PINK EYE DISEASE OF POTATO

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Pink eye disease of potato gets its name from the puffy, pink areas on and, sometimes, between the eyes of affected tubers. This early season symptom is observed only at harvest and usually disappears within hours after harvest. Unfortunately, a more serious late season tuber symptom often occurs soon after disappearance of the ephemeral pink eye symptom. The early season lesions on the periderm progress into thick, corky patches, a symptom that is referred to by the descriptive terms corky patch and bullhide. On russet cultivars, these later symptoms include flaking, cracking and sloughing of the periderm and has been called water scab. If a shallow cut is made below the affected patchy areas, a water soaked, rusty-brown area of discoloration can be seen extending only a small distance into the tuber. Even after extended storage, the corky, water soaked, discolored areas penetrate to a maximum depth of 5-10 mm. On rare occasions, a bud end rot can also be found in the field which may become invaded by Erwinia soft-rot bacteria and result in excessive tuber decay.

Pink eye has been found on many white cultivars and the russets Burbank and Norkotah. The disease has not been found on red-skinned cultivars. There are no vine symptoms, but there may be an association between pink eye and Verticillium wilt. The disease does not appear to be seed-borne and affected seed will germinate normally, provided the eyes are not killed by the corky patch symptom. Pink eye has only been reported from the northern United States and southern Canadian potato production areas, and is not known to occur in Europe.

The cause of this disease is unknown although many different possibilities have been investigated for over 20 years. We do not believe there is a connection between pink eye and nutrient imbalances or chemical agents such as herbicides or sprout inhibitors. The disease has been associated with a number of conditions in the field, but the most consistent association has been with wet conditions at or near harvest time. Over the last five years, we have isolated several common plant pathogenic fungi and bacteria from affected tuber tissue that could be responsible, including Verticillium, Rhizoctonia, Fusarium, Helminthosporium solani and Pseudomonas fluorescens. None of these pathogens have been isolated from pink eye tubers with repeatability or consistency, and often we may recover more than one pathogen. We are not certain that this is a disease caused by a plant pathogen or a physiological disorder.

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We, and others, have been unable to reproduce the symptoms of pink eye under controlled conditions in the greenhouse or field. Potato breeding programs in the northeastern United States have been selecting for resistance to this disease for the past 20 years.

Historically, pink eye has been classified as a minor disease that is economically unimportant. However, in the past ten years it has become increasingly more important and has occasionally caused severe economic losses in the fresh market and processing segments of the industry. The early and late symptoms detract from fresh pack appearance and have been a basis for rejection of fresh pack shipments. The late season corky patch, bullhide symptoms are a problem to the processing industry, especially the chip processors, because after prolonged storage the thickened patches cannot be removed during the normal peeling process and result in physical defects in the finished chips. Loads have been rejected from processing based on the presence of excessive pink eye.

One common characteristic of tubers that show the corky patch symptom is the presence of a translucent, water-soaked "stress zone" 1-10 mm thick in the tuber tissue beneath the patches. This stress zone is highly fluorescent under black light. The consistent occurrence of the fluorescent stress layer provided us with a new angle from which to study the disease.

Microscopic observations of the fluorescent tissue showed that the corky layer resembled the pattern we usually associate with wound periderm development, indicating that a wound healing or a wound response of some sort might have been responsible. We used high pressure liquid chromatography (HPLC) to investigate the fluorescent activity in order to determine the source of the compounds; tuber tissue or pathogen. Extracts were prepared from fluorescent tissues of pink eye affected tubers and compared to known fluorescent standards.

We detected significant amounts of three host-produced fluroescent compounds, chlorogenic acid, scopoletin and esculin. These are all phenolic compounds, specifically phenylpropanoids, and are part of the potato plant biochemical wound response to invasion by plant pathogens. Only one of the compounds, chlorogenic acid, was found in healthy tissue. None of the three compounds is produced by P. fluorescens, a bacterium found naturally occurring in plants that produces fluorescent compounds. The same compounds have been detected, in similar amounts, in potato tissue infected with both Fusarium sulphureum, the dry rot organism, and with Helminthosporium solani, the silver scurf organism. We also found small amounts of scopoletin in tubers with pink rot, a disease caused by Phytophthora erythroseptica.

The HPLC not only allowed us to identify the compounds, it also allowed us to get approximate concentrations of the compounds in tuber tissue. Just recently, we have been able to add these phenolic compounds, at normal physiological concentrations, to culture media and observe the effect on suspected pathogenic fungi. We have observed growth reductions in cultures of Fusarium but not in cultures of Helminthosporium.

Based on our previous information and these new studies, we have arrived at the following conclusions concerning the pink eye disease of potatoes:

1. Altered water relations causes the early season symptom and changes the susceptibility of the tuber to infection by pathogens.

2. A number of common pathogens can initiate infection.

3. In response to altered water relations and the presence of the pathogen, both the structural wound healing and the biochemical wound response is triggered in the tuber.

4. The deposition of heavy cork layers and the accumulation of fluorescent compounds occurs. The fluorescent compounds are produced to slow the invasion of the pathogenic agent and to participate in building new cork layers. However, the process does not stop with the establishment of a wound periderm layer, as under normal conditions. Due to the presence of the pathogen, cork continues to be produced to the outside of the pink eye site, resulting in the bullhide, water scab, corky patch symptom. The pathogen may be acting as an irritant in this case.

It seems clear that the entire process begins with altered water relations and concludes with a physiological response to a pathogen. The key to prevention and control appears to be proper water management late in the season.

We are closer, but we are still looking for the answer to the cause and control of this problem.