CANADIAN JOURNAL OF PLANT PATHOLOGY 7: 233-237, 1985

Disease progress and epidemiology of crown rot of spring barley in Utah

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Journal Paper 2969, Utah Agricultural Experiment Station

Accepted for publication 1985 05 25

The etiology and disease progress of a crown rot of spring barley in Utah were investigated. *Cochliobolus sativus* was the most frequently isolated pathogen from leading edges of necrotic lesions of infected plants. Of the diseased crowns examined each year, about 80% were infected with *C. sativus*. Time course studies of disease initiation, development, and progress were made throughout several growing seasons. Disease was first detected about 20 days after planting and reached maximum severity as the barley matured and dried. A positive correlation was found between soil inoculum density and amount of disease. The soil inoculum density increased as a function of the years the field was planted to small grains.

Kidambi, R.D., A.L. Lutz, and **N.K. Van Alfen.** 1985. Disease progress and epidemiology of crown rot of spring barley in Utah. Can J. Plant Pathol. 7: 233-237.

On a étudié l'étiologie et la progression d'une pourriture du collet chez l'orge de printemps en Utah. L'organisme pathogène qui fut isolé le plus souvent de la limite de taches nécrotiques etait le *Cochliobolus sativus*; environ 80% des collets malades examinés chaque année en étaient infectés. On a observé la chronologie du début, du développement et de la progression de la maladie pendant plusieurs saisons de culture. La maladie a été décelée environ 20 jours après le semis pour atteindre son intensité maximale lorsque l'orge murissait et séchait. On a relevé une corrélation positive entre la densité de l'inoculum dans le sol et l'intensité de la maladie. La densité de l'inoculum a augmenté avec le nombre d'années consacrées à la culture des céréales dans un champ.

Preliminary surveys of barley (*Hordeum vulgare* L.) fields in Utah indicated that a crown rot is prevalent on both irrigated and dryland spring barley. Symptoms on infected plants include brown, elongate lesions in the crown region of main stems and tillers, and brown lesions on subcrown internodes, when such internodes are present. Studies over a 5-year period showed that this crown rot limited yield significantly during some years (5).

Many fungi are known to be associated with root, crown, and seedling rot complexes of wheat and barley. The fungus found to be important in Utah is *Cochliobolus sativus* (Ito & Kurib.) Drechs. ex. Dastur (*Bipolaris sorokiniana* (Sacc. in Sorokin.) Shoemaker; syn. *Helminthosporium sativum* Pamm. King & Bakke) (7). This pathogen is widespread in wheat and barley crops in the prairie provinces of western Canada (6,9,11) and in most parts of the United States (13).

C. sativus is capable of attacking barley during all stages of its development (4). Reported symptoms include local lesions on seedlings, crowns, culms, leaves, floral structures, and kernels. Dickson (4) indicated that in dry, hot areas, seedling blight and crown rots were more common than in humid areas, where the pathogen damaged all tissues. In Utah, disease symptoms have been detected on crowns, stems, and frequently on leaves, but rarely on kernels or seedlings. The crown is the most commonly affected part of the plant in the Great Basin region.

The objectives of this study were to determine the etiology of this disease in the arid Great Basin region, and to initiate epidemiological studies. A companion study of the effect of this disease on yield parameters has been reported (5).

Materials and methods

Crown and basal stem symptoms were visually rated and expressed as a disease index of 0 to 3 where: 0 = no disease; 1 = slight, lesions are only on the outer leaf sheath; 2 = moderate, lesions occur in inner sheaths and into the basal stems; 3 = severe, lesions extend into center of basal stems, and some lower leaves severely infected.

Tissue pieces were taken from the leading edge of a necrotic lesion and placed onto potato dextrose agar (PDA) (Inolex Corp., Glenwood, IL) or acid PDA (acidified to pH 4 with lactic acid). Prior to plating, the tissue was disinfested in a 10% Clorox solution for 3 - 5 min and rinsed in sterile distilled water.

Two methods of inoculation were used when testing isolates for pathogenicity on barley. Either conidial suspensions containing 1×10^5 conidia/mL were sprayed onto the crowns and leaf sheaths, or a 23-gauge needle was rubbed across a 5-day-old *C. sativus* colony and then the needle inserted halfway through the crown of a barley plant. The latter procedure deposited a small conidial mat, containing approximately 1×10^5 propagules per mat, inside the wound. After inoculation

the pots were incubated at room temperature for 24 h in covered garbage cans, which served as moist chambers, and then transferred to a greenhouse. In all tests 3-week-old, greenhouse-grown Steptoe barley plants were used. All fungal isolates used in pathogenicity tests were derived from single spores.

The numbers of conidia of *C. sativus* in the soil were determined at the beginning, middle, and end of the growing season by taking five random soil samples per field. The flotation viability method of recovering conidia developed by Chinn et al. (2) was used, with the following modifications to improve efficiency of conidia extraction. Soil from the top 8.0 cm was collected with a soil auger and sieved successively through # 4 (4.75 mm) and # 8 (2.36 mm) screens. Next, 10 mL of mineral oil was added to 40 g of sieved soil in 500 mL erlenmeyer flasks. Tap water (85 mL) was added to the flasks and the flasks were agitated vigorously for 30 min using a Burrell mechanical wrist action shaker. The soil-water-oil emulsions were transferred to test tubes (28 × 300 mm). The test tubes were stoppered and manually tilted and shaken vigorously for 1 min to distribute the contents evenly throughout the tubes. After standing 20 min most of the soil had settled, leaving a two-phase system with the spores in the oil phase. The rest of the procedure was unchanged from that reported by Chinn et al. (2). Germinated and nonviable conidia of *C. sativus* were counted after 24-30 h incubation at 25° C.

The percentage of diseased plants was determined at least five times during the 1978-82 field seasons in six separate fields. Twenty locations in each barley field were selected randomly and five plants from each location were rated for disease severity. The transformations proposed by Van der Plank (12) were applied to the percentages of diseased plants as a function of time. After the transformation, the values $\ln 1/1 - X$ and $\ln X/1 - X$ were fitted to the linear regression equation $Y = Z + BX$, using number of days after planting as the independent variable. The coefficient of determination (r^2) for the regression of each corresponding linear transformation was then determined for both models.

Results

Tissue taken from the leading edges of necrotic lesions of infected plants contained the pathogen *C. sativus* more frequently than it did any other organism (Table 1). In some cases *Furasium* spp. were isolated alone or along with *C. sativus*. Of the *Fusarium* spp. isolated, *F. oxysporum* (Schlecht.) Snyd. & Hans. was isolated most frequently, with *F. roseum* (LK.) emend. Snyd. & Hans., cultivars

Table 1. Distribution of pathogens isolated from diseased barley plants

Field	*C. sativus*	*Fusarium* sp.	*C. sativus* + *Fusarium* sp.	Mixtures of fungi	None isolated
1	55*	10	15	15	5
2	68	12	12		8
3	65	25	10		
4	48	28		8	16
5	52		12	16	20
6	65		25	10	
7	70	15		15	
8	72	4	16		8
9	72		12		16
10	80	8	4		8
11	45	20	30	5	
12	88	4	4		4

*Percentage of total plants that were used for isolations, means of data from 2 years.

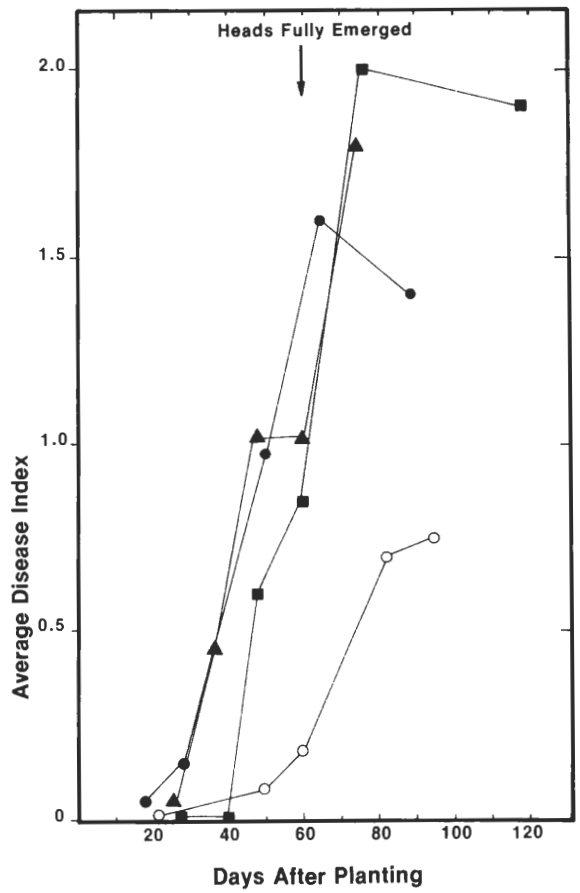

Figure 1. Disease index at times after planting in barley fields during three different years. The fields are all located in Cache Valley, Utah. The approximate time at which the heads were fully emerged is indicated. Data from two fields in 1980 are shown. One field had been cropped to barley continuously for at least 6 years (●). The other field was in its first year in barley after being cropped to alfalfa (○). Data collected from other nearby fields in 1982 (■) and 1983 (▲) are shown.

Table 2. Number of conidia of *C. sativus* in soil sampled at three different times during the 1980 growing season

	Relative sampling time[a]								
	Beginning			Middle			End		
Field	V[b]	NV[c]	T[d]	V	NV	T	V	NV	T
Barley									
1st year*	88[e]	40	134	75	30	105	95	27	125
2nd year*	115	25	138	130	33	163	136	36	172
5th year	124	33	159	122	34	157	128	28	156
6th year*	169	37	214	197	42	238	208	47	258
Corn									
7th year	43	15	58	46	17	63	45	15	60
Alfalfa									
8th year	73	15	89	78	15	93	72	11	82

[a]The three sampling times (beginning, middle, end) of season are compared to see if the three means are significantly different at P = 0.05 by the LSD test. Those fields marked with an asterisk (*) had conidia numbers that varied significantly through the growing season.

[b]V = Mean number of viable conidia per gram soil.

[c]NV = Mean number of nonviable conidia per gram of soil at time counted.

[d]T = Mean total number of viable + nonviable conidia per gram of soil.

[e]Average of 5 random soil samples per field and 3 subsamples per sample.

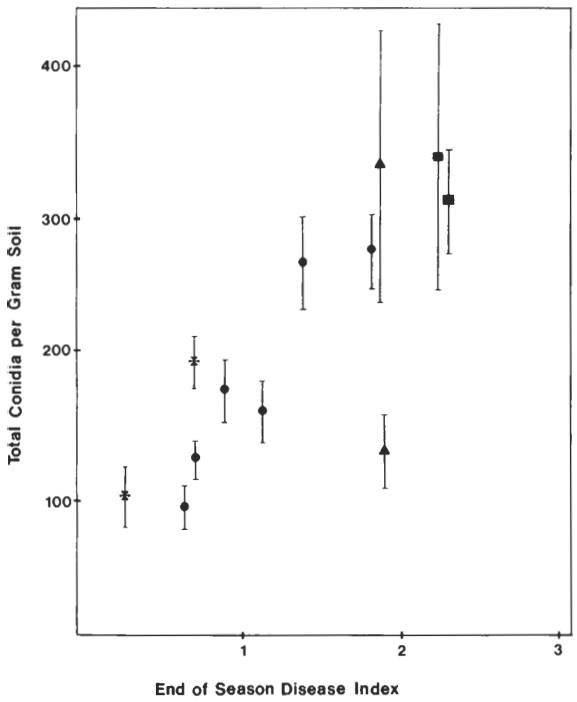

Figure 2. Mean end-of-season disease index as a function of number of conidia of *C. sativus* in the soil of a field. Data from four different years are presented: 1979 (*), 1980 (●), 1981 (■), and 1982 (▲). Each point represent a different field. Vertical bars represent twice the standard deviation.

'Culmorum' and 'Acuminatum', and *F. solani* (Mart.) Snyd. & Hans. detected to a lesser degree. Other fungi isolated included species of *Alternaria, Papularia,* and *Nigrospora* (Table 1).

Plants inoculated with *C. sativus* developed lesions in the crown region, basal stems, and lower leaves that were typical of symptoms seen in the field. Lesions that coalesced to form blotches frequently formed in the lower leaf sheaths. In one experiment, 59 of 60 plants sprayed with spore suspension of *C. sativus* developed symptoms, and 100% of the lesions examined contained the pathogen. All 15 of the plants injected with spores developed lesions and 98% of the lesions yielded the pathogen. All 25 isolates of the pathogen that were tested gave similar results.

Disease was first detected in field plants about 20 days after planting and reached a maximum between 60-95 days (Fig. 1). The disease index (DI) was highest as the crop approached maturity, with increase in disease severity occurring fastest at the time of heading. Data collected from selected fields in three different years are presented. Data from other years and fields are typical of those shown. The comparison of two fields in 1980 indicates that cropping history probably affects disease progression and severity. The field that was in barley for the first year had a slower increase in disease severity and a lower end-of-season DI than did the other field sampled in 1980, which had been cropped to barley continuously for at least 6 years. A comparison of disease progress in that first year field with an adjacent one that had been in barley 1 year indicates that the rate of increase in DI was the same in the two fields, but that the lag time was longer in the field in its first year in barley (data not shown). This observation that fields previously cultivated with barley have a higher incidence of disease than those with other cropping histories was evident in other years as well. For instance in 1979, fields planted with barley for the first time in 4 years had a disease index of 0.31 ± 0.06, whereas those that had been planted with barley at least twice in the past 4 years had a disease index of 1.13 ± 0.27 at the end of the season.

Numbers of *C. sativus* conidia in soil were greatest in the fields that have been continuously cropped to barley. However alfalfa and corn fields were also found to contain a relatively large number of conidia per unit weight of soil even though they had not been planted with barley for many years (Table 2). Some fluctuation in the number of conidia per gram of soil was detected at different times during the growing season. Some of these fluctuations were significant (P = 0.05), but there were no trends that would have suggested either an overall

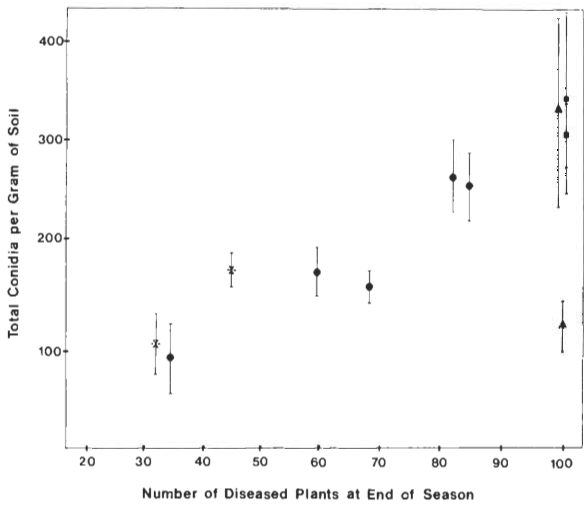

Figure 3. Number of diseased plants out of 100 sampled at end of season as a function of the number of conidia in the soil of a field. Data from four different years are presented: 1979 (*), 1980 (●), 1981 (■), 1982 (▲). Each point represents a different field. The vertical bars represent twice the standard deviation.

increase or decrease in the total number of conidia throughout the growing season (Table 2). Although Table 2 reports the data for 1 year, these results were typical of the 4 years during which we sampled conidia in soil. The changes in numbers of conidia over the winter of 1979-1980 were determined in two fields (2nd year barley and 6th year barley of Table 2). We found that the numbers in one field went from 95 ± 21 viable conidia/g soil at the end of the 1979 growing season to 115 ± 21 at the beginning of the 1980 growing season. In the other field the numbers were 148 ± 16 and 169 ± 16, respectively. A positive correlation was observed between the total number of conidia per gram of soil and end of season disease index (Fig. 2). Likewise the total number of diseased plants at the end of the season increased with increasing numbers of conidia in the soil (Fig. 3). The latter corelation had a slightly higher coefficient of determination ($r^2 = 0.64$) than the former ($r^2 = 0.61$).

Discussion

C. sativus and various *Fusarium* spp. were the organisms most frequently isolated from crown rot lesions of barley in the Great Basin area. The *Fusarium* spp. were not pathogenic to barley in greenhouse tests, nor was the most commonly isolated *F. oxysporum* antagonistic to *C. sativus* when the two were coinoculated (A. L. Lutz, and N. K. Van Alfen, unpublished).

Symptom expression under our conditions differ from that described in Canada (11). Most literature on this disease emphasizes the effect of the pathogen on subcrown internodes. Most barley in the Great Basin is sown shallowly because it is grown as an irrigated, spring planted crop. Subcrown internodes do not develop under these conditions. However wheat is generally planted in the fall and not irrigated, so seeds are sown deeply and, as a result, subcrown internodes develop on the emerging plants. The crown rot problem in the Great Basin, however, is most commonly associated with irrigated grains.

Chinn et al. (3) reported that in the prairie provinces of Canada the disease ratings of seedlings, but not of mature plants, correlated with the concentration of conidia of *C. sativus* in the soil. However we found correlations between conidia populations in the soil both with end-of-season disease index values and with the proportion of mature plants that were diseased. This correlation was generally observed during the 4 years that this relationship was investigated (Figs. 2,3). Only in one field in 1981 were data collected that did not closely fit the correlation curve. Seedling disease was never observed during these 4 years of study. Those fields not cropped to small grains in the previous 3 years had significantly lower end-of-season disease index values than those cropped to small grains more frequently. Spore concentrations were also lower in the fields that had not been cropped to small grains in the recent past (Table 2).

The years of this study included unusually dry as well as wet years. The disease index in all fields was lower during the dry year (1979) than in the other years. The conidia concentrations in soil were also lower that year. This relationship between conidia concentration in the soil and disease levels on a yearly basis supports the findings reported in Figures 2 and 3. The coefficients of determination for these relationships were not particularly high, but these data were collected during four different years by two different people. During these years disease incidence varied due to environmental factors. We think that this relationship between disease and soil conidia concentrations must be a strong one to be evident above all the variables that could have affected disease and inoculum densities. Our interpretation of these data is that the amount of disease is a function of the populations of soil conidia. However just the opposite may be the case, i.e. soil conidia numbers may be a function of disease severity. If this is the case, conidia numbers should show a detectable increase through the season as the lesions increase in number and size. Such a trend was reported by Chinn (1), but no such trend was

seen in our study. Although there were significant changes in spore numbers in some fields through the growing season, no discernible trends were observed. We did not sample conidia numbers as frequently as Chinn (1), but by sampling at the beginning and end of the growing season, we should have detected the highest and lowest values. In the two fields in which we monitored changes in conidia numbers from the end of the 1979 growing season to the beginning of the 1980 season, we found that little change in the number of viable conidia had occurred. The data we present here leave us with a dilemma. The data suggest that conidia numbers in soil do not change, either during the growing season or after the crop residue is incorporated into the soil. This conflicts with the data of Figures 2 and 3 which indicate that such changes do occur. The mean number of conidia increased from one season to another, but because of lack of precision in the assay, these changes were not significant. Perhaps changes in conidia numbers over several years would be detectable.

The disease progress curves were similar to those reported by others (8,10). We did not determine disease index frequently enough to enter the discussion between Stack (10) and Morrall and Verma (8) concerning the best model to describe disease progression. The lack of increase of conidia numbers in the soil through the growing season supports the simple-interest model. The compound-interest model predicts an increase in inoculum densities as the growing season progresses, and this was not observed. However, in greenhouse experiments (J. Kluck, and N. K. Van Alfen, unpublished) with sterile soil, we have observed that high levels of disease result from aerial distribution of conidia from plant to plant. If soil inoculum densities don't change significantly during the growing season, then we can only assume that in the field the aerial distribution of conidia must play a significant role in disease progress.

We wish to acknowledge the advice and assistance of James A. Hoffman and Rulon S. Albrechtsen in these studies. The reading of and advice regarding the manuscript by R. James Cook was appreciated.

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