Influence of infection by *Cochliobolus sativus* on yield components of spring barley

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Yield loss due to crown rot of spring barley in Utah caused by *Cochliobolus sativus* was investigated from 1978 to 1982. The intensity of the disease, based on the extent of crown and basal stem lesions, was correlated with yield. Losses were calculated relative to potential yields of noninfected plants. Variability in yield-limitation occurred during the 5-year period. When the disease affected yield, it did so primarily through lower kernel weights. During 1978 and 1980, kernel weights of severely diseased plants were only 36 to 46% those of healthy plants. In 1981 and 1982, however, no correlation between disease index and kernel weight was observed. During a year when disease did not affect yield, there was no apparent affect of the pathogen on the water relations of plants.

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On a étudié les pertes de rendement causées par la pourriture du collet, provoquée par le *Cochliobolus sativus*, chez l'orge de printemps en Utah de 1978 à 1982. Il y a eu une corrélation entre l'intensité de la maladie, établie d'après l'importance des lésions sur le collet et le pied, et le rendement. On a calculé les pertes par rapport aux rendements de plantes non- infectées. La limitation du rendement a été variable au cours des cinq années. Lorsque la maladie a réduit les rendements, ce fut principalement par le poids moins élevé des grains. Pour les années 1978 à 1980, le poids des grains des plantes gravement attaquées ne représentait que 36 à 46% de celui des plantes saines. On n'a toutefois pas relevé de corrélation entre le degré de maladie et le poids des grains en 1981 et 1982. Il n'y avait pas d'effet apparent du champignon sur les relations plante-eau durant une année où les rendements n'étaient pas réduits par la maladie.

The fungus *Cochliobolus sativus* (Ito & Kurib.) Drechs. ex. Dastur was found to be the primary cause of a crown rot of barley (*Hordeum vulgare* L.) in Utah. On a yearly basis, about 80% of all diseased crowns examined contained *C. sativus*. *Fusarium* spp. and other fungi were isolated at much lower frequencies. Symptoms of the crown rot were reproduced upon inoculation with *C. sativus* (1).

In Utah, the disease predominantly affects the crowns, basal stems, and sometimes the lower leaves of barley. Crown rot is prevalent throughout irrigated grain fields in the Great Basin and reaches maximum intensity as the plants approach maturity (1). Morphologically, however, the plants do not appear to be affected by the disease, i.e. there is no stunting or general chlorosis associated with it. As a result, the growers in this region are generally unaware of the presence of the disease in their fields.

Several yield components have been reported to be affected through infection of small grains by this pathogen. Ledingham et al. (2) found that plants infected with *C. sativus* had fewer heads and less kernel weight than healthy plants. Verma et al. (7) and Machacek (3) observed that infected wheat plants had fewer tillers and kernels. Piening et al. (4, 5) reported that the number of heads per barley plant were fewer as a result of infection. Our objective in this study was to determine the affect of this disease on barley yields in Utah.

Materials and methods

Disease severity was visually rated and expressed as a disease index (DI), ranging from 0 to 3 depending on the extent of lesion development in the crown. Plants rated 0 were healthy; those rated 1 had only the outer sheath with visible infection; a rating of 2 was given when the lesion extended into the basal stem; and a rating of 3 indicated that the crown, basal stems, and usually the lower leaves were severely infected. Once plants had begun to tiller, each tiller was indexed individuall for disease.

The influence of disease on yield was estimated by sampling 100 plants from 6-23 commercial fields every year except 1980. Twenty random locations per field were selected, and five adjacent plants from each location were rated for disease severity. During 1980 only one field was studied, and groups of five plants were selected at random until at least 100 plants in each disease-index category were obtained. In all cases, the number of fertile heads per plant, the number of kernels in the primary tiller, and the dry weight of the kernels in each tiller were determined. Dry weights of kernels were determined by hand threshing the grain from each head and then oven drying it at 80°C until no further weight reduction was detected.

The influence of disease on host-water relations was examined using a sprinkler-irrigated plot of Steptoe barley. The water potential of the leaves was determined using a pressure chamber (Soil

Moisture Equipment Corp., Santa Barbara, CA). Stomatal conductance was measured using a Delta-T automatic porometer (Delta T Devices, Burwell, Cambridge, England). Measurements were taken at the time of partial and complete flag leaf emergence and at plant maturation before plant senescence had begun. Flag leaves were used for the latter two sets of readings, and the leaf immediately below the flag leaf was used for the first set of measurements. Readings were made at 0500 h (pre-dawn), 1000 h, 1500 h, and 2000 h. Twenty plants were sampled at each of the four times. After each set of measurements, the plants were dug and the disease index recorded.

Results

Because the disease was widespread in 1981 and 1982, there were very few plants with a disease index of 0. Therefore plants of disease indices 0 and 1 were combined for calculations of the effect of disease on yield during these two years.

The disease had no significant effect on the average number of fertile heads per plant, except during 1982 when the coefficient of determination (r^2) between number of fertile heads and disease index was 0.92 (Fig. 1). No correlation existed between the number of kernels in the main tiller and the disease index, except in 1978 (Fig. 2) when, as the disease index increased, fewer kernels were present ($r^2 = 0.85$).

In general, when yields were limited because of disease, the lower yield resulted from a lower kernel weight per head. This effect, however, was not consistent during the years of our study (Fig. 3). Kernel weights were significantly lower during 1978–1980 and were a function of disease index. During 1978, the DI values of 1, 2, and 3 resulted in kernel weights of 75, 56, and 36% the weight of kernels from plants with DI of 0. During 1980 these values were 86, 65, and 46%, respectively. In both 1978 and 1980 there was a highly significant relationship between DI and kernel weight. In 1979 there was much less disease than in either 1978 or 1980. However it was still possible to detect significantly lower kernel weights with each corresponding increase in disease index. No correlation between disease index and kernel weight was observed in 1981 and 1982.

The influence of disease on host-water relations was examined in field plots in 1982. Although only a few healthy plants (DI = 0) were present for comparison, at no time was an increase in disease severity correlated with an increase in host water stress (Fig. 4). Leaf water potentials averaged for the summer showed that the plants uniformly exhibited little water stress at any hour readings were taken, regardless of DI. Stomatal conductance values

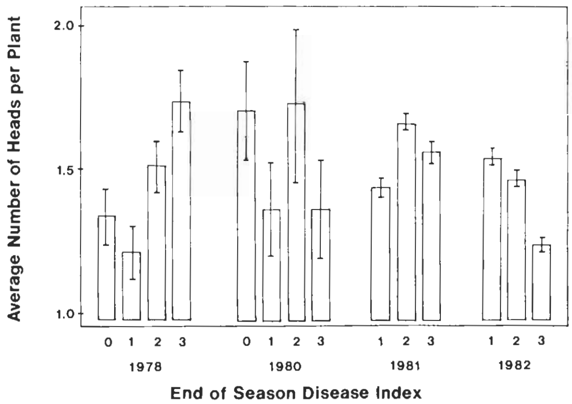

Figure 1. Average number of heads per plant as a function of disease index. DI classes range from no disease (0) to severe disease (3). Vertical bars represent twice the standard deviation.

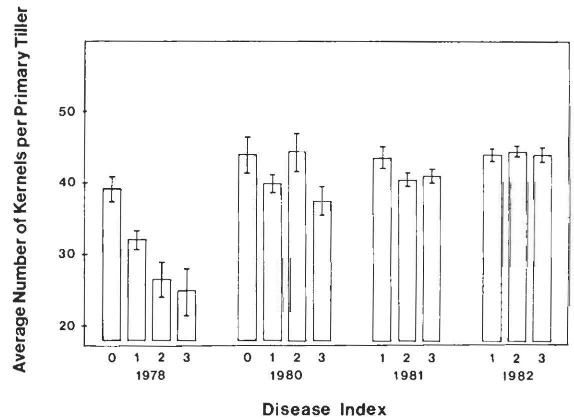

Figure 2. Average number of kernels per primary tiller as a function of disease index. DI classes range from no disease (0) to severe disease (3). Vertical bars represent twice the standard deviation.

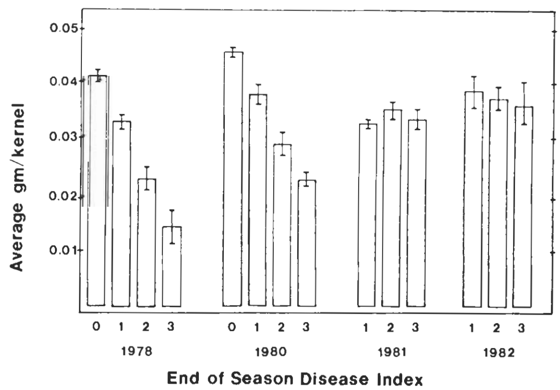

Figure 3. Average weight per kernel as a function of disease index. DI classes range from no disease (0) to severe disease (3). Vertical bars represent twice the standard deviation.

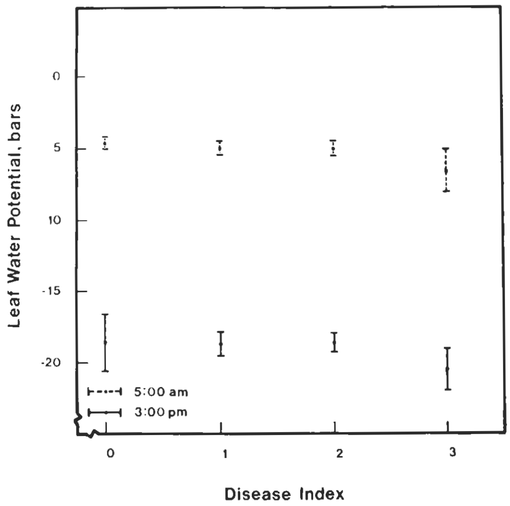

Figure 4. Leaf water potential of barley determined at two different times of the day as a function of disease index. DI classes range from no disease (0) to severe disease (3). Vertical bars represent twice the standard deviation.

expressed a similar lack of correlation with disease levels.

Discussion

Yield parameters of barley investigated in this study were variably influenced by infection with *C. sativus*. In three of the five years (1978-1980) the greatest effect of the disease was lower kernel weights. This may be the result of disease reaching maximum severity when kernel filling occurs, as was the case in the year of this study (1). The plants may also be under water stress at that time, which could result in greater susceptibility to the pathogen or increased physiological stress to the plants due to the pathogen's presence. In 1978, as Verma et al. (7) found in their study, kernel numbers were fewer as disease severity increased. This was not true of the other years of the study. Tiller numbers decreased as a function of disease severity of the surviving tillers only in 1982. An obvious explanation of the fewer tiller numbers in 1982 is that infections occurred earlier that year. However the data collected on progress of the disease in 1982 were no different from those of the other years of this study (1).

In four of the five years during which data were collected, parameters of barley yields were limited by this pathogen. During the years when yields were lower because of the disease, the most important effect was obviously because of lower kernel weights. Plants rated with a DI of 3, the most severe disease ranking, had kernel weights of only 36 to 46% those of healthy plants. However this effect was not consistent from year to year. A similar lack of consistent effect of the disease on yield parameters was also reported by Tinline and Ledingham (6). They suggested that the inconsistency may be artificial since a DI based only on subcrown internode lesions may be an inadequate method of assessing disease severity in barley. Our results suggest that the method of assessing disease may not be the problem since we had a similar lack of consistency using a different method of estimating DI.

Kernel weights during 1981 and 1982 generally remained high throughout all disease categories (Fig. 3), but were progressively lower with each increase in disease category during 1978 - 1980. *C. sativus* successfully invaded most plants during 1981 and 1982, but kernel weights were not reduced in those years. The only yield parameter affected by the disease during these two years was tillering, which was limited by disease during 1982.

Yearly variation of the effects of the pathogen on yield parameters may indicate that effects of the pathogen are greatest during years when the barley is additionally stressed, e.g. in years when the crop receives insufficient water at critical times of growth. The 1982 studies of the water status of healthy and diseased plants showed that in a year when disease had no effect on yield, the disease also had no effect on plant-water status. None of the plants was water-stressed at any of the sampling times since stomates remained at least partially open, even during the hottest part of the day. Flag leaf water potentials were never lower than -24 bars. Unfortunately we do not have similar data for years when the disease did reduce yields.

A correlation between decreased availability of water for cereal crops and limitation of yield caused by crown rot has been noted by other researchers. Ledingham et al. (2) have observed that high levels of infection may be tolerated by wheat without significant effect on yield as long as moderate temperatures and adequate rainfall prevail.

Our results from 1978 to 1980 and other researchers' work indicate that yield can certainly be limited due to crown rot. This is not a simple disease to understand, however, and it is difficult at this point to predict the influence of *C. sativus* on yield on a yearly basis. It is particularly puzzling that during 1981 and 1982, when disease incidence was very high, effects of the disease on barley yields were minimal. Before any recommendations can be made concerning crown rot control, much more information about environmental factors that may be acting synergistically to stress the host must be compiled and correlated with disease severity and yield data.

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