This is to certify that the

dissertation entitled

Epidemiology and Control of Anthracnose Incited by Colletotrichum graminicola (Ces.) Wils. on Annual Bluegrass

presented by

Tom Karl Danneberger

has been accepted towards fulfillment
of the requirements for

Ph.D degree in Plant Pathology

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Date May 19, 1983

EPIDEMIOLOGY AND CONTROL OF ANTHRACNOSE INCITED BY *COLLETOTRICHUM GRAMINICOLA* (CES.) WILS. ON ANNUAL BLUEGRASS

By

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A DISSERTATION

Submitted to
Michigan State University
in partial fulfillment of the requirements
for the degree of

DOCTOR OF PHILOSOPHY

Department of Botany and Plant Pathology

1983

ABSTRACT

EPIDEMIOLOGY AND CONTROL OF ANTHRACNOSE INCITED BY COLLETOTRICHUM GRAMINICOLA (CES.) WILS. ON ANNUAL BLUEGRASS

By

Tom Karl Danneberger

Anthracnose was observed during periods of warm weather after maximum seedhead production of annual bluegrass had occurred. A model predicting the initiation to total production of seedheads was developed for annual bluegrass (Poa annua var. reptans (Hauskins) Timm.) for the purpose of establishing a phenological starting point for anthracnose occurrence. The model was highly correlated (R^2 = .965) with degree-days starting on 6 April. The model was linear and of the form SD = -5.741 + 0.03 (DD), where SD = the logit (ln (% of accumulated seedheads/1.-% of accumulated seedheads)) of % accumulated seedheads and DD = degree-days with a base of 10 C.

Inoculum, temperature, and leaf wetness were significant (P=0.01) factors in anthracnose development. Increasing the inoculum concentration from 10^3 to 10^6 conidia/ml increased the percentage of infected plants at all leaf wetness - temperature treatments except at treatment combinations where 100% infection had previously been achieved. Increasing the temperature from 20 to 30 C increased the percentage of infected plants at all wetting periods except where 100% infection had previously been achieved. Increasing the wetting period from 12 to 72 hr increased the percentage of infected plants at all temperatures except where 100% infection had previously been achieved. Annual bluegrass plants stressed at four soil water potentials, -0.3, -1.0, -2.0, and -3.0 bars for 10 days preceding inoculation with Colletotrichum graminicola

had a significant (P=0.05) increase in disease at soil water potentials less than -1.0 bar.

A regression model relating leaf wetness and temperature to infection of annual bluegrass by C. graminicola was developed and validated in the field. The model is $ASI = 4.0233 - 0.2283LW - 0.5308T - 0.0013LW^2 + 0.0197T^2 + 0.0155(LWxT)$ in which ASI = anthracnose severity index, T = average daily temperature (C), and LW = hours of leaf wetness per day. The model successfully predicted 14 of 16 periods of disease increase when ASI value of 2 was taken as the minimum condition for infection.

Control of anthracnose by means of fungicide applications and nitrogen fertility were investigated. Field plots receiving the fungicide, triademifon, had little disease regardless of nitrogen application. In non-fungicide treated plots, application of nitrogen at 1.46 kg/are/year during June, July, August, September, and November had significantly (P=0.05) lower amount of disease than plots receiving 2.92 kg of nitrogen/are/year or nitrogen applications during April, May, June, August, and September.

ACKNOWLEDGEMENTS

I wish to express my appreciation to Drs. Jones, Rieke, and Stephens for their ideas, criticisms and support as committee members. Also, I would like to thank Ron Detweiler for his valuable assistance throughout this project and Drs. Eisensmith and Olson for teaching me the finer points of computer programming.

I wish to thank Marianne La Haine for typing this dissertation.

A special acknowledgement to the Michigan Turfgrass Foundation and the O.J. Noer Foundation for financial support of this project.

To Dr. Joseph M. Vargas, Jr., major professor and friend, a special thanks for guidance and direction given both professionally and socially, during the good times and bad.

Finally, to my parents, Tom and Evelyn, I will never be able to repay all you have done for me.

TABLE OF CONTENTS

PART IV
EFFECT OF WATER STRESS ON
ANTHRACNOSE OF ANNUAL BLUEGRASS

PART V
ANTHRACNOSE DEVELOPMENT ON ANNUAL BLUEGRASS
IN RESPONSE TO NITROGEN CARRIERS
AND FUNGICIDE APPLICATION

APPENDICES

LIST OF TABLES

Table Page

APPENDIX B

APPENDIX C

LIST OF FIGURES

INTRODUCTION

Annual bluegrass is a cool season grass best adapted to the northern United States and Canada. It is native to Europe and has been reported in South America, North Africa, Australia, and North Asia (11). Annual bluegrass forms a dense uniform quality turf under irrigated, close cut cultural conditions (1). The ability of annual bluegrass to adapt to low mowing heights makes it an excellent turfgrass species for golf course greens, tees and fairways (3,29).

Annual bluegrass is a member of the class - Monocotyledoneae, order - Poales, family - Poaceae, and tribe - Festuceae. The predominant types of annual bluegrass have been identified as an annual (Poa annua var. annua L. Timm) and a perennial type (Poa annua var. reptans (Hauskins) Timm). Annual types have lower leaf and node numbers and fewer secondary tillers and adventitious roots than perennial types (11). With frequent irrigation and mowing the perennial types usually predominate.

The major limitation of annual bluegrass as a turfgrass species is its inability to survive during warm weather periods. Based on limited research, turfgrass researchers and managers previously assumed that loss of annual bluegrass during warm weather was due to high temperature stress (1,2).

Researchers of the view that annual bluegrass dies in the summer due to high temperatures cite Carroll's (5) study on the effects of high soil temperatures and moisture at low and high nitrogen levels on the growth

of various turfgrasses. Carroll's experiment consisted of placing turf plugs in two water baths set at 50 and 60 C. The plugs were removed and percent plant survival was determined when the soil temperature equaled that of the water baths. Carroll reported 100% survival of annual bluegrass at 50 C and low nitrogen levels. Seventy percent of the annual bluegrass plants survived at high nitrogen levels whereas 100% of the Kentucky bluegrass (Poa pratensis L.) and 'Highland' creeping bentgrass (Agrostris palustris Huds.) plants survived. Sixty percent of the annual bluegrass plants survived for 6 hrs at 50 C under low nitrogen levels. By comparison, Kentucky bluegrass and 'Highland' creeping bentgrass had a plant survival rate of 80% and 70%, respectively. Under high nitrogen levels, the plant survival rates were 40% for annual bluegrass, 40% for Kentucky bluegrass and 55% for 'Highland' creeping bentgrass. The survival rate of annual bluegrass was less than 'Highland' creeping bentgrass but not to the degree expected if one was trying to explain the total die-out of annual bluegrass in the summer due to temperature effects.

Wehner and Watschke (24) updated Carroll's experiments using improved cultivars of Kentucky bluegrass and perennial ryegrass (Lolium perenne L.). They found that under intensive maintenance (94.4 kg/ha of nitrogen and 26% soil moisture) the annual bluegrass recovery rate at temperatures between 43 and 48 C was not significantly different from the Kentucky bluegrass recovery rate. Under low maintenance (11.8 kg/ha of nitrogen and 11-26% soil moisture) the survival rate of annual bluegrass was significantly lower than the survival rate of Kentucky bluegrass but not significantly different than the perennial ryegrasses. In field situations, where annual bluegrass is the predominant turfgrass species,

maintenance practices are similar to the high maintenance treatment reported by Wehner and Watschke.

Research done by Fischer (10) is frequently cited for the inability of annual bluegrass to survive in the presence of high temperatures. Fischer reported 50% kill of annual bluegrass plants after 2 hr at 42 C and 100% relative humidity. Fischer failed to include other turfgrass species in his experiment as comparisons to annual bluegrass.

High temperatures are a factor in annual bluegrass decline but cannot totally account for annual bluegrass dying in the summer at temperatures between 25-35 C. An important aspect that has been neglected in the past is the role that warm season diseases, specifically anthracnose, might have on the survival rate of annual bluegrass. The interaction between warm temperatures and anthracnose may explain the lack of summer survival of annual bluegrass.

Anthracnose

The significance of Colletotrichum graminicola (Ces.) Wils. as reported as a disease pathogen on turf varies widely throughout the literature. Sprague and Eval (20) first reported anthracnose as a severe disease of annual bluegrass in 1928. During the mid 1970's, Vargas (21) reported anthracnose as a serious disease of annual bluegrass in Michigan and Bolton and Cordukes (4) reported the disease as the main limiting factor in the growth of annual bluegrass in eastern Canada during 1978 and 1979. Before 1974 most researchers considered anthracnose a minor disease of turf (7,19,28). Interestingly, anthracnose of corn, incited by C. graminicola, was also considered a minor disease (8,9,26) until the

early 1970's when severe outbreaks occurred throughout the United States (14,15,23,25). Whether the increased severity of anthracnose on annual bluegrass coincided with the increasing severity of anthracnose on corn is not known.

Anthracnose first appears during warm weather as irregular patches of yellow-bronze turf ranging in size from a few centimeters to several meters. Leaf lesions initially appear as elongated reddish-brown spots. As the disease progresses, the turf fades to a light tan (7,21). Bolton and Cordukes (4) reported not all strains of annual bluegrass are susceptible to C. graminicola. They found 18 out of 20 strains were susceptible while one was highly resistant and one was immune.

Smith (19) described the fungus as producing spores abundantly on short squat conidiophores in acervuli. The dark brown, setose acervuli are erumpent and are to be found on shoot bases, leaf sheaths, and leaves. The setae are aseptate, dark colored, and tapering to a point from a swollen base. Conidiophores are unbranched, truncate-conic, and bear one spore. Spores are fuscoid, curved and hyaline. Appressoria which are one-celled, are produced on the surface of infected tissue. Smith (19) reported the optimum temperature for mycelial growth on yeast extract agar with 1% glucose included was 22 C. However, Bolton and Cordukes (4) reported that optimum infection occurred at 30-33 C.

The fungus was first described by Cesati (6) as Dicladium graminicola and in 1914 was renamed Colletotrichum graminicola by Wilson (27). Wilson included most forms of Colletotrichum with falcate conidia under C. graminicola. The preexisting species, C. cereale Manns and C. lineola Corda were included in the broad concept of C. graminicola. The anamorph state of this fungus is in the form-order Melanconiales. The

telemorph state of C. graminicola is Glomerella graminicola Politis, sp. nov. (18) and is described as follows:

> Perithecia (194-)215-450(-575) X (170-)200-450(-470) µm, erumpetia, rostrata, globosa, nigra, carbonario contextu, pseudoparenchymatica. Peridium ex cellulis brevibus, angularibus, porus periphysatus. Paraphyses longae, hyalinae, numerosae guttalatae cum septis. Asci 70-115(-125) X (9-)10-18(-19) µm, unitunicati, clavati, brevi pediculo, cum anulo refractivo ad apieem, octospori. Ascosporia (16-)18-26(-29) X (4-)5-8)-10) µm, hyalina, biseriata, unicellularia, gutulata, curvata. Habitat. Cultura in folis emortuis Zea mays. Mycelium homothallicum, status conidicus Colletotrichum graminicola.

Little is known of the life cycle of this fungus on turf. However, on corn, C. graminicola is a soil invader that colonizes plant debris as a saprophyte (22). Free water is necessary for successful penetration and disease severity is most often associated with periods of wet, windy weather (13). Spores of C. graminicola are embedded in a mucilaginous matrix in acervuli on infected tissue (16). The spore masses become dry which allow for wind dispersal. Nicholson and Moraes (16) found the spore matrix helped the pathogen to survive by protecting the spore against desiccation and increasing the efficiency of germination through invertase and hydrolase activity. Wheeler et al. (25) reported temperatures between 22-30 C had little, if any effect on disease severity. They found increasing inoculum levels and increasing periods of high humidity increased disease severity. Research has shown that low light intensities can increase the severity of anthracnose on corn (12,17,25).

The purpose of this research was i) to define the environmental conditions under which anthracnose infects annual bluegrass both in the greenhouse and in the field and ii) to determine disease management practices that control the disease.

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PART I

PREDICTING ANNUAL BLUEGRASS SEEDHEAD EMERGENCE AND NUMBER FROM DEGREE-DAYS

ABSTRACT

Seedhead production of annual bluegrass (Poa annua var. reptans (Hauskins) Timm.) during the spring reduces annual bluegrass root production and disrupts the aesthetic and playing qualities of the turf. Anthracnose has been observed most frequently after maximum seedhead production has occurred. A model to predict the number of annual bluegrass seedheads from emergence to total accumulation was developed from three locations in Michigan for the purpose of establishing a phenological starting point for anthracnose development. Seedhead emergence to total number accumulated was highly correlated (R^2 = .965) with degree-days starting on 6 April with a base temperature of 10°C. The seedhead model was linear.

INTRODUCTION

Annual bluegrass (Poa annua var. reptans (Hauskins) Timm.) is the predominant turfgrass species on most golf course fairways and greens in the northern United States and Canada. Under high maintenance (close cut, high nitrogen, frequent irrigation), annual bluegrass is capable of forming a dense, uniform turf (3). However, seedhead production of annual bluegrass in the spring disrupts aesthetic qualities of the turf and is associated with undesirable plant responses such as a reduction of the root system resulting in decreased water and nutrient uptake (3). An accurate prediction of the initiation to total production of seedheads would allow maintenance practices (i.e. irrigation, vertical mowing, and core cultivation) to be adjusted for best turf growth.

Heat accumulation models, sometimes referred to as growing degree day models, are useful in determining phenological stages in plant growth. Several models have been proposed for corn (7), sweet corn (1), leaf emergence of sour cherry (6) and peach bloom (9). For annual bluegrass no heat accumulation model has been proposed. Bogart (4) observed that annual bluegrass initiated growth in the spring when soil temperatures were above 12.7 C. In using seedhead formation as an indication of plant maturity, he observed that seedhead production occurred only after soil temperatures surpassed 15.5 C. Laboratory experiments either using thermogradient plates or growth chambers for base temperature determination have not been reported. The purpose of

this study was to determine the base temperature for annual bluegrass growth and to develop a heat accumulation model for predicting seedhead formation.

METHODS AND MATERIALS

Base temperature for annual bluegrass was determined under growth chamber conditions at temperatures of 5, 10, 12, and 15 C. Each treatment was replicated three times and the experiment was repeated twice. One-mo-old annual bluegrass plants growing in 700 cm^3 pots containing a 1:1:1 (soil:sand:peat, v/v) mix cut to a height of 1.2 cm were placed randomly in each growth chamber. Seven days later the plants were cut to 1.2 cm height and clippings were weighed on an oven dry basis (60 C).

Field data were collected on annual bluegrass (P. annua var. reptans (Hauskins) Timm.) seedhead emergence and number at three locations (Robert Hancock Turfgrass Research Center, East Lansing, MI, sandy loam soil; Michigan State Soils Research Barn, East Lansing, MI, sandy loam soil; and Meadowbrook Country Club, Livonia, MI, clay loam soil) for the year 1982. The annual bluegrass turf was mowed at 1.3 cm and irrigated as needed. Maximum and minimum daily temperature readings were taken from hygrothermographs (Belfort Leaf Wetness Recorder, Belfort Instrument Co., Baltimore, MD 21224) at all locations. The hygrothermographs were set 2 cm above the soil surface. Seedheads were counted every 1 to 4 days from 4 plots measuring 20 cm by 20 cm at all locations.

A FORTRAN V program was used to calculate and accumulate degree-days according to the Baskerville and Emin method (2), which assumes the sine curve as an approximation of the diurnal temperature curve. A Control

Data Corp. 750 computer and the Statistical Package for the Social Sciences Regression subprogram (8) were used to analyze the data and develop a model from initial seedhead emergence to total accumulated seedhead production based on degree-day accumulation.

RESULTS

It was established from the growth chamber study that annual bluegrass grew at a minimum of 10 C (Table 1). Thus 10 C was used as the base temperature for annual bluegrass.

Seedhead number at all three locations increased with time through the month of May peaking at May 21 (Figure 1A). For all three locations the general shape of the curve was in the form of a sine curve. Maximum seedhead number (>120/20 cm^2) occurred for a period of 14-17 days at the three locations.

The number of seedheads increased with degree-days (DD) at all locations (Figure 1B). Seedheads emerged between 50 and 80 DD with maximum seedhead number occurring between 200-250 DD. After 250 DD seedhead numbers rapidly decreased and leveled out around 330 DD. The curve for seedhead formation vs. DD was of the form of a sine curve similar to that of the seedhead vs. calendar date curve.

Seedheads at each location were accumulated then expressed on a percentage scale with 100% equalling the total number of seedheads produced. A graph of the percent accumulated seedheads vs. DD resulted in a sigmoid shaped curve (Figure 2).

Regression analysis of percent accumulated seedheads vs. DD accumulation using base temperatures of 9 to 13 C at 1 C intervals indicated that either 9 of 10 C base temperature with initial accumulation beginning April 6, 1982, resulted in the "best fit" for the observations

Table 1. Annual bluegrass clipping yields for four growth chamber temperatures.

Temperature (°C)	Clipping weight[1] (g)
5	0
10	.06
12	.08
15	.11
LSD (.01)	.03
LSD (.05)	.02

[1]After 7 days, the plants were clipped to 1.2 cm, the original height at the onset of the experiment. The clippings were oven dried at 60 C.

Figure 1. Number of annual bluegrass seedheads at A, time in days; and B, degree-days with a base of 10 C for 1982.

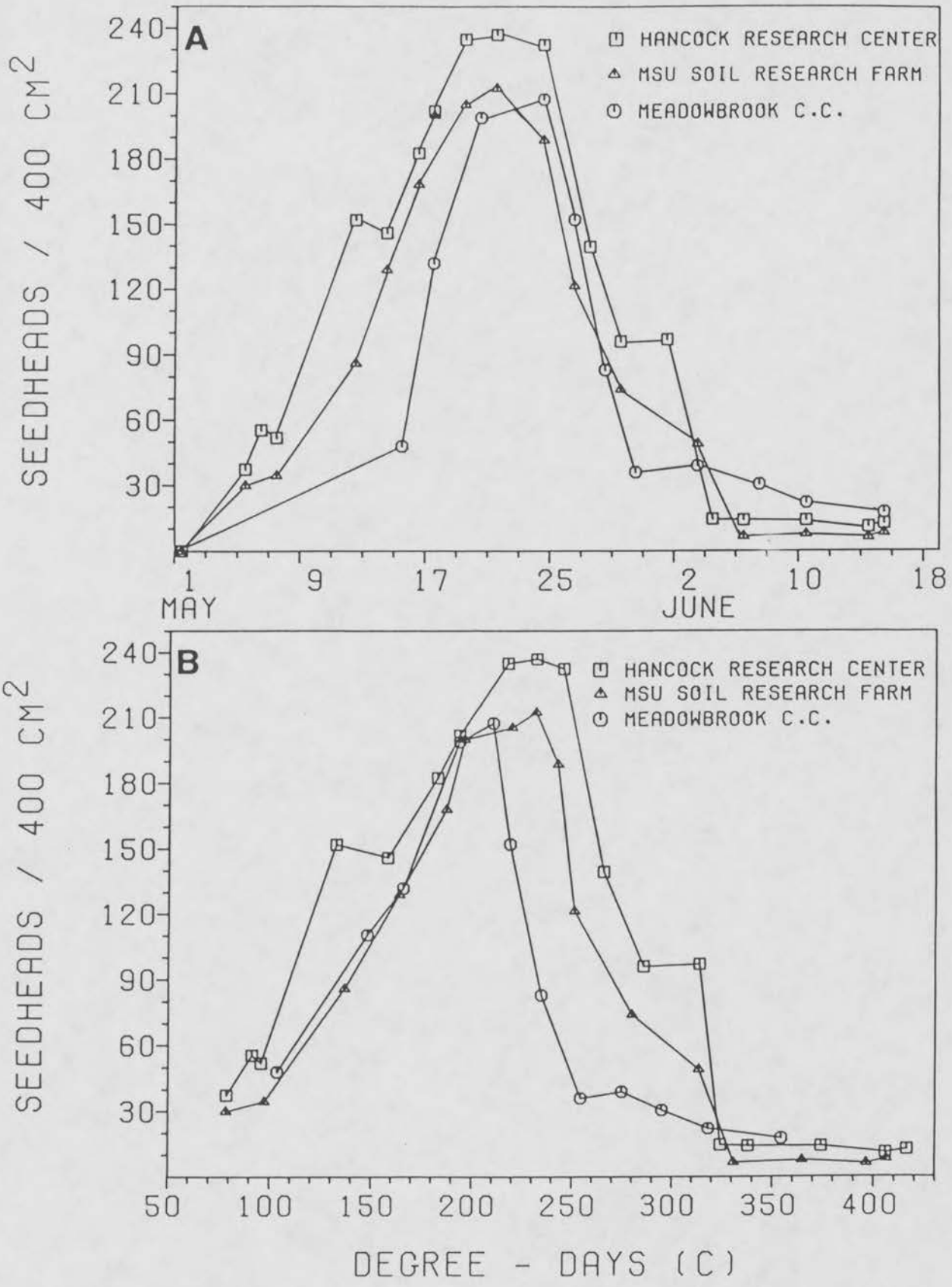

A
B
HANCOCK RESEARCH CENTER
MSU SOIL RESEARCH FARM
MEADOWBROOK C.C.
SEEDHEADS / 400 CM2
240
210
180
150
120
90
60
30
1
9
17
25
2
10
18
MAY
JUNE
50
100
150
200
250
300
350
400
DEGREE - DAYS (C)

Figure 2. Total percent of accumulated seedheads vs. degree-days (C) with a base of 10 C starting April 6, 1982.

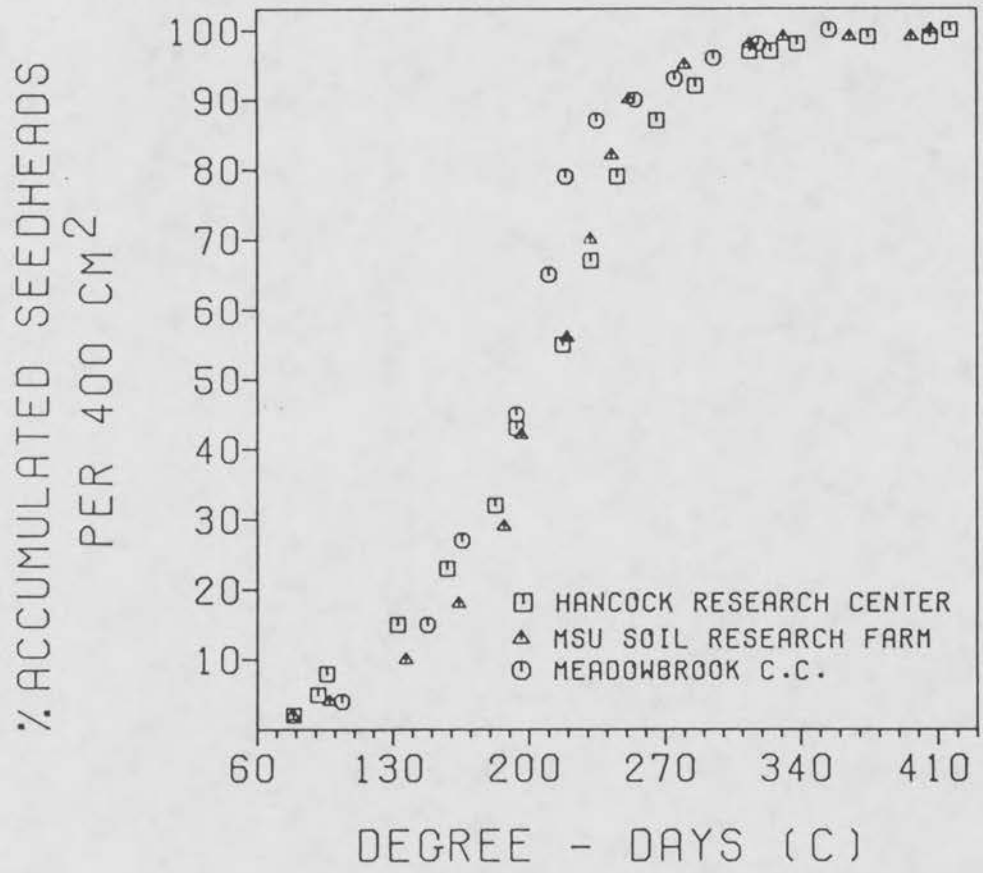
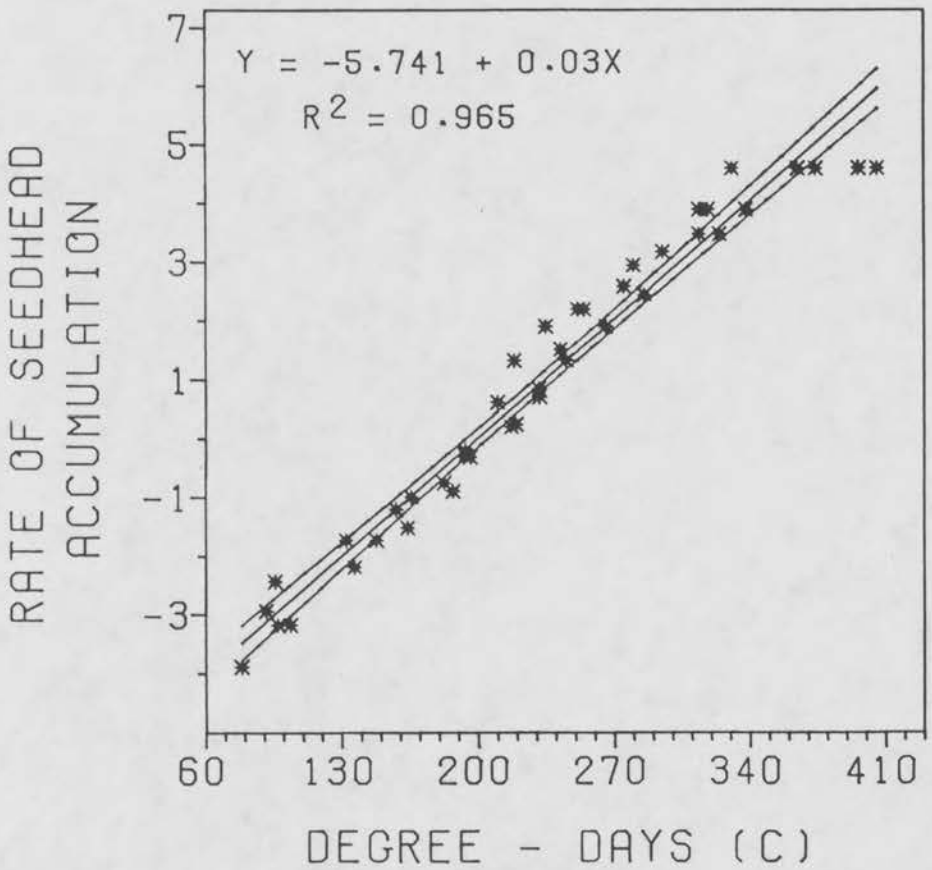

% ACCUMULATED SEEDHEADS PER 400 CM2
100
90
80
70
60
50
40
30
20
10
60
130
200
270
340
410
DEGREE - DAYS (C)
HANCOCK RESEARCH CENTER
MSU SOIL RESEARCH FARM
MEADOWBROOK C.C.

Figure 3. Regression analysis with corresponding 95% confidence belts of annual bluegrass seedhead number from emergence to total accumulated seedhead production based on degree-day accumulation with base 10°C starting April 6, 1982 for three locations. Logit (ln (percent of accumulated seedheads/1.- percent of accumulated seedheads)) transformation on % accumulated seedheads was performed before regression analysis.


Y = -5.741 + 0.03X
R2 = 0.965
RATE OF SEEDHEAD ACCUMULATION
7
5
3
1
-1
-3
60
130
200
270
340
410
DEGREE - DAYS (C)

(Table 2). Using 10 C as the base temperature, based on growth chamber results, and a logit transformation on the percentage of accumulated seedheads, a regression model was developed for the three locations (Figure 3). The model was linear and took the form:

$$\text{Percent accumulated Seedhead no. (logit)} = -5.741 + 0.03\,(DD)$$

with logit = ln (% accumulated seedheads/1.-% accumulated seedheads) and DD = degree-day accumulation above 10°C beginning April 6.

Table 2. Regression statistics for degree-day accumulation with base temperatures of 9-13 C beginning April 6 for relation to seedhead emergence to 100% accumulated seedhead production in annual bluegrass.

	Base Temperature				
Statistic	9	10	11	12	13
Coefficient of variance (%)	56.1	57.7	59.7	62.3	65.9
Coefficient of determination	0.967	0.965	0.962	0.959	0.954
Overall F value	1213.2	1146.9	1065.8	975.9	867.9

DISCUSSION

This model can be used to study the effect of rate and timing of application of certain growth regulators that suppress annual bluegrass seedhead formation, as well as an aid in defining annual bluegrass stage of growth as related to disease susceptibility. Certain growth regulators such as 1,2-dihydro-3-,6,-pyridazinedione (maleic hydrazide); N-3-(1,1,1-trifluoromethylsulfonyl)amino-4-methylphenyl acetamide; and 2,4-dimethyl-5-(trifluoromethylsulfonylamido)acetanilide(mefluidide) inhibit seedhead formation of certain turfgrasses (5,10). These growth regulators could be used for seedhead inhibition of annual bluegrass and the rate and timing of applications could be based on plant growth using the model and not on specific calendar dates.

An important part in disease development is the presence of susceptible tissue. The degree of susceptibility is often related to the stage of growth of the plant. Eisensmith and co-workers (6) proposed that a degree-day model for leaf emergence of cherry could be incorporated into disease models for cherry leaf spot to study host-pathogen interactions. Our seedhead model may serve similarly as a means of defining the phenological stage of annual bluegrass related to disease susceptibility (i.e. anthracnose) in any future disease models.

The model has the basic limitation of DD accumulation being based on a fixed calendar date and not some physiological parameter of the plant such as the breaking of dormancy. Possible improvements could be the use

of spring "green-up" or some internal plant function such as initiation of carbohydrate utilization in the spring as a starting point for the model.

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PART II

INTERACTION OF TEMPERATURE, LEAF WETNESS AND INOCULUM CONCENTRATION ON ANTHRACNOSE DEVELOPMENT ON ANNUAL BLUEGRASS

ABSTRACT

Growth chamber experiments were conducted to determine the importance of temperature, leaf wetness and inoculum concentration on anthracnose development of annual bluegrass (Poa annua L. Timm.). Increasing the inoculum concentration from 10^3 to 10^6 conidia/ml increased the percentage of infected plants at all leaf wetness-temperature treatments except at treatment combinations where 100% infection had previously been achieved. Increasing the temperature from 15 to 30 C increased the percentage of infected plants at all wetting periods except where 100% infection had previously been achieved. Increasing the wetting period from 12 to 72 hr increased the percentage of infected plants at all temperatures except where 100% infection had previously been achieved.

INTRODUCTION

Annual bluegrass (*Poa annua* L.) is the major turfgrass component on golf courses in the temperate region of the United States and Canada. Under heavy fertilization, adequate irrigation, and lack of fungicide use, annual bluegrass is susceptible to a number of fungal diseases with anthracnose being one of the most common (1).

Anthracnose, incited by the fungus *Colletotrichum graminicola* (Ces.) Wils., was first reported as causing severe damage to annual bluegrass in New Jersey (6). The disease was later reported in England and Canada (2,4,5). Reports in the United States have associated anthracnose with annual bluegrass decline during warm weather (6,7,8). The optimum growth of the fungus *in vitro* ranges between 21 and 31 C (5,6). No information is available on the environmental factors that favor anthracnose infection of annual bluegrass.

The purpose of this study was to investigate the effects of air temperature, leaf wetness and inoculum concentration on infection severity.

METHODS AND MATERIALS

Annual bluegrass plants were grown and maintained in a greenhouse at 22 C for 3 mo in clay pots each containing 700 cm^3 of a mix of sand, soil, and peat (1:1:1, v/v). The seeding rate was 0.5 g of seed per pot. Plants were fertilized with a total of 98 kg/ha each of nitrogen, phosphorus, and potassium and were maintained at a height of 2.5 cm by cutting the top growth weekly.

The isolate of C. graminicola used in this study was obtained from an annual bluegrass plant at the Robert Hancock Turfgrass Research Center, East Lansing, MI 48824. The isolate was grown on 4% potato-dextrose agar (PDA; Gibco Diagnostics, Madison, WI 53713) at 22 C. Spores from 18-day-old cultures were suspended in sterile distilled water and concentrations of conidia in the suspensions were determined with a hemacytometer. Lower spore concentrations were obtained by dilution with sterilized water. Spore suspensions were applied to the plants from a DeVilbiss hand atomizer. Viability of the spores was determined by atomizing spores onto blocks of PDA in Petri plates and incubating the plates at 22 C for 48 hr. Percent germination was determined by examining 200 conidia with a light microscope at 40X.

Four growth chambers (Sherer-Gillett, Marshall, MI 49068) were used to study the effect of air temperature, leaf wetness and inoculum concentration on infection severity of C. graminicola on annual bluegrass. Growth chambers were set for a 12 hr photoperiod. Four temperatures ±2 C

(15, 20, 25, and 30 C), five wetting periods (0, 12, 24, 48, and 72 hr), and four inoculum concentrations (10^3, 10^4, 10^5, and 10^6 conidia/ml were evaluated in a 4x5x4 factorial experiment. Each treatment was replicated three times and the experiment was repeated three times. Each run of the experiment was a replicate with chamber temperatures being randomly reset between replications to allow for possible chamber effects.

Wetting period treatments consisted of misting the plants then placing them in sealed plastic bags for the duration of the desired wetting period. Periodically, the plants were visually checked for the presence of free moisture on the leaf blades. The temperatures within each plastic bag was monitored with thermocouples connected to a temperature recorder (Yellow Springs Instrument Co., Yellow Springs, OH 45387). Growth chamber temperatures were adjusted so that the desired temperature was maintained within the plastic bags. After the wetting period, the plants were placed under wetted cheesecloth suspended 10 cm above the plants to maintain a relative humidity of about 80%. Percent infection was determined from fifty random plants per pot, selected 12 days after inoculation. Plants were considered infected if acervuli were present.

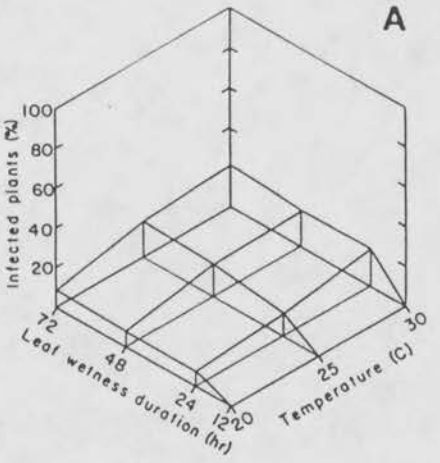
RESULTS

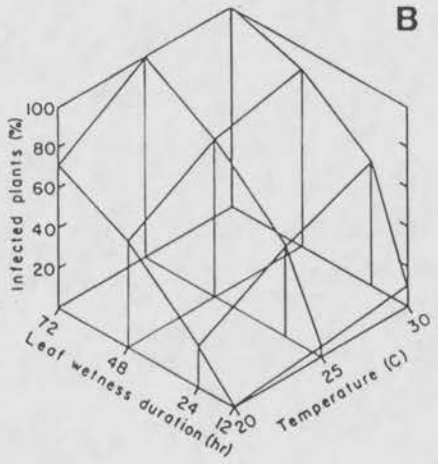
Spore germination was 90% or above in all experiments. No infection was detected at 15 C, at an inoculum concentration of 10^3 conidia/ml, or when the wetting period was 0 hr. These treatments were excluded from statistical analysis. No significant difference (P=0.01) was present between the three experiments.

The relationship of increasing inoculum concentrations from the 10^4 to 10^6 conidia per milliliter to the percentage of infected plants is illustrated in Figure 1. Inoculum concentrations of 10^4 conidia/ml resulted in minimal infection, even after a 72 hr wetting period at 30 C (Figure 1A). At 10^5 conidia/ml the percentage of plants infected increased over the level at 10^4 conidia/ml at all leaf wetness temperature treatments except no infection occurred after a 12 hr wetting period at 20 C (Figure 1B). At 10^6 conidia/ml, the percentage of plants infected was higher than at 10^5 conidia/ml at all leaf wetness-temperature treatments except where 0 to 100% infection was noted at 10^5 conidia/ml.

The relationship of increasing temperature from 20 to 30 C to the percentage of infected plants is illustrated in Figure 1. At 10^4 conidia/ml, minimal infection occurred at 25 and 30 C with no infection at 20 C (Figure 1A). Temperature treatments at 10^5 conidia/ml had higher percentage of infected plants than 10^4 conidia/ml except for the 20 C - 12 hr wetting treatment where no infection occurred (Figure 1B). Increasing the temperature within the 10^5 conidia/ml treatment resulted

Figure 1. Percentage of infected annual bluegrass plants by Colletotrichum graminicola at 12 combinations of temperature and leaf wetness at A, 10^4 conidia/ml; B, 10^5 conidia/ml; and C, 10^6 conidia/ml.


A
Infected plants (%)
100
80
60
40
20
72
48
24
12
20
Leaf wetness duration (hr)
25
30
Temperature (C)


B
Infected plants (%)
100
80
60
40
20
72
48
24
12
20
Leaf wetness duration (hr)
25
30
Temperature (C)

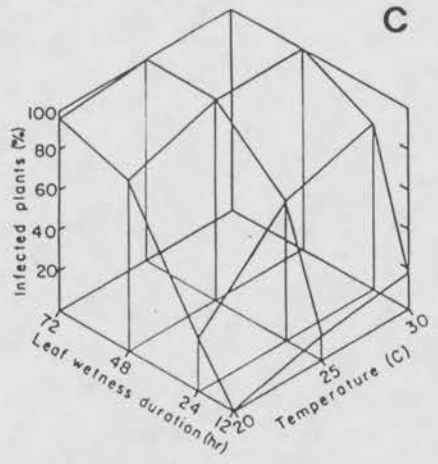

C
Infected plants (%)
100
80
60
40
20
72
48
24
12
20
Leaf wetness duration (hr)
25
30
Temperature (C)

Table 1. Partitioning treatment sum of squares for infection by temperature, leaf wetness, and inoculum concentration.

Source of variation	d.f.	Sum of squares	Percent of total	R^2
Treatment (infection by temperature)	2	21,528.00		
Linear	1	20,319.56**	94	.043*
Residual	1	1,208.68	6	
Treatment (infection by leaf wetness)	3	200,003.32		
Linear	1	172,086.00**	86	.360**
Quadratic	1	24,539.65**	12	.410**
Residual	1	3,377.67	2	
Treatment (infection by inoculum)	2	179,570.30		
Linear	1	162,580.91**	90	.330**
Residual	1	16,989.39**	10	

*Significant at the 0.05 level.

**Significant at the 0.01 level.

in increased percent of infected plants at all wetting periods except at 72 hr where 100% infection occurred at 25 and 30 C. At 10^6 conidia/ml, percentage of infected plants at each temperature was above that of 10^5 conidia/ml except where infection was 100% at 10^5 conidia/ml (Figure 1C).

The relationship of increasing hours of wetting from 12 to 72 to percentage of infected plants is similar in trend to that of temperature and is illustrated in Figure 1 and Table 1. At 10^4 conidia/ml, minimal infection occurred at 24, 48 and 72 hr with no infection occurring at 12 hr (Figure 1A). At 10^5 conidia/ml, the wetting period at each temperature had a higher percentage of infected plants than the corresponding wetting-temperature treatment at 10^4 conidia/ml. Increasing wetting periods within the 10^5 conidia/ml treatment resulted in increased percentage of infected plants except for 72 hr wetting period at 25 and 30 C where 100% infection occurred (Figure 1B). At 10^6 conidia/ml, each wetting period had a higher percentage of infection than the corresponding treatments at 10^5 conidia/ml, except where infection was 100% at 10^5 conidia/ml (Figure 1C).

DISCUSSION

In this study C. graminicola caused infection at temperatures similar to those reported for optimum growth of the fungus in culture (5,6). Greenhouse experiments by Bolton and Cordukes (2) showed anthracnose infection occurring between 27 and 33 C with severe infection between 30 and 33 C. This agreed with our results showing increasing infection with increasing temperature. Bolton and Cordukes did not report any results for temperatures below 27 C nor did they vary the wetting period or inoculum concentration. The results presented here are similar to those found for anthracnose on corn (9) which show the duration of the wetting period and the inoculum concentration are important factors in disease development.

These results may help turf managers minimize anthracnose severity on annual bluegrass turfs. For example, delaying irrigation from early-evening to early-morning would reduce the period free moisture is on the leaf blade. Also, dew or moisture removal by means such as "poling" would reduce the wetting period.

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PART III

A MODEL FOR FORECASTING ANTHRACNOSE ON ANNUAL BLUEGRASS BASED ON WEATHER DATA

ABSTRACT

A multiple regression equation relating hours of leaf wetness and temperature to amount of infection of annual bluegrass by conidia of Colletotrichum graminicola was developed from 2 yr of field data. The model is $ASI = 4.0233 - 0.2283LW - 0.5308T - 0.0013LW^2 + 0.0197T^2 + 0.0155(LWxT)$, in which ASI = anthracnose severity index, T = average daily temperature (C), and LW = hours of leaf wetness per day. ASI values of 1, 2, 3, 4, 5, and 6 were equal to < 10, 11-20, 21-30, 31-40, 41-50 and > 51% area infected, respectively. The predicted accuracy of the model was tested with data from three locations in 1982. The model successfully predicted 14 of 16 periods of disease increase when an ASI value of 2 was taken as the minimum conditions for infection. Average daily ASI values predicted from temperature and wetness data were related to rate of disease increase according to the Gompertz transformation.

INTRODUCTION

Anthracnose, incited by the fungus Colletotrichum graminicola (Ces.) Wils., causes severe damage to golf course greens and fairways of annual bluegrass (Poa annua var. reptans (Hauskins) Timm.) (2,6,7,10). The fungus overwinters in both dead and living plant material (4) and during the summer conidia produced in acervuli infect annual bluegrass plants. In Michigan, anthracnose is particularly severe during periods of warm weather in summer. The disease develops as irregularly shaped patches of yellow-bronze turf a few centimeters to several meters across. Infected leaves have elongated reddish brown lesions that expand to encompass the entire leaf blade (10). Laboratory studies report that optimum growth of the fungus occurrs between 27-33 C (6,7). Inoculation studies under greenhouse conditions report C. graminicola being pathogenic on annual bluegrass between 27-33 C (2).

The purpose of this study was to develop a model to predict anthracnose severity on annual bluegrass from leaf wetness and temperature data collected in the field. Many of the approaches used in this study were previously used to develop a prediction model for cherry leaf spot (3).

MATERIALS AND METHODS

At the Michigan State University Soils Research Farm, East Lansing, MI 48824, and the Glengary Country Club, Sylvania, OH 43560, 4 x 4 m plots were established in four replicated 100 m^2 areas of annual bluegrass during 1980 and 1981. Anthracnose was severe at both locations in 1979. The plots at both locations were clipped to a height of 1.25 cm by mowing three times a week, irrigated as needed to prevent wilt and fertilized with 112 kg/ha/yr nitrogen (urea) in May, June, August and September. Levels of phosphorus and potassium were adequate according to soil test results obtained from the Michigan State Soil Testing Laboratory.

Belfort leaf wetness recorders (Belfort Instruments, Co., Baltimore, MD 21224) were used to monitor air temperature and hours of leaf wetness at each location from 1 May to 10 October 1980 and 1981. Leaf wetness measurements included wetting periods from rain, dew, and irrigation. The recorders were located 1.3 cm above the soil surface.

Disease severity was estimated every 3-5 days as the percentage of area in each plot with symptoms of anthracnose. To detect the spread of inoculum at each location, 1-mo-old annual bluegrass plants in 700 cm^3 clay pots were placed in holes in the ground so that the soil in the pot was flush with the surrounding soil surface in the center of each plot. Pots were changed weekly with new plants maintained in a greenhouse. During the exposure period the plants were subjected to the same mowing

and moisture regime as the surrounding plot area. After the plants were removed from the plots, they were misted for 48-60 hr in a mist chamber at 25±5 C. Misted plants were placed on a greenhouse bench and examined periodically for 2 wk for symptoms of anthracnose. Isolations from leaf lesions on these plants were made periodically on potato dextrose agar to verify the presence of C. graminicola.

Model development

A model relating daily mean air temperatures and hours of leaf wetness to anthracnose severity was developed using regression techniques. The assumption was made, based on greenhouse data (Appendix C), that 10 to 12 days at 25±5 C were required for symptom development. Therefore, temperature and wetness data for 10, 11, and 12 days preceding the date of each disease rating were averaged and the means correlated with corresponding disease severity values according to Pearson's method as given in Statistical Package for the Social Sciences (4).

Validation of the model

From 1 May to 1 September 1982, the model was tested at three locations: Robert Hancock Turfgrass Research Center, East Lansing, MI 48824; Glengary Country Club, Sylvania, OH 43560; and Meadowbrook Country Club, Livonia, MI 48151. Anthracnose was severe at each location in 1981. Disease severity was monitored in three 4 x 4 m plots selected randomly within a 100 m^2 area at each location.

One Belfort leaf wetness recorder per location was set 1.3 cm above the soil surface to monitor hourly temperature and duration of leaf wetness. An anthracnose severity index (ASI) was calculated daily using the regression model. Disease severity in each plot was rated every 2 to 4 days.

Anthracnose severity vs. infection rate

To determine if the expected anthracnose severity index (ASI) values computed with the model were related to rate of disease increase, linear regression analysis was done on the data collected in 1982 from the Hancock Research Center and the Glengary Country Club. Infection rates were determined using the logistic (9) and Gompertz (1) methods. Tests for homogeniety of regression coefficients (5) were performed before pooling the two sets of data for regression analysis.

RESULTS

Model development. An acceptable second-order model relating temperature and duration of leaf wetness to disease severity took the form:

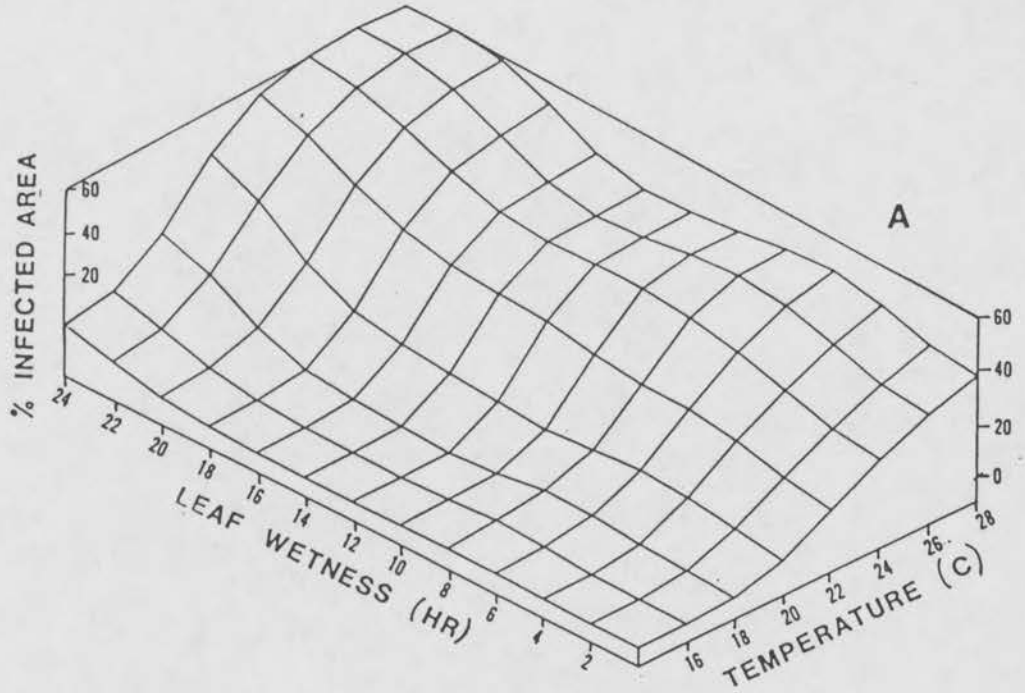
$$ASI = b_0 + b_1LW + b_2T + b_{11}LW^2 + b_{22}T^2 + b_{12}(LWxT) + e \quad \text{Eq. 1}$$

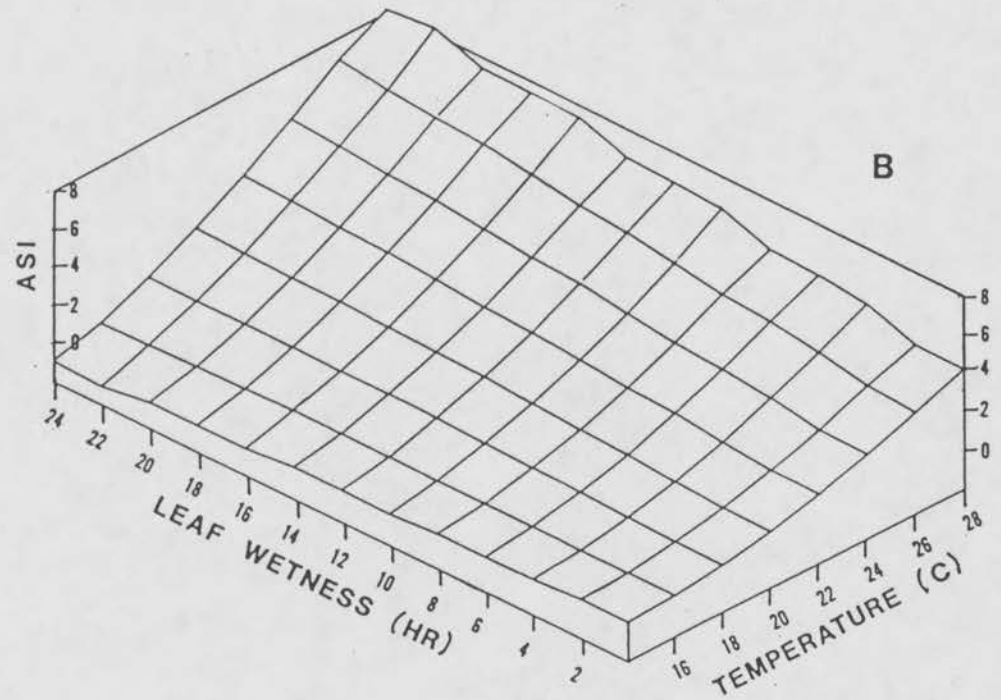
where ASI = anthracnose severity index, LW = hours of leaf wetness, and T = mean daily temperature (C). The b values are least-square estimates of the partial regression coefficients and e is a normally distributed random variable with mean zero and variance σ^2. The ASI values were 1, 2, 3, 4, 5, and 6 and represented 1-10, 11-20, 21-30, 31-40, 41-50, and > 51% of the area infected, respectively. This model accounted for 84% of the observed variation in disease severity and all estimated coefficients were statistically significant at $P=0.01$. The actual model was:

$$ASI = 4.0233 - 0.2283LW - 0.5308T - 0.0013LW^2 + 0.0197T^2 + 0.0155(LWxT)$$

The relationship of temperature and duration of leaf wetness to disease severity is shown in a computer generated surface (11) of the original data from the two monitoring sites (Figure 1A). A comparative surface generated from points predicted with the equation (Figure 1B) shows a good fit of the model for temperature values of 14 to 28 C and for wetting durations up to 24 hr. Examination of residuals, the difference between the original data points and those predicted by the model, supported the assumption that the error components are independent, have a mean of zero, and a constant variance.

Figure 1. Relationship of temperature and duration of wetting to infection of annual bluegrass by Colletotrichum graminicola. A, from actual field data. B, predicted from regression equation.


% INFECTED AREA
60
40
20
A
24
22
20
18
16
14
12
10
8
6
4
2
LEAF WETNESS (HR)
16
18
20
22
24
26
28
TEMPERATURE (C)
60
40
20
0


ASI
8
6
4
2
0
B
24
22
20
18
16
14
12
10
8
6
4
2
LEAF WETNESS (HR)
16
18
20
22
24
26
28
TEMPERATURE (C)
8
6
4
2
0

Model validation

To determine if ASI values calculated from daily temperature and wetness data accurately predicted disease development, ASI values were calculated from weather data for each of the three locations. To account for a latent period of 10 to 12 days, three consecutive ASI values were averaged and related to visual estimations of disease made 10 days later. The model predicted fourteen of sixteen periods of disease increase correctly (88%). Twice in mid-May, the model predicted disease when none appeared. Potted plants exposed at the Michigan State University Soils Research Laboratory in 1980 and 1981 failed to develop infections during May. Exposed plants did not exhibit infection until the week of 16 June in 1980 and June in 1981.

In addition, predicted ASI values were directly related to the actual percentage of diseased area observed 10 days later (Figure 2). However, low ASI values (1 to 1.8) predicted disease when none was present. Because 14 of 16 wetting periods suitable for infection had ASI values greater than 2, the assumption was made that an ASI value of 2 was the threshold value for infection to occur (Figure 3). Also, 90% of the wetting periods with no disease development fell on or below the line for ASI = 2.

Relationship of anthracnose severity index to infection rate

Anthracnose severity index (ASI) values were plotted for the Robert Hancock Turfgrass Research Center (representative of the two sites) (Figure 4). Disease was first predicted on 14 and 15 May and should have appeared in late May. Favorable disease weather was detected on 29-30 June and was followed about 10 days later by an increase in the

Figure 2. Regression and 95% confidence limits of anthracnose severity index (ASI) values predicted from mean temperature and wetness duration data collected in the field on actual estimate of infection in three locations used for validating the predictive model in 1982.

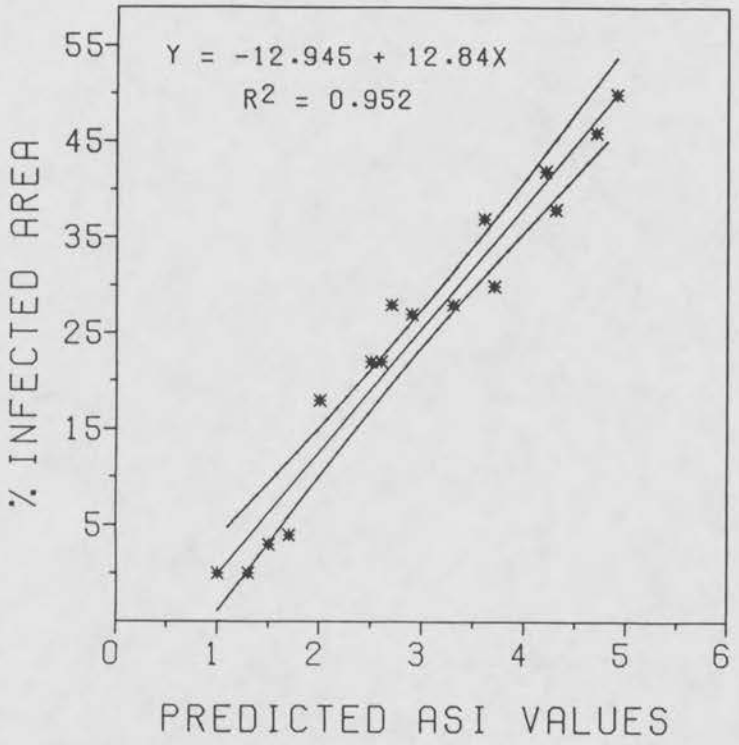

Y = -12.945 + 12.84X
R2 = 0.952
% INFECTED AREA
55
45
35
25
15
5
0
1
2
3
4
5
6
PREDICTED ASI VALUES

Figure 3. Comparison of leaf wetness periods monitored in the field to predict infection or non-infection events. An anthracnose severity index (ASI) of 2 was considered the minimum for infection.

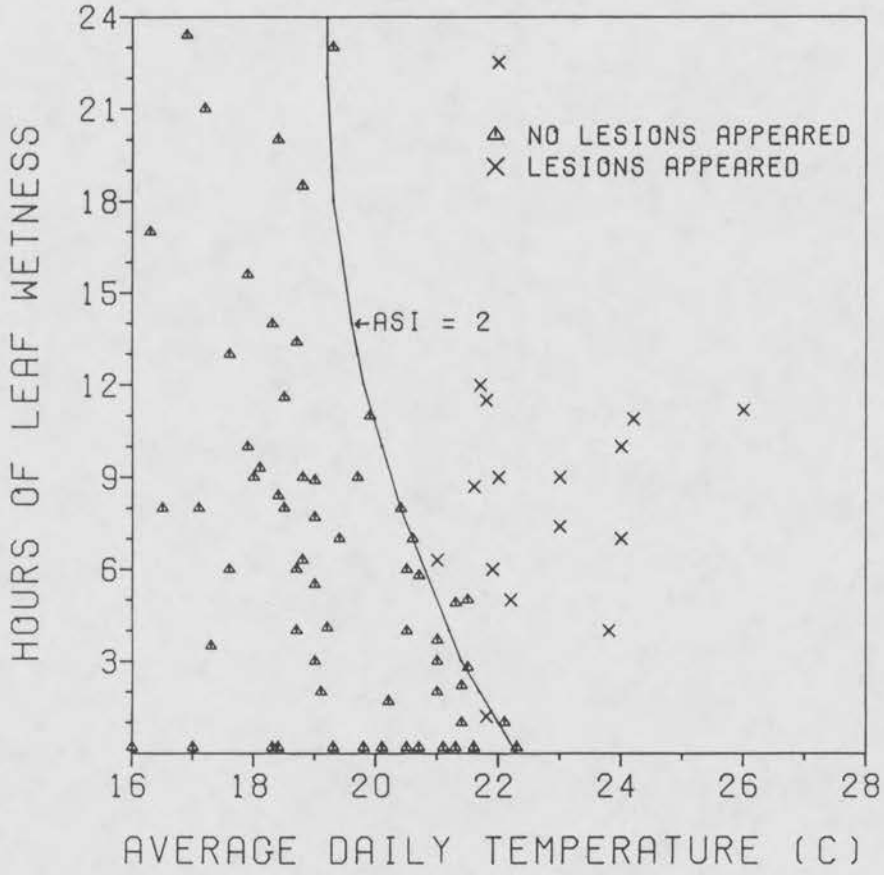
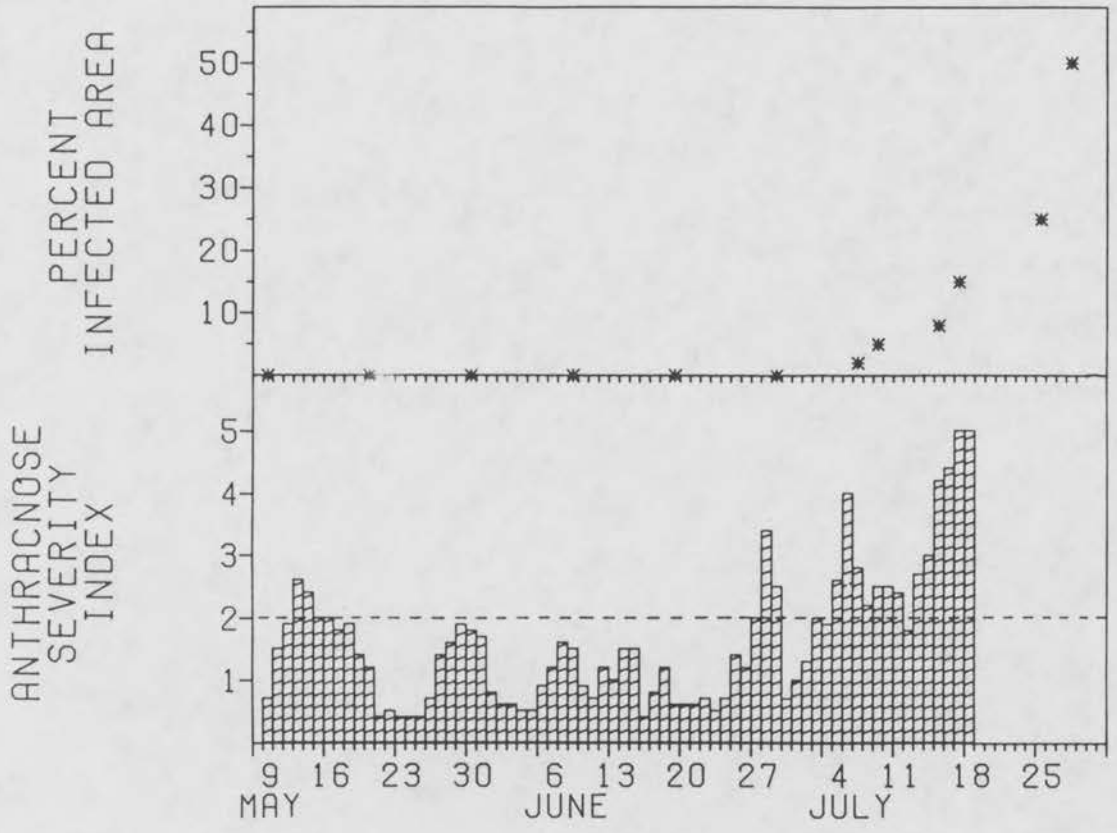

HOURS OF LEAF WETNESS
AVERAGE DAILY TEMPERATURE (C)
NO LESIONS APPEARED
LESIONS APPEARED
←ASI = 2
24
21
18
15
12
9
6
3
16
18
20
22
24
26
28

Figure 4. Progress of anthracnose on annual bluegrass at the Robert Hancock Turfgrass Center during 1982 and the favorability of temperature and leaf wetness as expressed by the anthracnose severity index (ASI). Dotted line represents threshold value for infection.


PERCENT
INFECTED AREA
50
40
30
20
10
ANTHRACNOSE
SEVERITY
INDEX
5
4
3
2
1
9 16 23 30 6 13 20 27 4 11 18 25
MAY
JUNE
JULY

area of annual bluegrass infected. Favorable periods on 6-12 July and 14-19 July were both followed by periods of disease increase.

Rates of disease increase and daily ASI values were calculated for two locations (Table 1). The resultant F-statistics for homogeniety of regression coefficients before pooling the data from Glengary County Club and Hancock Research Center were not significant ($\underline{P}$=0.05). Therefore, the data from the two locations were combined. When rates of disease increase were compared to daily ASI values, the Gompertz model resulted in a better fit (r^2 = 0.912) than the logistic model (r^2 = 0.646). The resultant regression showed that the predicted daily ASI values were related to the rates of disease increase (Figure 5).

Table 1. Rates of change in anthracnose severity and average daily anthracnose severity index (ASI) calculated with an infection model from temperature and leaf wetness data collected from two locations

Date	Infection area (%)		Infection Rate		Average
(t_1-t_2)	D_1[W]	D_2	Logistic[X]	Gompertz[Y]	ASI[Z]
GLENGARY					
6/22-6/24	5	10	.231	.088	3.27
6/24-7/06	10	15	.027	.015	2.13
7/06-7/09	15	40	.245	.182	4.02
7/09-7/15	40	70	.082	.135	3.26
HANCOCK					
6/27-6/29	3	5	.170	.053	2.60
6/29-7/05	7	8	.019	.024	1.71
7/05-7/07	8	15	.209	.096	3.13
7/07-7/15	15	25	.057	.035	2.68
7/15-7/17	25	50	.231	.231	4.50

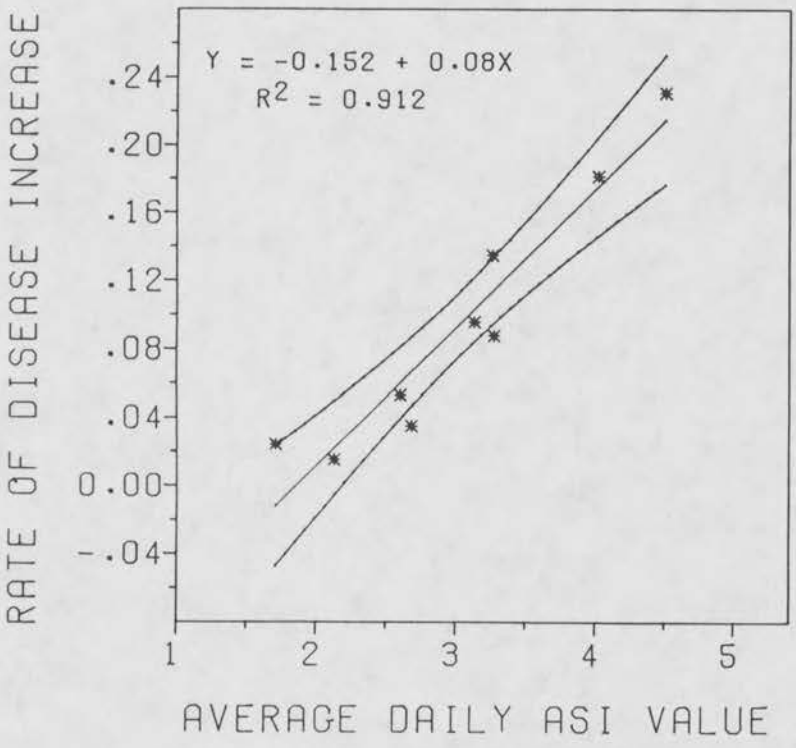
[W]D = percent infected area for the first (t_1) and second (t_2) dates of disease assessment.

[X]Logistic = $\ln (D_2) - \ln (D_1)/(t_2 - t_1)$

[Y]Gompertz = $-\ln (-\ln D_2) - (-\ln (-\ln D_1)/(t_2-t_1))$

[Z]Sum of ASI values from $t_1 - 12$ to $t_2 - 12$ divided by $t_2 - t_1$.

Figure 5. Fitted regression line and 95% confidence limits relating the proportional rate of change in disease as computed with a Gompertz transformation to an average daily anthracnose severity index (ASI) computed from mean temperature and wetness duration data (see Table 1).


Y = -0.152 + 0.08X
R2 = 0.912
RATE OF DISEASE INCREASE
.24
.20
.16
.12
.08
.04
0.00
-.04
1
2
3
4
5
AVERAGE DAILY ASI VALUE

DISCUSSION

A multiple regression model was developed for predicting the severity of anthracnose of annual bluegrass from daily mean temperatures and leaf wetness periods. The model assumed that adequate inoculum and a susceptible host were present.

The model accurately predicted that disease would increase once ASI values were 2 or above. Below this value, predictions were not followed by outbreaks of disease. Index values in early May sometimes predicted disease development but none occurred. This apparent failure of the model resulted from a lack of inoculum as determined by using live plants as spore traps. Testing of the model has been limited to average temperatures of 16 to 28 C. Although higher and lower temperatures should be studied, these temperatures fit the range at which the fungus is most active in culture (6,7) and is most damaging in the field (2,7).

The model may allow for the development of new disease management strategies for the anthracnose disease. Turf managers could reduce the severity of disease by reducing periods of leaf wetness at critical times. This could be accomplished by limiting the duration of leaf wetness periods from irrigation below the hours required for infection at prevailing temperatures. It may also be possible to time fungicides with this model. However, before fungicides can be combined with these predictions, it must be established whether fungicides with curative

activity are available. This is because spray treatments based on the model will be delayed until after the onset of infection.

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PART IV

EFFECT OF WATER STRESS ON ANTHRACNOSE OF ANNUAL BLUEGRASS

ABSTRACT

Greenhouse experiments were conducted to study the effect of pre- and post-water stress treatments on annual bluegrass (Poa annua var. reptans (Hauskins) Timm) infected with Colletotrichum graminicola (Ces.) Wils., the causal agent of anthracnose. Annual bluegrass plants pre-stressed at soil water potentials of -0.3, -1.0, -2.0 and -3.0 bars before inoculation resulted in increased disease severity once the water potentials were less than -1.0 bar. Annual bluegrass plants post-stressed at the different soil water potentials after inoculation had no differential response in disease development.

INTRODUCTION

Annual bluegrass (*Poa annua* var. *reptans* (Hauskins) Timm) is maintained as a desirable turfgrass species under adequately irrigated conditions in the cool season grass areas of North America. Water stress often occurs even under irrigated situations (1). Annual bluegrass is more readily injured from internal plant water deficits created by either atmospheric or soil drought than other cool season grasses such as creeping bentgrass (*Agrostis palustris* (Huds.) and Kentucky bluegrass (*Poa pratensis* L.) (2,5). Predisposing turf to water stress increases the disease severity of certain diseases (7,9,10), decreases the severity of others (6,11) and has no effect on some (3). Anthracnose caused by *Colletotrichum graminicola* (Ces.) Wils. is a disease that attacks annual bluegrass during the warm weather of July and August when water deficits would be expected (4,14,15). No information is currently available on the relationship between annual bluegrass and anthracnose as affected by water stress.

The purpose of this work was to determine the role of water stress on anthracnose development.

METHODS AND MATERIALS

One-mo-old annual bluegrass plants were subjected to four soil water potentials, -0.3, -1.0, -2.0 and -3.0 bars for 10 days, preceding or immediately after inoculating with conidia from Colletotrichum graminicola (Ces.) Wils. An uninoculated control was included for each soil water potential treatment. Each treatment consisted of four replicates and the experiment was repeated twice.

The soil used in the experiment was an Aubbenaubbee-Capac sandy loam complex, classified as a fine-loamy, mixed mesic Aeric Ochraqualfs, consisting of 53.3% sand, 27.2% silt and 19.4% clay. The soil water characteristic curve was obtained by using a pressure plate apparatus (12). Soil samples 4 cm in diameter and 1.0 cm in thickness were saturated for 24 hrs, placed on a ceramic plate and subjected to pressure potentials of -0.3, -1.0, -2.0, -3.0 and -15.0 bars for 48 hrs. The amount of water available in the soil at -0.3, -1.0, -2.0, and -3.0 bars was 23.0, 18.0, 12.3, 9.0 and 8.2%, respectively. A 393 g sample of air dry soil (1.7% moisture) was placed in each of 20 - 300 ml waxed cheese containers having a depth of 5.5 cm. Addition of 90, 55, 48 and 35 g of water resulted in soil moisture tensions of -0.3, -1.0, -2.0 and -3.0 bar, respectively. Distilled water was added every 6 hrs to raise the water content to the desired level. Maximum loss of water incurred during this period, on a weight bases, was 5.0, 4.0, 2.0 and 0.5% for -0.3, -1.0, -2.0 and -3.0 bars, respectively.

Annual bluegrass was seeded at a rate of 0.5 g per container. The plants were fertilized two weeks after seeding with 50 kg/ha each of nitrogen, phosphorus, and potassium and maintained at a height of 2.5 cm. The pre-stress treatments consisted of predisposing plants at the desired soil water potential for 10 days in a growth chamber set at 20±1 C. The photoperiod in each chamber was 12 hr. Following the stress period, plants in each container were inoculated with 3 ml of conidia suspension (60,000 conidia/ml) and placed in a continuous mist chamber for 48 hrs. Spore suspensions were applied to the plants from a DeVilbiss hand atomizer. The containers were placed on a greenhouse bench at 22±2 C in a completely randomized design following removal from the mist chamber. Infected plants were counted from 20 randomly selected plants per container 10 days later. Plants were considered infected if acervuli were present on the leaf surface.

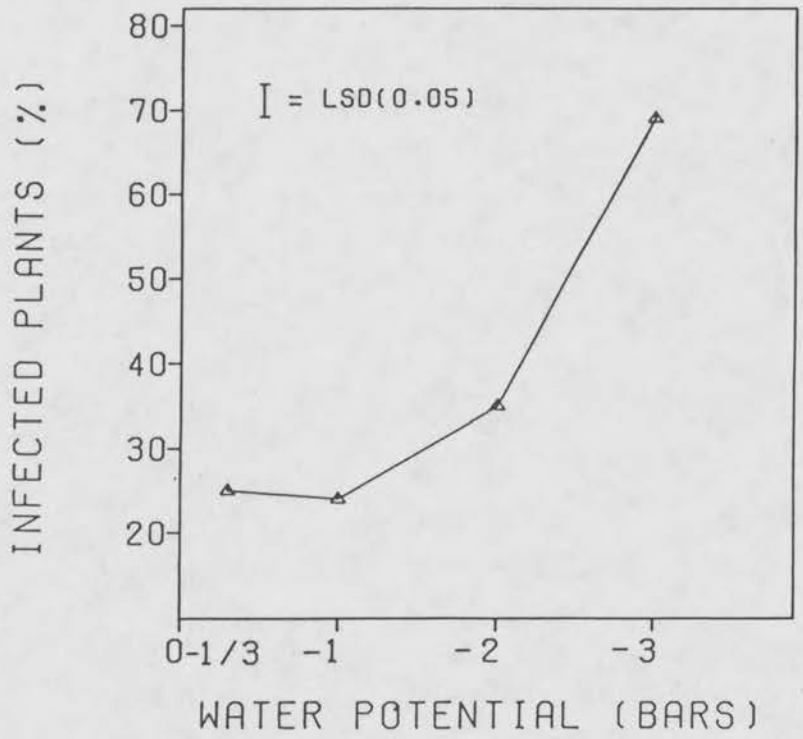
The post-stress treatments consisted of inoculating the plants in each container with 3 ml of conidia suspension (60,000 conidia/ml), misting for 48 hr, then stressing the plants at the desired soil water potential for 10 days in a growth chamber set at 20±1 C. Immediately following removal from the growth chamber infected plants were counted from 20 randomly selected plants per container.

RESULTS

Infection occurred at all soil moisture levels. However, soil water potentials less than -1.0 bar significantly increased the amount of disease present on annual bluegrass plants subjected to the water stress treatments before inoculation with C. graminicola (Figure 1). The plants that were pre-stressed at -0.3, -1.0, and -2.0 bars before inoculation visually appeared similar in color and overall health. The plants at -3.0 bar appeared spindly and chlorotic and were severely infected and chlorotic after inoculation.

The post-stress experiment that consisted of inoculating the plants with C. graminicola before stressing the plants at the various soil water potentials, resulted in no difference in the amount of disease present.

Figure 1. Effect of pre-stressing annual bluegrass to four soil water potentials of anthracnose infection.


80
70
60
50
40
30
20
I = LSD(0.05)
INFECTED PLANTS (%)
0
-1/3
-1
-2
-3
WATER POTENTIAL (BARS)

DISCUSSION

An increase in the amount of anthracnose at soil water potentials less than -1.0 bar occurred on annual bluegrass plants stressed preceding inoculation with C. graminicola. The pre-water stress appeared to be the major factor in increasing the susceptibility of annual bluegrass to C. graminicola since post-water stress treatments after initial inoculation resulted in no significant disease differences at any of the soil water potentials. This may not be an uncommon occurrence because the same effect has been reported for Phytophthora root rot in sunflower (8). The effect of the preinoculation water stress treatments that lead to the increased amount of anthracnose infection was probably a result of increased host susceptibility due to the unhealthy appearance of the plants at the lower soil water potential, rather than increased pathogen virulence. Although effects of stress on the host-pathogen interaction is often difficult to separate (13,16).

Management practices that minimize the time annual bluegrass plants are subjected to moisture stress could reduce the severity of anthracnose. Turf managers may need to irrigate frequently to maintain soil moisture levels at or near field capacity. Due to the fact that moisture levels are lowest at mid-day (1), turf managers may also be required to irrigate or syringe during mid-day if moisture stress occurs when conditions are favorable for infection by C. graminicola.

Continued research is needed to determine the relationship between our growth chamber results and results obtained under actual field conditions. Future research should also examine the importance of changes in leaf water potentials related to disease development. The major reason being C. graminicola is a foliar pathogen during warm weather. Thus, fluctuations in leaf water potentials would greatly influence any host-pathogen interaction that might occur when environmental conditions are optimum for infection by C. graminicola. However, the importance of leaf water potentials might play regarding disease development is directly related to soil moisture levels.

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PART V

ANTHRACNOSE DEVELOPMENT ON ANNUAL BLUEGRASS IN RESPONSE TO NITROGEN CARRIERS AND FUNGICIDE APPLICATION

ABSTRACT

Anthracnose caused by Colletotrichum graminicola (Ces.) Wils., is a serious disease of annual bluegrass (Poa annua L.) turf. In the northern and pacific-northwestern United States, annual bluegrass is the predominant golf course turfgrass and in some instances the main turfgrass species of home lawns. In turf, cultural practices are effective ways of controlling many turfgrass diseases. However, no reports are available on cultural practices that may reduce or control anthracnose development on annual bluegrass. The purpose of this study was to look at one cultural practice, nitrogen fertilization, along with fungicide treatments for controlling anthracnose. A field study was initiated in November of 1979. Three nitrogen carriers (isobutylidene diurea, sulfur-coated urea, and urea), applied at two rates (1.46 kg N/are/year and 2.92 kg N/are/year) and two timings spring (April initiation) and summer (June initiation), with or without fungicide treatments were evaluated for anthracnose control. Triademefon [1-(4-Chlorophenoxy)-3,3-dimethyl-1-(1H-1,2,4-triazol-yl)-2-butanone] fungicide treatments provided the most effective management of anthracnose. Fungicide treated plots averaged 1.9 and 1.7% infected area for 1980 and 1981 whereas non-fungicide treated plots were 29.6 and 30.6% infected, respectively. Type of nitrogen carrier, whether isobutylidene diurea (IBDU), sulfur-coated urea (SCU) or urea, had no effect on anthracnose development. Moderate nitrogen levels (1.46 kg/are/year)

were associated with less disease incidence than the higher level of nitrogen (2.92 kg/are/year). Nitrogen applications during the months of June, July, August, September, and November resulted in less disease than nitrogen applied in April, May, June, August, and September. Growth chamber inoculation studies showed the number of acervuli formed decreased with increasing nitrogen at 22 C. At 32 C, the number of acervuli decreased with increasing N to 0.90 kg/are but increased at the 1.80 kg/are nitrogen rate. In conclusion, nitrogen fertilization program of applying moderate levels of nitrogen (1.46 kg/are/year) with applications beginning in June followed with applications in July, August, September, and November at 0.24, 0.24, 0.24, 0.24, and 0.48 kg nitrogen/are, respectively, resulted in less anthracnose damage than the higher level of nitrogen (2.92 kg/are/year) or the alternate application schedule (April, May, June, August, September). If the nitrogen program was combined with fungicide applications, anthracnose was effectively controlled.

INTRODUCTION

Anthracnose caused by Colletotrichum graminicola (Ces.) Wils., is a serious disease of annual bluegrass (Poa annua L.) golf course fairways, greens, and home lawns in the northern and pacific-northwestern United States.

Anthracnose was first reported as a disease of annual bluegrass in 1954 (12). Since then the disease had not received much attention until the 1970's when outbreaks occurred in Michigan (14). Anthracnose appears as irregular shaped patches varying in size from 15 cm up to several meters, eventually covering entire fairways, greens, or lawns. Leaf spots first appear yellow, turning quickly to bronze during hot humid weather (1). The fungus produces acervuli, which are characteristic signs of this disease. Anthracnose has been reported on other turfgrass species such as Canada bluegrass (Poa pratensis L.) (4) and perennial ryegrass (Lolium perenne L.) (5).

Information regarding the influence of nitrogen fertilization on anthracnose development is not available. However, previous work with other turfgrass diseases has shown nitrogen fertilization to be an important factor in disease development. High nitrogen levels have been shown to increase the severity of Drechslera leaf spot [Drechslera sorokiniana (Sacc.) Subram. and Jain] on Kentucky bluegrass (Poa pratensis L.) (3) and red fescue (Festuca rubra L.) (10). Also, high nitrogen levels have been associated with increased browth patch

(Rhizoctonia solani Kuhn) (2), Ophiobolus patch (Gaeumannomyces graminis (Sacc.) Arx and Oliver) (7), Drechslera melting-out [Drechslera poae (Baudys) Shoem.] and Fusarium blight [Fusarium roseum (LK.) Snyd. & Hans f. sp. cerealis and F. trincinctum (Cda.) Snyd & Hans f. sp. poae] (6). In contrast, low nitrogen levels can also increase disease severity, such as dollar spot (Sclerotinia homoeocarpa F. T. Bennett) on creeping bentgrass (Agrostris palustris Huds.) (11), and red thread [Corticium fuciforme (Berk.) Waket.] on fescues (Festuca spp.) (8). The purpose of this study was to evaluate the effect of nitrogen carriers, timing and rate interactions with and without fungicide treatments for anthracnose control.

METHODS AND MATERIALS

Field experiments

A field experiment was conducted in Sylvania, Ohio, from November of 1979 to November of 1981. The experimental design was a randomized complete block with three blocks. Each plot measured 1.8 X 2.7 m and contained at least 90% annual bluegrass [*Poa annua* var. *reptans* (Hauskins) Timm.]. The turf area was maintained at a height of 1.3 cm and irrigated when needed.

Nitrogen (N) fertilizers evaluated included soluble (urea, 45-0-0), slow release sulfur coated urea (SCU, 32-0-0), and insoluble isobutylidene diurea (IBDU, 31-0-0). Fertilizers were applied at rates of 1.46 and 2.92 kg N/are/year. Application of N fertilizers followed two programs. One program was initiated in April (spring), the other in June (summer). The spring treatment consisted of applications in the months of April, May, June, August, and September. For the 1.46 kg N spring treatment the rates were applied at 0.37, 0.37, 0.24, 0.24, and 0.24 kg N/are, respectively, for the corresponding months. The summer treatment consisted of applying the N fertilizers in June, July, August, September, and November. The 1.46 kg N/year rates were applied at 0.24, 0.24, 0.24, 0.24, and 0.48 kg N/are, respective, for the summer treatment.

The 2.92 kg N/year treatments were double that of the 1.46 kg/N/year and applied at the same time. All fertilizer treatments were applied the first of each month.

Each plot received either a fungicide treatment or no fungicide. The fungicide used was triademefon [1-(4-Chlorophenoxy)-3,3-dimethyl-1-(1H-1,2,4-triazol-1-yl)-2-butanone; Bayleton®, Mobay Chemical Corp.][1] which was applied the first of June and every 14 days thereafter up to the first of September. The fungicide was applied at 0.6 g/m^2 active ingredient (a.i.). The experiment contained two controls. The first had no fertilizer or fungicide applications and the second contained no fertilizer but did receive the fungicide treatment. Plots were evaluated 10 days after initial symptoms appeared (24 June 1980 and 8 July 1981). Percent infected area was determined by evaluating anthracnose damage in each plot by visual observation. The experiment was analyzed as a 3X2X2X2 factorial.

The visual ratings of the 1981 non-fungicide treated plots were converted into number of multiple infections per 1,000 plants. Van der Plank's (13) transformation equation was used.

$$m = -\log_e(1-y)$$

Where m is the mean number of infections per plant and y is the proportion of diseased area.

Laboratory experiment

In vitro study was performed to measure the growth of *C. graminicola* on media containing various concentrations of N. Eighteen-day-old cultures of *C. graminicola* growing on 4% potato dextrose agar (PDA) were used. Plugs of the fungus measuring 2 mm in diameter were transferred to water agar media containing 0, 10, 100, 500, 1,000, 2,000, and 5,000

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[1]Mention of a commercial product by name does not imply endorsement to the exclusion of others which may be suitable.

µg/ml of technical grade urea [$(NH_2)_2CO$] and allowed to grow for 10 days at 22 C. Diameters of the cultures were then measured. Each treatment was replicated four times and the experiment was repeated once.

Greenhouse experiment

An experiment was conducted using 8 week old annual bluegrass plants. The plants were grown in 18 cm diameter pots containing a greenhouse mix of sand, soil, and peat (1:1:1). Each pot received one of four treatments. Treatments used were 0, 0.23, 0.45, 0.90, and 1.80 kg N/are applied as urea. Adequate levels of P and K were maintained. Ten days later the plants were spray inoculated with 325,000 spores/ml of C. graminicola. Each pot received 3 ml of inoculum. The plants were continuously misted for 72 hours, at 22 C. The plants were then placed in growth chambers set at 22 and 32 C. Acervuli were counted 5 days later from 20 random leaf blades in each pot. The treatments were replicated four times and the experiment was repeated once.

RESULTS

Field study

Fungicide application had the greatest effect on reducing anthracnose development (Table 1). Fungicide treated plots averaged 1.9 and 1.7% infected area across all N treatments for 1980 and 1981 whereas non-fungicide treated plots contained 29.6 and 30.6% infected area.

The type of N fertilizer had no effect on anthracnose development (Table 1 and 2). Across all treatments urea averaged 17.7 and 17.3% for 1980 and 1981, regardless of the other factors (fungicide, rate or timing) while IBDU averaged 15.2 and 12.0, and sulfur-coated urea averaged 15.1 and 18.0, respectively. Rate of N had an effect on anthracnose development which showed that high levels of N (2.92 kg/are/year) caused more disease damage than a lower level of N (1.46 kg/are/year) (Table 1, Figure 1). In 1980, non-fungicide treated plots receiving 1.46 kg N/are/year averaged 25.0% infected area whereas treatments receiving 2.92 kg N/are/year contained 36.1% diseased area. In 1981, non-fungicide treated plots receiving the 1.46 kg N/year rate averaged 16.0% anthracnose development whereas the 2.92 kg N rate contained 39.8% infected area (Figure 1). The fungicide treated plots for both years were not significantly different. Interaction between fungicide and rate of N was significant. The application of 1.46 kg N/are/year with fungicide resulted in the least amount of disease development.

Table 1. The effect of rate, type, and timing of nitrogen fertilization and fungicide applications on anthracnose development.

	Fungicide				No fungicide			
	Spring[a]		Summer[a]		Spring		Summer	
N source	1980	1981	1980	1981	1980	1981	1980	1981
Urea:								
1.46 kg/year	1.7[b]	0.0	0.0	0.0	25.0	23.3	26.7	13.3
2.92 kg/year	6.7	3.3	0.0	0.0	46.7	61.7	35.0	29.3
IBDU:								
1.46 lbs/year	3.3	0.0	0.0	0.0	31.7	13.3	21.7	10.0
2.92 lbs/year	0.0	8.3	1.7	0.0	31.7	55.0	31.7	19.0
SCU:								
1.46 lbs/year	1.7	2.7	0.0	0.0	21.7	24.3	23.3	11.7
2.92 lbs/year	3.3	4.3	1.7	3.3	46.7	42.0	25.0	41.7
Control	2.3	2.0	2.3	2.0	45.3	52.3	45.3	52.3

[a]Spring refers to N being applied in April, May, June, August, and September. Summer refers to N being applied in June, July, August, September, and November.

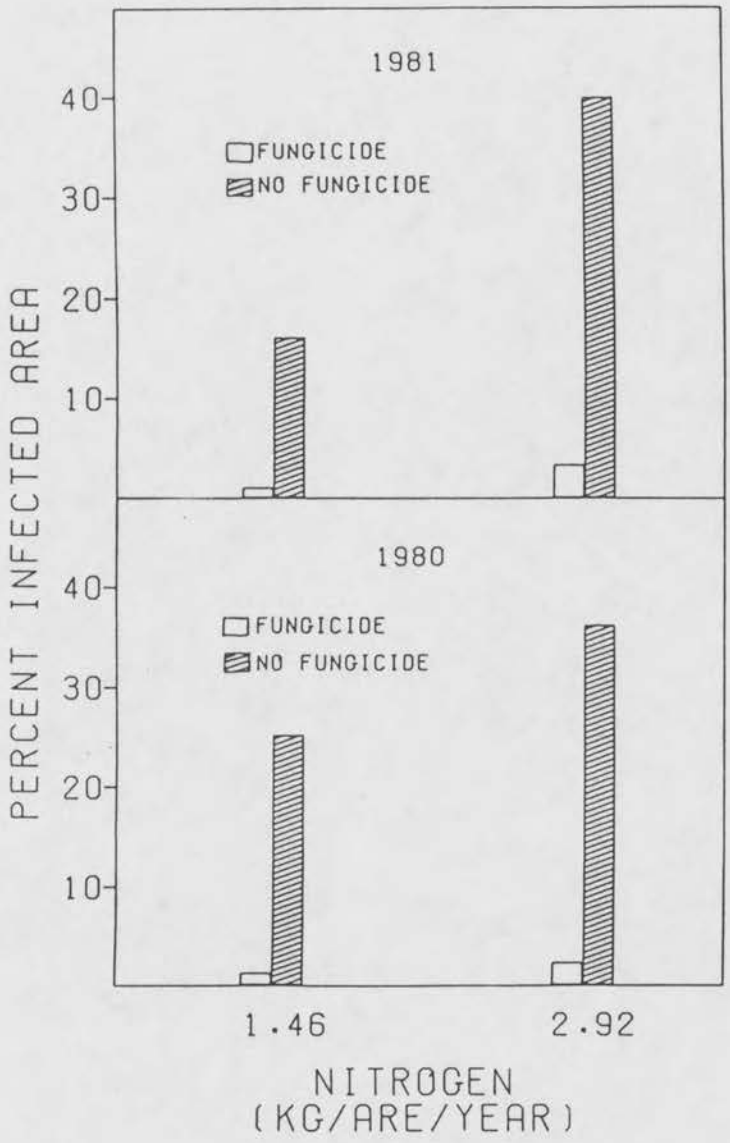
[b]Values based on a percent infected area.

Table 2. Analysis of variance of anthracnose damage influenced by rate, type, and timing of nitrogen fertilization and fungicide application.

Source of variation	df	Mean squares 1980	Mean squares 1981
Fungicide (Fu)	1	15,022.22*	12,746.72*
Fertilizer (Fe)	2	46.18	68.85
Rate (R)	1	672.22*	3,726.72*
Timing (T)	1	355.56*	1,605.56*
Fu x Fe	2	25.35	117.10
Fu x R	1	450.00*	2,426.72*
Fe x R	2	75.35	.85
Fu x T	1	88.89	854.22
Fe x T	2	12.85	89.26
R x T	1	88.89	304.22
Fu X Fe x R	2	19.79	7.09
Fu x Fe x T	2	21.18	68.35
Fu x R x T	1	88.89	107.56
Fe x R x T	2	162.84	342.93

*Significant at the 0.05 level.

Figure 1. Effect of two different nitrogen rates on anthracnose development of annual bluegrass.


1981
FUNGICIDE
NO FUNGICIDE
40
30
20
10
1980
FUNGICIDE
NO FUNGICIDE
40
30
20
10
PERCENT INFECTED AREA
1.46
2.92
NITROGEN
(KG/ARE/YEAR)

The time of application of N was critical in disease development (Figure 2). In both 1980 and 1981, spring application of N resulted in higher anthracnose incidence than the summer treatment of N. In 1980, spring treatment of N in the non-fungicide treated plots averaged 33.9% diseased area compared to the summer average of 27.2. In 1981, the spring diseased area in the non-fungicide treated plots was 36.6% compared to the 19.2 for the summer. The fungicide treated plots showed the same trends as the non-fungicide treated plots. Spring N application in 1980 resulted in 2.8% anthracnose while the summer treatment contained 0.6, and in 1981, anthracnose was 3.1 and 0.6%, respectively.

Non-fungicide treated plots of 1981 calculated for multiple number of infections (Table 3) showed similar trends as mentioned previously in Table 1. The type of N carrier was not significant. However, plots receiving N at 1.46 kg/are/year were associated with fewer number of multiple infection (31.0) than the 2.92 kg/are/year treatment (276.1). The summer treatment resulted in 53.9 multiple infections/1,000 plants compared to 253.2 multiple infections/1,000 plants for the spring treatment.

Laboratory results

Increasing the amount of N in the growing medium above 1000 µg/ml restricted growth of the pathogen (Figure 3). Between 0 and 100 µg/ml no difference in fungal growth occurred.

Figure 2. Effect of nitrogen timing on anthracnose development of annual bluegrass.

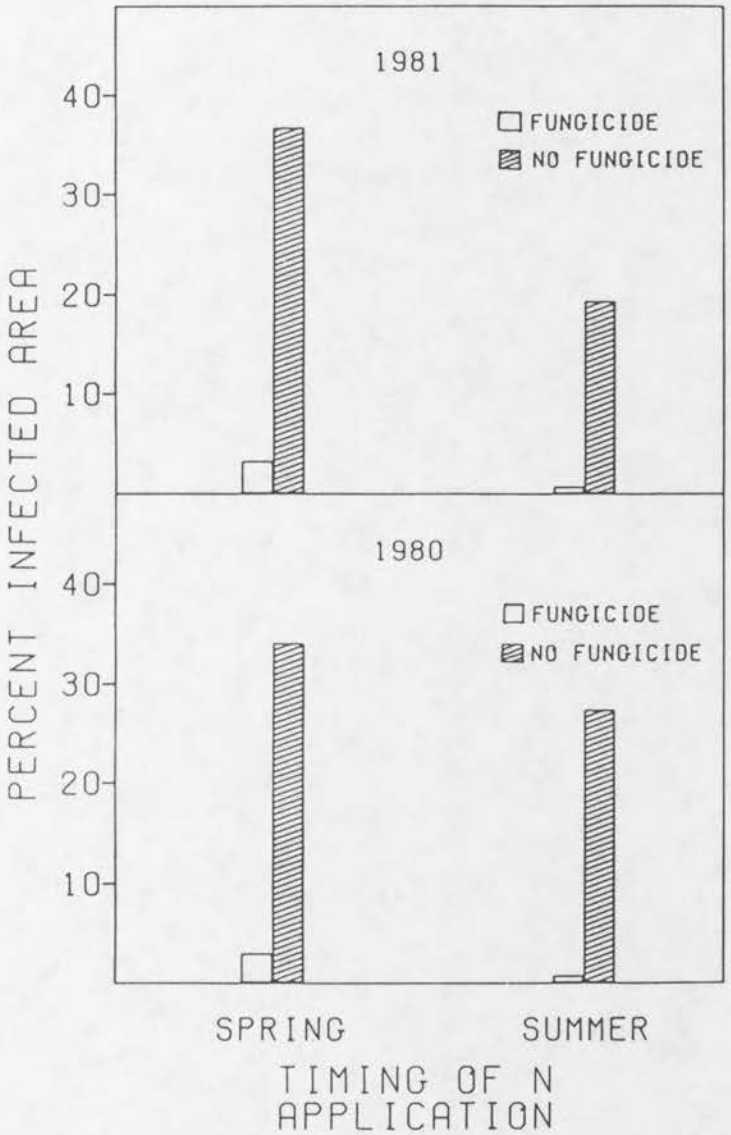

1981
FUNGICIDE
NO FUNGICIDE
40
30
20
10
1980
FUNGICIDE
NO FUNGICIDE
40
30
20
10
PERCENT INFECTED AREA
SPRING
SUMMER
TIMING OF N APPLICATION

Table 3. The numbers of multiple infections calculated by Van der Plank's equation $m = -\log_e (1-y)$ for non-fungicide treated plots in 1981.

N source	Spring[a]		Summer[a]	
	Infected area[b] (y)	No. of multiple infections/1,000 plants[c]	Infected area (y)	No. of multiple infections/1,000 plants
Urea:				
1.46 kg/year	0.233	35.0	0.133	15.3
2.92 kg/year	0.617	477.7	0.293	84.0
IBDU:				
1.46 lbs/year	0.133	16.3	0.100	8.0
2.92 lbs/year	0.550	576.7	0.090	21.7
SCU:				
1.46 lbs/year	0.243	106.7	0.117	4.7
2.92 lbs/year	0.420	306.7	0.417	189.7
Control	0.523	841.7		

Source of variation	df	Mean squares
Fertilizer (Fe)	2	2061.4
Rate (R)	1	616,486.7*
Timing (T)	1	300,121.4*
Fe x R	2	1,567.7
Fe x T	2	38,320.5
R x T	1	174,584.7
Fe x R x T	2	79,937.2

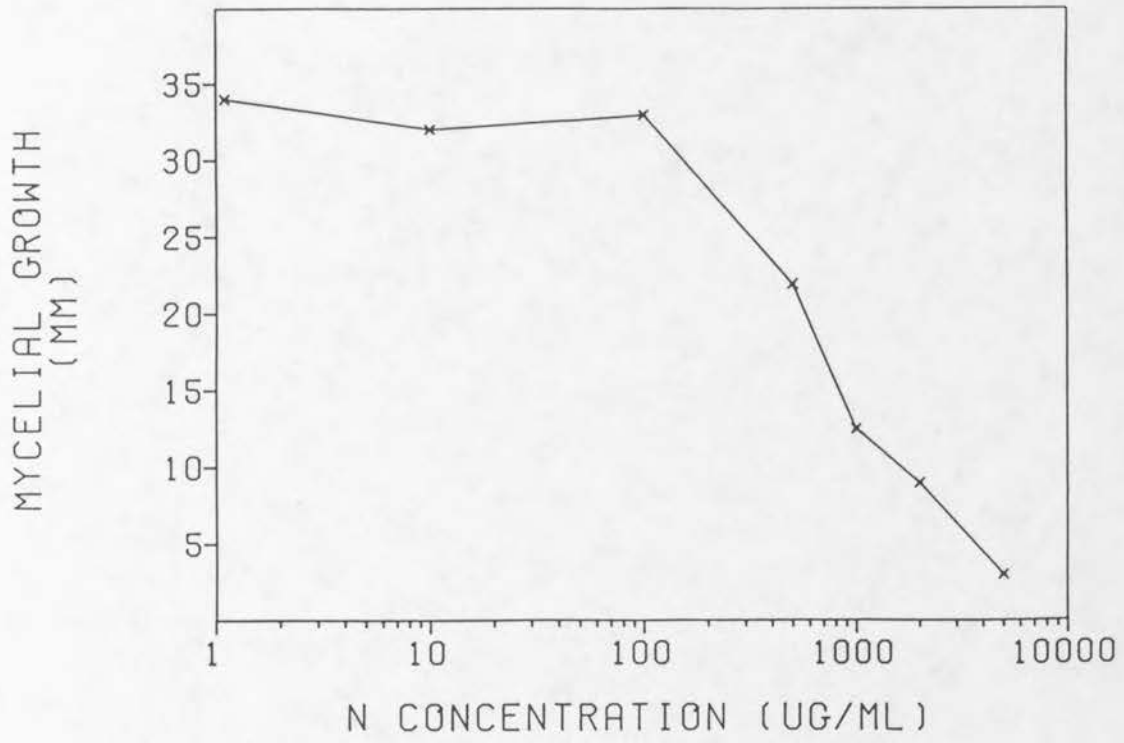
*Significant at the 0.05 level.

[a]Spring refers to the months April, May, June, August, and September when the N was applied. Fall refers to the months June, July, August, September, and November when N was applied.

[b]Expressed as a decimal of percent infected area.

[c]Multiple infections/1,000 plants = $-\log_3(1-y) \times 1{,}000$.

Figure 3. The effect of different concentrations of nitrogen on *Colletotrichum graminicola* growth *in vitro*.


MYCELIAL GROWTH
(MM)
35
30
25
20
15
10
5
1
10
100
1000
10000
N CONCENTRATION (UG/ML)

Greenhouse results

Inoculation studies showed that under 22 C increasing N resulted in decreasing acervuli formation (Table 4). Under warmer temperature (32 C) acervuli development decreased with increasing N to a point (0.90 kg/are) but increased at the higher rate of N (1.80 kg/are).

Table 4. Effect of different rates of nitrogen and temperature on the number of acervuli found on annual bluegrass leaf blades.

N rate	Number of acervuli	
	22.2 C	32.0 C
(kg/are)		
0	10.0	12.0
0.23	7.0	7.0
0.45	6.3	5.0
0.90	3.7	9.7
1.80	2.3	15.3
LSD (0.05)	1.6	3.1

DISCUSSION

Fungicide application was the most effective means of controlling the incidence of anthracnose regardless of N treatment. However, in situations where fungicide was not used, differential disease severity did occur with differing rates and timings of N. The type of N carrier had no sigificant effect on disease development.

Reduction in severity of anthracnose was observed when N was applied at 1.46 kg/are/year compred to 2.92 kg/are/year. The effect of N on fungal growth *in vitro* using varying N levels was inconclusive. However, greenhouse stidies showed the number of acervuli was reduced by increasing N levels at temperatures of 22 C. At 32 C, the number of acervuli on annual bluegrass leaf blades was the lowest at moderate N rates of 0.23 and 0.45 kg/are. The reduction in acervuli at 22 C with increasing N may reduce initial inoculum development whereas in warmer situations a decrease in acervuli numbers with moderate levels of N could result in a subsequent reduction in secondary inoculum and thus multiple infections. Reduction of multiple infections calculated from the field data was observed under moderate levels of N (1.46 kg/are/year) applied as the summer treatment compared to the higher level (2.92 kg/are/year) spring treatment schedule.

Timing of N application influenced the amount of disease that developed. Less disease was observed for summer N application vs spring applications. During summer months, the depth of rooting of annual

bluegrass can be one-third that of creeping bentgrass (Agrostris palustris Huds.) (9). The restricted root system of annual bluegrass may limit the amount of N uptake (especially when competing against creeping bentgrass) which, during environmental stress periods, may help predispose the plants to infection. Therefore, by applying moderate levels of N during the summer months, N availability and uptake may not be limited, thus less disease developed would occur.

In conclusion, our work shows that moderate levels of N (1.46 kg/are/year) applied during the months of June, July, August, September, and November (summer treatment) at 0.24, 0.24 0.24, 0.24, and 0.48 kg N/are, respectively, was the most effective N program for reducing the amount of anthracnose damage. If the N program is combined with fungicide applications, anthracnose was effectively controlled.

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APPENDIX A

THE EFFECT OF TEMPERATURE, LEAF WETNESS AND INOCULUM CONCENTRATIONS ON ANTHRACNOSE INFECTION SEVERITY OF ANNUAL BLUEGRASS

Table A1. The effect of temperature, leaf wetness and inoculum concentrations on anthracnose infection severity of annual bluegrass.

Leaf wetness (hrs)	Temperature (°C)											
	20				25				30			
conidia/ml	12	24	48	72	12	24	48	72	12	24	48	72
10^4	0.0[a]	7.7	8.3	9.0	0.0	11.0	16.7	18.3	1.0	19.3	17.0	22.0
10^5	0.0	20.3	53.7	71.7	4.7	45.0	79.0	100.0	11.0	63.0	89.1	100.0
10^6	0.0	26.0	85.0	96.0	11.7	69.0	100.0	100.0	17.7	82.0	98.6	100.0

Analysis of Variance

Treatment	d.f.	Mean Square
Experiment	2	22.87
Temperature (T)	2	10,764.12*
Leaf Wetness (LW)	3	66,667.77*
Inoculum (I)	2	89,785.15*
T x LW	6	1,021.77*
T x I	4	877.70*
LW x I	6	10,210.99*
T x LW x I	12	400.98*

[a]Percent infected plants based on 50 plants per replicate.

*Significant at the 0.01 level.

APPENDIX B

ALTERNATIVE FORMS FOR ANTHRACNOSE SEVERITY INDEX MODEL

Table B1. Alternative forms for anthracnose severity index equation (A.L. Jones, personal communication)

$$ASI = a + bLW + cT + dLW^2 + eT^2 = f(LW \times T)$$

$$T = \frac{c - fLW + (fLW-c)^2 - 4 \times 3 \times (2.0233 - b \times LW - (d \times LW)^2}{.2(e)}$$

---

a = 4.0233

b = -0.2283

c = -0.5308

d = -0.0013

e = 0.0197

f = 0.0155

LW = hours of leaf wetness

T = average daily temperature (C)

APPENDIX C

DETERMINATION OF THE PERIOD NEEDED FOR MAXIMUM SYMPTOM EXPRESSION TO OCCUR AT THREE TEMPERATURES FOR ANTHRACNOSE ON ANNUAL BLUEGRASS

One-mo-old annual bluegrass plants were grown in 700 cm^3 clay pots containing a mix of sand, soil, and peat (1:1:1, v/v). Plants were fertilized with 50 kg/ha each of nitrogen, phosphorus, and potassium and were maintained at a height of 2.5 cm by cutting the top growth weekly.

The isolate of C. graminicola used to inoculate the plants was obtained from an annual bluegrass plant at the Crops Field Laboratory, East Lansing, MI 48824. The isolate was grown on 4% potato-dextrose agar (PDA; Gibco Diagnostics, Madison, WI 53713) at 22 C. Spores from 18-day-old cultures were suspended in sterile distilled water and a spore concentration of 10^5 spores/ml was determined with a hemacytometer. Two milliliters of spore suspension was applied to the plants from a DeVilbiss hand atomizer. Viability of the spores, determined by atomizing spores onto blocks of PDA in Petri plates and incubating at 22 C for 48 hr, was above 90%.

The plants were continuously misted for 48 hr then placed on a greenhouse bench. The greenhouse temperature was 22±2, 25±2, or 30±2C depending on the temperature treatment. Initial appressoria formation was noted by microscopic observation. The day maximum disease expression occurred was determined by lesion counts per 20 randomly selected leaf blades. If no increase in lesion number occurred, maximum disease development was considered complete.

Each temperature treatment was replicated four times and the experiment was repeated twice. The results of the two experiments were averaged and are presented in Table 1C.

Table C1. Stages of anthracnose development on annual bluegrass in days from initial inoculation

Average temperature (± 2C)	Appressoria formation (day)	Maximum disease development (day)
22	2*	12.5
25	2	11.9
30	2	9.2
average	2.0	11.20

*days after inoculation