An Epidemic of Sorghum Downy Mildew in Nebraska in 1987

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ABSTRACT

Jensen, S. G., Doupnik, B., Jr., Wysong, D., and Johnson, B. 1989. An epidemic of sorghum downy mildew in Nebraska in 1987. Plant Disease 73:75-78.

Sorghum downy mildew was found in 17 of 34 counties surveyed in south central and southeastern Nebraska in 1987. Grain sorghum, sudangrass, and shattercane were infected, but no maize was found with symptoms. The most common symptoms observed were leaf spots caused by conidial infections and systemic stripes on tissues that developed after the main stalk. In some grain sorghum fields in south central Nebraska, however, up to one-third of the plants had early systemic infection of the primary stalk, resulting in significant yield reduction. We hypothesize that the exceptionally early and warm spring, with soil temperatures 5–8 C above normal, contributed to the epidemic, and we offer a theory to account for the presence of inoculum where the disease had not been seen before.

Additional keywords: epidemiology, *Peronosclerospora sorghi, Sorghum bicolor*

Sorghum downy mildew, caused by *Peronosclerospora sorghi* (Weston & Uppal) C. G. Shaw, was first observed in the United States in 1961 (6) in southern Texas. It caused little concern until 1967, when it was found to be widely distributed in Texas and elsewhere in the southern corn- and sorghum-producing areas. The disease was first observed in Kansas in 1967 (6), but major outbreaks did not occur until 1978 (9) and 1979 (16). The disease was discovered for the first time in Nebraska in 1978 (10) and has been observed since then only on rare occasions. Prior to 1987, it had not been an economic problem in the state.

The pathogen overwinters as a thick-walled oospore capable of surviving for several years in the soil (6). Given proper environmental conditions (11,12,14) and in the presence of roots, the oospore germinates, penetrates the root, and grows intercellularly toward the apical meristem (7,13). Once it has reached the differentiating tissue, it keeps pace with the plant growth and induces characteristic systemic mosaic or striping symptoms. Conidia formation is triggered by external environmental conditions (6), and the conidia produced can cause several cycles of localized infections in a season. Oospores are produced, usually in large quantities, only in the systemically infected tissue. Systemically infected tissue dies prematurely and becomes

Accepted for publication 31 August 1988.

friable, and the leaves shred and release oospores into the soil for the next cycle of disease.

Early in the 1987 growing season, conidial infections were observed on a number of sorghum genotypes in a breeding nursery at the University of Nebraska Agricultural Research and Development Center (ARDC) at Mead in eastern Nebraska. A few tillers, showing systemic symptoms, were the probable source of the conidia.

Nearby grain sorghum nurseries and sorghum-sudangrass plots were free from disease, and the grain sorghum nurseries remained free from sorghum downy mildew through the season. The sorghum-sudangrass plots were part of a regrowth study, and with each mowing throughout the summer, regrowth showed progressively more systemic symptoms. By late fall, all of the plants in the majority of the plots were showing systemic downy mildew symptoms.

Additional sorghum downy mildew observations were made at the ARDC. One was on late-developing shattercane in a soybean field that had been the sorghum breeding nursery in 1986. The second, a newly seeded alfalfa field, had a heavy infestation of shattercane, with over 50% of the plants infected with sorghum downy mildew. This field had a history of more than 10 yr of continuous cropping to maize and was rotated to alfalfa to control the shattercane infestation.

Sorghum downy mildew was also observed in breeding materials planted in isolated sites throughout the 3,600 ha of ARDC. These same isolation plots are used annually for random intermating sorghum populations and are located away from other sorghum fields to prevent contamination from windborne sorghum pollen. Each isolation plot contained approximately 6,500 plants in a 730-m^2 area. From one to three plants in six of eight isolations were observed to have systemic downy mildew, typically seen on tillers and secondary tissue.

Sorghum and sudangrass have been continually grown at the ARDC since 1967. In spite of intensive observations in previous years, sorghum downy mildew had never been observed on sorghum, maize, sudangrass, or shattercane in any of these research fields before 1987.

MATERIALS AND METHODS

Regrowth of grain sorghum. Because of the frequency of systemic symptoms on late-developing and regrowth tissue, a simple experiment was conducted to corroborate these observations. In the breeding nursery, there was a block containing 32 paired rows of various grain sorghum genotypes. At approximately 14 days after anthesis, one row of each pair was cut off at approximately 30 cm to stimulate regrowth of the basal and lateral buds. Sixty days later, notes were taken on the number of plants showing sorghum downy mildew on any part of the plants in either row.

Field survey. By mid-August, we received confirmed reports of sorghum downy mildew in producers' fields from several locations in south central and southeastern Nebraska. A systematic survey was conducted in 34 counties between late August and early September. The objectives of the survey were to inspect three to five randomly selected fields per county to determine: 1) the geographic distribution of the disease, 2) the type of symptoms found, and 3) the economic losses.

Pathotype designation. Soil from the infected plot at ARDC was used to grow susceptible sorghum seedlings, and infected plants were used to produce conidia and infect seedlings according to the methods of Craig (2,4). Seedlings (1–3 cm tall) of cultivars Tx412, CS3541, Tx430, and QL-3 were the differentials used for inoculation with conidia. The seedlings were held at 25 C for 6 days and examined for systemic symptoms and conidia production.

RESULTS

Regrowth of grain sorghum. In the regrowth study involving 32 paired rows of grain sorghum, five uncut rows had

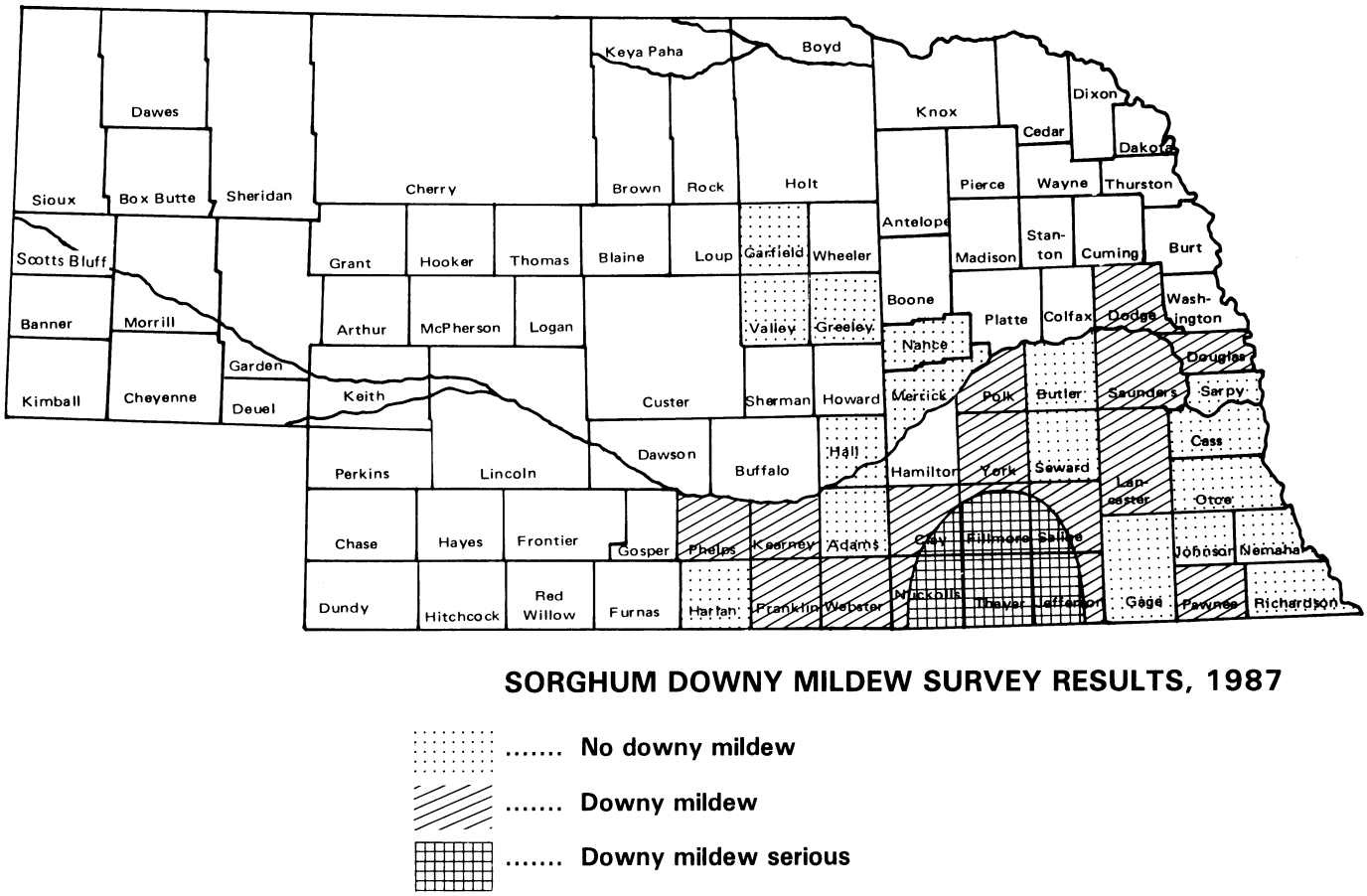

Fig. 1. Occurrence of sorghum downy mildew in Nebraska based on 1987 survey results.

one plant each with systemic symptoms on late growth or regrowth tissues. In the cut rows, 21 of 32 had one to eight plants per row showing systemic symptoms on the regrowth tissues. There were 80 total plants showing symptoms in the cut rows, compared with five in the uncut rows. Often, but not always, all of the regrowth shoots on a cutoff plant would show symptoms. Shoots without symptoms were always the upper shoots on the plant. The amount of regrowth varied considerably from genotype to genotype, and downy mildew incidence was not correlated with the amount of regrowth. Two of the rows with the greatest amount of regrowth had no sorghum downy mildew.

Field survey. The results of the survey are shown in Figure 1. Sorghum downy mildew was found in 17 of the 34 counties surveyed. In these 17, the disease was found in 26 of 61 fields surveyed. In the 17 counties where no sorghum downy mildew was found, 59 fields were surveyed. Typical disease observations were the appearance of a few conidial infections on leaves and/or systemic symptoms on a few late-growth or regrowth tillers. Only one or two systemically infected plants were found in several of the inspected fields. However, in Clay, Fillmore, Nuckolls, and Thayer counties, several fields were observed where the incidence of disease was much higher and the systemic symptoms were on the main stem. In the worst of these fields, three pathologists made independent stand counts in different parts of the field. The estimates of the plants that were systemically infected and therefore did not produce heads ranged from 25 to 35%. The producer estimated a 30–40% reduction in yield. Throughout the survey there was no correlation between downy mildew incidence and previous cropping, as estimated from plant residue in the field.

Pathotype designation. In the pathotype identification studies, the cultivar Tx412 was highly susceptible, showing nearly 100% infection, while Tx430, CS3541, and QL-3 were resistant, having 20% or less infection in three experiments. This confirms the identification of the pathogen at ARDC as pathotype 1. Pathotype 1 was also identified from shattercane collected in Nebraska near the Republican River in 1981 (*unpublished*).

DISCUSSION

Sorghum downy mildew was first reported in Nebraska in 1978 (10) when the disease was epidemic in Kansas (9). Normally, the disease is rare in Nebraska. In 1984, however, two fields in Clay County were found with heavy conidial infections. In 1985, a chance observation was made of systemic symptoms in regrowth of 25% of broken grain sorghum plants in a commercial field in Saline County.

With such rare observations of the disease in Nebraska, the question naturally arises about the source of inoculum for its widespread appearance in 1987. In most of the producers' fields, it is possible that an occasional symptomatic plant escaped detection and a new but low level of inoculum was periodically added to the soil. However, this was probably not the case in the experimental fields at ARDC, where trained observers would not likely have overlooked the striking systemic symptoms of sorghum downy mildew.

The pathogen might have been introduced into a new area via a few seedborne (1,5) or windborne (3) oospores. However, an infestation as severe as 20–50% of the main stems, as seen in some commercial fields, would have required a high level of oospore inoculum.

Systemic invasion can result from conidial infection but only with seedlings less than 2 wk old (8,17). In a field planting, a single seedling infected early would not produce conidia before the rest of the crop was too mature to develop systemic symptoms from conidial infection.

Systemic infection of regrowth could, in theory, result from local conidial infections that might spread to a dormant lateral bud. If that bud was stimulated to grow, it might show systemic infections. From our observations in Nebraska, that mode of systemic infection seems unlikely. In 1984, we intensively surveyed a 100-acre field of sorghum that had a high incidence of conidial infections without observing systemic symptoms on a single main tiller or lateral shoot. In 1985, in two widely separated spots at the edge of one field where traffic had broken many plants, about one-quarter of the broken plants had systemic symptoms on the regrowth. There were no systemic symptoms on unbroken plants or conidial infections on any broken or unbroken plants. In this case, systemic infection was probably not caused by conidial infections.

We speculate that the most probable source of oospore inoculum for the 1987 epidemic in Nebraska was from the latent infections that occur in sorghum and shattercane in most years. Because of environmental constraints, the fungus does not reach the zone of differentiation in meristematic tissue, so no typical systemic symptoms result. The tissue is invaded by the mycelium, however, and sufficient numbers of oospores are produced to maintain a significant oospore load in the soil. In support of this theory, Jones (8) observed the hyphae of *P. sorghi* in the stems of symptomless plants.

This theory would be consistent with the observations in Kansas and Texas, where the disease was first seen and reported in low levels in isolated areas. Oospores may have been spread at low levels by wind, seed, or other sources, but no significant amount of disease was seen. Then, several years later, with the occurrence of environmental conditions such as warm soils and optimum moisture that favor the disease, downy mildew became very widespread and economically threatening.

If these considerations are correct, the key to the 1987 sorghum downy mildew outbreak in Nebraska was the weather. The winter of 1986–1987 was very mild, and the spring was warm and very early. In south central Nebraska, soil temperatures at planting depth were above 20 C by mid-April and above 25 C by the end of April, well before any sorghum was planted. During planting and early growth of the seedlings, the average soil temperature at 4 cm was 5–8 C above normal. Specific measurements of soil moisture were not available, but soil moisture reserves and accumulated precipitation were about 7 cm above normal at the beginning of the growing season and stayed about that far above normal through mid-June. The sorghum seedlings had an unusually early, fast, and vigorous start.

Environmental conditions favorable to oospore germination and infection have been carefully described by laboratory studies (12,14), but the environmental factors identified in laboratory studies did not correlate with field observations in Texas (15). Principal component analysis, a complex mathematical model that combines environmental factors in various ways, was required to account for a significant amount of the variation in field infection. Pratt (11) observed that several weeks of preconditioning were required before overwintering spores were fully infective. Preconditioning may be a critical factor in Nebraska, where soil temperatures warm rapidly in the spring.

Factors leading to the sudden widespread outbreak of sorghum downy mildew in Nebraska require further investigation, but this 1987 epiphytotic parallels those occurring in Kansas in 1978 and Texas in 1967, where sorghum downy mildew remains an economic problem.

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