RING ROT OF POTATO - AN OVERVIEW

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Ring rot has probably been present in potatoes for hundreds of years but was not serious enough to be reported until 1913 in Germany. It soon became a problem everywhere potatoes were grown and was reported in the United States in 1934. This disease has never been a widespread epidemic in the United States. There are many other diseases of potatoes that play a larger role in reducing total production but few other diseases cause such devastation in individual fields. The sporatic but potentially severe nature of this disease and the seemingly unpredictable way it appears in fields has made this disease perhaps the most feared of all in the Western United States.

In Washington, there are documented cases where over 75% of the plants in large areas of the field were infected with this organism. Crops over 50% infected are often not harvested because the crop is worth less than the cost of harvest. However, most common reported losses range from one to twenty-five percent and many infestations of less than one percent are not reported. Loss due to ring rot is definitely not limited to the reduced yields. The per unit value of the harvested crop is greatly reduced because at times the entire field cannot be sold due to tuber rot or discoloration that is not visible from the exterior and cannot be sorted out by hand.

For the past five or six years the incidence of this disease has been rapidly increasing in the state of Washington. Reasons for this increase are not known. The incidence of this disease took a dramatic drop in the state last year. This could be taken as a sign of better disease control. However, one year does not make a trend. There are new techniques and programs being developed and if this research is carried to completion the seed industry may be able to identify seed lots with latent or trace infection so that field problems can be avoided.

The causal agent of this disease is a short, non-motile rodshaped bacteria called Corynebacterium sepidonicum. It can be identified by a number of methods: 1) potato plant symptoms, 2) size and shape of the bacteria, 3) a positive reaction in a gram stain and 4) sereolical methods. A positive identification of this pathogen usually depends on the combined use of a number of the above methods. This highly infectious organism survives in living potato plants. However, it can also over-winter as a contaminant on any cool, dry surface. It will not live in a free state in soil under normal cultural conditions for the potato. Infection takes place when the bacteria are brought into contact with an injured surface of a potato plant usually the cut surface of a seed piece. The bacteria infests the vascular system of the plant and can quickly multiply. The plant is damaged by the partial blocking of its vascular system and the disruption of the distribution of water throughout the plant. However, the most serious disruption for the potato plant comes from a toxic substance the bacteria produce. The plugging of the vascular system, combined with the toxin, causes the potato plant to undergo a number of characteristic symptoms. The extent of these symptoms and how quickly they progress depend on temperature, length of growing season, potato variety, plant vigor and moisture stess. If infected plants maintain a vigorous growing condition through proper fertilization and watering and are under little moisture and temperature stress, symptoms may be delayed, light, or even non-existent. On the other hand, if plant growth is retarded due to lack of fertilization or stressed due to hot weather and a high water demand, symptoms progress rapidly. Growing conditions in the Columbia Basin can cause severe symptom expression in ring rot infected plants. Prior to blooming, infected plants are slightly stunted with shortened upper inner nodes, somewhat reduced leaf size and rossetting and yellowing of the younger terminal leaves. The "green dwarf" symptoms last only a short time and are difficult for even a highly trained person to see in the field. Within a few days to several weeks after the first bloom, the infected plants develop yellow mottling and chocolate brown dead areas between the veins

and along the upward cupped leaf margins. Sap squeezed from the lower stem at this time will often appear milky due to high bacteria numbers. Some of the tubers may begin to show the characteristic discoloration and light browning and separation of the vascular ring. Tubers under an infected hill will be in various stages of rotting as the disease progresses. Some tubers may rot completely and become a paper shell due to secondary organisms while others remain symptomless. A few of the tubers of a hill may show the classic "ring rot" stage where the xylem ring seen with a cross section through the tubers has separated and is brown, rotted and filled with a viscous cheesy liquid. Some tubers may have shallow dark cracks as the advancing rot breaks through the tuber skin, a condition that is especially common in the Norgold Russet. The plant may die and become overgrown by neighboring plants or blend in with those dead or dying from other causes. Low infestations in the field can go unnoticed until harvest.

This organism can live over the winter as a contaminant on cool, dry surfaces, however, it is most commonly believed to be maintained as a low-level contaminant in potato seed. The potential severity of even low levels of this organism in seed potatoes is recognized by all seed certification agencies and no level of this disease is tolerated if identified in seed fields or storages. All conscientious potato seed growers conduct strict sanitation procedures to reduce a chance contamination of their crop. However, due to the highly infectious nature of this organism, a number of potato seed fields are rejected for certification each year.

Despite diligent sanitation and inspection, ring rot contaminated potatoes continue to be certified and sold. Disinfectants are never 100% effective in cleaning up all contamination and due to the highly infectious nature of this organism it takes only a few infected plants in a seed field to cause a ring rot problem. A few infected plants can be easily missed by inspectors and growers. Almost all potato seed is grown in cool, short season areas and the bacteria can remain latent in symptomless plants. Infected tubers can seldom be detected in storage since low temperatures slow or even stop the further progression of ring rot and other secondary rot symptoms inside the tubers.

In the spring most seed is bulk loaded into trucks and transported to a buyer in a production area. Bacterial cross contamination of seed lots can occur unless the truckbed, tarp and other handling equipment are carefully cleaned and disinfected. The incidence of ring rot in a field planted to whole uncut infected seed would be considerably more if the seed had been cut prior to planting. This point is illustrated by the observation that those areas of the world that always use uncut single drop seed rarely have a severe outbreak of ring rot.

When an infected seed potato is cut in a mechanical seed cutter, the knives and all other surfaces that come into contact with the cut seed become contaminated. Much of the clean seed that follows will be infected as it is cut or as it rubs against other contaminated surfaces such as belts, other seed pieces, workers gloves and truck beds. Seed piece contamination will drop as more clean seed is run through the machine. The more infected tubers in the seed lot the higher the bacterial contamination and the higher the infection in the field. Poor or no sanitation between lots of seed result in contamination of healthy lots of seed subsequently cut. Careful sanitation procedures during seed handling and cutting can reduce but will not eliminate losses due to ring rot.

All concerned should hope that losses will not continue on in the future at the same rate as they have in the past. Perhaps we need to take a fresh look at our ring rot control program further researching new techniques and seed screening programs for ring rot detection in seed. Working together we could make this disease a curiosity rather than the disaster it can be today.

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