Relation Between Foliar Symptoms and Systemic Advance of *Cephalosporium gramineum* During Winter Wheat Development

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Contribution from the Montana Agricultural Experiment Station. Journal Series Paper 983.
Accepted for publication 1 March 1980.

ABSTRACT

MORTON, J. B., D. E. MATHRE, and R. H. JOHNSTON. 1980. Relation between foliar symptoms and systemic advance of *Cephalosporium gramineum* during winter wheat development. Phytopathology 70:802-807.

Systemic spread of the vascular pathogen, *Cephalosporium gramineum*, was characterized relative to anatomical and developmental features of its winter wheat (*Triticum aestivum*) host. Pathogen restriction was associated with xylem maturation gradients between internodes, within nodes, and within leaves. Restriction of symptom development also was attributed to increased gelation and gummosis within the xylem and/or to inhibition of fungal sporulation. All evidence suggested that this pathogen is incapable of penetrating living cells at any time during the disease cycle. The number and pattern of vascular bundles in different winter wheat cultivars and the temporal association between pathogen colonization and symptom induction formed the basis for a disease index rating system. Both the movement and distribution of *C. gramineum* and the disease index rating system have practical implications in a germplasm development program. The former emphasizes the need to separate rate of disease development from rate of host development in evaluating winter wheat genotypes. The latter provides a valuable tool for monitoring symptom expression within and among plants of different genotypes.

Cephalosporium gramineum Nisikado & Ikata (= *Hymenula cerealis* Ell. & Ev.), a facultative soilborne pathogen, is the causal agent of the only reported fungal vascular disease of winter wheat. Although *C. gramineum* has a wide host range within the Gramineae, environmental conditions restrict infection to fall-sown crops or perennials (3,14). The pathogen enters the vascular system of the roots through wounds made by soil-frost heaving or by wireworms (3,16). Subsequently, the fungus moves systemically through the vascular network and is confined there until the host is moribund (18,19). The rate and extent of disease development is dependent upon the movement and multiplication of conidia in the xylem (3,18).

Symptoms appear as discrete chlorotic stripes on leaves and leaf sheaths which ultimately coalesce and result in chlorosis and necrosis of all foliar tissue followed by blighting of the heads (2,3,9).

Colonized vascular bundles are discolored and become occluded with conidia, mycelia, and gels (2,3,17,18). Interruption of the lateral flow of water has been suggested as the cause of stripe formation (15,17,18). While the events leading to impairment of lateral water transport are unresolved, it is known that colonization by the pathogen precedes foliar symptom development (18). In addition to occlusion by the fungus and byproducts of the host-pathogen interaction, a polysaccharide produced by *C. gramineum* in culture has been hypothesized as a major contributor to vascular dysfunction (15,17).

To date, studies of the relationship between pathogen localization and foliar symptom expression have involved analysis of leaves from field-grown plants sampled late in the season or from nonvernalized, wound-inoculated seedlings in the greenhouse (17,18). Even though symptomatic leaves are obvious indications of infection, they represent only a portion of the integrated vascular network within the entire plant body. Furthermore, a nonvernalized winter wheat plant is an atypical physical and chemical environment for *C. gramineum*, since infection is believed to occur in the spring after vernalization has occurred. In this study, we used controlled inoculum levels of *C. gramineum* to aid in examining disease development of naturally infected winter wheat plants throughout their development. A standardized disease index rating system based on anatomical features of a wheat leaf and on patterns of fungal movement in relation to symptom expression also was developed for use in an ongoing resistant germplasm development program (9).

0031-949X/80/08080206/$03.00/0

MATERIALS AND METHODS

Plant materials. The disease index rating system for measuring symptom severity was developed by using six winter wheat (*Triticum aestivum* L.) cultivars which demonstrated differential yield responses when infected with *C. gramineum*. Marias (CI 17595) and Lancer (CI 13547) were highly susceptible, Winalta (CI 13670) and Crest Line Row Component (LRC) 40 (MT 7579) were intermediate, and PI 094424 and PI 278212 were resistant (9). Marias and Crest LRC 40 were used to compare the movement and distribution of *C. gramineum* with symptom expression in infected tillers.

Field planting and inoculation procedures. All cultivars were planted in single rows 3.1 m long which were spaced 30.5 cm apart and seeded in early September at the rate of 200 seeds per row. In addition, 20 g of *C. gramineum*-infested oat kernels were added with the seed as an inoculum source (8). This experiment had a randomized block design replicated four times and was planted on the Montana Agricultural Experiment Station near Bozeman, MT. Seeding early in September facilitated fall root growth which maximized infection which occurred naturally, probably during spring soil-frost heaving.

Histological processing. Standard paraffin methods were used in preparing excised tissue segments for light microscopic analyses (6). The segments were fixed in FAA (40% formaldehyde-glacial acetic acid-ethanol [5:5:90, v/v]) for 48 hr, dehydrated in a graded series of *t*-butyl alcohol solutions, embedded in paraffin (Paraplast, Scientific Products Div., American Hospital Supply Corp., Evanston, IL 60201), softened in a glycerol, water, Tween-20 solution (30:69:1, v/v) for 5 days, and frozen on dry ice prior to sectioning to prevent tearing of the tissue segments. Transverse sections 10- to 11-μm thick were cut with a Model 820 Microtome (American Optical, Buffalo, NY 14215) and placed on glass slides coated with Haupt's adhesive (2% gelatin). The sections were deparaffinized in xylene, and then stained with safranin (1.0%

aqueous) and fast green (0.5% in 95% ethanol).

Disease rating system for infected leaves. Sixty leaves, one per plant, were selected randomly from different locations on primary tillers of six cultivars at heading. Each leaf exhibited a different striping pattern ranging from one discrete stripe to complete chlorosis. Leaves were photographed with back-lighting illumination which was necessary to distinguish translucent appearing veins from the more opaque lamina. Hence, veins associated with chlorotic stripes could be identified readily (Fig. 1). A segment of each leaf was excised for histological processing and subsequently examined for (i) anatomical comparisons of vascular bundle distribution between cultivars, and (ii) identification of vascular bundles colonized by *C. gramineum* and their distribution relative to chlorotic striping.

Systemic movement of *C. gramineum* between consecutive nodes. Fifty randomly selected diseased plants of the susceptible cultivar Marias were tagged in the spring when stem elongation of the main tillers began. Observations were restricted to the developmental stages between penultimate leaf emergence and anthesis (Feekes' scale 7.5 to 10.5) (7). At each of five collection dates corresponding to a change of 0.5 on the Feekes' scale, 10 main tillers were harvested and the upper four leaves of each were rated for symptom severity. Each node and its attached leaf were then excised and cultured on acidified cornmeal agar (HCMA) (Difco) to test for the presence of the fungus.

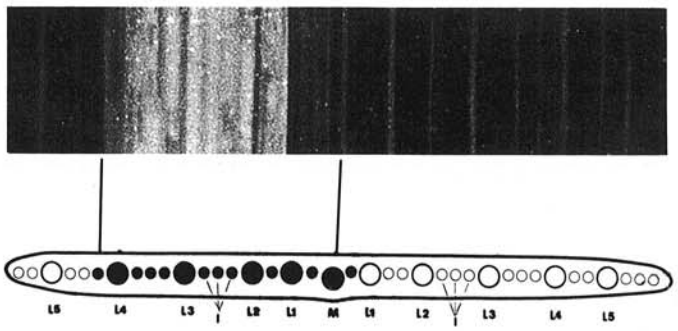

Fig. 1. Chlorotic striping pattern on a *Cephalosporium gramineum*-infected winter wheat leaf and diagrammed transverse section of that leaf showing the distribution of vascular bundles colonized. M = median vein, L = lateral vein, and I = intermediate vein. Ten lateral bundles were observed in all wheat leaves examined.

Anatomical features of the node and pulvinus and the pattern of fungal movement within these regions were studied by excising successive nodes and attached leaves from six primary tillers of both Marias and Crest LRC 40 at each growth stage. Serial sections were cut from the base of each node to midway through the pulvinus. Presence of the fungus in vessel elements was established by microscopic identification of conidia, mycelia, gel accumulations, or any combination of the three. Within each node, vascular bundles and interconnecting xylem strands colonized by *C. gramineum* were identified in relation to leaf of origin as described by Patrick (12). Vertical movement of the fungus could then be traced between successive nodes and leaves of infected tillers at each growth stage.

RESULTS

Disease rating index system. Patrick (12,13) classified wheat leaf vascular bundles into three categories. The order of earliest to latest maturing and of largest to smallest bundles were: the median bundle, the lateral bundles, and the intermediate bundles (Fig. 1). Five lateral bundles were observed consistently on each side of the median bundle in all cultivars. The number of intermediate bundles was variable, ranging from 33 to 43 per leaf.

The distribution of colonized vascular bundles was closely related to the pattern of external striping. Small quantities of the fungus in the xylem of both the stem and leaves produced no visible damage to surrounding tissues. With increased colonization, however, disruption of protoplasts in phloem and mesophyll cells bordering the vascular bundles was detected (Fig. 2A-C), as well as gels and gums within the xylem. A marked decrease in chloroplast numbers within affected mesophyll cells was associated with external manifestations of chlorotic stripes. Zones of mesophyll disruption coalesced when adjacent vascular bundles were colonized.

Each stripe appeared to result from localized vascular dysfunction caused by fungal colonization of the xylem. Hence, the striping pattern was used as an indication of the extent of systemic infection by *C. gramineum*. The disease index quantified the spread of these chlorotic stripes on a leaf. A range of one to eleven was chosen, since all wheat leaves possessed a single median vein and ten lateral veins. The intermediate bundles, being numerous and variable between leaves, were not included. A score of one denoted a single stripe on a leaf, which corresponded to invasion of a single lateral bundle and adjacent intermediates. A score of eleven

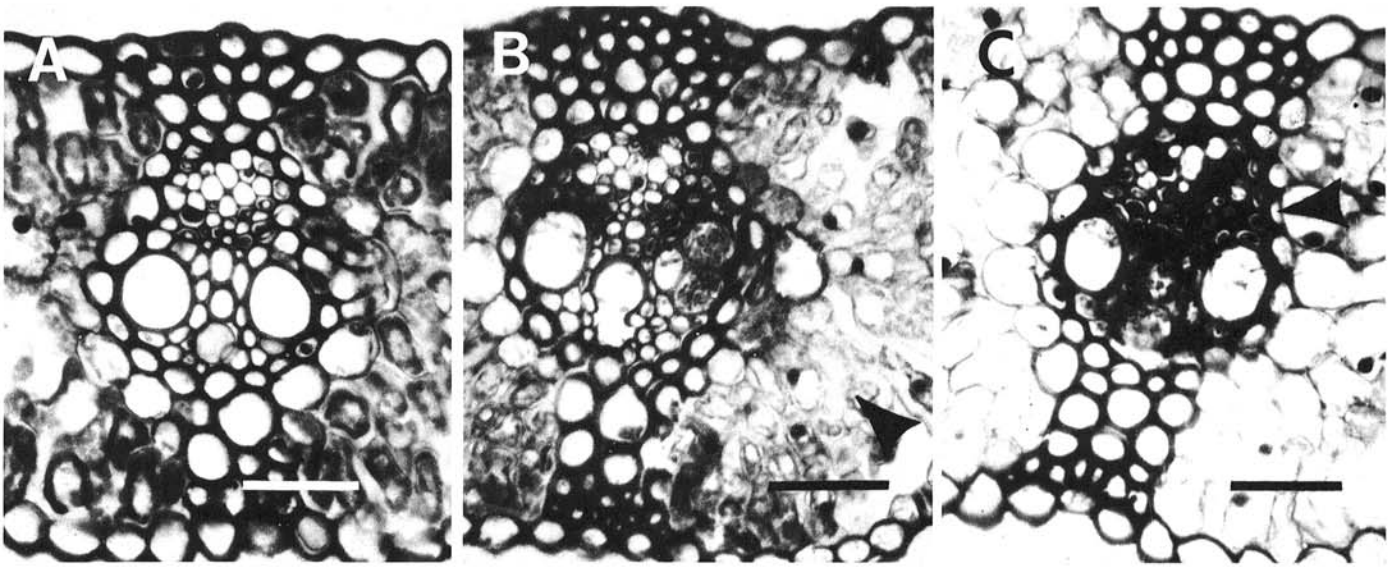

Fig. 2. The pathological effects of vascular bundle colonization by *Cephalosporium gramineum* in infected winter wheat leaves. **A,** An uninfected vascular bundle. **B,** An infected vascular bundle in the early stages of colonization. Some mesophyll deterioration is evident (arrow). **C,** An infected vascular bundle in late stages of colonization. Extensive deterioration of phloem (arrow) and surrounding mesophyll cells. Bars represent 50 μm.

indicated complete chlorosis, which corresponded to fungal establishment in most, if not all, vascular bundles of a leaf.

Systemic movement of *C. gramineum* in winter wheat. Both the pattern of foliar stripe formation on successive leaves of infected winter wheat tillers and the rate of pathogen advancement were affected by the stage of host development (Table 1). *C. gramineum* was isolated on HCMA only from fully expanded leaves attached to well developed nodes.

Close histological examination revealed that *C. gramineum* consistently invaded nodes only after maturation of the xylem strands leading into them. Moreover, the fungus demonstrated selectivity for vascular bundle types dependent upon their sequence of development within each node. The order of invasion followed closely the order of differentiation (Table 2). The fungus gained ingress first into the large L bundles which contain fully mature vessel elements (Fig. 3). *C. gramineum* was able to move into the L1 bundles at the base of the node only after maturation of the interconnecting bridging strand network which interconnects L and L1 bundles (Fig. 4). With the acropetal maturation of additional bridging strands within the node, the fungus gained access to the L2 bundles situated near the apex of the node.

The pith plexus develops independently of all other bundle types (13). Hence, contact must be made with the bridging strands before the fungus can invade it. After successful colonization of the pith plexus, the fungus has ready access to all other vascular bundles and linking strands throughout the node which ultimately leads to complete systemic invasion. Macroscopically, this sequence appears as initial discrete stripes (L bundles) on leaves, followed by slowly widening stripes as the adjacent intermediate bundles become invaded. Stripes may coalesce depending upon the pattern of L bundle colonization in the node. Eventually, all lateral and intermediate bundles become colonized and blighting occurs. This sequence is repeated in each node and attached leaf as the fungus moves upward in the plants.

The pattern of pathogen distribution observed histologically within and between nodes was mirrored macroscopically in the pattern of stripe formation (Table 1). At each consecutive growth

TABLE 1. Relationship between foliar striping patterns and movement of *Cephalosporium gramineum* in consecutive leaves of infected Marias winter wheat plants at different developmental growth stages

	Severity Score[a]				
	Feekes' scale[b]				
Leaf[c]	7.5	8.0	8.5	9.5	10.5
1	0	0	0	0	1.4†[d]
2	0	0	0.1†	2.0†	4.4†
3	0	1.7†	4.1†	6.9†	10.1†
4	2.3†	9.4†	9.9†	10.8†	11.0†

[a] Each value represents the mean of 10 tillers at each growth stage. Each leaf was assigned a severity score based on the number of stripes visible on a scale from one to eleven. 1 = one stripe per leaf, 11 = totally blighted leaf.
[b] Each growth stage is represented by the Feekes' scale, which partitions winter wheat development into a numerical scale from 1 to 11.
[c] Leaf 1 represents the flag leaf, leaf 2 the penultimate leaf, leaf 3 the third leaf below the flag, and leaf 4 the fourth leaf below the flag.
[d] Dagger (†) indicates successful isolation of the fungus from the node to which the leaf was attached.

TABLE 2. Distribution of *Cephalosporium gramineum* in each bundle type within consecutive nodes of two winter wheat cultivars at different growth stages

	Proportion of bundles colonized[a]							
	Cultivar Marais				Cultivar Crest LRC 40			
Growth Stage (Feekes' scale) and node	Bundle type[b]				Bundle type			
	L	L1	L2 & BS	PP	L	L1	L2 & BS	PP
7.5								
Node 1	0	0	0	0	0	0	0	0
Node 2	0	0	0	0	0	0	0	0
Node 3	0	0	0	0	0	0	0	0
Node 4	1.7	0.8	0	0	1.2	0.2	0	0
8.5								
Node 1	0	0	0	0	0	0	0	0
Node 2	0	0	0	0	0	0	0	0
Node 3	1.5	0.5	0	0	1.0	0.2	0	0
Node 4	2.0	1.2	0.7	0	1.3	0.8	0.3	0
9.5								
Node 1	0	0	0	0	0	0	0	0
Node 2	1.2	0.8	0.2	0	0.3	0	0	0
Node 3	2.3	2.1	1.5	0.5	1.0	0.5	0.3	0
Node 4	3.2	2.5	2.1	1.2	2.1	1.5	0.5	0
10.5								
Node 1	1.1	0.4	0	0	0.2	0.2	0	0
Node 2	2.0	1.7	1.2	0.3	1.0	1.0	1.0	0
Node 3	3.9	3.3	2.7	2.0	1.5	1.0	1.0	0
Node 4	4.0	3.5	3.2	2.2	2.5	1.8	1.3	1.0

[a] Proportion of vascular bundles colonized within each bundle type. Each value represents the mean of 6 tillers. 1 = 0–25% bundles colonized, 2 = 25–50% bundles colonized, 3 = 50–75% bundles colonized, 4 = 75–100% bundles colonized.
[b] Bundles identified by leaf of origin in relation to each node. L = leaf attached to node, L1 = lateral bundles of leaf above attached leaf, L2 = lateral bundles of the second leaf above attached leaf, BS = bridging strand network, comprised of intermediate bundles from the leaf above attached leaf, PP = pith plexus, arising independently within the node.
[c] Node 4 is the lowermost node and node 1 is the flag node.

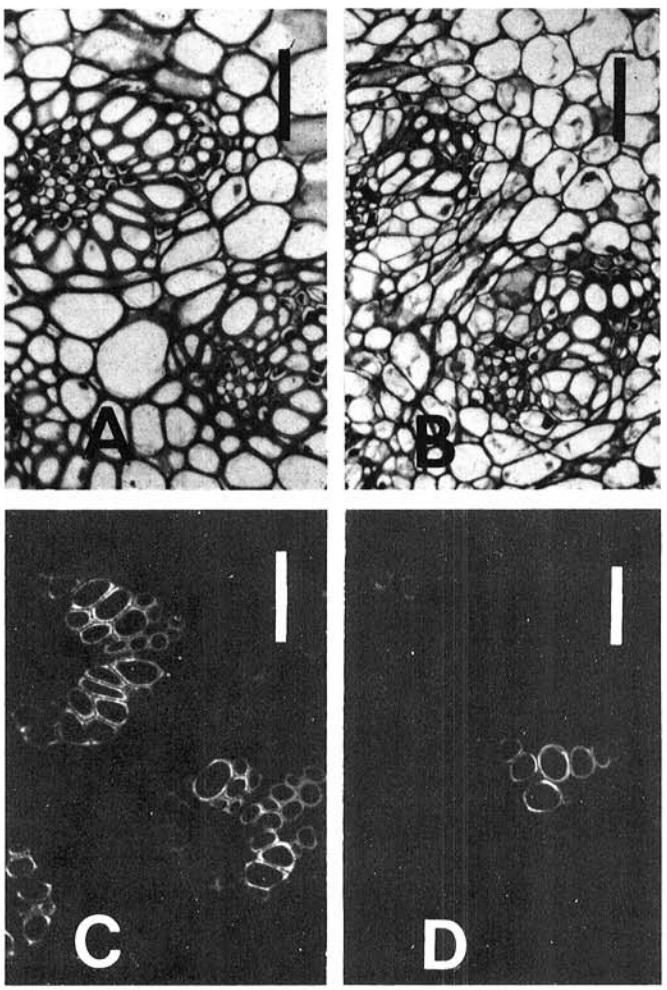

Fig. 3. Light micrographs showing xylem differentiation in the stem of a winter wheat plant. Bright-field optics of **A,** mature and **B,** immature vascular bundles in the stem of the pulvinus. Fluorescent optics of **C,** the same mature and **D,** the same immature vascular bundles showing the extent of lignification in vessel cell walls. Bars represent 50 μm.

stage after 7.5, the average number of stripes on leaves attached to recently matured nodes did not exceed two stripes, whereas the leaves immediately below averaged between four and 10 stripes. Leaves further down the stem were usually completely blighted.

Differential responses of two cultivars to the spread of *C. gramineum*. Marias and Crest LRC 40 were compared with respect to the rate of symptom expression throughout their growth and development. Crest LRC 40 was chosen because it appeared to possess some mechanism by which complete blighting of all leaves occurred later than in Marias within the same time span (10,11). Consequently, histological studies similar to those performed on Marias were conducted to determine any visual dissimilarities which might characterize this phenomenon. The fungus invaded the different bundle types of Crest LRC 40's nodes in the same sequence observed in Marias, but its rate of invasion lagged considerably (Table 2).

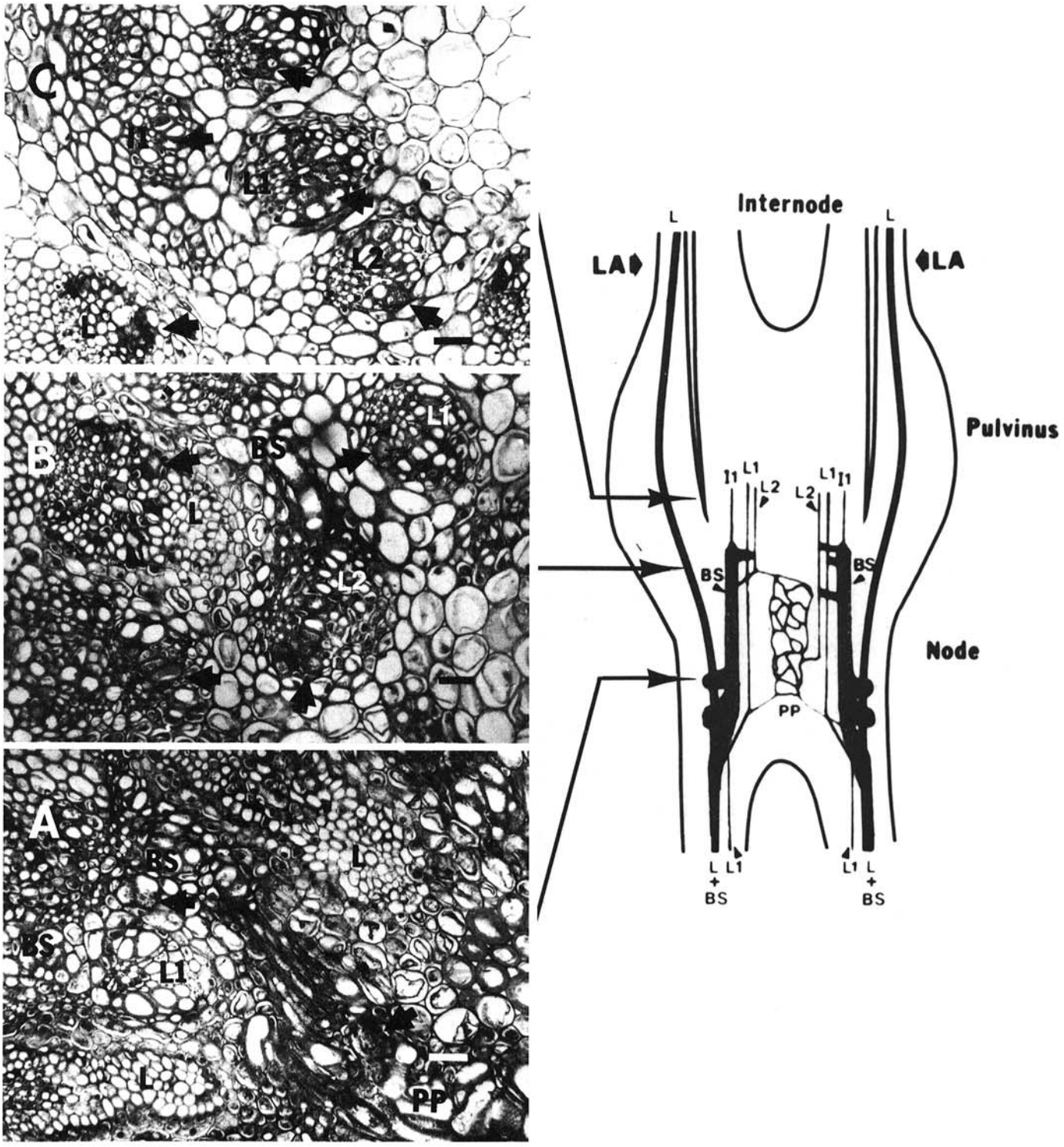

Fig. 4. *Left:* Light micrographs of cross-sections through a winter wheat node infected with *Cephalosporium gramineum,* showing vascular bundle types in their distribution at levels A-C indicated by arrows. L = lateral bundles from leaf of attachment, L1 = lateral bundles from leaf above leaf of attachment, L2 = lateral bundles from second leaf above leaf of attachment, BS = bridging strand network, PP = pith plexus. Arrows indicate colonization by *C. gramineum*. Bars represent 50 μm. *Right:* Diagram of the vascular bundle types and their course through a winter wheat node. Redrawn from Patrick (12). L = lateral and intermediate bundles from leaf of attachment (LA), L1 = lateral bundles from leaf above LA, L2 = lateral bundles of second leaf above LA, BS = bridging strand network, PP = pith plexus.

To discern differences in host response to pathogen colonization, conidial, mycelial, and gel accumulations were quantified in colonized vascular bundles by rating each on a scale of one to three indicating trace (1–30% vessels/bundle filed), moderate (30–70% vessels per bundle filled), and abundant (70–100% vessels per bundle filled) quantities. The number of vessels in each vascular bundle within a node ranged from a minimum of 24 in L1 bundles upward to 139 in L bundles. No tyloses were ever observed in infected vessels, ruling them out as a significant host response. Only those nodes at various growth stages in which the fungus had progressed into the L1 and L2 bundles, but had not yet invaded the pith plexus, were scored.

Substantial differences in conidia, mycelia, and gel accumulations appeared to exist between Marias and Crest LRC 40 (Table 3). If Marias was assumed to lack any capacity for active restriction of pathogen movement, then Crest LRC 40 possessed the ability to elicit either greater gel production or inhibit conidial proliferation, or both.

DISCUSSION

All host-pathogen interactions may be characterized by examining components of the disease pyramid singly and together (1). This study placed emphasis on pathological effects of *C. gramineum* on the host and on physiological and morphological effects of the winter wheat host on pathogen movement. The temporal relationship between these two components was investigated by following disease development during the ontogeny of infected wheat plants.

Our study confirmed earlier reports that pathological effects in and around infected vascular bundles did not occur until after abundant colonization by *C. gramineum*. The fungus was detected prior to external symptom development, but chlorotic striping was rarely visible in vascular regions devoid of the fungus. Impairment of lateral water transport resulting from vascular dysfunction has been postulated as the mechanism of stripe formation (15,17,18). As adjacent vascular bundles became infected, the regions of tissue degeneration overlapped. Ultimately, when all bundles were colonized, the leaf became blighted.

This pattern of pathogen movement in relation to symptom expression formed the basis for a disease index rating system which may be a useful tool for visually monitoring the rate and extent of pathogen spread upward through an infected winter wheat plant. Individual readings may then be extended to a population of plants within a single cultivar for evaluating differential responses between genotypes as well as selecting plants from a segregating population.

Winter wheat is distinctive in its requirement for a vernalization period to initiate flowering. In comparing symptom development within wound-inoculated vernalized and nonvernalized winter wheat plants, Bruehl (3) observed that the latter were resistant to stripe formation. He suggested that "it is probable that the fungus is not particularly active until the host passes a certain stage of development." In our studies, we found that stripe formation in nonvernalized winter wheat was evident only in the mature outer leaves (11). While Wiese's (18) observations regarding events leading to symptom development within maturing leaves is similar to ours, his study did not consider the impact of host ontogeny on disease development.

The unique nature of *C. gramineum* as a plant pathogen was discovered after studying the mutual interaction of host ontogeny and pathogen movement. The speed of fungal growth and sporulation, the structure and function of vessels in the plant, and the speed of host response all interacted to determine the rate and extent of systemic infection. In the highly susceptible cultivar Marias, systemic movement was associated with the stage of vascular differentiation. Since the fungus could not be detected in xylem in which any of the individual vessel elements retained protoplasmic components, it apparently lacked any capability to parasitize living cells. Other aspects of disease etiology substantiate the inability of *C. gramineum* to penetrate living cells. These include the pathogen's requirement for root wounding for successful ingress and its restricted presence within the xylem until after all other plant tissues have senesced.

The pattern with which *C. gramineum* moves through its winter wheat host has important practical application to a germplasm development program. Because of the close temporal association between symptom expression and xylem maturation gradients, all parental and progeny lines require close monitoring of heading dates so that selection for late-maturing genotypes is not favored. Furthermore, selection for differential responses to symptom development is effective only after heading, since symptoms do not appear on the flag leaf until head emergence.

The rate of systemic spread of *C. gramineum* may be affected in two ways. The first, which is related to the capability for gaining successful ingress into new territory, is governed by the rate of growth and maturation of xylem tissue as discussed above for Marias. The second, which is related to the ability of the pathogen to overcome host defenses after successful ingress, is governed by genetic differences between winter wheat cultivars. Such an interaction appeared to exist in Crest LRC 40, in which complete systemic infection by the fungus is delayed much longer than in Marias.

Two responses which could explain this resistance are: the physical blockage of vertical and/or lateral movement of conidia by tyloses, gums, and gels; and the synthesis of an inhibitory substance which slows sporulation. The first response has been implicated as a resistance mechanism in several vascular diseases (5). However, unlike most of these host-vascular pathogen interactions, no tyloses or vessel collapse were observed in vessels infected with *C. gramineum* (4,5). Moreover, both gelation and gummosis were rarely detected in advance of conidial invasion, but occurred simultaneously with or subsequent to fungal proliferation in infected vessels.

Crest LRC 40 exhibited increased gelation and gummosis compared to Marias. It is possible that these reactions precluded or retarded the lateral movement of conidia from colonized L and L1 bundles into the bridging strand network and pith plexus, which would limit systemic spread into all bundles of a node. Movement of *C. gramineum* into this interconnecting network of xylem strands lagged considerably behind the maturation gradient.

The apparent inhibition of conidial proliferation within colonized bundles of Crest LRC 40 suggest an interaction at the molecular level. Possibly, a toxic compound is synthesized by the cultivar which inhibits sporulation sufficiently to retard systemic infection. If this phenomenon is responsible for the retardation of systemic invasion by *C. gramineum*, it should be subjected to rigorous biochemical analyses to determine its relative role in pathogenesis.

TABLE 3. Reaction of two winter wheat cultivars to infection by *Cephalosporium gramineum* as related to accumulation of gels, conidia, and mycelia within the lumina of infected vascular bundles

Cultivar	Extent of accumulation[a]		
	Gels	Conidia	Mycelia
Marias	1.0	2.4	1.1
Crest LRC 40	2.2	1.2	1.8

[a] Each value represents the mean of 42 nodes. Readings of all L and L1 bundles in each node were averaged. Only nodes in which *C. gramineum* had not yet progressed into the pith plexus were scored. Rating scale: 1 = trace amounts (1-30% vessels per bundle), 2 = moderate amounts (30-70% vessels per bundle), and 3 = abundant amounts (70–100% vessels per bundle).

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