Development of Sudden Death Syndrome of Soybean in Relation to Soil Temperature and Soil Water Matric Potential

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ABSTRACT

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Sudden death syndrome, caused by strains of *Fusarium solani*, has been recognized as an important disease of soybean (*Glycine max*) in the southern United States for more than 20 years, but has only recently become more prevalent and severe in northern soybean production areas. Little is known about environmental factors that influence root infection and above-ground symptom expression in this pathosystem. We established quantitative relationships between soil temperature, the matric component of soil water potential (ψ_m), and disease development in controlled conditions. Soil temperature in the range of 15 to 30°C was manipulated in pots equipped with heating tapes, and soil moisture was manipulated by withholding irrigation water until ψ_m reached threshold values of –0.003, –0.02, –0.05, –0.1, –0.2, –0.4, or –0.8 MPa; when ψ_m for a given moisture treatment had reached its threshold, the pots belonging to this treatment were watered until the soil was saturated. The most important results were that i) soil temperature differentially influenced the development of root symptoms and foliar symptoms, with the former being most severe at low temperatures (15°C) and the latter being most severe at intermediate temperatures (22 to 24°C); ii) the severity of foliar symptoms decreased rapidly with decreasing soil moisture and was negatively correlated with the number of days the average ψ_m was lower than –0.01 MPa; and iii) there was no close correlation between root disease severity and foliar disease severity. The relationships between ψ_m and disease derived in controlled conditions were confirmed in a field irrigation trial. The information provided here, when used together with long-term temperature data and regional soil moisture maps, should be valuable in assessing the risk that sudden death syndrome will continue to expand into previously unaffected production areas.

Sudden death syndrome is a mid- to late-season disease of soybean (*Glycine max* (L.) Merrill) caused by blue-pigmented, slow-growing strains of *Fusarium solani* (Mart.) Sacc. (28,30) that are similar to *F. solani* f. sp. *phaseoli*](Burkholder) W. C. Snyder & H. N. Hans. (25). Although the pathogen is soilborne, the disease causes characteristic foliar symptoms that are generally first observed during flowering or pod development as mottling or mosaic on the upper leaves. Within a few days, chlorotic blotches develop on leaves, which rapidly become necrotic and coalesce to form interveinal necrotic streaks. In severe cases, the leaflets drop off leaving the petioles attached to the stem; pod abortion and pod drop also occur. Below-ground symptoms consist of crown necrosis and lateral root rot (32). Despite recent progress in understanding the ecology and epidemiology of sudden death syndrome (22,31,45), it has remained difficult to explain within-field patterns or seasonal variations of the disease based on environmental factors, soybean growth stage, or management practices.

Over the past 20 years, sudden death syndrome has become an important problem in the southern soybean production areas of the United States, from northern Mississippi to southern Illinois and southwestern Indiana (27,32). In recent years, however, the disease has been observed with increasing prevalence and severity in soybean fields in more northern states: on localized scales in Kansas (17) and on larger scales in central and northwestern Indiana (1), east-central Illinois (12), and southeastern Iowa (46,47). In east-central Illinois in 1993, yield losses in fields with high incidence of the disease ranged from 20 to 46% (12). It is not known whether strains of *F. solani* that cause sudden death syndrome have just recently been introduced into northern production areas or whether the disease has been present there without being recognized and its increasing prevalence was because of unusual weather (e.g., midwestern floods in 1993) or other factors (e.g., changes in management practices).

Based on the apparent expansion in the geographical range of sudden death syndrome and the potential yield losses associated with this disease, there is concern among producers in the North Central region, which yields approximately 80% of the U.S. soybean harvest (9), that sudden death syndrome might become a widespread problem there. Therefore, risk assessment studies are needed to determine if and where environmental conditions are suitable for consistent occurrence of the disease. For such studies, quantitative information is needed on the relationships between environmental and edaphic factors and disease development. Such information is presently not available for sudden death syndrome.

Sudden death syndrome is thought to be most severe under cool and wet conditions (32), but the precise relationship between soil temperature, soil moisture, and disease development has remained elusive. For example, in field surveys in Arkansas, Mississippi, Illinois, and Tennessee, Hirrell (14) noted that disease onset generally followed below-normal temperatures and above-normal rainfall. In contrast, McLean and Lawrence (21), based on results from microplot experiments in Mississippi, suggested that years with high soil moisture and low temperatures during the early part of the growing season followed by relatively high temperatures during reproductive development of the soybean crop are optimal

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for symptom expression. Rupe and Gbur (31) reported that sudden death syndrome in field plots in Arkansas followed a two-phase disease progress curve: early epidemic development appeared to correlate with accumulated heat-units (degree-days), whereas later development seemed to be related to rainfall and soil moisture. Relationships between soil moisture and disease intensity were not presented in their study, however.

In this report, we document experiments designed to i) establish quantitative relationships between soil temperature, the matric component of soil water potential (ψ_m), and disease development in controlled conditions; and ii) validate the relationships between ψ_m and disease derived in controlled conditions using a field irrigation trial. A brief report of parts of this work has been published (34).

MATERIALS AND METHODS

Fungal isolates. Three isolates of *F. solani* known to cause sudden death syndrome were used: isolate 171, recovered from a symptomatic soybean plant in Lee county (Arkansas) in 1991; and isolates BH-F2-13 and W-F1-19, recovered from symptomatic soybean plants in Black Hawk and Washington counties (Iowa), respectively, in 1994. The isolates were stored on silica gel at 4°C (44). Colonized silica gel particles were transferred to potato-dextrose agar (PDA) for inoculum production. For growth chamber experiments, mass production of inoculum was initiated by streaking the fungus onto 9-cm petri plates containing PDA. The plates were incubated for 2 to 3 weeks at 23°C under a 12-h photoperiod (cool, white fluorescent tubes; photon flux density 12 μmol m^{-2} s^{-1}). Inoculum slurries were made by homogenizing colonized media from streak-inoculated plates with media from noninoculated plates (ratio 1:9) and sterile, distilled water (20 ml per plate) in a blender. Inoculum concentrations in the slurries were determined using a hemacytometer.

For the field experiment, inoculum was mass-produced on autoclaved oat grains (39). We used two 1-cm plugs of PDA colonized by *F. solani* (one plug each of isolates BH-F2-13 and W-F1-19) per 150-ml batch of water-soaked, autoclaved oats to initiate fungal growth. Inoculated oat grains were incubated, with periodic shaking, in the dark at 23°C for 3 weeks before use.

Inoculation and plant growth. A mixture of steam-sterilized sand and soil (ratios 1:1 and 2:1 [vol/vol] for the soil temperature and moisture experiments, respectively) was passed through a screen and mixed with PDA-based inoculum at a ratio of 50 to 60 μl of slurry per gram of soil. This resulted in macroconidial densities of 4.2 to 5.1 × 10^4 and 2.8 to 3.8 × 10^4 per gram of soil for the temperature and moisture experiments, respectively. Control soils were prepared similarly with slurries made from noninoculated PDA. Clay pots with a diameter of 10.2 cm (temperature experiments) or 12.7 cm (moisture experiments) were filled with the soils; cultivar BSR 101 soybean seeds were planted at a rate of six per pot. All pots were maintained at 23°C until seedlings began to emerge (4 days after planting), at which time they were moved into a growth chamber with an air temperature of 15°C (temperature experiments) or 20°C (moisture experiments) and a 10-h photoperiod (metal halide lamps; photon flux density 500 μmol m^{-2} s^{-1} at plant height). Seedlings were thinned to four (temperature experiments) or five (moisture experiments) per pot within 1 week after emergence. Liquid fertilizer (N-P_2O_5-K_2O = 20-20-20) was applied at 2-week intervals at a rate of 200 mg of fertilizer per pot (285 μg of fertilizer per gram of soil).

Soil temperature experiments. Four days after planting, silicone rubber heating tapes (Barnstead/Thermolyne, Dubuque, IA) were wrapped around the pots and fastened to them. The heating tapes were connected to stepless input controllers (Barnstead/Thermolyne) adjusted to provide target soil temperatures of 20, 25, or 30°C in the centers of the pots; pots without heating tapes had a target soil temperature of 15°C, equal to the air temperature in the growth chamber. Soil temperatures were monitored continuously in one pot per treatment with thermistors connected to a data logger (Campbell Scientific Inc., Logan, UT). Mercury-in-glass thermometers were used to monitor soil temperatures in additional pots. All soils were kept wet (ψ_m = –0.003 MPa) for the duration of the experiment. There were three pots (replicates) per soil temperature treatment for each of three inoculum treatments (isolates 171 and BH-F2-13 and noninoculated control) arranged in a completely randomized design. The experiment was conducted twice (referred to as repeats 1 and 2).

Soil moisture experiments. Because it is impossible to keep ψ_m at constant levels drier than field capacity in trials with potted plants (7), we employed fluctuating soil moisture regimes. All soils were initially kept wet for 4 days after planting until seedlings began to emerge. Subsequently, plants were subjected to different soil moisture treatments by withholding water until ψ_m reached target threshold values of –0.003, –0.02, –0.05, –0.1, –0.2, –0.4, or –0.8 MPa; when ψ_m for a given treatment had reached its threshold, the pots belonging to this treatment were watered until the soil was saturated. Values of ψ_m were monitored continuously in one pot per treatment with gypsum or Watermark soil moisture blocks (Campbell Scientific; sensor models 227 and 257) connected to a data logger. (Watermark blocks are accurate between 0 and –0.2 MPa, whereas gypsum blocks are accurate from –0.05 to –1.5 MPa.) There were three pots (replicates) per soil moisture treatment for each of three inoculum treatments (isolates 171 and BH-F2-13 and noninoculated control) arranged in a completely randomized design. The experiment was conducted twice.

Disease assessment and data analysis. Foliar disease severity (percent chlorotic or necrotic leaf area) was assessed visually with a Horsfall-Barratt scale (15), with individual plants as sampling units. The assessments were made at 3-day intervals, beginning 21 days (temperature experiments) or 13 days (moisture experiments) after planting. Root disease severity (percent root area with reddish-brown discoloration) was assessed using the same methodology, except that only one assessment was made at the end of the trials (41 and 28 days after planting for the temperature and moisture experiments, respectively). After conversion to percentages using the midpoint rule (5), disease severity values were averaged over plants (subsamples) to give an estimate of disease severity per pot (replicate). The pathogen was reisolated from symptomatic roots by plating discolored root segments onto modified Nash-Snyder medium (37); representative colonies were confirmed on PDA.

Selected variables were plotted against each other, and regression analyses were performed where appropriate. These included i) disease severity against soil temperature, ii) disease severity against ψ_m, iii) disease severity against the number of days the average ψ_m was lower than –0.01 MPa, and iv) foliar disease severity against root disease severity. The relationship between foliar disease severity (y, in percent) and soil temperature (T, in degrees Celsius) was described by fitting the data to a biological growth model (33,35) using nonlinear regression analysis:

$$y = \frac{\rho_{25}\,\frac{T+273.2}{298}\exp\left[\frac{\Delta H_A}{1.987}\left(\frac{1}{298}-\frac{1}{T+273.2}\right)\right]}{1+\exp\left[\frac{\Delta H_H}{1.987}\left(\frac{1}{T_{1/2H}}-\frac{1}{T+273.2}\right)\right]}$$

This equation describes a temperature response curve that is unimodal and skewed to the left. A possible biophysical interpretation of the four parameters, ρ_{25}, ΔH_A, ΔH_H, and $T_{1/2H}$, is discussed by Schoolfield et al. (35). All other relationships were described using linear regression analysis. Regression models were fitted to pooled data after tests for heterogeneity of slopes (19) had shown that regression coefficients were not significantly different between repeats ($P \leq 0.05$). All analyses were carried out using SAS/STAT procedures (SAS Institute Inc., Cary, NC).

Field experiment. A trial was conducted during the 1995 growing season at the Hinds Research Farm near Ames, IA, to validate the relationships between ψ_m and disease derived in the growth chamber experiments. The experimental site, which had no previous history of sudden death syndrome, was a 120-m^2 area of sandy loam soil, covered by an automatic rain-out shelter to exclude rainfall and allow for the experimental manipulation of soil moisture by irrigation. The site, which had been planted to corn during the previous 4 years, harbored low to moderate levels (2,100 eggs per 100 cm^3 of soil) of the soybean cyst nematode, *Heterodera glycines*, which has been implicated as a cofactor for sudden death syndrome (21,22,27,28,29,32). The experiment had six treatments arranged in a randomized complete block design with three replicates. There were two inoculum treatments (non-inoculated and inoculated) for each of three irrigation treatments (irrigation when ψ_m reached target threshold values of –0.008, –0.08, or –0.8 MPa). The 1995 growing season in central Iowa was wetter and cooler than normal in late May, early June, and mid-July; unusually hot and humid in early July; and drier and warmer than normal in August.

Cultivar BSR 101 soybean seeds (maturity group I) were hand-planted on 22 May in microplots that were 1.74 m long and 1.22 m (two plant rows) wide, with an in-row spacing of 3 cm. All plots were separated by border rows and by 10-cm-high dams of soil to prevent movement of irrigation water among plots. A tank mix of herbicide (bentazon [Basagran], clethodim [Select], and imazethapyr [Pursuit]) was applied on 15 June. Plants were grown under natural rainfall until they reached the V7 growth stage (10), at which time they were inoculated and the automatic rain-out sheler was turned on. Inoculations were made on 6 and 7 July (45 and 46 days after planting, respectively) by placing 15 oat grains colonized by *F. solani* (mixture of isolates BH-F2-13 and W-F1-19) next to the stem of each plant at a depth of 1 to 2 cm below the soil surface (39), taking car not to wound the taproots. Control plots were treated similarly by inoculating them with water-soaked, autoclaved oat grains. Immediately after inoculation, all plots were sprinkler-irrigated until the soil was close to saturation. Subsequently, plots were subjected to different soil moisture treatments by withholding irrigation water until ψ_m for a given treatment reached its threshold value, at which time the plots belonging to this treatment were irrigated with a watering can until the soil was close to saturation. Values of ψ_m were monitored continuously in two plots per treatment with gypsum or Watermark soil moisture blocks, and soil temperatures were monitored with thermistors. The sensors were placed at a depth of 10 cm below the soil surface. Irrigation treatments were ended on 28 August (R6 growth stage) when plots in the dry treatment (irrigation threshold –0.8 MPa) began to mature rapidly; at the same time, the rain-out shelter was removed to allow for natural rainfall until harvest. Plants were harvested on 13 October with a single-plant thresher, and grain yields were adjusted to a moisture content of 13%.

Foliar disease severity (percent chlorotic or necrotic leaf area) was recorded three times, on 9, 20, and 27 August at growth stages R5, R6, and R6, respectively. Twenty plants per plot were selected arbitrarily, and the five uppermost leaves of each plant were assessed visually using a Horsfall-Barratt scale, with individual leaves as sampling units. After conversion to percentages using the midpoint rule, disease severity values were averaged over leaves and plants to give an estimate of disease severity per plot. Root disease severity was not assessed in this experiment, but the pathogen was reisolated from symptomatic roots by plating discolored root segments onto modified Nash-Snyder medium; representative colonies were confirmed on PDA.

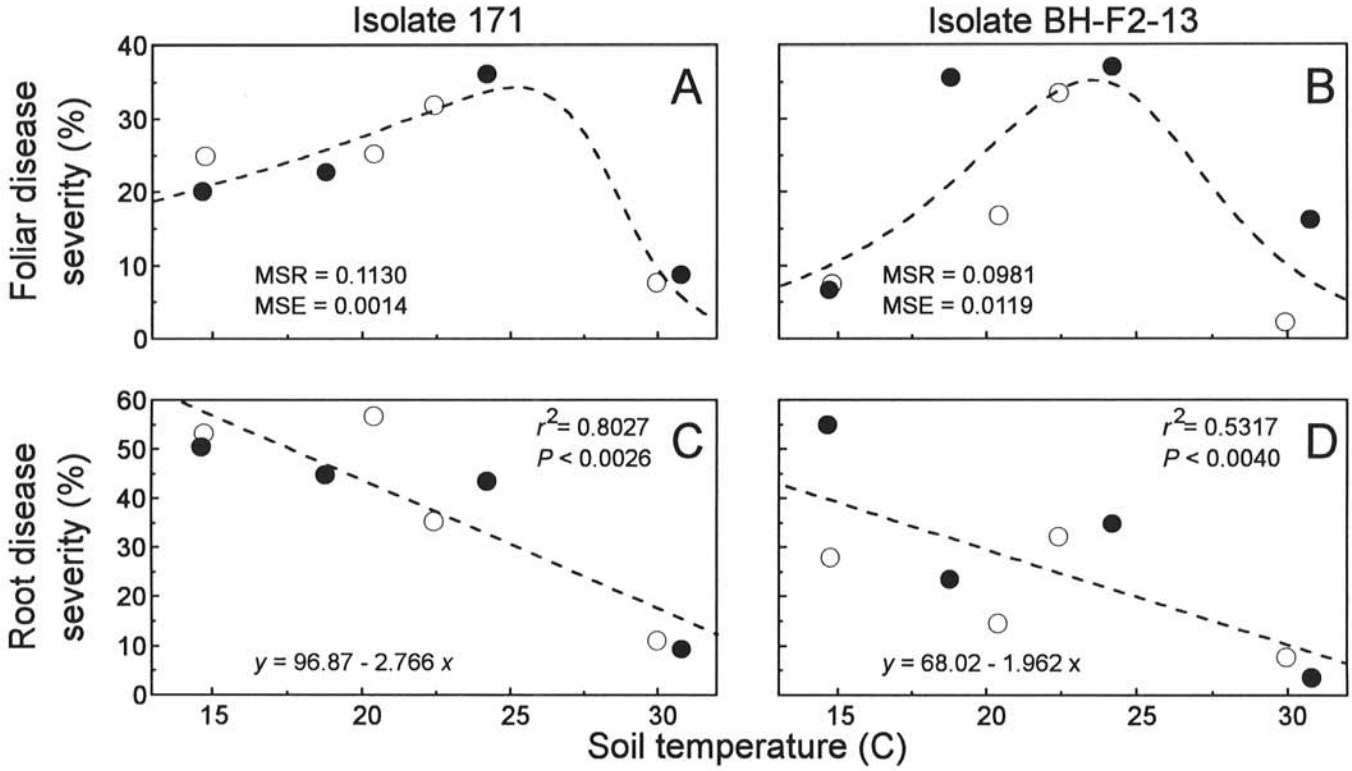

Fig. 1. Relationship between soil temperature and foliar disease severity of sudden death syndrome on potted soybean plants after inoculation with two isolates of *Fusarium solani*: **A,** isolate 171 and **B,** isolate BH-F2-13. Relationship between soil temperature and root disease severity of sudden death syndrome on potted soybean plants after inoculation with two isolates of *Fusarium solani*: **C,** isolate 171 and **D,** isolate BH-F2-13. Closed and open symbols represent results from repeats 1 and 2 of the experiment, respectively. Each point is the mean of three replicates. The curves in **A** and **B** were obtained by nonlinear regression of disease severity on temperature using the equation in the text, yielding the parameter estimates $\rho_{25} = 0.3612$, $\Delta H_A = 8827.7$, $\Delta H_H = 155078$, and $T_{1/2H} = 301.52$ for isolate 171; and $\rho_{25} = 0.6918$, $\Delta H_A = 31327$, $\Delta H_H = 94840$, and $T_{1/2H} = 297.96$ for isolate BH-F2-13. MSR = regression mean squares. MSE = error mean squares.

RESULTS

Soil temperature. The four treatments resulted in average soil temperatures of 14.7 ± 0.84, 18.8 ± 1.54, 24.2 ± 1.28, and 30.8 ± 2.29°C (repeat 1) and 14.8 ± 0.99, 20.4 ± 0.66, 22.4 ± 0.94, and 29.9 ± 2.38°C (repeat 2). (Values are means plus or minus standard deviation.) Plants grew less vigorously at lower temperatures, but there was no evidence of low-temperature plant damage or injury.

Plants in noninoculated pots did not develop foliar symptoms of sudden death syndrome, regardless of temperature. For both isolates of *F. solani*, foliar disease severity in inoculated pots was greatest between 22 and 24°C (Fig. 1A and B); foliar symptoms were light at 15 and 30°C for isolate BH-F2-13 and at 30°C for isolate 171. In contrast to foliar disease severity, maximum root disease severity occurred at the lowest temperature tested (15°C) and decreased with increasing temperature (Fig. 1C and D); root symptoms were light at 30°C for both isolates. Some roots in noninoculated pots were slightly discolored (considered normal for soybean roots), with a maximum severity of 2.3%. *F. solani* could be reisolated from inoculated roots but not from noninoculated roots.

Soil moisture. Differential watering of the treatments resulted in values of ψ_m that varied over time (Fig. 2), not unlike moisture conditions in field soils. The wettest treatment had daily averages of ψ_m that remained constant at –0.003 MPa, whereas the driest treatment (irrigation threshold –0.8 MPa) had daily averages of ψ_m that varied between –0.003 and –0.45 MPa. Plants grew less vigorously in the treatments with irrigation thresholds of –0.2, –0.4, and –0.8 MPa than in the other treatments.

Plants in noninoculated pots did not develop foliar symptoms of sudden death syndrome, regardless of ψ_m. Foliar disease severity in inoculated pots decreased rapidly with decreasing values of ψ_m. For both isolates, the decrease followed a power-law relationship when disease severity was plotted against ψ_m (Fig. 3A and B); the decrease was linear when disease severity was plotted against the number of days the average ψ_m was lower than –0.01 MPa (Fig. 3C and D). Both isolates caused up to 100% disease incidence in the wettest treatment (data not shown), but final disease severity was greater for isolate 171 than for isolate BH-F2-13 (Fig. 3).

Roots in noninoculated pots showed some discoloration, particularly in the treatments with irrigation thresholds of –0.2, –0.4, and –0.8 MPa. In inoculated pots belonging to these treatments, it was therefore difficult to clearly distinguish disease symptoms from discoloration due to water stress. Discoloration in inoculated pots (maximum severity 51.0%) was much greater than in noninoculated pots (maximum severity 3.2%), however. Further, *F. solani* could be reisolated from inoculated roots but not from noninoculated roots. In contrast to foliar disease severity, maximum root disease severity was similar for isolates 171 (51.0%) and BH-F2-13 (45.5%). There were no clear relationships between ψ_m and root disease severity (data not shown), or between root disease severity and foliar disease severity (Fig. 4). Most of the data points in Figure 4 fell below the 1:1 line, indicating that both isolates of *F. solani* caused more severe root symptoms than foliar symptoms.

Field experiment. The three irrigation treatments resulted in values of ψ_m that varied over time (Fig. 5). The "wet" treatment had daily averages of ψ_m that remained constant at –0.008 MPa, whereas the "dry" treatment (irrigation threshold –0.8 MPa) had daily averages of ψ_m that varied between –0.006 and –1.05 MPa. Average values of ψ_m in the "intermediate" treatment (irrigation threshold –0.08 MPa) ranged from –0.006 to –0.114 MPa. Plants matured earlier in the dry treatment and developed foliar symptoms of brown spot (caused by *Septoria glycines*), a disease prevalent on soybeans in Iowa during late growth stages.

Plants in noninoculated plots did not show foliar symptoms of sudden death syndrome, regardless of irrigation. In inoculated plots, plants in the wet treatment developed high levels of sudden death syndrome (incidence 86.7% ± 4.41%; severity 41.5% ± 2.28%). (Values are means plus or minus standard error.) Symptoms were much less severe in the intermediate treatment (incidence 86.7% ± 6.67%; severity 5.3% ± 1.96%) and absent in the dry treatment (Fig. 6A). In the wet treatment, grain yields were significantly reduced in inoculated compared with noninoculated plots (yield loss 36.8% ± 7.93%), whereas there was no difference in yield between inoculated and noninoculated plots in the intermediate and dry treatments (Fig. 6B).

DISCUSSION

Our most important results were that i) soil temperature in the range of 15 to 30°C differentially influenced the development of root symptoms and foliar symptoms of sudden death syndrome, with the former being most severe at low temperatures and the latter being most severe at intermediate temperatures; ii) the severity of foliar symptoms decreased rapidly with decreasing soil moisture and was negatively correlated with the number of days the average ψ_m was lower than –0.01 MPa; and iii) there was no close correlation between root disease severity and foliar disease severity.

As for soil temperature, the present study provided experimental support for McLean and Lawrence's (21) observation that low temperatures during the early part of the growing season (which according to our growth chamber data would maximize root infection) followed by intermediate temperatures (which would maximize the expression of foliar symptoms) are optimal for the development of sudden death syndrome in the field. Indeed, increased foliar disease was observed after soybeans had been planted in cold soils, for example by early planting (13) or by planting in no-till fields (43,45). Rupe (29) was able to isolate strains of *F. solani* causing sudden death syndrome from soybean roots as early as 3 weeks after planting in the field, also indicating that early-season infection, and presumably environmental conditions favoring or limiting it, may be important for later symptom development. Further, because toxins are involved in the development of foliar symptoms of sudden death syndrome (18), the most severe above-ground symptoms would be expected after the appearance of conditions favorable for root colonization and infection during early growth stages followed later by conditions favorable

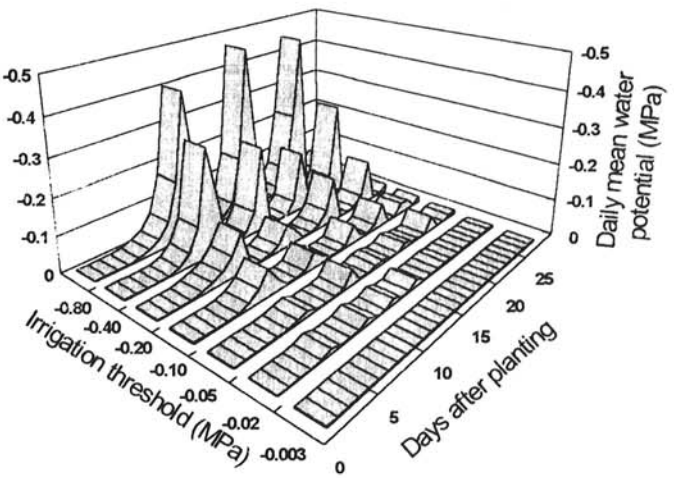

Fig. 2. Soil water matric potential (ψ_m) regime during repeat 1 of the soil moisture experiment. Potted soybean plants in a growth chamber were subjected to fluctuating soil moisture treatments by withholding water until ψ_m reached target threshold values of –0.003, –0.02, –0.05, –0.1, –0.2, –0.4, or –0.8 MPa; when ψ_m for a given treatment had reached its threshold, the pots belonging to this treatment were watered until the soil was saturated. Values of ψ_m were measured continuously with soil moisture blocks. The soil moisture regime during repeat 2 of the experiment was similar.

for plant growth (such as intermediate to warm temperatures and high moisture); this would lead to high transpiration rates and, presumably, rapid translocation of toxins from infected roots to the foliage. Comprehensive controlled-environment experiments with variable temperature treatments (such as low temperatures early followed by intermediate temperatures later), with treatments testing for interactions between temperature and ψ_m, and with disease assessments over time are needed to confirm this hypothesis.

As for soil moisture, our results corroborated earlier observations on the importance of conditions close to saturation for the development of sudden death syndrome. For example, in field trials (22), disease intensity was higher in irrigated microplots than in nonirrigated microplots. In greenhouse trials (28), sudden death syndrome reduced plant height, root and shoot dry weight, and yield in continuously irrigated pots but not in periodically irrigated pots. Soil moisture was not measured in these studies, however, nor were attempts made to develop quantitative relationships between irrigation frequency or amount and disease intensity.

The lack of a close correlation between root symptoms and foliar symptoms, as documented in our growth chamber experiments and by Rupe (29) in a field study, may be because of a differential response of the two types of symptoms to environmental factors (e.g., temperature). This does not exclude the possibility of a correlation at other points in time, however, perhaps between root disease severity in the seedling stage and foliar disease severity at harvest. An alternative explanation for the lack of a clear relationship between root symptoms and foliar symptoms is that conditions favorable for toxin production by the pathogen, or environmental or physiological conditions that render the host more susceptible to the toxin(s), may be more important for the expression of above-ground symptoms in this pathosystem than conditions favorable for root colonization and infection. Indeed, the susceptibility of soybeans to environmental stresses during reproductive development is well documented (20), and any deviation from optimal growth conditions may result in stresses that trigger symptom expression during this critical period.

It is interesting and important to compare the soil temperature and moisture relationships of sudden death syndrome with those of other *Fusarium*-incited diseases of soybean and related leguminous crops. Similar to the results presented here, low temperature increased root rot of soybean by *F. oxysporum* (11), of dry bean (*Phaseolus vulgaris*) and mung bean (*Vigna radiata*) by *F. solani* f. sp. *phaseoli* (3,4,36), and of lentil (*Lens culinaris*) by *F. avenaceum* (16). According to Burke's data (3,4), bean root rot in soil naturally infested with *F. solani* f. sp. *phaseoli* was greatest at 16°C and decreased almost linearly with increasing temperature. French (11), in a study of root rot of soybean caused by *F. oxysporum*, noted that symptoms were most pronounced at 14°C and absent at temperatures greater than 26°C. Based on different temperature optima for the growth of the host and the pathogen, it was concluded that "enhancement of the disease by lower temperatures probably results from lowered resistance of the host." A similar mechanism, i.e., low-temperature predisposition to root infection by isolates of *F. solani* causing sudden death syndrome, may have acted in our study. As for the influence of temperature on the expression of foliar symptoms, there is less agreement among the results from related pathosystems: according to Tu (42), foliar symptoms on pea (*Pisum sativum*) in soil naturally infested with *F. solani* f. sp. *pisi* and *F. oxysporum* f. sp. *pisi* in-

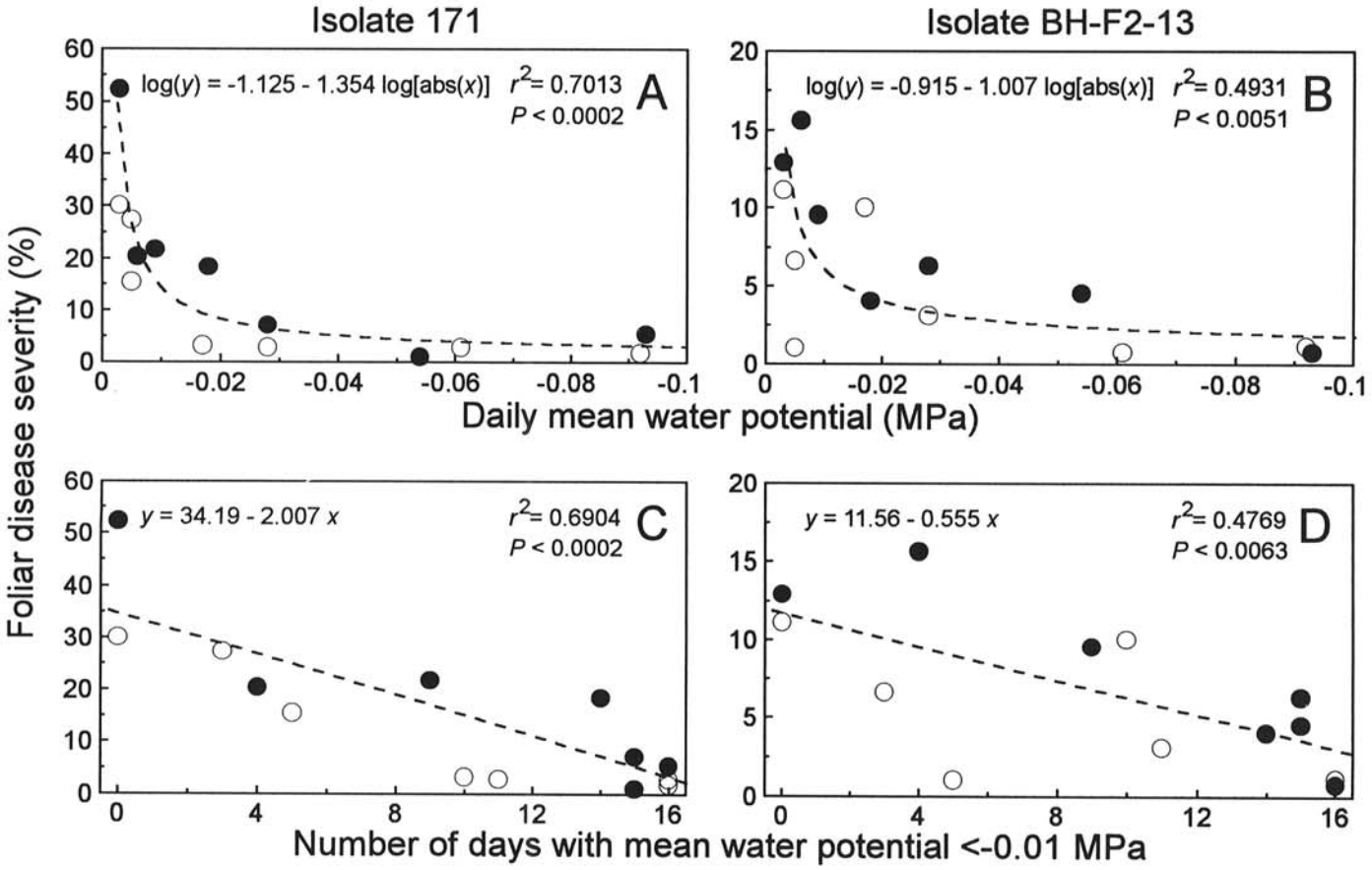

Fig. 3. Relationship between soil water matric potential (ψ_m) and foliar disease severity of sudden death syndrome on potted soybean plants after inoculation with two isolates of *Fusarium solani*: **A,** isolate 171 and **B,** isolate BH-F2-13. Relationship between the number of days the average ψ_m was lower than –0.01 MPa and foliar disease severity of sudden death syndrome on potted soybean plants after inoculation with two isolates of *Fusarium solani*: **C,** isolate 171 and **D,** isolate BH-F2-13. Closed and open symbols represent results from repeats 1 and 2 of the experiment, respectively. Each point is the mean of three replicates. The curves in **A** and **B** were obtained by linear regression of $\log_{10}$-transformed values of disease severity on $\log_{10}$-transformed values of ψ_m.

creased with increasing temperature from 10 to 30°C, whereas our study showed that foliar symptoms on soybean by strains of *F. solani* causing sudden death syndrome were most severe at intermediate temperatures (22 to 24°C). These differences may be because of dissimilarities in the physiological response of aboveground plant parts of the two host species to root infection.

Similar to the results presented here, high soil moisture (greater than –0.005 MPa) increased root rot of bean by *F. solani* f. sp. *phaseoli* (38) and of chickpea (*Cicer arietinum*) by *F. solani* f. sp. *pisi* (2). Miller and Burke (24) implicated reduced soil aeration as a predisposing factor for bean root rot in wet soils. In contrast, root rot of pea by *F. solani* f. sp. *pisi* was most severe at intermediate levels of moisture (6,42), although temporary (1 to 5 days) flooding also increased disease in that pathosystem (42). Interestingly, growth and survival of *Fusarium* spp., including *F. solani*, in the absence of host plants was greatest in soil with low moisture content (15% saturation) and decreased almost linearly with increasing soil moisture (40). This supported Rotem and Palti (26) who, in a review of the effects of irrigation on plant diseases, concluded that "the fact that different levels of soil moisture are optimal to *Fusarium* spp. on various plants may be attributed mainly to the response of the host rather than to that of the pathogen." As for the influence of soil moisture on the expression of foliar symptoms, we did not observe a predisposing effect of water stress in our study; Mengistu et al. (23) noted such an effect for brown stem rot (caused by *Phialophora gregata*), a root and lower stem disease of soybean with foliar symptoms similar to sudden death syndrome.

The relationships between soil temperature, soil moisture, and disease intensity derived here should be valuable in risk assessment studies to determine whether sudden death syndrome has reached the limits to its geographical range (set by climatological and, presumably, edaphic factors) or whether the area affected by the disease will expand further. An approach similar to that used in the CLIMEX computer program (41), which predicts the geographical distribution of a pest (insect, weed, or plant pathogen) based on relationships between temperature, moisture, and growth and survival of that pest, could be used to assess whether isolates of *F. solani* causing sudden death syndrome could become established in northern production areas of the soybean belt. Predictions from such studies will be important for researchers, commodity groups, and the seed industry to allocate scarce resources, e.g., for resistance breeding or disease management research.

The information presented here could also provide guidelines for disease management in soybean production areas where the disease is already established. Because high soil moisture is very important for the development of sudden death syndrome, management practices that reduce moisture in the root zone, such as ridge-tillage (8), could provide a means of reducing the disease in fields harboring strains of *F. solani* causing sudden death syndrome. Indeed, in a 4-year field experiment by Wrather et al. (45), the disease tended to be lower in ridge-tilled fields than in no-till or disk-tilled fields. Clearly, more research on this and similar aspects is needed before detailed management recommendations for sudden death syndrome can be given.

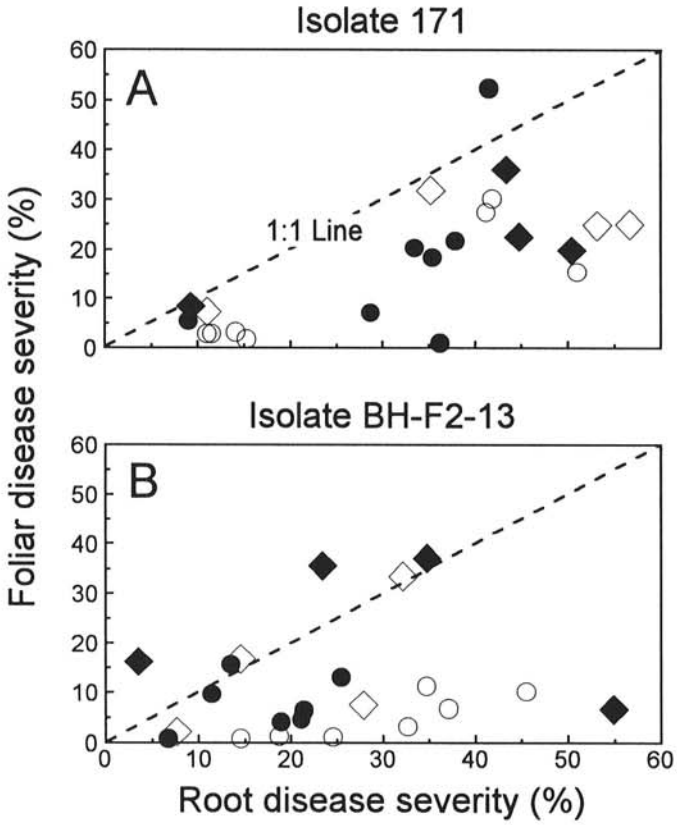

Fig. 4. Relationship between root disease severity and foliar disease severity of sudden death syndrome on potted soybean plants after inoculation with two isolates of *Fusarium solani*: **A,** isolate 171 and **B,** isolate BH-F2-13. Data are pooled from the soil temperature and soil moisture experiments and are represented as squares and circles, respectively. Closed and open symbols represent results from repeats 1 and 2 of the experiments, respectively. Each point is the mean of three replicates.

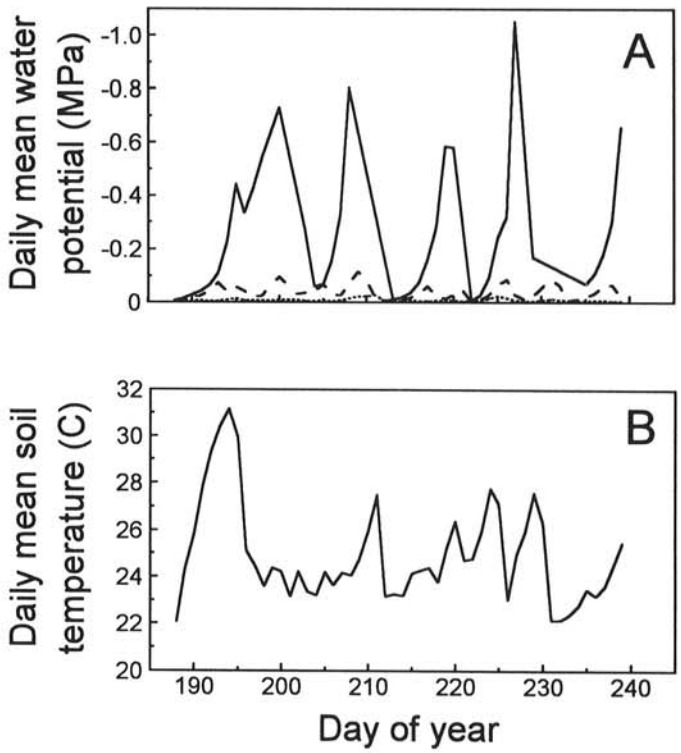

Fig. 5. A, Soil water matric potential (ψ_m) regime and **B,** soil temperature regime during the field experiment. Plots were subjected to fluctuating soil moisture treatments by withholding irrigation water until ψ_m reached target threshold values of –0.008 (dotted line), –0.08 (broken line), or –0.8 (solid line) MPa; when ψ_m for a given treatment had reached its threshold, the plots belonging to this treatment were watered until the soil was close to saturation. Values of ψ_m were monitored continuously with soil moisture blocks. Treatments began on day 188 (soybean growth stage V7) and ended on day 239 (growth stage R6); foliar symptoms of soybean sudden death syndrome were first observed on day 221.

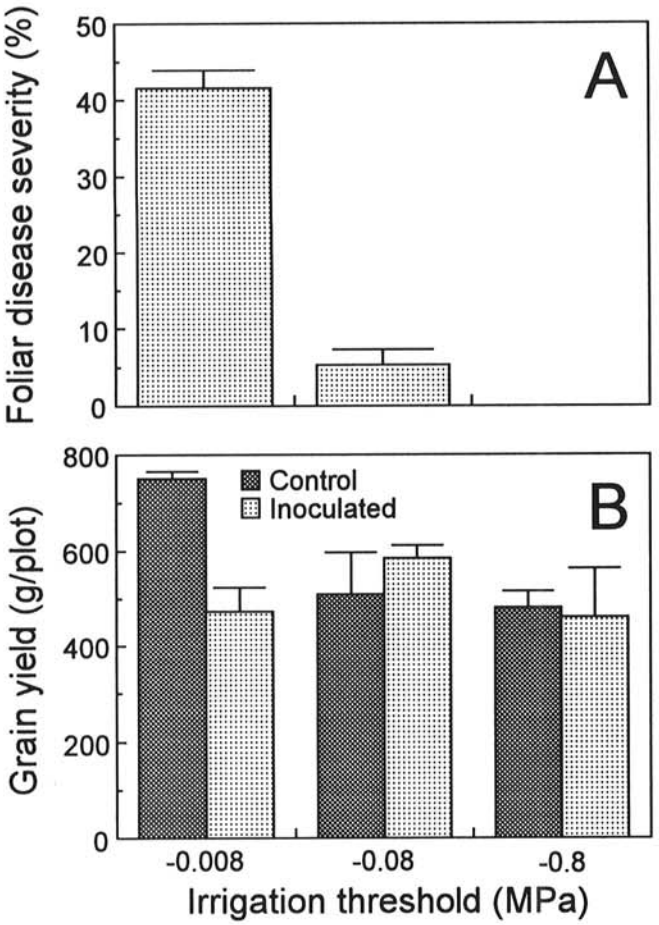

Fig. 6. A, Foliar disease severity of sudden death syndrome and **B,** soybean grain yield in relation to soil water matric potential in the field experiment. Plots (area 2.12 m^2) were either inoculated or not inoculated with *Fusarium solani*; noninoculated plots did not show symptoms of sudden death syndrome. Data are means of three replicates. Error bars represent one standard error.

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