IPM Integrated Pest Management

# VEGETABLE/ HORTICULTURAL CROPS

CORNELL COOPERATIVE EXTENSION

## Rhizoctonia Disease of Potato

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*Rhizoctonia solani* is a fungus that attacks tubers, underground stems, and stolons of potato plants. Although it probably occurs wherever potatoes are grown, it causes economically significant damage only in cool, wet soils. In temperate production areas, losses from *R. solani* are sporadic and occur only when weather is cold and wet in the weeks following planting. In northern areas, where growers often must plant in cold soils, Rhizoctonia is a more consistent problem. Poor stands, stunted plants, reduced tuber number and size, and misshapen tubers are characteristic of the Rhizoctonia disease.

### Symptoms and Signs

The phase of the disease called black scurf is common on tubers produced commercially and in home gardens. The irregular, black to brown hard masses on the surface of the tuber are sclerotia, or resting bodies, of the fungus (fig. 1). Although these structures adhere tightly to the tuber skin, they are superficial and do not cause damage, even in storage. They do perpetuate the disease and inhibit the establishment of a plant from the tuber if it is used as seed.

Black scurf is the most noticeable sign of Rhizoctonia. But the most damaging phase of the disease occurs underground and often goes unnoticed. The fungus attacks underground sprouts (fig. 2) before they emerge from the soil. Stolons that grow later in the season can also be attacked (fig. 3). The damage varies. The fungal lesion, or canker, can be limited to a superficial brown area that has no discernible effect on plant growth. Severe lesions are large and sunken, as well as necrotic. They interfere with the normal functioning of stems and stolons in translocating starch from leaves to storage in tubers. If the fungal lesion expands quickly, relative to the growth of the plant, the stolon or stem can be girdled and killed.

Figure 1.

Figure 2.

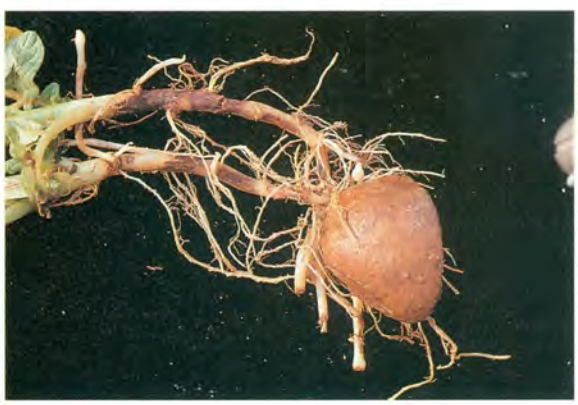

Figure 3.

Figure 4.

Figure 5.

Figure 6.

Damage is most severe at cold temperatures, when emergence and growth of stems and stolons from the tuber are slow relative to the growth of the pathogen. Wet soils also contribute to damage because they warm up more slowly than dry soils and excessive soil moisture slows plant development and favors fungal growth. If Rhizoctonia damage is severe and lesions partially or completely girdle the shoots, sprouts may be stunted or not emerge above the soil. Stolon cankers reduce tuber numbers and size and are identical to shoot cankers in appearance.

Poor stands may be mistaken for seed tuber decay, caused by *Fusarium* species or soft rot bacteria, unless the plants are excavated and examined. Rhizoctonia does not cause seed decay; its damage is limited to sprouts and stolons. Poor stands and stunted plants can also be caused by blackleg, a bacterial disease that initiates from the seed tuber and progresses up the stems, causing a wet, sometimes slimy, rot. In contrast, Rhizoctonia lesions are always dry and usually sunken.

Late season damage to plants is a direct result of cankers on stolons and stems causing problems with starch translocation. Tubers forming on diseased stolons may be deformed. If stolons and underground stems are severely infected with Rhizoctonia canker, they cannot carry the starch produced in the leaves to the developing tubers. In this case, small, green tubers, called aerial tubers, may form on the stem above the soil (fig. 4). Formation of aerial tubers may indicate that the plant has no tubers of marketable quality below ground.

At the end of the growing season, the fungus produces its sexual state, *Thanetephorus cucumeris,* on stems just above the soil line. It appears as a superficial delicate white mat which is easily removed (figs. 5 and 6). The fungus does not damage the tissue beneath this mycelium.

*R. solani* is a specialized pathogen. Only a subset of the isolates of this fungal species can cause cankers on potato. Isolates are grouped by the ability of their hyphae to fuse; isolates that can fuse, or anastomose, are in the same anastomosis group (AG). Isolates that are pathogens of potato are in AG-3. Rarely, isolates in other AG groups can form sclerotia on tubers and mycelial mats on stems. Though not damaging to potato, other AGs of *R. solani* cause diseases on sugar beet, beans, crucifers, and rice. In the absence of host plants, *R. solani* can exist by deriving its nutrients as a soil saprophyte from organic debris.

## The Disease Cycle

The disease cycle is very straightforward. Inoculum usually is introduced into fields on potato seed tubers, although it may be introduced via contaminated soil. Sclerotia in soil or on seed tubers germinate, and the resulting mycelium colonizes plant surfaces where nutrients are available. Seed inoculum is particularly effective in causing stolon damage because it is so close to developing sprouts. The fungus penetrates young, susceptible tissue, causing cankers that slow or stop the expansion of the infected stem or stolon. Cankers can sever the stolon or shoot from the plant or kill the growing point (fig. 2). The plant's resistance to stolon infection increases after emergence, eventually limiting expansion of lesions. Sclerotia form on tubers and in soil, providing inoculum for other growing seasons.

## Disease Management

Getting potato plants to emerge quickly in the spring is key to minimizing damage to shoot and stolon cankers because plants are more susceptible before emergence. Planting seed tubers in warm soil and covering them with as little soil as possible will speed the emergence of the shoots and increase resistance to canker infection. Plant fields with coarse-textured soils first because they are less likely to become waterlogged and will warm up faster.

Crop rotation reduces inoculum that can cause cankers because those *R. solani* isolates are specific to potato. Sclerotia are relatively resistant to degradation in the soil, however, and may survive for several years in the absence of potato. The fungus can also exist as a saprophyte in soil by colonizing organic debris. The longevity of the population is determined by the initial density of sclerotia at the start of the rotation period, the soil conditions, and the amount of microbial activity in the soil. Planting sclerotia-free seed is an excellent management strategy. Fungicide treatments applied to tubers may help suppress tuber-borne inoculum but are not a replacement for clean seed.

Black scurf, or sclerotia, can be minimized by harvesting soon after vines are killed. Sclerotia begin to form on tubers as vines senesce and become larger and more numerous over time. Therefore, harvesting tubers as soon as possible after skin set reduces tuber scurf significantly. Sclerotia do not form and grow in storage, and there is no increase in tuber storage rot.

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