, advances in biotechnology and continued improvements in biocontrol systems should eventually lead to their use in agriculture.

References

1. Cappaert, M.R., M.L. Powelson, G.D. Franc and M.D. Harrison. 1988. Irrigation water as a source of inoculum of soft rot erwinias for aerial stem rot of potatoes. Phytopathology 78:1668-1672.

2. Davis, J.R. 1985. Approaches to control of potato early dying caused by Verticillium dahliae. Am. Potato J. 62:177-185.

3. Gross, D.C. 1988. Maximizing rhizosphere populations of fluorescent pseudomonads on potatoes and their effects on Erwinia carotovora. Am. Potato J. 65:697-710.

4. Martin, M.J., R.M. Riedel and R.C. Rowe. 1982. Verticillium dahliae and Pratylenchus penetrans: causal agent of early dying of potato in organic soil in Ohio. Phytopathology 72:640-644.

5. Powelson, M.L. 1985. Potato early dying disease in the Pacific Northwest caused by Erwinia carotovora pv. carotovora and E. carotovora pv. atroseptica. Am. Potato J. 62:173-176.

6. Rowe, R.C. 1985. Potato early dying -- a serious threat to the potato industry. Am. Potato J. 62:157-161.

7. Rowe, R.C., J.R. Davis, M.L. Powelson and D.I. Rouse. 1987. Potato early dying: causal agents and management. Plant Dis. 71:482-489.