Temperature and Moisture Influences on Development of White Mold Disease (*Sclerotinia sclerotiorum*) on Great Northern Beans

A. WEISS, Assistant Professor of Agricultural Meteorology, and E. D. KERR, Professor of Plant Pathology, University of Nebraska Panhandle Station, Scottsbluff 69361; and J. R. STEADMAN, Associate Professor of Plant Pathology, Department of Plant Pathology, University of Nebraska, Lincoln 68583

ABSTRACT

WEISS, A., E. D. KERR, and J. R. STEADMAN. 1980. Temperature and moisture influences on development of white mold disease (*Sclerotinia sclerotiorum*) on Great Northern beans. Plant Disease 64:757-759.

Great Northern dry edible beans (*Phaseolus vulgaris*) inoculated with a suspension of mycelial fragments of *Sclerotinia sclerotiorum* were incubated in a constant-temperature chamber at 100% relative humidity for periods up to 268 hr at 10–30 C. Percentage of diseased foliage was measured and expressed as a function of time and temperature. Air temperatures favorable for disease development ranged from 10 C to the optimum 25 C. Plants incubated at 30 C did not develop white mold. White mold did develop, although more slowly, on plants incubated at 25 C until symptoms first appeared, at 30 C for 24 hr, and then at 25 C. In a field experiment, air temperatures at 10-cm height were measured in Great Northern bean plots under normal and heavy irrigation treatments. Temperatures were favorable for white mold development 82 and 87% of the time in normal and heavy irrigation treatments, respectively. Disease incidence was about 13 times greater in the heavily irrigated plot than in the normally irrigated plot, indicating that duration of leaf moisture, rather than air temperature, limits white mold disease development in western Nebraska.

White mold disease, caused by *Sclerotinia sclerotiorum* (Lib.) deBary (=*Whetzelinia sclerotiorum* (Lib.) Korf and Dumont [8]), attacks more than 360 plant species, including most oilseed, legume, and vegetable crops. In western Nebraska, where dry edible beans (*Phaseolus vulgaris* L.) are grown under irrigation, this disease can reduce yields significantly (7). Microclimate modification, eg irrigation scheduling and/or choosing cultivars with differing canopy architectures, is one way to ameliorate the effects of white mold disease on dry edible beans. This method is easy to use, but it requires an understanding of the quantitative interactions of microclimate and white mold development.

White mold disease of bean generally can be divided into three stages: 1) germination of sclerotia in the soil and subsequent release of ascospores from apothecia, 2) ascospore germination and mycelial colonization of senescent flowers, and 3) infection of healthy plant tissue and subsequent deterioration of the plant. The influence of environment on survival and germination of sclerotia and the importance of senescent tissue as an energy base for initial infection have been described (1–6, 9–12). Less attention has been given to the third stage, infection of healthy plant tissue, which we investigated in a controlled environment as a function of time and temperature. Our results were combined with field data to determine the relative importance of temperature and moisture as possible parameters for microclimate modification.

Published as Journal Series paper 5921, Nebraska Agricultural Experiment Station.

MATERIALS AND METHODS

An isolate of *S. sclerotiorum* from Great Northern bean was grown on potato dextrose broth (PDB). After 1 wk at 22 C, the fungus covered the surface of 250 ml of PDB in a 1,000-ml Erlenmeyer flask. This mycelial mat was rinsed in distilled water and comminuted for 15 sec at a low speed. The mycelial fragments were washed through a 60-mesh (250-μm opening) screen into 600 ml of distilled water, and 12 g of dextrose were added to serve as an energy base for the fungus until infection occurred.

Great Northern beans, cultivar UI 59, were grown three plants to a pot in a 1:1 sand/peat moss potting mixture (supplemented as needed with a modified Hoaglands solution) in a greenhouse for 5–6 wk. After flowering, the plants were sprayed with the inoculum suspension until all leaves were covered to the point of runoff. Inoculated plants were placed in a polyethylene chamber (41 × 41 × 61 cm) kept at 100% relative humidity in an incubator maintained at a constant temperature. The temperatures in the chamber were set at different 5-degree intervals between 10 and 30 C, and each temperature treatment was replicated three times. Plants were exposed to light 12 hr per day.

Twelve plants were placed in the chamber in each experiment. Two plants were removed every 24 hr after symptoms were first observed. The leaves were placed in a plant press until dry, and the leaf area was measured by a Li-Cor area

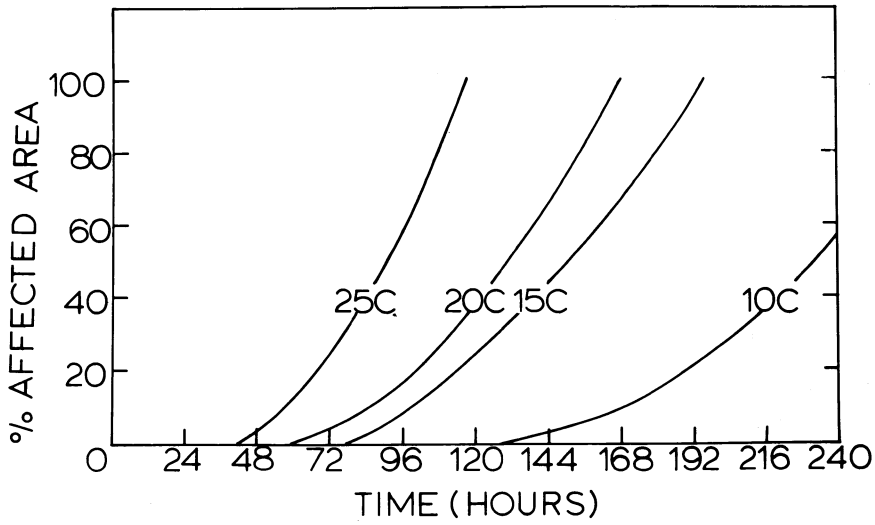

Fig. 1. Percentage of leaf area affected by white mold (*Sclerotinia sclerotiorum*) of dry edible bean plants as a function of time after inoculation and temperature.

0191-2917/80/08075703/$03.00/0
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meter (model Li-3000) with a transparent belt conveyor (Li-3050A) (Lambda Instruments Corporation, Lincoln, NE 68504).

Because infection was not observed at 30 C, another experiment was performed and replicated twice to determine how a previously infected plant reacts to 30 C. The temperature in the chamber was kept at 25 C until symptoms were observed (48 hr after inoculation), then changed to 30 C for 24 hr, and finally returned to 25 C until all 12 plants were consumed. Percent leaf area with symptoms was then determined by measuring the leaf before and after excision of the infected portion.

Air temperatures recorded in a field experiment relating white mold disease in dry edible beans to microclimate (13) during the period when the disease was first observed and approached its maximum severity (20 August–31 August 1976) were reexamined. These temperatures were measured at a height of 10 cm in two plots (18 × 20 m) of the Great Northern cultivar Tara, which has a vigorous, indeterminate growth habit. One plot received a normal irrigation treatment of 5.5 cm of water every 10 days; the other plot received 5.5 cm every 5 days, a heavy irrigation treatment.

RESULTS

The development of white mold in bean plants was approximately an exponential function of time (Fig. 1). In general, the rate of development increased with temperature. For example, at 20 C, 20% of the leaf area was affected after approximately 98.4 hr, and 60% was affected after 138.6 hr. At 25 C, about 67.2 hr and 97.2 hr were required for the same proportions of affected leaf area. That is, the same change in percent affected leaf area was reached about 10.2 hr faster at 25 C than at 20 C.

At 25 C, white mold symptoms were observed 48 hr after inoculation and had spread over the entire leaf area by 120 hr (Fig. 1). At lower temperatures, the disease developed progressively more slowly, as indicated by the slopes of the disease development curves. At 10 C, symptoms first appeared after approximately 127 hr, spread to 57% of leaf area after 240 hr, and reached 100% after approximately 268 hr. Severe injury to leaf petioles and upper stems also contributed to leaf damage at this temperature. Petiole damage was less evident at higher temperatures; however, the total exposure was shorter.

At 30 C, leaf lesions did not appear, although occasional minor infections were observed on some petioles and stems. The leaves turned yellow and abscised within 48–72 hr of inoculation. In inoculated plants incubated for 48 hr at 25 C, then at 30 C for 24 hr, and then returned to 25 C for the duration of the experiment, disease development was again approximately exponential (Fig. 2). The rate of development, however, was much slower than in the 25-C experiment.

Figure 3 shows the distribution of hourly averages of air temperatures in 5-C intervals measured during the period of greatest disease development (20–31 August) in the heavily irrigated plot. Seventy-three percent of the observations fell between 10 and 25 C, the temperature range favorable for disease development. This value rose to 87% when the 25–30 C interval was included. The data summarized in Fig. 2 suggest that the 25–30 C interval should be considered in calculating the percentage of time that temperatures are compatible with disease development.

In the normal irrigation treatment plot, temperatures were similarly distributed, with 69% of the observations falling between 10 and 25 C and 82% falling between 10 and 30 C. On 31 August, only 3% of the plants in the normal irrigation plot had white mold infection, while 40% were infected in the heavy irrigation plot.

DISCUSSION

Infection of bean leaves by *S. sclerotiorum* mycelium and spread of symptoms were favored by a wide range of temperatures. The relationship between temperature and symptom development agrees closely with that found for excised leaves by Abawi and Grogan (1), except that they reported that lesion diameter developed at about the same rate at 20 and 25 C for 48 and 68 hr. Our optimum temperature for disease development, 25 C, corresponds to their optimum temperature for mycelial growth and sclerotial production on PDA. Abawi and Grogan (1) reported 48–72 hr of free moisture before plants inoculated with ascospores showed signs of infection; we found a similar pattern for mycelial inoculum.

The continued development of the disease, though at a slower rate, during and after the step change in temperature to 30 C suggests that short exposure to high temperatures in the field may not effectively curb the disease.

We conclude that air temperatures in the plant canopy were favorable for disease development 82 and 87% of the time for the normal and heavy irrigation treatments, respectively. Yet disease incidence on 31 August was 3% under normal irrigation and 40% under heavy irrigation. Spread of white mold apparently was limited not by air temperature, but by the availability and duration of free moisture on the leaves, as concluded by Abawi and Grogan (1). Whereas precipitation provided leaf moisture in their New York State study, irrigation was the moisture source in our study. Thus, irrigation scheduling may reduce the severity of white mold in beans grown in a semiarid region such as western Nebraska.

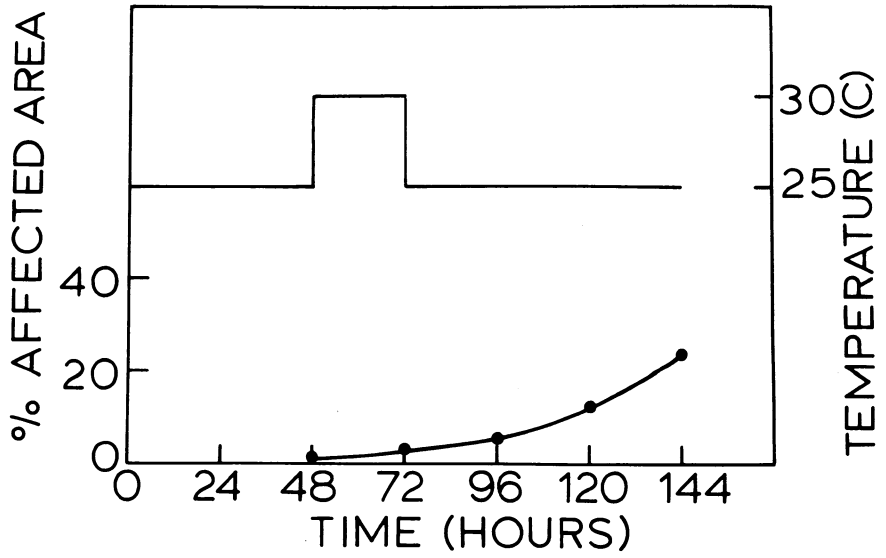

Fig. 2. Influence of a step change in temperature of limited duration on percentage of leaf area affected by white mold (*Sclerotinia sclerotiorum*) of dry edible bean plants.

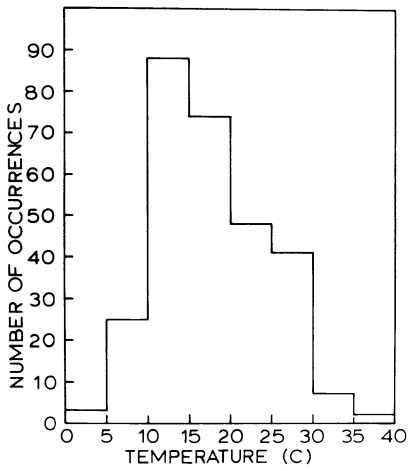

Fig. 3. Distribution of hourly average air temperatures (in 5-C intervals) at 10 cm above ground in Great Northern cultivar Tara heavy irrigation plot during 20–31 August 1976.

ACKNOWLEDGMENTS

J. L. Weihing provided encouragement and advice during this study. N. J. Rosenberg and D. S. Wysong made valuable comments in reviewing the manuscript. R. B. Suverkrubbe provided valuable technical assistance, and A. Muldoon analyzed and prepared the data for computer calculation.

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