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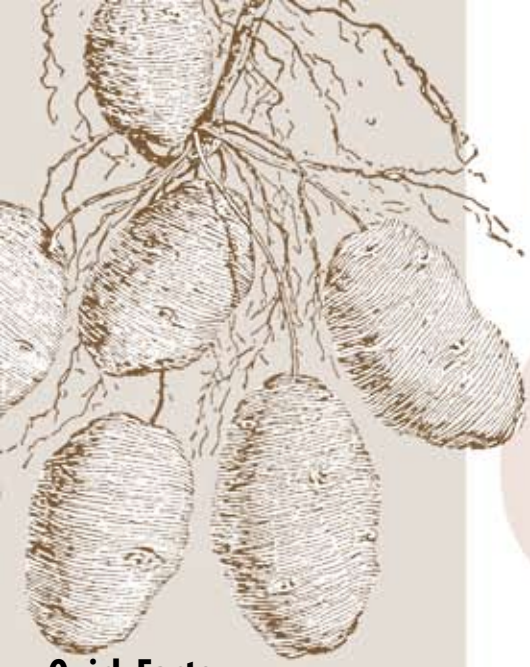

Rhizoctonia Canker and Black Scurf of POTATO

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Quick Facts

Rhizoctonia canker and black scurf caused by the fungus ***Rhizoctonia solani*** is one of the most common potato diseases.

Disease causes non-uniform emergence, weakened plants, uneven tuber-size distribution in the harvested crop, and deformed and scurfy tubers of poor quality.

Tuber-borne and soil-borne inoculum cause disease development. Crop rotation and planting high-quality scurf-free seed will help reduce disease.

Plant seed tubers under environmental conditions that promote rapid germination and emergence. Fungicide applied to seed and to soil in the seed bed may reduce infection and suppress disease that develops from seed-borne inoculum.

Black scurf forms on maturing tuber surfaces and decreases quality of both table stock and seed potatoes. Harvest tubers as soon as vines are dead to reduce the time available for black scurf formation.

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Introduction

Rhizoctonia canker and black scurf occurs worldwide and is one of the most common potato diseases. Rhizoctonia can affect potato development at any time from planting until harvest. Total tuber yield usually is not significantly reduced except under conditions of unusual disease severity; however, Rhizoctonia routinely reduces marketable yields by adversely affecting tuber size distribution while causing plants to produce a greater percentage of "rough" tubers that are misshapen, knobby, and grooved. The black scurf stage of Rhizoctonia present on tubers at harvest decreases value of both table stock and seed potatoes.

Symptoms and Signs

Reddish-brown to black lesions form underground on sprouts, stolons, roots, and stems. When lesions form near the sprout tip, the growing point is often killed, thereby preventing or delaying emergence, which results in uneven plant stands and/or weakened plants. Stem lesions often appear as sunken cankers confined to cortical tissues that may ultimately girdle the stem (**Figure 1-left**).

Above-ground symptoms resulting from stress induced by stem lesions include general stunting, leaf yellowing, leaf-tip rolling, purple pigmentation of leaves, and swollen nodes. Small aerial tubers may form where leaves branch from stems (**Figure 1-right**). After row closure and under a dense canopy of foliage, the fungus may develop a white, powdery, moldy growth on stems near the soil line (**Figure 2**). Although this growth is superficial and causes no damage to the plant, it is believed to indicate fungal activity below the soil surface.

Figure 1: *Sunken reddish-brown cankers typical of Rhizoctonia solani infection (left). If severe cankering occurs below ground, aerial tubers may form in leaf axils (right).*

Perhaps the most recognized sign of Rhizoctonia disease is the dark brown to black sclerotia present on tuber surfaces. These sclerotia are irregular in shape and can range from pinpoint, small, flat and barely noticed structures to large, raised lumps that cover most of the tuber surface (**Figure 3**). The sclerotia do not penetrate the skin but adhere tightly and are often mistaken for dirt that won't wash off. Mature tuber symptoms include malformation, knobs, creases, cracks, and surface blemishes may be present, as well as shallow, brown lesions around lenticels.

Causal Organism

Rhizoctonia stem canker and black scurf of potato is caused by the fungus *Rhizoctonia solani* Kuhn. This fungus has a very broad host range that includes dry beans sugar beets and many other crops. Populations of the fungus exist as biotypes or genetically isolated anastomosing groups (AGs) that generally attack a narrow range of host plant species. For example, *R. solani* AG-3 mostly infects potatoes causing stem cankers, stolon lesions, and black scurf on maturing tubers, with AG-5 and several other AGs sometimes detected depending on temperature and other influencing factors. The perfect (sexual) stage of the Rhizoctonia fungus, *Thanatephorus cucumeris* (A.B.

Figure 2*: This powdery-gray growth (circled) is the sexual stage of Rhizoctonia solani. This harmless growth occurs on stem surfaces near the soil line and should not be confused with late blight or white mold.*

Frank) Donk, will appear at the soil line on potato plant stems as a thin film of powdery-gray growth easily removed by gentle rubbing with a fingertip (**Figure 2**). Although this growth indicates presence of the fungus, it appears to have no direct effect on potato plant development.

Disease Development

The Rhizoctonia fungus can survive several years as mycelium on tuber surfaces, in decomposing plant tissues, or as dormant resting structures called "sclerotia" in soils, organic matter, and in association with crop debris. The fungal sclerotia also survive as black scurf on the surface of seed potatoes (**Figure 3**), where it is often mistaken for dirt that won't wash off. Soil populations of *R. solani* decline over several years in the absence of a susceptible host, although the rate of decline is affected by soil type, rotational crops, and the amount of organic matter present in the soil.

Inoculum important for disease development can either be seed-borne or soil-borne, and effects of Rhizoctonia will vary depending on the growth stage when the plant is attacked and prevailing environmental conditions. Maximum disease development generally occurs in wet soils at soil temperatures less than 54 degrees Fahrenheit. Potato plants are most

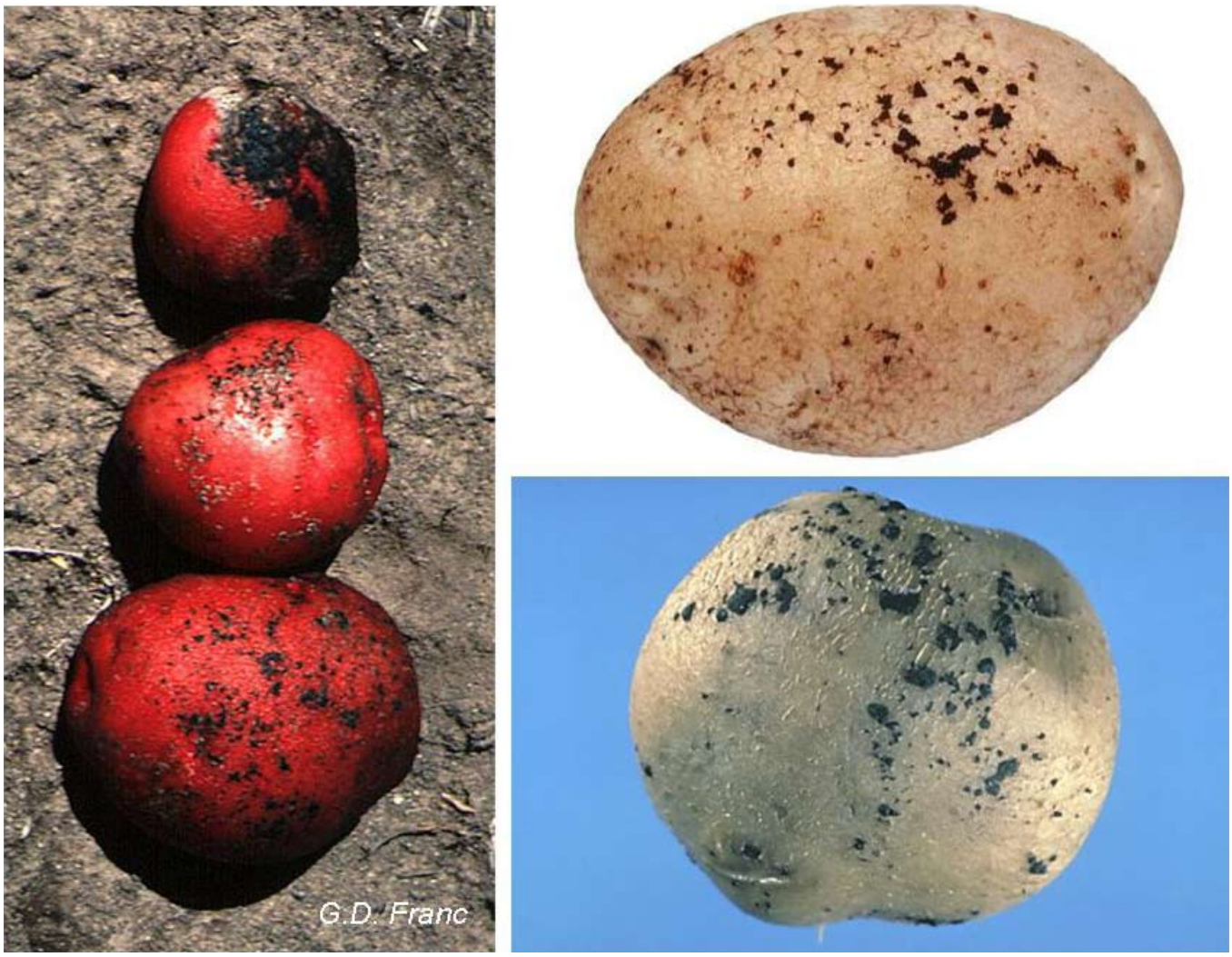

Figure 3. *Black scurf forms on the surface of tubers late in the growing season, and they are survival structures of Rhizoctonia solani. Plant seed tubers free from black scurf to decrease risk of infection and disease development in the new crop.*

severely affected in the spring when underground sprouts are killed prior to emergence. Emerging sprouts are usually attacked by inoculum carried on the surface of seed tubers. In cold, wet soil, the fungus invades and kills the growing tip which stops elongation and prevents emergence, or, alternatively, secondary sprouts may form that eventually emerge. Secondary sprouts are generally less vigorous and emerge much later causing irregular, uneven stands and weakened plants. Cold, wet conditions slow sprout development and increase the time available for germination of sclerotia and the infection processes that cause lesions to develop on young, underdeveloped tissues. Tuber-borne inoculum is very important in this phase of the disease, while soilborne inoculum is believed to be more important for later stem and stolon infection.

Once green leaves develop, stem tissues become less susceptible to infection. Stem cankers, stolon infections, and sclerotia on tubers usually develop when these tissues grow in proximity to sources of the Rhizoctonia fungus in soil. Rhizoctonia infection of older plants very seldom leads to girdled stems and dead plants; however, infected plants are stressed and become more susceptible to other diseases, especially early blight. Depending on the extent of the lesion, above-ground portions of the plant may appear yel-

lowed (chlorotic) with some purpling and upward curling of the foliage. Although these symptoms may be confused with psyllid yellows, leaf roll, or purple top, Rhizoctonia can be diagnosed because it also forms aerial tubers in the lower leaf axils in addition to stem lesions below the soil surface.

Stolon infection frequently results in pruning that prevents tuber formation or may cause tubers to abort early in their development. Stolon pruning often triggers the formation of secondary stolons, which are often shorter and set tubers closer to the stem. As a result, tuber malformation is increased when tubers expand and "grow" around the stem base. Cool, moist conditions, with moisture being the most critical factor, also favor disease development on stems and stolons.

The mechanisms that trigger sclerotia formation on daughter tubers is poorly understood, but they may include products related to plant aging. Sclerotia form on the surfaces of mature tubers under cool, moist conditions, generally after the vines have begun to die. Low soil temperatures in the fall favor formation of sclerotia on tubers. Daughter tubers that form on mother plants infected by Rhizoctonia do not always become infested with sclerotia. Although some factors important for black scurf formation are known, it remains difficult to predict when black scurf formation on daughter tubers will be problematic.

Management Approaches

Integration of management practices is essential because no single practice is completely effective. Reliance on a single approach to disease suppression frequently results in failure.

Crop rotations of at least two years or longer between potato crops generally will reduce the amount of inoculum present in the soil to economic levels. Cereal crops such as oat, barley, and corn are generally regarded as being good rotations with potatoes for the reduction of soil-borne inoculum rather than dry beans and sugar beets. The AG-3 group was recently reported to attack sugar beets, further suggesting that sugar beet is a poor rotational crop.

There is a trend that Rhizoctonia declines more rapidly in sandier soils compared to loamy soils that typically contain more organic matter. Avoid planting into soil with heavy residue from previous crops, as this seems to increase problems with Rhizoctonia. If conditions are typically favorable for disease development, longer rotations may be necessary to reduce inoculum to economic levels. The fungus is able to survive deep in the soil profile, suggesting that deep tillage prior to planting should be avoided because it may reintroduce inoculum into the root zone.

Although difficult to quantify, natural enemies of *R. solani* present in the soil environment seem to affect disease development in some production areas; therefore, if seed tubers have black scurf present, avoid planting in soil that has been recently fumigated since beneficial effects of antagonistic micro-organisms may be temporarily eliminated. Seed treatments that suppress growth of Rhizoctonia may partially compensate for the loss of antagonists early in plant development.

Plant certified seed tubers that are free of sclerotia. Collecting seed samples and washing tuber surfaces to enable sclerotia detection may be necessary to properly assess seed quality. One sclerotium per square inch (considerably less than 1-percent) can significantly increase disease pressure under environmental conditions that favor disease development. Potato varieties will vary in their reaction to black scurf, and highly susceptible varieties should be avoided if possible. Apply fungicide treatments to seed or to soil in-furrow to suppress growth of Rhizoctonia and to protect plants during the early stages of growth.

Plant seed in well-prepared soils under conditions that promote rapid germination and emergence. Cultural practices such as pre-cutting and pre-sprouting seed, shallow hilling or hill drag-off, and delayed planting until soil temperatures are considerable warmer than 45 degrees Fahrenheit, will encourage emergence and will also reduce sprout pruning by the fungus. It is also important to avoid high soil moisture during this period, and irrigation prior to emergence should be avoided.

Disease is most severe in moderately wet soils, rather than waterlogged or dry soils. Infection of young plants is most severe when plant growth is slow due to adverse growing conditions, and rapidly growing plants seem to escape infection even when environmental conditions appear favorable for infection. Adverse conditions that slow plant growth are most frequently associated with cold, wet soils; however, immature seed, soil-applied herbicide and other factors also may provide the scenario that slows seed germination and plant emergence and increases the risk of infection by Rhizoctonia. Do not plant if soil is cooler than 45 degrees Fahrenheit at planting depth and, when soil temperatures are borderline, shallow planting (two inches from the surface) will help compensate by reducing the time to emergence. Rapid emergence will not necessarily reduce root and stolon infection during the growing season.

Sclerotia (black scurf) of the fungus form as the potato periderm matures. Harvest tubers promptly after vines are dead to avoid development of sclerotia on the surfaces of tubers while still in the soil. Generally, harvest tubers within two to three weeks of vine desiccation to reduce the amount of sclerotia formation, and waiting longer than three weeks after vine desiccation generally leads to increased black scurf. Avoid destruction practices that kill vines rapidly such as burning and cutting, as these seem to enhance black scurf formation. Vine pulling to remove stems will reduce black scurf formation.

Chemical Suppression

Fungicides applied as seedpiece treatments or as in-furrow soil treatments may reduce Rhizoctonia stem canker and black scurf. These chemical suppression methods typically target inoculum that is seed-borne and may offer additional protection from soil-borne inoculum. Several different fungicide formulations are available to growers, and labels must be carefully reviewed for application rates, specialized equipment requirements for application of liquid formulations, whether or not the fungicide may be used on the seed potato crop, as well as for plant back restrictions following fungicide use.

Although a number of different fungicide seedpiece and in-furrow treatments have been tested for their efficacy, azoxystrobin, fludioxonil, and flutolanil have been reported to suppress Rhizoctonia disease development. Disease suppression provided by fungicides applied to seedpieces and in-furrow to soil are not consistent, probably due to the confounding effects of soil-borne inoculum and changing environmental conditions that influence infection processes and disease development. Fungicide will be most effective when combined with integrated cultural practices that reduce both seed- and soil-borne inoculum.

Additional Information

Banville, G.J., and D.E. Carling. 2001. “Rhizoctonia canker and black scurf.” Pages 36-37 In: *Compendium of Potato Diseases.* W.R. Stevenson, R. Loria, G.D. Franc and D.P. Weingartner. (Eds.). APS Press, St. Paul, MN. (106 pp.).