Physiological Effects of *Cephalosporium gramineum* on Growth and Yield of Winter Wheat Cultivars

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ABSTRACT

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The pattern of stripe formation on *Cephalosporium gramineum*-infected flag leaves of the susceptible winter wheat cultivar Marias was closely correlated with depression of relative water content, stomatal conductance, net photosynthesis, and chlorophyll content based on measurements from paired healthy and infected plants in the field. Regression analysis indicated that all four physiological parameters were interrelated, providing evidence that stripe formation coincides with localized water stress, reduction in transpiration rate, suppressed photosynthetic activity, and loss of chlorophyll. Chlorosis around colonized vascular bundles is therefore attributed to effects of localized restriction of lateral H_2O movement rather than to a readily diffusible toxin. The influence of pathogenesis on vegetative and reproductive growth patterns was measured during growth and development of three winter wheat cultivars, Marias, Crest LRC 40, and PI 278212. Internode elongation was inhibited, but leaf expansion remained unaffected by disease. Spikelet number was unaltered by disease, seed number was reduced in Marias and PI 278212, and thousand kernel weight was sharply reduced in Marias and PI 278212 but only moderately reduced in Crest LRC 40. Thus, the effects of pathogenesis are not pronounced until after anthesis during grain filling. Duration of photosynthesis, as measured by averaging CO_2 exchange of 10 flag leaves of each cultivar for 35-days after anthesis, appeared to play a major role in seed weight reduction.

Alteration of water relations has been implicated in the interaction between the vascular pathogen, *Cephalosporium gramineum* Nisikado and Ikata (= *Hymenula cerealis* Ell. and Ev.) and its winter wheat host (5,6). All of the disease symptoms (including blighted leaves and heads, stunted plants, and greatly reduced yields) are responses that can be attributed in part to disruption in water economy (14,21). Disease symptoms caused in winter wheat by root and crown-infecting fungi occasionally have been confused with those of Cephalosporium stripe because of their resemblance to the white head stage which invariably occurs late in disease development (2,5,23). In such instances, symptoms were thought to be associated with water deficits imposed as a result of severe infection (23).

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Most attempts at defining the disease physiology of the Cephalosporium stripe-winter wheat interaction have been indirect. Wiese (32) histologically evaluated pathogen movement relative to foliar symptom development. He observed that invasion and colonization of leaves by the fungus preceded induction of foliar stripes. Moreover, there seemed to be an association between the appearance of gums and gels, which accumulated as fungal colonization became more extensive, and the disruption of the phloem within vascular bundles and surrounding mesophyll cells (22). Stripes appeared in conjunction with cell collapse in these regions. Examination of transport of eosin dye in excised infected leaves revealed that both vertical and lateral translocation of water were inhibited in symptomatic regions (29,32). Some investigators have suggested that accumulation of a low-molecular-weight polysaccharide produced by *C. gramineum* in culture is responsible for stripe formation (25,29). Others have implicated a tetronic acid

toxin, Graminin A, in vascular browning, although they do not postulate its mode of action (18).

The only attempt to directly determine the effects of pathogenesis on the water relations of winter wheat infected with *C. gramineum* was that of Spalding et al (29). They measured the relative moisture content of all regions of winter wheat shoots after heading and found that desiccation was more pronounced in diseased than in healthy tissues. Their study, while substantiating that water deficits are induced during pathogenesis, did not evaluate the physiological effects of these water deficits on the host throughout disease development or determine whether the disease syndrome was due wholly or in part to water stress.

The purpose of this investigation was to obtain a more comprehensive analysis of disease physiology in relation to symptom expression and to determine its application to selection for disease resistance. The effects of pathogenesis on plant metabolic processes, on plant growth patterns, and ultimately on yield components from the onset of stem elongation through the grain-filling period were examined in three differentially responding winter wheat cultivars.

MATERIALS AND METHODS

Three winter wheat (*Triticum aestivum* L.) cultivars, Marias (CI 17595), Crest Line Row Component (LRC) 40 (MT 7579), and PI 278212 were used in this study. They were planted in early September 1978 at the Montana Agricultural Experiment Station near Bozeman, MT, in a randomized block design with four replications. Each cultivar was seeded in paired rows 3.1 m long and spaced 71 cm apart. Twenty grams of oat kernels infested with *C. gramineum* (20) was added to one row with the seed. A seeding rate of 200 seeds per row was used to maximize the number of infected seedlings. In the spring, diseased plants were identified in each row by striping on lower leaves and leaf sheaths; the main tillers were tagged. Each row was thinned manually until infected plants were spaced at least 15 cm apart. Seedlings in adjacent noninoculated check rows were similarly thinned so that every infected plant was paired with a healthy plant.

Relative water content (RWC), stomatal conductance, net photosynthesis, and chlorophyll content were measured to evaluate the physiological relationships between water stress and symptom expression at various degrees of striping on flag leaves of diseased winter wheat plants. RWC and stomatal conductance were used as indicators of water stress; the former being a measure of total water potential in a leaf and the latter being a measure of transpiration. Net photosynthesis and chlorophyll content were used as indicators of total photoassimilatory activity. For each infected flag leaf, a healthy flag leaf was sampled concurrently. Thus, all four parameters could be expressed as percent of control to help cancel out environmental factors common to both healthy and diseased plants in the field. Implicit in the results, therefore, was the assumption that observed differences are caused primarily by pathological responses. Symptom severity (number of stripes per leaf) was based on a disease index rating system previously described (22).

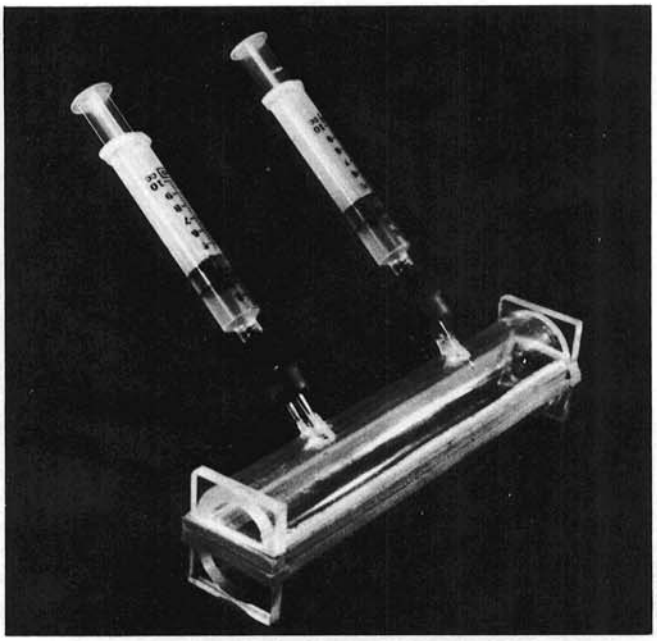

Fig. 1. Tubular Plexiglas chamber for measuring carbon dioxide exchange in winter wheat leaves in the field. Two gas samples were collected in plastic 10-ml syringes at the beginning and end of a 2-min interval and brought back to the laboratory for injection into an IR gas analyzer.

Leaves were sampled between 0900 hours and 1200 hours on calm, clear days. Between 10 and 15 infected and healthy flag leaves were evaluated on each sampling date. The leaves ranged in symptom severity from one stripe to complete chlorosis. Ultimately, 10 leaves of each severity rating were analyzed for all four physiological parameters. Measurements were made in the following sequence: Net photosynthesis, stomatal conductance, and RWC. Chlorophyll content was determined from a different, though equal sized, sample population, since RWC determinations were destructive.

Net photosynthesis. Carbon dioxide flux was measured on attached leaves in the field using a portable closed system adapted from a technique developed by Clegg et al (9). The sample chamber was constructed of tubular plexiglass with a diameter of 2.6 cm and a length of 19 cm. It consisted of two halves sealed by closed-cell insulation tape (Fig. 1). Gas samples were taken in plastic syringes (B·D multifit, 10 ml) inserted in portals positioned in the upper half of the tube. The angle of the sample chamber was continually adjusted so that the leaf blade was positioned perpendicular to the sun's incident rays. A 10-ml aliquot of gas was collected prior to sealing the leaf within the sample chamber to represent initial CO_2 levels around the leaf. The chamber was then clamped shut with a self-closing spring brass test tube wire clamp, and after 2 min, a second 10-ml aliquot of gas was taken from within the chamber. The halves of the chamber were left open several minutes between leaf measurements to allow equilibration with ambient air. After gas sampling, the syringes were stored for 2–3 hr in a cool, shaded container until they could be transferred to the laboratory. The gas samples were injected into a Beckman IR 215 Infrared Gas Analyzer to determine their CO_2 concentrations. A carrier gas of known CO_2 concentration (320 μl/L) was passed through the system at a rate of 1L/min. A 15.2-cm (6-in) drying column packed with drierite was inserted between the injection site and the IR analyzer. The IR analyzer was calibrated to read from 0 to 200 μl/L CO_2 using standard gases at 170 μl/L and 320 μl/L. Differential CO_2 concentrations were recorded as peaks on a Beckman Model 93500 Recorder. Peak heights were measured to determine differential CO_2 assimilation rates between paired healthy and diseased leaves.

In converting μl/L CO_2 to $mgCO_2/dm^2/hr$, leaf area measurements were estimated by using the formula, $0.905\ L \times W$ in which L = leaf length and W = width of the leaf midway from the tip (16).

Conductance. Diffusive resistance to water vapor loss was measured with a diffusive porometer (Model LI 60, Lambda Instruments Co., Lincoln, NE 68504). The recommended precautions were observed to reduce sampling variation (17). Three readings were taken from the adaxial leaf surface, near the base, in the middle, and near the tip and the mean was used to estimate overall diffusive resistance for each leaf. Readings were made after completion of photosynthetic measurements, which allowed at least 30 min for a leaf to return to a steady-state condition.

Leaf conductance was calculated as the reciprocal of adaxial leaf diffusive resistance.

Relative water content. The leaves were severed below the ligule after diffusive resistance had been measured. A healthy leaf was paired with an infected leaf, and both excised leaves were immediately sealed together in a small plastic bag, which was placed on ice in an insulated cooler chest.

In the laboratory, a specific sequence for handling each leaf was employed to minimize sampling error. Only one leaf was removed from the bag at a time, the infected leaf first. Removal took place within an enclosed chamber lined with saturated paper towels to maintain high humidity. Four 1.5-cm segments were cut from each leaf using a pre-cut template. One segment was from the basal region, two from the middle region, and one from near the tip of each leaf.

RWC was measured and calculated similar to procedures reported by Catsky (8). A major modification included the use of saturated polyurethane foam strips to accommodate square leaf segments.

Chlorophyll content. Chlorophyll was extracted from a separate population of paired healthy and diseased leaves by Arnon's procedure (1). Optical density readings of the chlorophyll-acetone supernatant were obtained from a Beckman Model 25 Scanning Spectrophotometer. Chlorophyll content was computed as milligrams of chlorophyll per unit leaf area. Leaf area was estimated as described above.

RESULTS

Flag leaves infected with *C. gramineum* did not exhibit generalized wilting typical of other vascular diseases (11). Rather, reductions in water content, leaf conductance to water vapor diffusion, CO_2 uptake, and chlorophyll content were linearly correlated with successive increases in the number of chlorotic stripes per infected leaf (Fig. 2). Correlations between all of the parameters were highly significant with respect to symptom severity with correlation coefficients ranging from 0.76 to 0.92. This suggested that the effects of vascular dysfunction led to localized water deficits only in the regions around heavily colonized vascular bundles. Noninvaded portions of the leaf continued to function in a normal manner.

No change in net photosynthesis was observed until after foliar symptoms appeared. The CO_2 compensation point was attained when >90% of the vascular bundles in a leaf were colonized (Fig. 3). CO_2 exchange was expressed as a negative value in completely blighted leaves. Thus, as symptom severity increased, respiratory activity rose in relation to photosynthesis until it predominated.

Internode elongation was severely restricted by *C. gramineum* throughout development of cultivars Marias and PI 278212 (Table 1). Stunting was more pronounced during elongation of the internode between the penultimate and flag nodes and in the peduncle, suggesting that the cumulative effects of disease were most severe during head emergence. The response of Crest LRC 40 reflected a more moderate increase in rate of pathogen spread. Unlike stem elongation, there were no significant effects of pathogenesis on leaf areas in the three winter wheat cultivars.

The heads of 20 primary tillers of each cultivar were examined to determine the effects of disease development of the yield components. No change in spikelet number among the three cultivars indicated that the movement of *C. gramineum* had not progressed enough in the early stages of vegetative growth to evoke

TABLE 1. Percent reduction with respect to healthy controls of consecutive internode lengths of winter wheats infected with *Cephalosporium gramineum*

	Internode[a]					
Cultivar	1-4	1-3	1-2	1-1	Peduncle	Mean
Marias	35[b]	34	32	42	41	37
Crest LRC 40	4	6	11	17	16	11
PI 278212	28	28	27	47	40	35

[a] Internodes are numbered from below the flag node (1-1) downward.
[b] Mean percent reduction from healthy controls based on a sample size of 50 primary tillers of each cultivar.

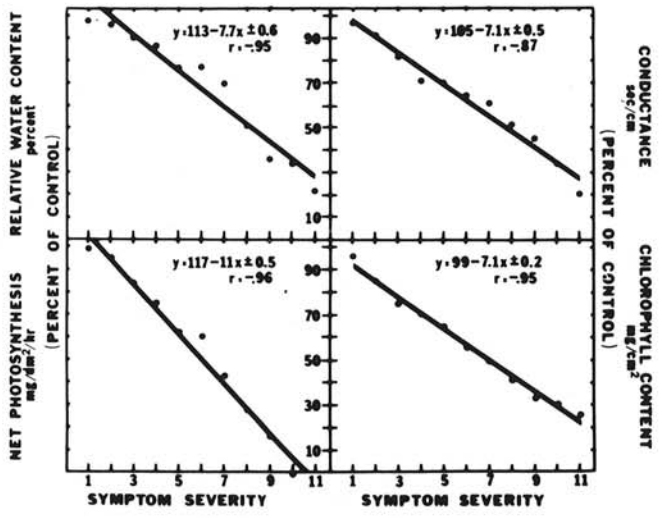

Fig. 2. The relationship between stripe formation in *Cephalosporium gramineum*-infected flag leaves and net photosynthesis, relative water content, stomatal conductance, and chlorophyll content. Symptom severity was based on the number of stripes per leaf. 1= one stripe per leaf, 11 = complete chlorosis. Each point is the mean of ten leaves. Absolute values for net photosynthesis for controls ranged 9–15 mg $CO_2/dm^2/hr$. All values are averaged across the three cultivars.

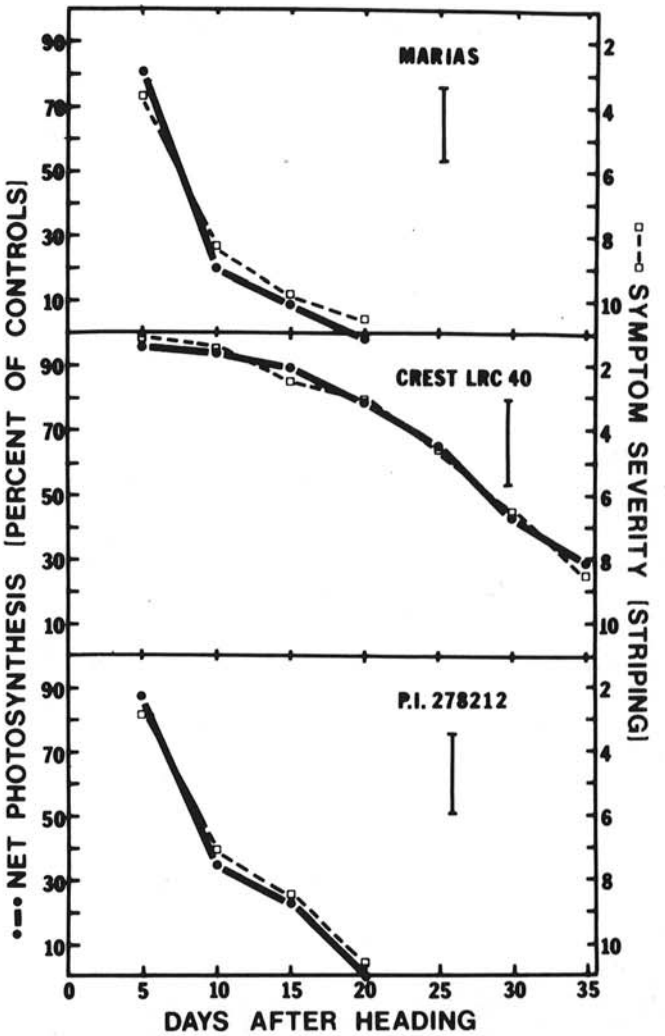

Fig. 3. Relationship between stripe formation and net photosynthesis in *Cephalosporium gramineum*-infected flag leaves of three winter wheat cultivars. Ten leaves of each cultivar were monitored for 35 days after heading. Symptom severity was scored by the number of stripes per leaf. 1 = one stripe per leaf, 11 = complete chlorosis. Vertical bars represent one standard deviation.

a stressed condition (Table 2). By the time of flowering, however, disease severity had increased sufficiently to impose water stress on late-maturing florets located near the apex and base of the heads, which was associated with decreased seed set. The most dramatic effect observed was a reduction in grain weight at plant maturity.

DISCUSSION

Disease-induced plant water deficits may be generated by alteration in the total plant water potential either directly, as in the case of increased resistance to water movement within the water conducting system, or indirectly as in the case of toxin-induced changes in membrane permeability, which alter solute potentials within affected cells (12). While vascular dysfunction in *C. gramineum*-infected plants has been implicated in disease symptomatology (6,25,29,32), a toxin, Graminin A, also has been suggested as a cause of symptom development. Graminin A caused vascular discoloration and some chlorosis when it was administered to healthy plants (18).

The results of this study suggest that a diffusible toxin may not play a major role in disease development. The linear relationships between symptom expression and net photosynthesis, relative water content, stomatal conductance, and chlorophyll content, as well as the significant correlations between the four parameters throughout progressive stripe formation, indicate that only pronounced localized effects develop around extensively colonized vascular bundles. The decline in RWC and stomatal conductance indicates internal water stress (4,12,28). Since both of these parameters were affected similarly by disease development, the main factor causing water deficits appears to be reduced water supply. A diffusible toxin that altered membrane permeability might have resulted in a poor correlation between RWC and conductance due to abnormal stomatal opening or closure (12,28). Even so, toxin activity confined to the region around each colonized vascular bundle cannot be ruled out completely. It is doubtful, however, that the low-molecular-weight tetronic acid derivative isolated by Kobayashi and Ui (18) would be as spacially restricted as the chlorotic stripes. The temporal association between accumulation of fungal cells, gels, and gums with internal cell collapse and external chlorosis (22,32) suggests that the low-molecular-weight polysaccharide isolated from culture extracts of *C. gramineum* could be a more logical incitant of restricted lateral water movement (25,29).

Net photosynthesis can be suppressed by pathogen-induced water deficits in two ways. The first involves an increase in stomatal resistance, which restricts both the outward diffusion of water vapor and the uptake of carbon dioxide (4,28). The second involves localized disruption of chloroplasts, which would effectively alter the photochemical machinery of a leaf such that the Hill reaction, photophosphorylation, and the reductive pentose phosphate cycle are inhibited (4). Based on the highly significant correlation between net photosynthesis, RWC, conductance, and chlorophyll content with respect to stripe formation, both responses may be involved. Lawlor (19) determined that photosynthesis in wheat may be completely suppressed at a water potential of only −18 bars. Thus, localized water stress due to blockage of lateral water transport out of colonized vessels could be responsible for the drop in photosynthetic activity.

The linear relationships between symptom severity and the four physiological parameters provided indirect evidence that an interaction between successive leaves was not a significant host response to disease. The penultimate and flag leaves of 15 primary tillers of the susceptible cultivar Marias were examined for decline in net photosynthesis and RWC in relation to symptom severity. Both leaves responded as predicted from the relationships shown in Fig. 2. No physiological disorder was evident in asymptomatic upper leaves, even when lower leaves were completely blighted. Such a response suggests that high-molecular-weight substances are not contributing to water imbalance in this host-vascular pathogen interaction, since the extremely short vessel elements in the nodal regions (Morton, *unpublished*) would greatly facilitate a wilting response similar to that observed in elm trees treated with the high-molecular-weight toxin produced by *Ceratocystis ulmi* (31).

The collective disruptive effects of Cephalosporium stripe development on the physiological processes of a wheat leaf were accurately reflected in the leaf's symptom severity score. In comparing net photosynthesis with symptom development concurrently in three cultivars, the close relationship that existed between the physiological measurements and visual scoring of symptom expression makes either method suitable for delineating cultivar differences in disease severity. Evaluation of germplasm for resistance to Cephalosporium stripe, therefore, does not require the more complex and time-consuming procedures involved in measuring physiological responses. Rather, a direct visual scoring of symptom severity after heading suffices to accurately reflect the host's phenotypic response to infection.

The effect of Cephalosporium stripe infection on different stages of head development pinpointed the period in which pathogenesis most severely affected yield potential. Full expression of potential spikelet number per head is contingent upon the duration of photosynthetic area on lower leaves (10). The lack of disease alteration in this yield component supported visual and histological evidence that pathogen movement and distribution was linked to host maturation gradients (22). Source-sink relationships between consecutively expanding leaves and the differentiating head apparently kept pace with stripe formation. Complete genetic expression of grain number per head is dependent upon successful self-fertilization of each mature floret. Since floret development progresses from the middle of the head toward each end (3), the location of any abortive florets is an indicator of the stage during flowering at which detrimental effects of pathogenesis occur. Reduction in seed number occurred only at both ends of the heads, indicating that the effect caused by disease was introduced only during late anthesis. Substantial reduction of carbohydrate storage in the grains of each diseased head suggested that the most severe effects of pathogenesis are expressed after flowering when grain filling takes place. This verified earlier reports which concluded that yield reduction resulted from pathological responses late in host development (15,26). The flag leaf, peduncle, and head contribute up to 80% to carbohydrate production for grain filling (7,13,24,30), most of which accumulates within the first 4 wk after anthesis (27). It is not surprising, therefore, that the decrease in net photosynthesis concomitant with an increase in foliar striping of diseased flag leaves was largely responsible for the dramatic decline in thousand-kernel weight of two susceptible cultivars. In addition, transport of assimilates to the head was undoubtedly affected along with inhibition of photosynthesis, both from the standpoint of reduced carbohydrate synthesis and also because of extensive phloem disruption in colonized vascular bundles (22).

Selecting on the basis of seed size could be an effective, yet simple, means of identifying and evaluating resistance to Cephalosporium stripe in infected plants of winter wheat germplasm lines.

TABLE 2. The effects of Cephalosporium stripe symptom development on winter wheat yield components and their relationship to the duration of photosynthesis of flag leaves following anthesis

	Percent of healthy control[a]			
Cultivar	Spikelets per Head	Seeds per Head	Thousand-kernel Wt	Duration of photosynthesis[b]
Marias	100 a[c]	96 a	31 a	35 a
Crest LRC 40	100 a	100 a	65 b	72 b
PI 278212	100 a	85 b	33 a	27 a

[a] Mean of 20 primary tillers.
[b] Mean percent net photosynthesis over a 35-day period following anthesis (from the data presented in Fig. 3).
[c] For each column, values with the same letter are not significantly different, $P = 0.05$, according to Duncan's multiple range test.

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