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# Root Rot of Sweet Corn in Western Oregon



In the past decade, Oregon produced on average 344,000 tons of sweet corn for processing on 43,000 acres annually, which ranked the state fourth nationally. Most Oregon sweet corn is grown in the Willamette Valley where, along with green beans, it is a mainstay of the processed vegetable industry. However, reduced yields in sweet corn plantings over the past several years have led to declining profits for many growers.

While many factors contribute to the productivity of a sweet corn crop, root rot was identified in the 1990s as a major contributor to yield decline. Soil fumigation and pasteurization studies in soils infested with the root rot pathogens have documented losses of up to 50 percent from this disease. All cultivars currently grown in the Willamette Valley, including the industry standard, Golden Jubilee, are regarded as susceptible.

The purpose of this publication is to describe the causal agents and disease cycle of root rot of sweet corn in the Willamette Valley.

## Causal agents of root rot

Root rot occurs in all corngrowing regions of the world and, to the best of our knowledge, is a disease complex caused by several soilborne organisms, including species of *Pythium*, *Fusarium*, *Phoma*, *Helminthosporium*,

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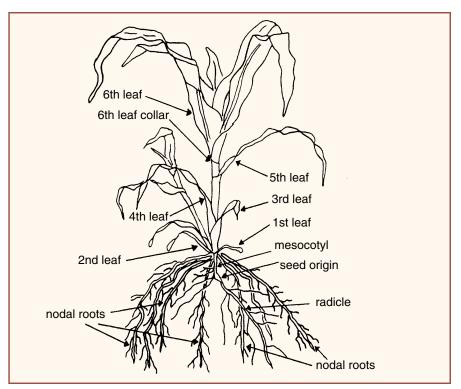


Figure 1. Sweet corn seedling at the sixth leaf stage (about 6 weeks after emergence) showing the three components of the root system: the radicle (also known as the primary root), mesocotyl (or subcrown internode), and nodal (or secondary) roots.

*Rhizoctonia, Phialophora*, and *Trichoderma*. All are fungi, except *Pythium*, which is a water mold.

In pathogenicity tests at Oregon State University, *Pythium arrhenomanes*, *Phoma terrestris*, *Drechslera* sp., and *Fusarium graminearum* caused symptoms of root rot on sweet corn. Specifically, *P. arrhenomanes* caused rot of nodal roots and reduced root biomass by "pruning" root tissue (see Figure 1 for components of the sweet corn root system). This pathogen also has been reported as causing root rot of field corn, wheat, rice, sugarcane, and annual, perennial, and cereal ryegrass. *Phoma terrestris* and *Drechslera* sp. caused extensive rot on both the radicle and nodal roots. *P. terrestris* 

Beth Hoinacki, research assistant; Mary L. Powelson, professor; and Robin Ludy, senior research assistant; all of Oregon State University, Department of Botany and Plant Pathology. is considered the primary pathogen in a complex of organisms causing red root rot of field corn and also causes root rot of perennial ryegrass. Species of *Drechslera* and the related genera *Helminthosporium, Exserohilum,* and *Bipolaris* cause root rot of a number of crops, including field corn, sorghum, wheat, barley, and annual, perennial, and cereal ryegrass. *F. graminearum* primarily causes rot of the mesocotyl. This pathogen also has been reported as causing root rot of field corn and wheat.

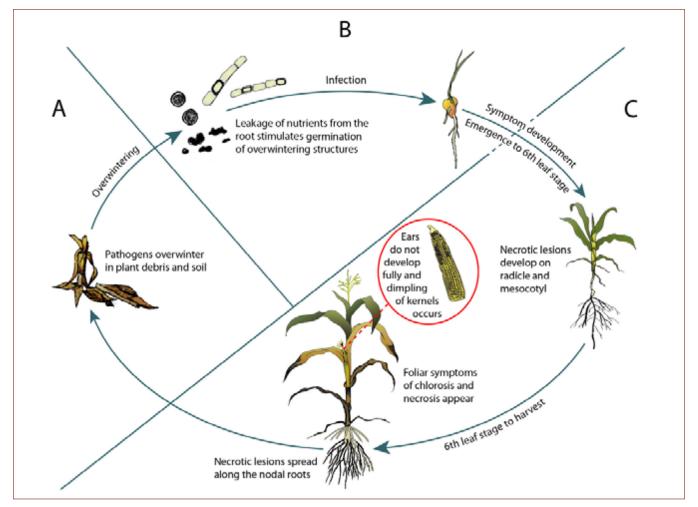
These root rot pathogens exist in most Willamette Valley soils with a history of corn production.

## **Disease cycle**

There are three basic stages in the development of root rot: (a) overwintering of pathogen propagules (infective units), (b) the infection process, and (c) symptom development (Figure 2).

(a) Overwintering of the pathogens. This stage refers to the survival of the pathogens in the absence of their host. The pathogens overwinter in plant debris as strands of fungal cells and in both soil and plant debris as specialized thickwalled survival structures (e.g., oospores, chlamydospores, or sclerotia). Many of these structures can survive in soil for several years in the absence of a susceptible host.

(b) Infection process. The first step in the infection process is inoculation, i.e., the initial contact of a pathogen with the corn root. Root infection occurs when the root makes contact with the overwintering structures. These survival structures germinate in response to the leakage of nutrients from the root of the sweet corn seedling. After germination, they can quickly penetrate and colonize the root. The root is then diseased. This process is



*Figure 2. Disease cycle of root rot of sweet corn caused by* Pythium arrhenomanes, Phoma terrestris, Drechslera *sp., and* Fusarium graminearum. *Stages in the disease cycle include: (a) overwintering of pathogen propagules, (b) infection process, and (c) symptom development.* 

repeated numerous times over the season as roots come in contact with the infective units, leading to disease symptoms.

(c) Symptom development. The initial or "primary" symptom occurs as dark brown to black dead areas (lesions) on the radicle (the primary root originating from the seed, Figure 1) as early as 3 to 4 weeks after emergence (Figure 3). In severely affected fields, 50 to 100 percent of the radicle may be rotted by 6 weeks after emergence.

As the nodal roots develop (6 to 12 weeks after emergence) they too become diseased and rotted. Typically, lesions on the nodal roots develop slowly until silking, at which time lesions expand and whole roots become rotted (Figure 4). In fields with high disease potential, the entire root system may be rotted by harvest (Figure 5). The mesocotyl (or subcrown internode, located just below the crown and above the seed origin, Figure 1) may or may not become diseased.

Secondary symptoms appear later in tissues other than the root. They include leaf "firing" (chlorosis, or yellowing, and necrosis of leaves starting at the base of the plant and progressing upward), small ears, poor ear tip fill, and dimpled kernels (Figures 6 and 7, back page).

In the field, diseased plants occur in patches, which often correspond to low or poorly drained areas.

## **Evaluating risk**

Root rot is widely distributed throughout the Willamette Valley. In some cases, growers may not be aware there is a root rot problem in their fields. For growers interested in knowing the root rot potential of a field, scouting is an option.

Roots of plants at the sixth leaf stage (Figure 1), or approximately 6 weeks after emergence, can be assessed for how severely the radicle is rotted. If the radicle is more than 50 percent rotted at this growth stage, the field has a high root rot potential, and the corn crop is at high risk for low yield, small ears, and poor-quality kernels. If the radicle is less than 50 percent rotted, the field has a relatively low root rot potential and the risk of reduced yields and ear quality is minimal to moderate, depending on the degree of necrosis of the radicle and whether lesions are beginning to develop on the nodal roots.

Environmental conditions such as soil moisture and ambient air temperature influence the development of the disease on both the radicle and nodal roots as well as the extent of secondary symptoms.



Figure 3. Necrotic lesions developing on the radicle. Lesions expand until roots are completely rotted.



Figure 4. Root system of a plant at the tenth leaf stage. Note that entire roots are blackened with disease.



Figure 5. Healthy (left) versus diseased (right) root systems at harvest.

### Management of root rot

Currently, no effective management strategies for suppression of root rot have been developed. Conventional seed treatments can protect emerging seedlings against pathogens causing seed rot and preand postemergent damping-off; however, they rarely are efficacious against root rot because these pathogens attack roots at some distance from the seed.

Soil fumigation (67 percent methyl bromide, 33 percent chloropicrin at 400 lb/acre or metam sodium at 75 gal/acre) can effectively reduce the severity of root rot and increase yield. However, these chemicals are not regarded as economical.



Figure 6. Leaf firing is a secondary symptom of root rot. It starts at the plant base and progresses upward. It usually appears after silking.



Figure 7. Small ear size, poor fill of ear tips, and dimpled kernels of ears are additional secondary symptoms of root rot. Ears on the left are from diseased plants; healthy ears are on the right.

Generally, root rot is best suppressed with the manipulation of cultural practices. For example, choice of herbicide can influence the severity of root rot. In greenhouse trials, the soil-applied preemergent herbicides Dual and Eradicane increased severity of root rot at the sixth leaf stage. Other soil-applied herbicides (atrazine and Dual II) neither increased nor reduced the severity of root rot.

Current research at Oregon State University is examining the effects of irrigation timing during the 6 weeks after planting, crop rotation (number of years between corn crops), and seed- and soil-applied biological control agents on suppression of root rot. In addition, the vegetable breeding program at OSU is screening corn germplasm for both resistance and tolerance to root rot and acceptable processing traits.

Because root rots traditionally are difficult to suppress with a single disease management tactic, the integration of several tactics may be necessary. It is expected that a successful disease management strategy may combine the planting of resistant or tolerant cultivars with production practices that enhance the quality of the soil environment.

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For questions or comments concerning this publication, please address e-mail to hoinackb@science. oregonstate.edu or write to Beth Hoinacki, Department of Botany and Plant Pathology, Cordley 2082, Oregon State University, Corvallis, OR 97331.

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