Development of Stem Lesions on Slash Pine Seedlings Infected by *Cronartium quercuum* f. sp. *fusiforme*

J. E. Lundquist and Thomas Miller

Research officer, Plant Protection Research Institute, Private Bag X5017, Stellenbosch, 7600 South Africa, formerly graduate research assistant, Department of Plant Pathology, University of Georgia, Athens 30602; and research plant pathologist, USDA Forest Service, Southeastern Forest Experiment Station, Gainesville, FL 32611, respectively.

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ABSTRACT

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Pigmentation was the first macroscopic symptom of fusiform rust (caused by *Cronartium quercuum* f. sp. *fusiforme*) detected on infected stems of slash pine (*Pinus elliottii* var. *elliottii*) seedlings. Epidermal cells responded first by producing a red substance that filled their lumens by 14 days after inoculation (d.a.i.). Subsequently, cortical cells developed lesions, which varied in color, size, and shape and followed a typical sequence of development. In this sequence, a cortical lesion began as a water-soaked area (14–18 d.a.i.), progressed sequentially into an orange area surrounded by a water-soaked ring (18–25 d.a.i.), an orangish-red lesion (25–31 d.a.i.), and finally, a solid, dark-red lesion (31–65 d.a.i.). Water-soaked areas contained cells with granular cytoplasm and high concentrations of phenolic compounds. Orange lesions were associated with the development of an impermeable layer of cells that prevented the movement of substances from noninfected to infected tissues. Increasing quantities of red pigment was related to the increasing numbers of phellem cells that formed between infected and noninfected regions of the cortex. The major differences in lesion type between resistant and susceptible seedlings were observed 42 d.a.i. when susceptible seedlings had a greater proportion of large, irregularly shaped lesions, with a less uniform color pattern, that appeared relatively late in the developmental sequence.

Additional key words: disease resistance, stem pigmentation.

The symptoms of fusiform rust, caused by *Cronartium quercuum* (Berk.) Miyabe ex Shirai f. sp. *fusiforme*, include distortions in growth and alterations in pigmentation. Most studies have concentrated on the former (4,8), but few have dealt in detail with the latter. Some studies have attempted to find correlations between pigmentation and resistance (5,7,9,12,19), but much remains unknown about the relationship.

Miller et al (13) described tissues beneath pigmented lesions as composed of necrotic cells separated from noninfected tissue by a layer of thin-walled rectangular cells. Jewell et al (7) found tannin formation common in cortical cells of stem lesions on both susceptible and resistant seedlings. They also determined that cellular enlargement and disorganization were common in susceptible seedlings, whereas "definitive resistance zones" bordered by what they refer to as "pseudoperiderm cells" were common in lesions on resistant seedlings. Walkinshaw (19) found no correlation between the amount of necrotic tissue beneath stem lesions and resistance, but showed that lesions on resistant seedlings were bordered by a periderm, whereas lesions on susceptible seedlings were not.

In general, studies that have dealt with alterations in pigmentation as an early response to infection have not defined the relationship between pigmentation and colonization of host tissues. This study was designed to examine the relationship between macroscopic pigmentation patterns of stem lesions, and the microscopic, histopathological reactions of the underlying stem tissues on slash pine (*Pinus elliottii* Engelm. var. *elliottii*) seedlings following infection by *C. quercuum* f. sp. *fusiforme*, and to determine whether resistant seedlings could be identified on the basis of pigment color or pattern of development.

MATERIALS AND METHODS

Two weeks after seeds were planted in sterile soil, slash pine seedlings were transplanted into polypropylene tubes (17 × 100 mm) containing medium-grain vermiculite and placed into test tube racks (40 seedlings per rack). A modified Hoagland's solution (100 μg N, 75 μg P, and 100 μg K/ml) (16) was applied at the rate of 25 ml per seedling twice weekly for the duration of the experiment. Six-week-old seedlings were inoculated with a water suspension containing 2×10^6 basidiospores per milliliter (determined by using a Coulter Counter [Coulter Electronics, Franklin Park, IL 60131]), that was applied with a chromatography sprayer at a rate of ~0.25 ml per seedling. Immediately after inoculation, seedlings were placed in a mist chamber at 20 C for 24 hr, moved to an air-conditioned headhouse at 20–25 C for 24 hr, then moved to a greenhouse.

To determine how individual symptoms change with time, a single developing stem (epicotyl) lesion was marked on 60 inoculated seedlings of one susceptible (S-118) and 60 of one resistant (Jones 18) open-pollinated family of slash pine and analyzed individually at intervals beginning 12 days after inoculation (d.a.i.). Twelve days was chosen because results of previous studies (11) showed that with this experimental system, differences between symptoms on resistant and susceptible families could be detected first at 10 and 14 days after inoculation. Each lesion was viewed under a dissecting microscope equipped with fiber optic illumination, and color of pigment and pigmentation pattern were recorded.

In a separate study, lesions showing different colors and patterns of pigmentation were sectioned at 15 μm with a freezing microtome, stained with various histochemical indicators (10), and observed microscopically. Stains used for detecting phenolic compounds were: 0.05% toluidine blue in acetate buffer, pH 4.4, with sections treated for 2.5 min; and Hoepfner-Vorsatz reagent (HVR) as described by Ling-Lee et al (10). To detect suberin in phellem cells, sections were stained in a saturated solution of Sudan Black B in 70% ethanol for 5–10 min and placed in 70% ethanol to remove excess stain (10).

The permeability of infected tissues was studied with a modification of Mullick's technique (14). Infected stem sections were split longitudinally on a plane through apparently healthy tissue between the innermost portion of a lesion and the pith. The exposed surface of the part containing the intact lesion was placed

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face-downward onto a drop of 10% $FeCl_3$. The solution was allowed to permeate the sections for several hours. After treatment, tissues were either sectioned and viewed under a microscope or left intact and viewed with a dissecting microscope. Tissues penetrated by the $FeCl_3$ solution were stained brown to black.

In another study, the frequency distribution of different types of lesions was studied by inoculating groups of 60 seedlings each of Jones 18 and S-118. Each lesion appearing at various times of measurement from 18 to 65 d.a.i. was classified into one of several categories according to pigmentation patterns.

RESULTS

The first macroscopic response to infection was the development of red pigment on the epicotyls of inoculated seedlings within 14 d.a.i. Not all seedlings had developed pigment by this time.

Nonpigmented seedlings. Either stem pigment was not present or was not discernible without the aid of a dissecting microscope. When pigment was present, it occurred in widely scattered epidermal cells and with no easily identifiable macroscopic pattern. There was no macroscopic marker useful in locating colonized tissues beneath the epidermis. However, the point of infection and stem areas colonized were located by sectioning that portion of the stem that was most likely to have become infected following inoculation. When different seedlings were sectioned 14 d.a.i., the volume and pattern of tissue colonized varied considerably. Small to moderate-size haustoria were common (Fig. 1). Intercellular hyphae were present, but difficult to see. Although there were areas in the cortex where the fungus was present in abundance, it was concentrated mainly near the cambium-phloem region and around the epithelial cells of resin ducts. No morphological alterations of the host cells or changes in phenolic content were detected. No periderm was observed.

Often, seedlings that showed no pigment by 14 d.a.i. subsequently produced lesions between 42 and 65 d.a.i.

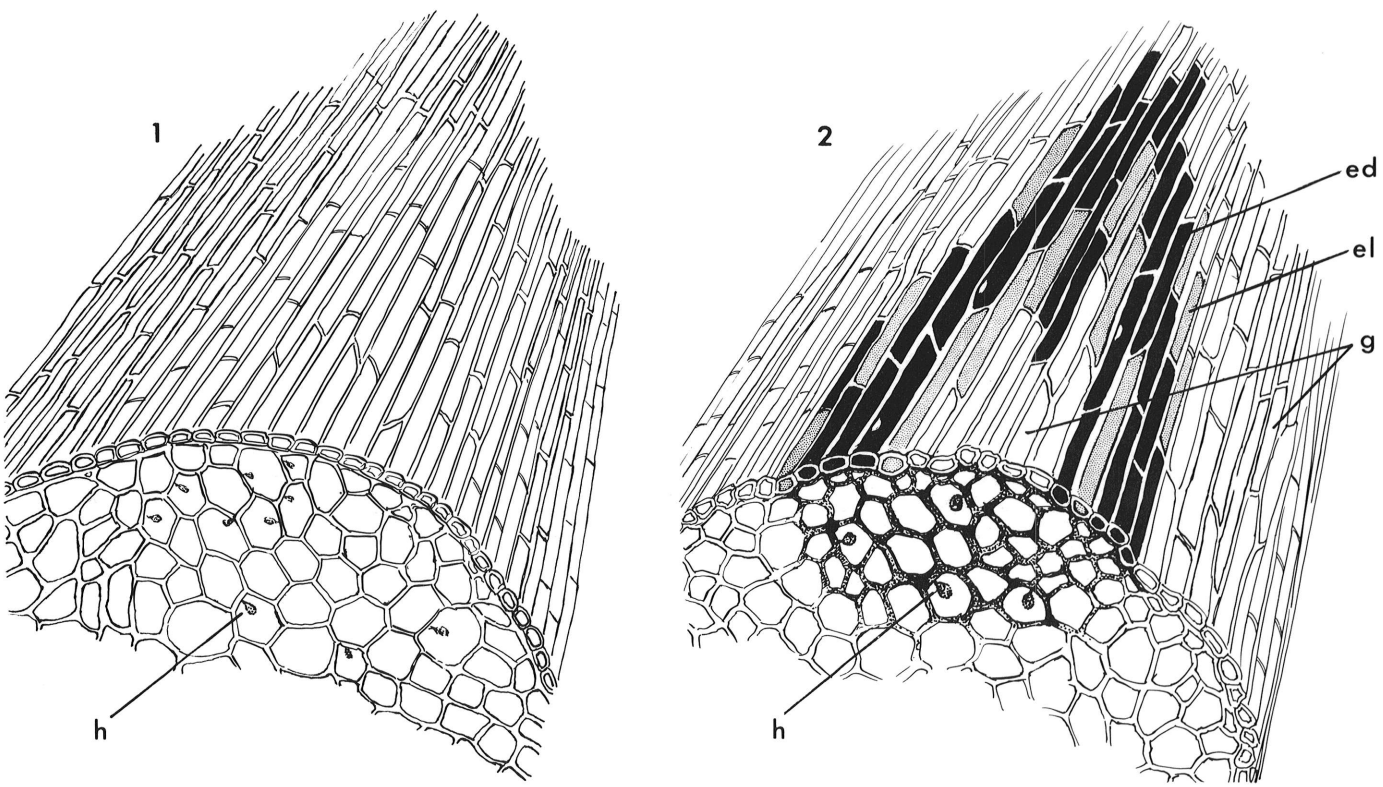
Pigmented seedlings. Stem pigment was easily discernible without the aid of a dissecting microscope and could be seen in lesions on the epicotyl surface. On many seedlings that showed pigment by 14 d.a.i., lesions underwent a typical sequence of development, which, for convenience, we divided into five phases: pigmented epidermal cells, water-soaked, orange-centered, orange-red, and dark-red lesions. Some seedlings did not show pigmented lesions by 14 d.a.i., but by 41–60 d.a.i. had developed lesions that differed from any of the other types. We refer to these as late-developing lesions.

Within 14 d.a.i., certain stem areas had developed pigmented epidermal areas (Fig. 2). A common pattern was a ring of red pigment surrounding a green center. Usually the fungus was observed only in cells beneath the red epidermal cells. Haustoria were relatively less abundant than intercellular hyphae and were usually much larger than in seedlings that were nonpigmented at 14 d.a.i. These pigmented epidermal areas were not included in the frequency distribution study since they seldom developed as distinct lesions that could be individually counted.

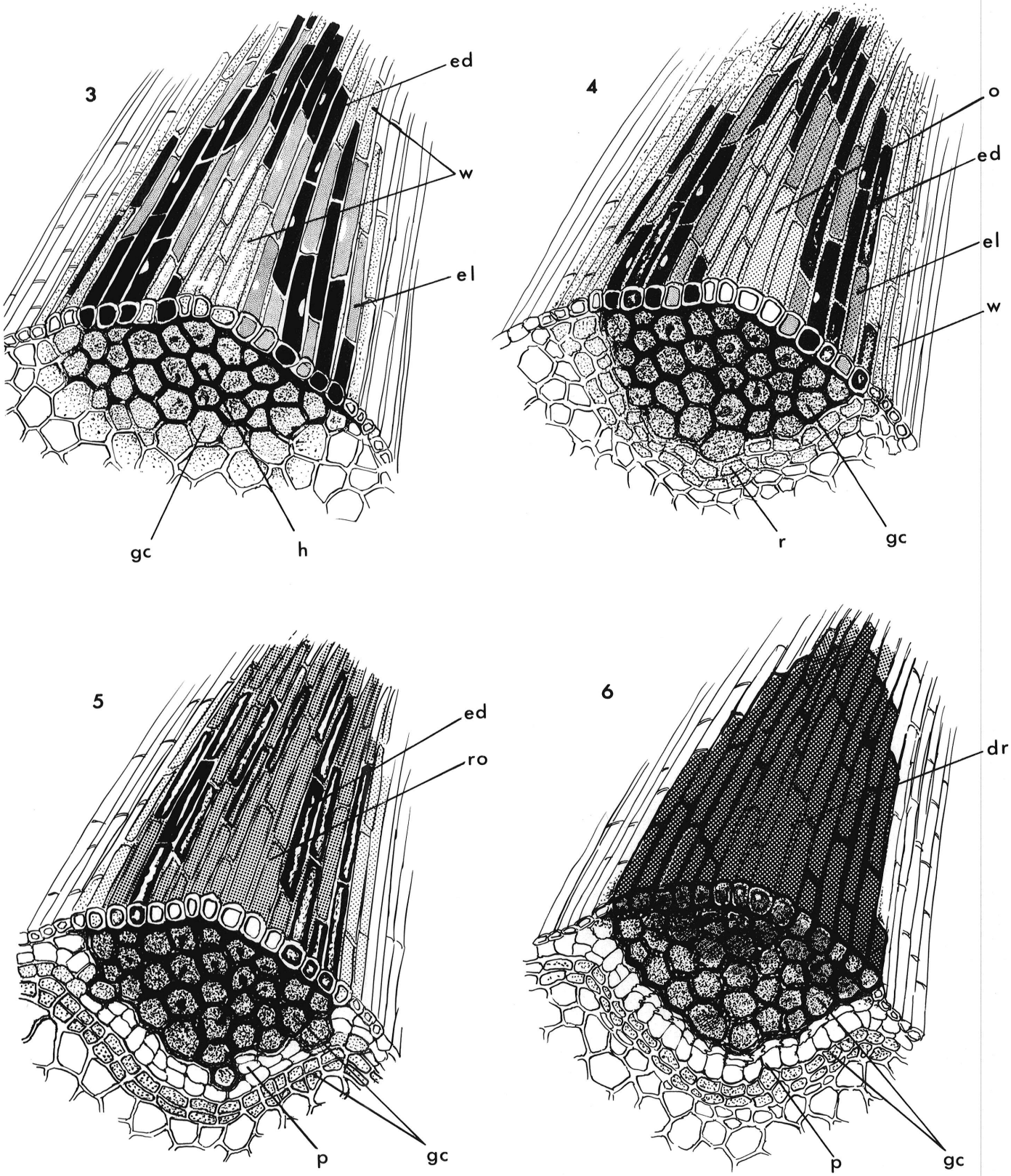
Water-soaked lesions (Fig. 3) were usually distinct spots that were circular, appeared dark green or water-soaked, and generally developed directly beneath areas of the stem with pigmented epidermal cells. Substances that stained positive with phenol stains were detected in high concentrations in lumens of cells within infected tissues and often in cells located beyond direct contact with hyphae or haustoria.

Water-soaked lesions were not the dominant type at any time in either of the two slash pine families (Table 1). The highest proportions of this type of lesion occurred at 18 d.a.i., when it represented 8% of the lesions on Jones 18 and 6% on S-118.

Orange-centered lesions (Fig. 4) were characterized by a circular-to-oblong, light- to dark-orange area surrounded by a ring of water-soaked tissue of variable width. When stem sections that had intact orange-centered spots were permeated with $FeCl_3$ solution, the orange-pigmented area remained orange, but the water-soaked ring became somewhat darker than the surrounding tissue, indicating the presence of an $FeCl_3$-impermeable barrier between the water-soaked region and orange-pigmented area. The fungus was no more widely distributed in this type of lesion than it was in the



Figs. 1–2. Diagrams of epicotyl of 8-wk-old slash pine seedlings infected with *Cronartium quercuum* f. sp. *fusiforme* 14 days after inoculation showing **1,** nonpigmented section of susceptible seedling and **2,** pigmented section of resistant seedling. Abbreviations: h = haustoria; ed = epidermal cells containing dark-red pigment; el = epidermal cells containing light-red pigment; and g = green region of epicotyl surface.



Figs. 3–6. Diagrams of epicotyl of 8- to 11-wk-old resistant slash pine seedlings infected with *Cronartium quercuum* f. sp. *fusiforme* showing the distribution of pigment, the anatomy of infected tissue, and the location of the fungus for **3,** water-soaked lesion between 14 and 18 days after inoculation (d.a.i.), **4,** orange-centered lesion 18 d.a.i., **5,** reddish-orange lesion 25 d.a.i., and **6,** dark-red lesion 31 d.a.i. Abbreviations: ed = epidermal cells containing dark-red pigment; el = epidermal cells containing light-red pigment; h = haustoria; gc = granulated cytoplasm; o = orange region of epicotyl surface; r = boundary layer of rectangular cells; w = water-soaked region of epicotyl surface; ro = reddish-orange region of the stem surface; p = phellem cells of the periderm; and dr = dark red.

water-soaked type. Large haustoria were abundant, but the main fungal component was intercellular hyphae. Granular substances that stained positive with stains for phenolics were present throughout the lesion. By later stages of development of this lesion type, a boundary layer of roughly rectangular cells (probably a cork cambium) was observed subtending the infected area.

Orange-centered lesions represented the predominant type at 18 days in both Jones 18 (69%) and S-118 (48%) (Table 1). Their numbers decreased after 18 days until none could be found in the later stages of disease development.

Orange-red and reddish-orange lesions (Fig. 5) arose from orange-centered spots due to increased development of red pigment. The water-soaked ring, noted above, became faint and in many cases disappeared. During the early stages of development of this lesion type, an incomplete layer of scattered, suberized phellem cells formed at some distance from the innermost growth of the fungus. By the time the lesions became orangish-red, this phellem layer was complete and one to several cells thick.

Red-orange lesions predominated at 25 days when they represented 68 and 70% in Jones 18 and S-118, respectively (Table 1). The incidence of this lesion type decreased after 25 days.

Dark-red and purple lesions (Fig. 6) were characterized by a dark-red coloration that became progressively darker, finally turning purple. They were ~0.5–1.5 mm in size and circular to oblong. Infected tissue was surrounded by a layer of phellem cells that was complete and continuous with the normal periderm. In most sections, the phellem layer was two to four cells thick. Infected cells within the lesion and uninfected cells bordering the lesions centripetally stained positive for phenolics. Dark-red lesions predominated at 31, 42, and 50 d.a.i. for Jones 18 and at 31 d.a.i. only for S-118 (Table 1).

Late-developing lesions appeared first in the later stages of pre-gall symptom development as relatively large (>1.0 mm) orange spots with irregular, diffuse borders and were often associated with needle axils. The pattern and extent to which the pathogen was distributed in the tissue varied greatly among lesions. Large haustoria were present in cells along with relatively large amounts of intercellular hyphae. Commonly, fungal growth was concentrated in the vascular cambium-phloem region and in epithelial cells around resin canals. Within the affected tissues, host cells often had thickened cell walls and were rounded. There was frequently a proliferation of abnormal parenchyma in the phloem region. When stained for phenolics, positive staining substances were present, but not uniformly distributed, among infected cells of the lesion as in previously described lesion types. When sections were stained with Sudan Black B, phellem cells were usually detected, but seldom continuously surrounded the infected region. Cells of the cortex, phloem and pith, and the epithelial cells of the resin ducts all demonstrated the capacity to dedifferentiate and produce peridermlike cells. Layers of these cells often extended from the epidermis to the vascular cylinder.

Late lesions never reached more than 16% in Jones 18 (Table 1). For S-118, this spot type predominated at 32 d.a.i. (43%) and shared dominance with red-orange (35%) and dark-red (32%) at 50 d.a.i. and with red-orange (39%) at 65 d.a.i.

DISCUSSION

Symptom expression in seedlings infected with *C. quercuum* f. sp. *fusiforme* is dependent on time. In the early stages of disease development, infected host tissues exhibit a spectrum of macroscopic responses. At one extreme there is no readily detectable macroscopic reaction and the pathogen ramifies freely within the host tissues. At the other extreme, there are small, circular lesions that clearly identify areas where the pathogen has been restricted. Between these two extremes of host response, there are larger, more irregularly shaped lesions in which the pathogen has been restricted to varying degrees.

Seedlings from both resistant and susceptible families have the capacity to produce pigmented stem lesions. Most lesions undergo a distinct sequence of morphological development that corresponds to microscopic events occurring in the epidermis and the cortex. Not all lesions begin this developmental sequence at the same time, nor do all complete the entire sequence. As a result, there is much variation in the appearance of lesions.

Previous workers have not distinguished between pigment located in the epidermis and pigment occurring in the cortex. Although several past studies have described cortical lesions (6,7,13,17,19), none has shown that these lesions undergo an orderly sequence of morphological development that involves progressive changes in color and patterns of pigmentation.

Even though the first macroscopic symptom of a resistant response is the formation of epidermal pigment, we believe that this pigment may have little to do with initially stopping or reducing the rate of fungus spread; it appears at the wrong time and in the wrong place. By the time epidermal pigment appears, the fungus has grown past the epidermis and has infected and colonized a portion of the cortex.

The appearance of water-soaked tissue on the stem surface

TABLE 1. Percentage of lesion types occurring on slash pine seedling stems of a resistant (Jones-18) and a susceptible (S-118) family at different times after inoculation with *Cronartium quercuum* f. sp. *fusiforme*[a]

		Percentage at indicated day after inoculation					
Family	Lesion type[b]	18	25	31	42	50	65
Jones-18	Water-soaked	8	1	3	1	0	0
	Orange-centered	69	19	1	0	0	0
	Reddish-orange	0	68	29	20	11	15
	Dark-red	18	12	66	68	79	68
	Late-developing lesions	4	1	1	1	10	16
S-118	Water-soaked	6	2	0	1	0	0
	Orange-centered	48	9	8	0	1	0
	Reddish-orange	3	70	22	26	35	39
	Dark-red	29	12	59	31	32	20
	Late-developing lesions	13	7	11	43	32	41

[a] Proportion of total number of lesions on 60 seedlings of S-118 and 60 seedlings of Jones-18.
[b] Each lesion type is described in detail in the text.

TABLE 2. Relation between macroscopic patterns of pigmentation and microscopic events in resistant lesions on the stems of slash pine seedlings infected with *Cronartium quercuum* f. sp. *fusiforme* at different times after inoculation

Days after inoculation	Lesion type[a]	Microscopic events
0–7	None	Initial resistance response, fungus growth slowed or stopped, and few haustoria develop
8–18	Epidermal pigment	Red pigment fills epidermal cell lumen
14	Water-soaked	Granular cytoplasm develops in cortical cells
18	Orange-centered	Orange pigment develops in cortical cells; impermeable cell layer formed
25	Reddish-orange	Phellem cells develop and form a discontinous boundary around infected area
31	Orangish-red	Phellem cells form a continuous boundary around infected area
42	Dark-red	Phellem cells press into and crush infected tissues toward the epidermis
50	Purple	Continuing phellem development

[a] Each lesion type is described in detail in the text.

corresponds to the development of granulated cytoplasm in the cortical cells. Granulation is a common response of host cells to infection and has been used as an indicator of cell death (20). Cytoplasmic granulation has been studied in white pine (*P. strobus* L.) tissue infected with blister rust (*C. ribicola* Fisch.) (2) and has been noted in slash pine infected with fusiform rust (13). Baur and Walkinshaw (1) found that during the normal process of senescence in slash pine tissue cultures, cells undergo cytoplasmic degeneration resulting in increased tannin content and granulation of the cytoplasm, but that granulation does not necessarily signify cell death. However, they were unable to observe mitosis in granulated cells. In contrast, cells with granulated cytoplasm observed in this research retained the ability to develop into cells of a periderm.

Granulation in the intact tissues of slash pine affected by fusiform rust occurs both within and beyond tissues colonized by the pathogen. Similarly, Robb et al (15) found that tissue culture cells of white pine infected with the blister rust fungus become granulated as much as 1–2 mm beyond the advancing pathogen. They coined the phrase "action at a distance response" to describe this phenomenon.

The appearance of orange pigment is correlated with development of decreased permeability. Impermeability as a response to wounding has been studied by Mullick (14). He identified a nonpermeable layer of tissue that surrounds the injured region and develops before suberized phellem cells appear. He referred to it as the "nonphellem impervious tissue." In the fusiform rust system, impermeability similarly develops prior to the formation of a phellem layer. With the development of impermeability, water and nutrients are prevented from passing into the infected region. As a consequence, infected cells are isolated from healthy cells and experience a stress that may profoundly alter their health and lead eventually to their death.

The presence of a periderm has been demonstrated in previous studies of fusiform rust-affected trees (4,6,13,17). More recently Walkinshaw (19) found that lesions occurring on resistant seedlings are surrounded by a periderm, whereas a periderm is absent in lesions on susceptible seedlings. In our study, a periderm was found in lesions on both resistant and susceptible seedlings.

The role played by the periderm in resistance has been the object of much speculation in the past. Struckmeyer and Riker (18) suggested that the periderm is part of the resistance reaction in white pine blister rust, but that host responses occurring before development of the periderm act to slow the spread of the fungus. Hoff and McDonald (3) identified wound periderm formation as one of the resistance mechanisms in Armand pine (*P. armandii* Franch.) infected with *C. ribicola*. Jackson and Parker (4) concluded that a periderm retarded but did not halt growth of *C. quercuum* f. sp. *fusiforme* in loblolly pine. Our results support Jackson and Parker's (4) contention that the periderm is not always impenetrable. On several occasions haustoria were found where the fungus had apparently grown from the infected region through previously formed layers of phellem into uninfected cortical cells.

The amount of red pigment developing in lesions appears to be directly correlated with the completeness of the phellem layer. By the time the phellem layer is complete, lesions are dark red. The relationship, if any, between phellem formation and red pigment formation could not be determined from this study.

Under dark-red or purple lesions, cells located between the phellem layer and epidermis were frequently distorted. These cells may have been affected by the continual production of phellem cells that press into the lesion and create enough pressure to cause cells to collapse. Supporting this hypothesis is the observation that stem surfaces often bulge over lesions. Furthermore, high concentrations of phenolic compounds occur in the cortex of dark-red and purple lesions. As the phellem cells expand, the volume of the infected region possibly decreases, producing an effective increase in the concentration of substances. This increase in concentration may create an additional stress on the fungus within this region.

An hypothesis of the relationship between macroscopic stem spot pigmentation patterns and microscopic histological reactions of the underlying stem tissues is presented in Table 2. This model proposes that pigmented stem spots signify that the host has reacted to prevent the spread of the fungus but that pigmentation alone is not the complete mechanism of resistance. Pigmentation may be symptomatic of several physiological and anatomical responses that work in concert to resist the spread of the pathogen. Resistance in slash pine to fusiform rust is probably a quantitative feature that can be seen as a contest decided by the rate at which the pathogen spreads in comparison to the rate at which the host can mobilize its physiological and anatomical defense mechanisms to stop the fungus.

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