Factors Affecting Symptom Appearance and Development of Phymatotrichum Root Rot of Cotton

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Accepted for publication 11 July 1984.

ABSTRACT

Rush, C. M., Gerik, T. J., and Lyda, S. D. 1984. Factors affecting symptom appearance and development of Phymatotrichum root rot of cotton. Phytopathology 74:1466-1469.

Soil temperature and moisture, sclerotial placement, and flowering were evaluated as factors affecting the initial appearance and subsequent development of Phymatotrichum root rot (PRR) of cotton. In a greenhouse study, nonflowering cultivar of cotton, T-25, and a flowering cultivar, GP-3774, which was defruited, both died from PRR. This indicated that flowering as such is not a prerequisite for development of PRR. Additional greenhouse studies showed that sclerotial placement and soil temperatures can affect the initial appearance of PRR symptoms. When soil temperature was maintained at 27 C and sclerotial inoculum was placed at a depth of 5 cm, 50% of the test plants had died 40 days after emergence. When the inoculum was placed at 60 cm, a depth at which sclerotia are commonly found in the field, it took 21 additional days to reach the same disease level. Reducing soil temperature delayed plant death even more. No foliar disease symptoms developed until the soil temperature at the depth at which the inoculum was placed was above 22 C. Soil moisture was the main factor affecting PRR development, and soil moisture levels between −12 to −16 bars reduced the rate of disease development in 1982 and 1983.

Additional key words: *Gossypium hirsutum*, soil water potential.

Initial symptom appearance of Phymatotrichum root rot of cotton (PRR), and its subsequent development during the year, is influenced by a number of physiological and environmental factors. These include host maturity, quantity and location of primary inoculum, soil temperatures, and soil moisture. In the field, first symptom development is highly correlated with the flowering process (1,6,15). Whether this is due to a hormonal change in the host, a change in carbohydrate sink from root to developing boll, or some other factor is unknown. Rush et al (14) showed that foliar symptoms of PRR are not expressed until the root cortex is sloughed 18–25 days after emergence (DAE). Squaring, the development of the flower bud, occurs 25–35 DAE. Because of the proximity of these two events, the importance of their individual roles in initial symptom expression has not been determined.

In greenhouse studies, symptoms of PRR began to appear soon after the cortex is sloughed 30–35 DAE, but in the field, symptoms appear later, at about 40–55 DAE depending on time of planting (2). In the greenhouse, conditions favoring growth of the fungus are usually near optimal. Quantity and placement of sclerotial inoculum are arranged to encourage optimum disease development, and soil moisture and temperature can be monitored daily and easily controlled to ensure favorable growth conditions for *Phymatotrichum omnivorum* (Shear) Duggar.

Numerous reports have been published concerning optimum moisture and temperature conditions for *P. omnivorum* and disease development. Taubenhaus and Dana (16) were among the first to study the effects of moisture and temperature on PRR. They related the amount and time of rainfall and air temperature to PRR severity. Ezekiel studied air temperatures and rainfall patterns in relation to the range of *P. omnivorum* (5) and annual severity of the disease (3,4). Lyda and Burnett (8) and Rogers (12) determined the optimum soil temperatures for sclerotial germination and strand development, and Wheeler and Hine (18) reported on the affects of soil moisture and temperatures on strand growth and survival.

The majority of these studies have been conducted in vitro or in the greenhouse. Actual field studies have usually addressed the affects of rainfall and air temperature on PRR as opposed to moisture and temperature conditions of the soil. Taubenhaus (16) stated that air and soil temperatures differ by only a few degrees. This may be true in the top few centimeters of the soil, but with increasing soil depth, the variation between air and soil temperatures increases, especially in the heavy clay soils in which *P. omnivorum* is normally found. Likewise, measurement of rainfall is not indicative of available soil moisture or, more importantly, soil water potential. This study was conducted to determine the effects of soil water potential (ψ_s), soil temperature, placement of sclerotia, and the flowering process on the onset and development of PRR symptoms in cotton.

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MATERIALS AND METHODS

Effects of flowering on symptom expression. The purpose of this study was to determine whether the flowering process affects the onset of PRR symptoms. Containers 10 cm wide and 60 cm deep were filled with nonsterile Houston black clay and placed in a temperature tank maintained at 27 C. Temperatures were regulated in the tank with an air conditioner and heating cables. Two genotypes of cotton (*Gossypium hirsutum* L.) were used in the study, T-25, a short-day, nonflowering line (from J. Quisenberry, USDA-ARS, Lubbock, TX) and GP-3774, a long-day, flowering cultivar commercially available. Seeds were planted and ~2 g of sclerotia of *P. omnivorum* were placed as a single clump in each container at a depth of 15 cm. Sclerotia were produced in the laboratory as previously described (7,8). Following emergence, plants were thinned to one per container. Each treatment was replicated four times with each replication containing 18 plants; six T-25, six defruited GP-3774, and six GP-3774 allowed to fruit normally. The first disease count based on typical foliar wilt symptoms was made 30 DAE and at 5-day intervals thereafter for 60 days.

Effects of sclerotial placement and soil temperature. A greenhouse study was conducted to evaluate effects of sclerotial placement and soil temperature on PRR symptom development. Plants were grown in containers and temperature tanks as previously described. Two separate temperature tanks were used in this study. In Tank I, soil temperatures were maintained at 27 C for the duration of the experiment and sclerotial depth was the dependent variable. Sclerotia were placed at one of three depths at time of planting in each container, 5, 30, or 60 cm. There were four replications with eight containers for each treatment per replicate. Disease counts began 31 DAE and were taken at weekly intervals up to 66 days. Plants were counted as diseased when typical foliar wilt symptoms appeared.

In Tank II, sclerotia were placed at the same depths as in Tank I and the effect of varying soil temperature on PRR symptom expression was evaluated. A vertical soil temperature gradient was established in Tank II to simulate gradients found in field soils. This was achieved by placing a thermostat in the middle of the temperature tank and allowing the air conditioner to run only when the temperature rose above the desired setting. The initial setting was 17 C. It was altered three times during the experiment to obtain progressively warmer temperatures (Table 1). Soil temperatures in eight containers were measured every 2 days at 5, 30, and 60 cm by using previously placed copper-constantan thermocouples. Disease counts were first taken 45 DAE and then at weekly intervals up to 101 DAE.

Field studies. Measurements of soil temperature and moisture, and disease symptom development were taken during the growing seasons of 1982 and 1983. At the Blackland Research Center, Temple, TX, measurements were taken each year in a 2.5-ha field that had been continuously planted to cotton for 10 yr and had a known history of PRR. The same cultivar of cotton, GP-3774, was planted both years. Cotton was planted on 25 April 1982 and 12 April 1983. The soil type was a Houston black clay. Soil moisture measurements were taken gravimetrically and by the neutron scattering technique (9). These readings were converted to ψ_s using a moisture release curve for Houston black clay (11). Soil temperatures were measured with copper-constantan thermocouples. Temperature and moisture measurements were made at six locations within the field. Rate of PRR development was measured as the percentage of plants dying each week in six two-row plots 30 m long.

TABLE 1. Soil temperature gradients simulating vertical gradients in the field produced in a temperature tank during a greenhouse study of Phymatotrichum root rot of cotton

Days after plant emergence	Soil temperatures[a] at depths of:		
	5 cm	30 cm	60 cm
1–22	22.7 ± 2.7	17.0 ± 0.67	14.2 ± 0.32
23–45	23.13 ± 1.7	19.5 ± 0.61	17.2 ± 1.1
46–66	25.9 ± 1.6	22.2 ± 0.56	20.6 ± 1.2
67–100	29.0 ± 1.3	27.1 ± 0.43	24.4 ± 1.4

[a]Each value represents the mean and standard deviation of eight measurements of soil temperature over the period indicated. Measurements were taken at 2-day intervals.

RESULTS

Flowering study. Nonflowering as well as defruited and flowering control plants died from PRR (Fig. 1). The T-25 plants, which were generally larger than those of GP-3774, were the first to die from PRR and had the highest disease percentage at the end of the experiment. Removing young squares as they formed on GP-

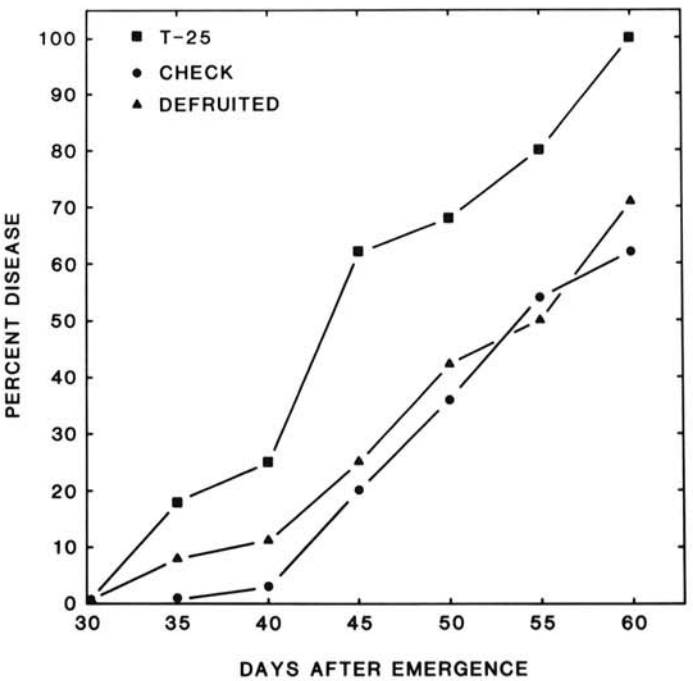

Fig. 1. Incidence of Phymatotrichum root rot (PRR) symptoms in plants of the nonflowering cotton genotype, T-25, and in fruited (control) and defruited plants of GP-3774. Soils were infested with sclerotia at time of planting and plants with typical wilt symptoms of PRR were counted as diseased.

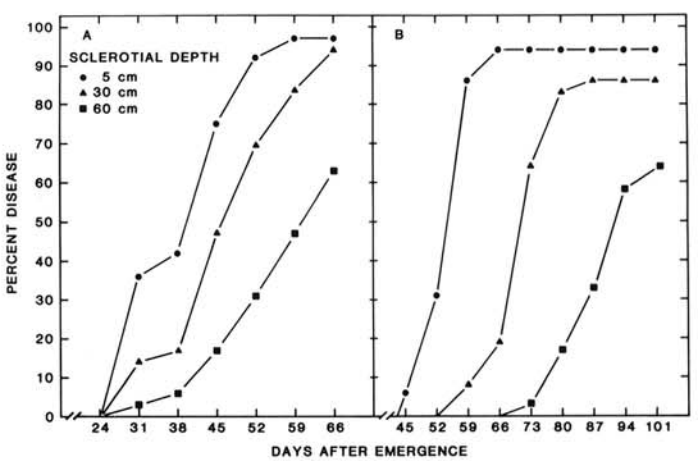

Fig. 2. Effects of sclerotial depth and soil temperature on Phymatotrichum root rot disease development in cotton. **A,** Tank I. Soil temperatures maintained at 27 C. The mean standard deviation for each disease curve was: 5 cm, ±7%; 30 cm, ±9.6%; and 60 cm, ±7.1%. **B,** Tank II. Soil temperatures were altered three times during the experiments beginning at 17 C and ending at 27 C. The mean standard deviation for each disease curve was: 5 cm, ±9.2%; 30 cm, ±10.5%; and 60 cm, ±10.9%.

3774 had no effect on disease development or final percentage of plants killed, as compared to flowering plants.

Sclerotial placement and temperature study. The effects of sclerotial placement and soil temperature on PRR development are shown in Fig. 2. Several aspects of disease development in Tank I and Tank II were similar. In both tanks the initial rate of disease development was faster in containers with sclerotia at 5 cm and slower with sclerotia at 60 cm. At every counting date, mortality was higher in containers infested at 5 cm and lower in containers infested at 60 cm. The final percentage of disease was similar in Tanks I and II between plants growing in containers with inoculum placed at similar depths.

In Tank I (Fig. 2A), PRR had appeared in all treatments by 31 DAE, but the mortality was greatest in containers infested at 5 cm. After 66 DAE, disease development on plants with sclerotia at 60 cm was ~18 and 23 days behind plants with sclerotia at 30- and 5-cm levels, respectively. In Tank II (Fig. 2B) no disease symptoms appeared until 45 DAE. However, once plants began to die in Tank II, the rate of PRR development appeared to be faster with all treatments than in Tank I. Disease development at 94 DAE in plants in Tank II with sclerotia at 60 cm was ~21 days behind plants in containers with sclerotia at 30 cm and 35 days behind plants with sclerotia at 5 cm. Comparison of PRR development (Fig. 2) and soil temperatures at the different levels in Tank II (Table 1) shows that little disease occurred in any treatment until soil temperatures exceeded 22 C at the level at which the inoculum was placed.

Field studies. Early-season soil temperatures were warmer in 1982 than in 1983, but for the remainder of the growing seasons temperatures were similar in 1982 and 1983 (Table 2). The mean soil temperature at 45 cm 1–30 DAE in 1983 was too low for PRR development as indicated by our greenhouse study, but after 30 DAE in both years, soil temperatures were well within the range suitable for PRR development.

In both years, ψ_s at 30 cm fluctuated around −1 bar until ~50 DAE when the ψ_s rapidly decreased. A reduction of the ψ_s to −12 to −15 bars at about 67 DAE resulted in a reduced rate of PRR development (Fig. 3). In 1982, there was no appreciable precipitation during the summer months, and from 67–110 DAE, ψ_s was <−13 and the mean disease incidence never exceeded 17%. The 1983 growing season was also extremely dry until August, and between 98–116 DAE, 138 mm of rain increased the ψ_s at 30 cm from <−20 bars to ~−0.7 bars. This resulted in an increased rate of disease development which exceeded that at the beginning of the season.

TABLE 2. Soil temperatures taken in 1982 and 1983 at two depths in a research plot continuously cropped to cotton for 10 yr at the Blackland Research Center, Temple, TX

Days after plant emergence	Mean soil temperatures[a]			
	1982		1983	
	15 cm	45 cm	15 cm	45 cm
1–22	24.5 ± 1.11	22.32 ± 1.41	20.1 ± 1.76	19.6 ± 0.91
23–45	24.8 ± 0.88	23.7 ± 4.50	24.7 ± 1.67	22.9 ± 1.20
46–66	27.5 ± 0.96	25.7 ± 0.83	28.1 ± 1.19	25.7 ± 0.63

[a] Each value represents the average and standard deviation of soil temperatures over the period indicated. Measurements were made twice weekly at six locations within the study area.

DISCUSSION

During the normal development of a cotton plant grown in the Blackland soils of Texas, the root cortex sloughs 19–25 DAE, first squares appear 25–35 DAE, and blooming commences 55–65 DAE. Symptoms of PRR usually began to appear 40–55 DAE, and for this reason symptom expression of PRR has been associated with the flowering process. A second observation, which supports the association between flowering and PRR symptom expression, is that field-grown plants which remain vegetative, either because of insect pressure or excessive nitrogen fertilizer, either fail to die or the disease progresses at a slower rate than with flowering plants (15). The reasons for this are unknown, but the results of this study suggested that flowering is not a prerequisite for disease development or symptom expression. Plants of cultivar T-25, which remained vegetative during the entire experiment, showed the faster rate and higher percentage of disease. The results of this study support the previous report (14) that cortical senescence and sloughing are the primary physiological factors affecting initial PRR symptom expression in cotton. These factors, however, cannot explain the delay in appearance of PRR symptoms in the field compared to greenhouse-grown plants. The cortex of field-grown plants is sloughed and plants are susceptible by 25 DAE, but plants in the field usually do not start to die until about 40–55 DAE.

In greenhouse studies, relatively high densities of sclerotial inoculum are used. These are uniformly mixed in the soil or placed in clumps 5–10 cm below the soil surface (7,14) resulting in rapid root-fungus contact. The majority of sclerotia in field soils, however, are found 45–75 cm below the soil surface (13). This

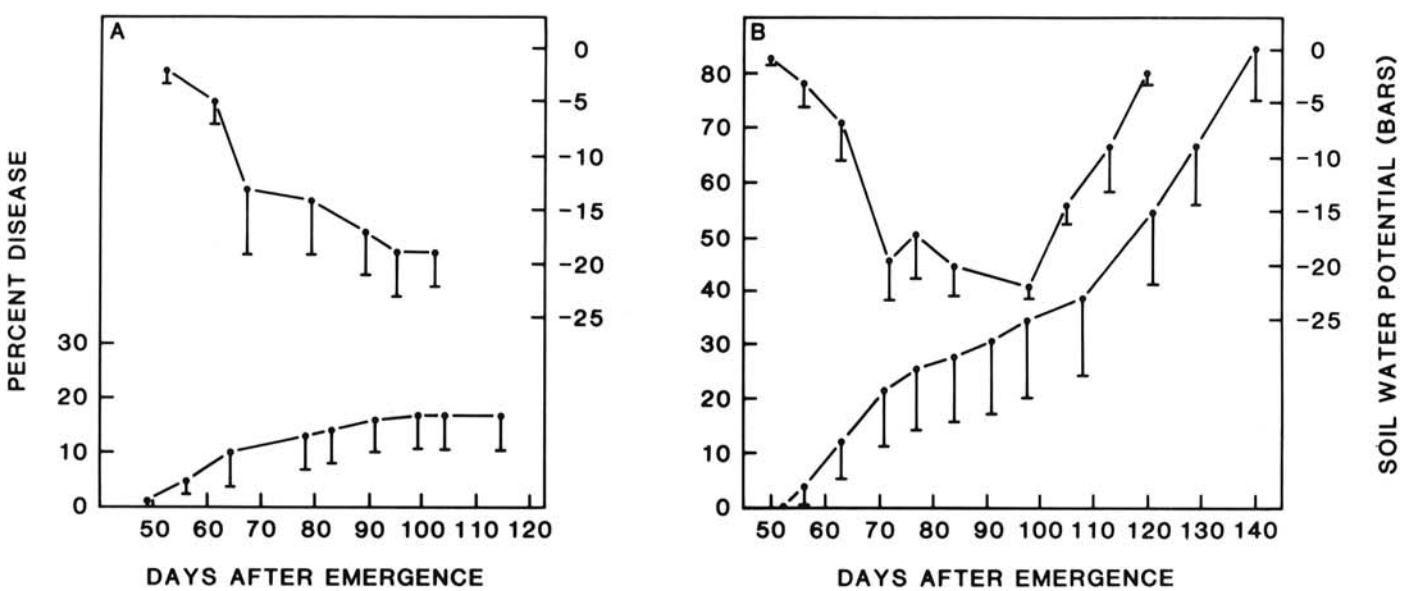

Fig. 3. Effects of soil water potential measured at a depth of 30 cm on Phymatotrichum root rot development in cotton at the Blackland Research Center in Temple, TX. **A,** 1982 was an exceptionally dry year and disease incidence never exceeded 17%. **B,** August rains in 1983 approximately 100 days after emergence resulted in an increased rate of root rot development.

depth, as indicated by the sclerotial placement study, is sufficient to cause the observed delay in field symptoms of PRR. Fifty percent of the plants in Tank I with inoculum placed at 5 cm had died by 40 DAE, but this level of disease incidence was not reached until 21 days later when inoculum was placed at 60 cm.

Although sclerotial depth alone can explain the delay of symptom expression in the field, the affect of soil temperatures must also be considered. The results presented in Fig. 2B and Table 1 show that reduced temperatures markedly increase the time of initial PRR appearance. It was interesting that no disease symptoms were observed in Tank II until the soil temperature exceeded 22 C at the level at which the sclerotial inoculum was placed. Once symptoms appeared, however, the rate of disease development apparently was more rapid than in Tank I maintained at 27 C. *P. omnivorum* can grow at soil temperatures <20 C, but at a reduced rate (15). When temperatures exceed 22 C, the rate of fungal growth increases rapidly (13,18). We believe that the effect of temperature on fungal growth through the soil can explain the long delay of symptom expression and the rapid rate of disease development that eventually occurred in Tank II.

In the Blackland soils of Texas, the mean soil temperature is normally >22 C after 30 DAE. The root cortex is often not completely sloughed until 25 DAE and therefore, plants seldom die before 30 DAE. Because of these two facts, we concluded that soil temperature is not an important factor in determining the initial onset of PRR in the field. By the time the cortex has sloughed, temperatures are warm enough for PRR to develop rapidly. With increased research on the effects of soil temperature and depth of sclerotial inoculum on disease development, it should be possible to estimate sclerotial depth in the field by measuring soil temperature and recording the initial appearance of disease symptoms.

Cortical senescence and sclerotial placement are important in the initial appearance of PRR symptoms, but soil moisture is the predominant factor affecting disease development during the year (10,17). This has been recognized by scientists and producers alike, but because of a lack of information on water potential, practically all of the data concerning moisture effects on PRR development is technologically obsolete. We are aware of only one study (18) concerning *P. omnivorum* which relates percent soil moisture to water potential. Because of this shortcoming, conflicting reports have resulted. In Arizona, a soil moisture content of 22% in sandy soils supporting *P. omnivorum* was equivalent to −0.33 bar (18) while 24% soil moisture in a Houston black clay is equivalent to −16 bars (11). Therefore, it is obvious why results concerning the effects of moisture on PRR in Texas and Arizona often differ. It is also clear that the procedure of measuring rainfall and correlating it with PRR severity can be improved on by using ψ_s measurements instead. Ezekiel (3) reported that rainfall in August did not affect the final percentage of PRR, but this was reported in 1937 when considerable precipitation was recorded during June and July. Our 1983 data show conclusively that rainfall in August can affect the final percent of PRR. Conflicting reports can be eliminated if ψ_s is used instead of rainfall or soil moisture measurements. This will allow accurate comparisons of results from different locations despite variations in farming practices, soil types, and climatic conditions.

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