Halo Blight of Rye: Multiplicity of Symptoms Under Field Conditions

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ABSTRACT

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Several types of symptoms were caused by *Pseudomonas coronafaciens* on rye in Georgia. Elliptical to linear brown lesions, surrounded by mild chlorosis, and often in association with frost injury, were observed on leaves during the winter when temperatures rarely exceeded 15 C. Halo symptoms were prominent in early spring when temperatures were mild (maximum 15-22 C). Chlorotic lesions were less common after heading. Halo lesions turned buff-colored within 1-2 days giving the lesions a "scalded" appearance. Scald symptoms were most common from flowering until maturity, but easily recognizable halo symptoms persisted on leaf sheaths nearly until host maturity. Minute halos sometimes were found on the glumes but more commonly the entire floret blighted, dried, and became white. Failure to recognize all symptoms may lead to an underestimation of losses from the disease or to an inaccurate diagnosis of the causal agent.

Halo blight, caused by *Pseudomonas coronafaciens* (Elliott) Stevens, was first reported on oats (*Avena sativa* L.) by Elliott in 1920 (4). Subsequently this disease has been noted in all oat-growing regions of the world (3, 8, 10). Recently we reported that halo blight was widespread on rye (*Secale cereale* L.) in Georgia (2). Symptoms were similar to those found on oats; ie, lesions with small brown necrotic centers surrounded by large chlorotic halos. During the late fall, we observed that halo symptoms did not always develop following inoculation. This investigation reports the variation in disease symptoms associated with differing environmental conditions. Ability to recognize all symptom types is important for diagnosing the disease and understanding its epidemiology.

MATERIALS AND METHODS

Symptomology.—Observation of symptoms was made in plots of rye cultivars 'Vitagraze' and 'Athens Abruzzi' at Experiment, Georgia, among lines in an experimental breeding nursery at Tifton, Georgia, and in commercial rye fields in Georgia. The plants were inoculated either by injecting a bacterial suspension with a hypodermic syringe or by atomizing the suspension onto leaves while simultaneously wounding them mechanically. A combination of *P. coronafaciens* strains collected from rye in Georgia was used. Natural infection also was present. Daily temperature and relative humidity were recorded with a hygrothermograph from 1 November through harvest on 1 June.

Isolations.—Leaf or glume segments with lesions were surface-sterilized for 5 min in 1% sodium hypochlorite, rinsed for 1-2 min in 9 ml of sterile distilled water and blotted dry. The tissue was ground in 0.1 ml sterile distilled water on a spot plate and a loopful of the suspension was streaked on King's B (KB) medium (7). Plates were incubated at 25 C for 48-72 hr. Single colonies suspected to be *P. coronafaciens* were streaked onto KB to obtain pure cultures. Cultures were stored on KB slants at 2C and transferred every 6 wk, or were lyophilized. Approximately 20 leaves showing each lesion type were collected and assayed for *P. coronafaciens* when suspected halo blight symptoms were first observed. To determine whether pathogenic fungi might be involved, five leaves representing each lesion type were surface-sterilized in 1% sodium hypochlorite solution and fragments of symptomatic tissue were transferred to potato-dextrose agar.

Cultures used for pathogenicity tests were grown overnight in liquid medium 523 (6) on a rotary shaker at 23-26 C. Bacterial suspensions were adjusted to 50 Klett units (Klett-Summerson colorimeter, green filter) and diluted to 10^{-2}. Leaves of Vitagraze rye seedlings (3-5 leaf stage) were injected with a few μliters of the bacterial suspension with a 0.51-mm diameter (25-gauge) needle attached to a 1-ml hypodermic syringe. Plants were incubated at 22-25 C in a greenhouse with supplemental fluorescent light when the irradiance was less than 220 $\mu W\ cm^{-2}$. Symptoms were recorded 4-7 days after inoculation.

Mutant strain.—A spontaneous mutant resistant to rifampicin was isolated from late log phase cells of *P. coronafaciens* strain C-107 on KB agar containing 100 μg/ml rifampicin. The strain (C-107 Rfm^R) was morphologically and pathologically indistinguishable from three wild-type rifampicin-sensitive strains. The strain retained its rifampicin-resistant phenotype after repeated subculturing on yeast-dextrose calcium carbonate agar (12) in the absence of rifampicin and retained its pathogenicity as tested by inoculations on rye.

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RESULTS

Detection of atypical symptoms.—Strain C-107 Rfm^R was selected to facilitate recovery of the pathogen from field-inoculated plants. Inoculations were made with C-107 Rfm^R to Athens Abruzzi rye in mid-November. Symptoms consisting of dull yellow, partly water-soaked, elliptical lesions were observed in mid-December. The highest temperature in the field during this time was 17 C. The pathogen was isolated from these lesions on KB plus rifampicin (25 μg/ml) medium. Halos developed on rye following inoculation with these strains in the greenhouse. The field symptoms were not typical of the disease.

Several types of symptoms occurred as the season progressed. The lesion types were: (i) circular to linear brown lesions, (ii) halo lesions, and (iii) scald lesions. Distinctive lesions were found on heads and leaf sheaths. Symptoms are described in chronological sequence as observed during the season.

Leaf symptoms.—Two types of lesions were observed in mid-February when plants were in the tillering stage. The temperature 5 cm above the soil surface during the 60-day period prior to these observations exceeded 15 C for short periods on only 3 days, and did not exceed 10 C on at least half of the days. The first lesion type often was found at the interface of white-to-gray frost-damaged leaf tips and healthy tissue (Fig. 1-2). The lesion was irregularly circular to elliptical, buff to dark brown, and 3-6 mm in diameter (Fig. 3). The brown center was surrounded by a 1- to 2-mm-wide band of faded dull yellow tissue unlike the bright yellow-green color of typical halo lesions. The chlorotic area often had a half-moon appearance because of the necrotic frost-killed tissue above the lesion.

The second lesion type was linear, 2-4 mm wide, light to dark brown and extended for 5-10 cm along the leaf (often the entire length of the blade) (Fig. 4). The lesions often followed the midvein. Sometimes one or more small buff-colored sunken areas approximately 2 × 4 mm were seen near the center of the darker brown area. These sites were the sites of initial infection. Some linear lesions resulted from the coalescence of several lesions. The brown necrotic tissue was surrounded by a narrow (1-3 mm) zone of faded yellow tissue. The linear lesions sometimes appeared to be adjacent to frost-killed leaf tips, but in many instances there was green tissue above and below a lesion. Temperatures never exceeded 18 C when these symptoms were observed.

Typical halo lesions appeared in early March, when temperatures ranged as high as 15-22 C (Fig. 5). Halos were observed from jointing stage nearly until maturity. Halo lesions had a small sunken necrotic center that varied in color from white to buff in new lesions to dark brown in old lesions. Centers of lesions ranged from less than 1 mm in diameter in new lesions to 2-4 mm wide in older lesions. The central area was surrounded by a bright yellow-green halo 2-8 mm wide. The halo often extended from the lesion center along the entire length of the blade because of the rapid diffusion of toxin from the infection site.

Several weeks after halos were found nearly all of the tissue in new lesions collapsed rapidly and became buff to light brown (Fig. 6). Older lesions became darker brown.

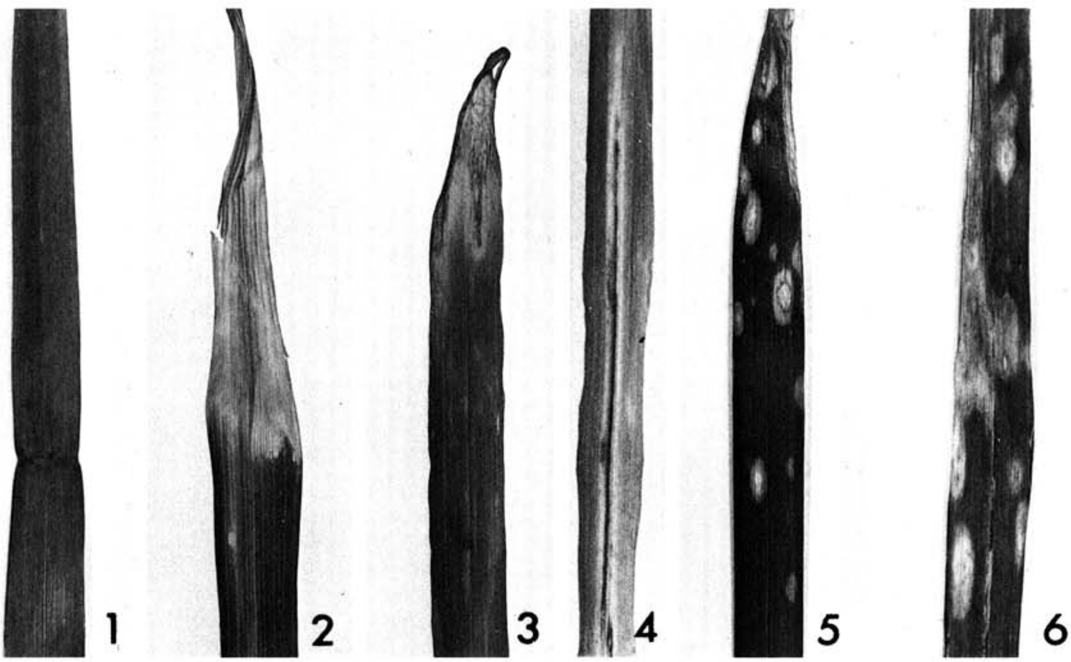

Fig. 1-6. Leaf symptoms of halo blight of rye. **1**) Healthy leaf. **2**) Leaf with frost-injured tip. **3**) Irregular to elliptical lesions below frost-injured tip. **4**) Linear brown lesion surrounded by dull chlorosis. **5**) Chlorotic halos typical of the disease. **6**) Buff-colored, small, coalescing scald lesions. Toxin-injured tissue dries out rapidly.

Sometimes a narrow band of chlorosis less than 1 mm wide was seen along the edge of lesions which seldom exceeded 1-2 cm in length. These lesions often coalesced and eventually encompassed the entire leaf. Numerous lesions of this type gave the leaves a "scalded" appearance or a pattern somewhat resembling chemical injury.

Sheath symptoms.—Halo and scald symptoms were prominent on leaf sheaths until plant maturity. Halos were similar to those observed on leaves (Fig. 7). In some lesions most of the affected tissue became buff-colored, similar to the scald symptoms on leaves. The lesions did not penetrate the thick cuticle of the epidermis on the adaxial side of the sheath so that the culm beneath was unaffected. Halo lesions at the base of the leaf blade often spread onto the sheath. These lesions frequently extended across the sheath and caused the blade to wither and die. Later, when the dead leaves had broken off, the dry sheath remnants retained the buff color at the upper end.

Floral symptoms.—Shortly after emergence of the heads, small chlorotic halos 2-3 mm in diameter with minute chocolate-colored centers were seen on the upper portion of the lemma just below the awn. The chlorosis quickly faded to white (Fig. 8). Infections prior to or during flowering resulted in death of the flower. Glume infection which occurred after fertilization had no apparent effect on the seeds.

Isolations and reinoculations.—*Pseudomonas coronafaciens* was isolated from two of 20 leaves exhibiting only frost injury and from 65% of lesions exhibiting elliptical and linear browning in late winter (Table 1). The pathogen also was isolated readily from halo lesions, scald lesions, and blighted glumes. These strains of *P. coronafaciens* caused halos when reinoculated onto rye. All fungi isolated from rye lesions were nonpathogenic.

DISCUSSION

One of the characteristics of the cereal halo blight pathogen is the production of a water-soluble toxin which causes a chlorotic halo around the infection site. Chlorosis is extensive and easily recognized. The pronounced chlorotic symptom seen on oats (4) also is observed on rye. Emphasis has been placed on the halo symptom in numerous researches on the disease (3, 4, 8), but this approach has ignored the importance of other symptoms. Roane and Kuriger (9) stated that the halo symptom on oats may be only one way in which the disease is expressed. They associated *P. coronafaciens*

TABLE 1. Frequency with which strains of bacteria isolated from various rye plant parts exhibiting different symptoms caused halo lesions when reinoculated onto rye

Plant part	Symptom	Number of isolations attempted	No. of strains causing halo symptoms[a]
Leaf	Frost injury	20	2
Leaf	Irregular to elliptical lesion	19	10
Leaf	Linear brown lesion	21	16
Leaf	Halo	20	18
Leaf	Scald	25	17
Glume	Halo and white bleaching	16	9

[a]Inoculated onto Vitagraze rye seedlings.

Fig. 7-8. Symptoms of halo blight of rye: **7**) halo lesion on leaf sheath, and **8**) individual glumes infected during flowering stage and turning white (arrow).

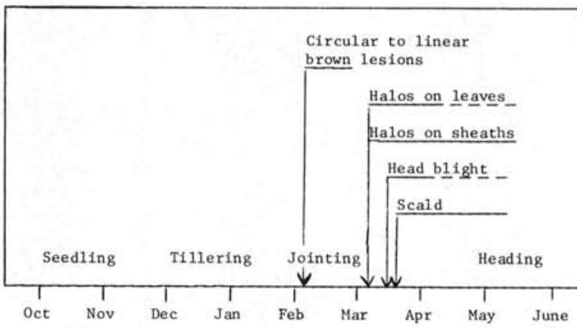

Fig. 9. Occurrence of the various symptoms of halo blight of rye during the growing season.

with crown rot and blighting of leaves and tillers during cool weather; however, a second bacterium also may be involved. Hagborg (5) isolated *P. coronafaciens* from oats in Manitoba over a 40-yr period. Of 118 strains isolated, only 36 were from leaves showing halo symptoms. Müller (8) described yellow-brown necrotic streaks and necrotic leafspots caused by *P. coronafaciens* on oats. He cited other workers who had made similar observations. Tominaga (11) reported that *P. coronafaciens* f. sp. *atropurpurea* caused diverse symptoms on various forage grasses in Japan. Many of these symptoms resemble those we found on rye.

Pseudomonas coronafaciens grows at 0 C in vitro [(4) and authors, *unpublished*]. Arny et al (1) reported that *P. syringae* and *P. coronafaciens* induced frost injury on maize leaves by acting as ice nuclei. If this also is true for rye, *P. coronafaciens* may help induce frost wounds which aid its entry into the suscept.

Halo lesions, although easily recognized, are not the most common symptom of halo blight on rye during the span of the season. Symptoms vary, apparently in response to different temperatures during the season (Fig. 9). From the milk stage of growth until maturity the scald symptom constitutes at least 95% of the lesions found in the field. The ability to recognize all symptoms of the disease is important in epidemiological studies and in the evaluation of germplasm for resistance. Because of the failure to recognize the diverse symptoms, halo blight probably has caused greater losses of rye than previously realized.

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