AN INOCULATION TECHNIQUE USED TO IDENTIFY RESISTANCE TO SORGHUM DOWNY MILDEW

By

QUENTIN B. KUBICEK II

Bachelor of Science

Louisiana State University and

Agricultural and Mechanical College

Baton Rouge, Louisiana

1976

Submitted to the Faculty of the Graduate College
of the Oklahoma State University
in partial fulfillment of the requirements
for the Degree of
MASTER OF SCIENCE
December, 1981

AN INOCULATION TECHNIQUE USED TO IDENTIFY

RESISTANCE TO SORGHUM DOWNY MILDEW

Thesis Approved:

Dale E. Weibel
Thesis Adviser

J. P. Worrill

Charles E. Denmon

Norman N. Durham
Dean of the Graduate College

ACKNOWLEDGMENTS

My sincere appreciation and gratitude is extended to my major adviser, Dr. Dale E. Weibel, for his extreme patience, friendship, invaluable assistance, constructive criticism, and constant encouragement throughout my stay at Oklahoma State University. I would also like to thank my advisory committee members, Prof. C. E. Denman and Dr. L. G. Morrill, for their valuable time taken to review the manuscript.

To my parents, Mr. and Mrs. Quentin and Olga Kubicek, I wish to express my deepest gratitude and love for their generous assistance, encouragement, and moral support throughout the course of my education.

I am indebted to the Fundacion Gran Mariscal de Ayacucho for their financial support. Special thanks is expressed to the faculty of and to the Agronomy Department of Oklahoma State University for providing the facilities which made this study possible.

TABLE OF CONTENTS

LIST OF TABLES

FIGURE

CHAPTER I

INTRODUCTION

Control of diseases in sorghum, Sorghum bicolor (L.) Moench, is essential since losses due to diseases reduce net profits. The incorporation of disease resistance into experimental breeding material and commercial hybrids would be beneficial. Fortunately, sorghum downy mildew, Peronosclerospora sorghi (Kulkarni) Weston and Uppal, although present in Oklahoma has been of no real concern in sorghum fields. Susceptible cultivars can be planted with little fear of production losses. As expected, cultivars are often selected for their yield potential and not for their resistance to sorghum downy mildew or any other diseases. However, this is not the case in South Texas where sorghum downy mildew may limit profitable sorghum yields. Susceptible sorghum breeding germplasm should not be used in South Texas as parental lines in the production of hybrids. Selection of resistant lines in segregating populations will allow for the development of resistant hybrid parental lines.

The objectives of this study were to develop a sorghum downy mildew screening technique similar to that developed by Craig (9), to identify resistant lines from segregating populations, and to get an indication of the mode of inheritance of resistance to sorghum downy mildew.

CHAPTER II

LITERATURE REVIEW

Peronosclerospora sorghi (Kulkarni) Weston and Uppal, the causal agent of sorghum downy mildew (SDM), is a destructive disease of sorghum (Sorghum bicolor (L.) Moench) and corn (Zea mays (L.)). SDM was first reported by Butler in India in 1907 (45). Weston and Uppal studied the genus Sclerospora and revised the status of certain members of this genus (49). Based on morphological and physiological comparisons Sclerospora graminicola var. Andropogonis sorghi (Kulkarni) the causal agent of what is now known as SDM, was recognized as being a distinct species from S. graminicola. They recognized the fungus as Sclerospora sorghi (Kulk.) Weston and Uppal (49). Shaw (44) elevated the subgenus Peronosclerospora of Sclerospora to a generic rank and transferred S. sorghi and other species into the genus Peronosclerospora. Thus S. sorghi is now referred to as Peronosclerospora sorghi (Weston and Uppal) C. G. Shaw, and comb. nov. Sclerospora sorghi (Weston and Uppal) is a synonym.

The host range of this disease is limited to a few members of the Gramineae family. Several authors have reported some of the known hosts of this fungus (3, 7, 12, 45, 48). The host range of SDM consists of sorghum, corn, teosinte, broomcorn, johnsongrass, sudangrass, sweet sorghum, S. arundinaceum, S. verticilliflorum, S. almum, and Heteropogon contortus.

SDM is found worldwide wherever sorghum and corn are grown (23). The disease was first reported in the United States in College Station and Chillicothe, Texas in 1961 (35). In the United States it is found in the following 16 states: Texas, Oklahoma, New Mexico, Kansas, Nebraska, Illinois, Indiana, Kentucky, Tennessee, Arkansas, Missouri, Mississippi, Alabama, Florida, Georgia, and Louisiana (16). Soilborne oospores or sexual spores are believed to cause primary infection in the disease cycle of SDM (7, 20, 27, 32, 45). Frederiksen (14) considers that infection occurs over a fairly wide range of environmental conditions. Oospores are the resting stage of the pathogen (8). Oospores can persist in the soil for several years and serve as the primary inoculum for succeeding crops (8, 25). Seed transmission of SDM is not very common but may occur in seed of certain diseased plants and may explain the appearance of an occassional systemically infected seedling in sorghum fields where SDM has not been known to occur (5). Seed transmission of inoculum is probably the primary means of long distance dissemination of oospores (19). There is no transmission of SDM mycelium by seed from infected plants if the seed is dried below 20 percent moisture (15). Young seedlings become infected at or soon after germination (13). The disease is initiated by hyphae from germinating soil- or seedborne oospores or airborne conidia (51). Susceptibility to systemic infection decreases with age (26, 28).

Sorghum downy mildew causes different symptoms and occurs in either an early season systemic, a late season systemic, or a localized form in sorghum. Early season systemically infected seedlings incited by oospore inoculum show a characteristic green mottling pattern on the first few leaves. Subsequent emerging leaves are bleached in streaks and stripes.

These bleached tissues do not support conidial production and are packed with oospores (50). Oospores are produced somewhat linearly arranged in the mesophyll cells, parrallel to the veins (45). Later these tissues will become red or purple and then turn necrotic, the leaves will be shredded by the wind and oospores will be liberated and fall to the soil. The life cycle of the fungus is then completed. In addition to leaf symptoms the internodes are shortened and the leaves stand out stiffly. Some genotypes show the characteristic bunched, stiff leaves, but are somewhat taller than healthy plants (45). Early season systemic infection is more damaging than late season systemic infection. A second symptom is that of late season systemic infection. Late season systemic infection occurs on plants after a period of apparently healthy growth. The lower portion of plants appear normal while the upper portion displays systemic symptoms. Late season systemic infection can be caused by either oospores or conidia (46). A third type of symptom is due to foliar or local lesion infection. Local lesions generally are considered to be caused by conidial inoculum produced from plants infected either systemically or locally (28, 29).

In the fields heavy dews provide the suitable saturated atmospheric conditions necessary for the abaxial surfaces of infected portions of leaves to produce the characteristic white bloom or down (31). This down composed of conidia and conidiophores is the asexual inoculum of the fungus. These conidia are important in the secondary spread of the disease (26). Local lesions are usually confined to interveinal tissue and are long and narrow (19). This type of infection produced strippled necrotic areas on the leaves and may remain localized or become systemic and produce the characteristic symptoms of striped leaves, shredding and

sterility (51). No sorghum cultivar appears resistant to local lesions (20).

Conidia are fragile, asexual spores produced on the tips of conidiophores by the fungus mycelium during the haplophase stage of its life cycle. Sporulation or production of conidia is primarily influenced by high relative humidity and cool temperatures. Darkness promotes sporulation but exposure of the infected plant to light is a prerequisite for sporulation (12, 38). Craig (9) placed systemically infected plants under 2000 ft-c light 10 hr before the leaf pieces were collected for the inoculation chamber. Bonde et al. (6) exposed plants to supplemental light for 12 hr but did not specify light intensity. Schmitt and Freytag (40) reported getting good sporulation when plants received incandescent light (125 ft-c) during the night. No sporulation occurred on leaf pieces from systemically infected plants kept for 8-9 hr in the dark before placing in the inoculation chamber (40). Light regimes of 600-20,000 ft-c for three or more hours on plants at 26 C induced very acceptable sporulation (40). By the proper timing of artificial light, temperature, and humidity, Craig (8) could secure conidia at any time, day or night. Conidia are hyaline, ephemeral propagules, having a duration of 3-4 hr under ideal conditions (2, 6, 8, 16, 20, 26, 39, 40). The short life of conidia necessitates the continuous production of inoculum. Conidia are not considered to play a role in the long range distribution of inoculum (16), although, they are considered to account for the secondary spread of the disease (14, 27). Miller (31) considers that a few systemically infected seedlings or plants can lead to a heavily diseased field. The optimum temperature for sporulation is around 21 C (20, 38). A wide optimum temperature range for sporulation

has been reported for SDM. Schmitt and Freytag (40) report it to be from 13 to 26 C. Frederiksen (20) did not plot conidial production curves, but obtained infections in plants incubated at temperatures from 15 to 30 C. Infection has been obtained as low as 10 C and as high as 33 C (6). Bonde et al. (6) working with precise laboratory equipment and specific environments, consider that the optimum temperature for conidia germination was 15 C with an optimum temperature range of 10 to 19 C. They reported the optimum temperature for germ tube growth was 22 C with an optimum range of 14 to 22 C (6). They do not report an optimum temperature for sporulation. The total time required for sporulation to occur from the time knob-like outgrowths of conidiophores begin to protrude through stomata until conidia matured was 7½ hr at 20 C (47). Germination, penetration, and establishment of infection on sorghum leaves occurs within a few hours (6, 26). Sorghum downy mildew must germinate and penetrate very rapidly due to the ephemerality of its conidia. Conidia are suborbicular, reportedly varying from 15 to 28.9 u X 15 to 26.9 u (17, 20, 38, 45).

The inheritance of resistance to oospores appears to be dominant (18, 30, 36, 37). Rao (34) reports resistance to be partially dominant and apparently quantitative based on F_3 progenies of resistant X susceptible crosses. Malaguti (30) reported that resistance is polygenic. Rosenow et al. (37) indicates more than one gene is involved. Frederiksen (20) believes that more than one gene is involved and in studies of the inheritance in a line derived from IS 2816, concludes resistance to be controlled by multiple genes or one major dominant gene with modifiers. Given the dominant nature of inheritance of resistance, Rosenow et al. (37) postulate that it should be easily transferred to

any genotype using the backcross method with selection for resistance. Anahosur (1) reports that additional doses of resistant parents in backcrosses increases the level of resistance proportionately. Riccelli (36) states that only one resistant parent is needed to obtain a resistant hybrid. Immunity is unknown and resistance is relative to inoculum potential (17).

Frederiksen et al. (19) indicates that resistance to conidia is closely related with average incidence of systemically diseased plants. The relationship between reactions to conidial inoculum and reaction to natural infection are complex and are not necessarily correlated since oospores constitute the primary inoculum in the field (51). Kenneth and Shahor (29) imply resistance to oospores is different from resistance to conidia. Resistance in corn to conidial infection and to oospore infection appears to be under a similar genetic system (42). Correlations between reactions of resistant hybrids to conidial inoculation in the greenhouse and their reactions are good, but less resistant hybrids exhibited much higher levels of infection in response to artificial inoculations than those observed in the field (8). Frederiksen (20) reports that SDM

> . . . field resistance did not hold up under laboratory conidial inoculations, although in a test with converted sorghum lines, those that were resistant in the field showed a comparatively lower degree of infection with artificial conidial inoculations and there was a positive correlation between infection in the field and infection from artificial inoculations for the same lines (p. 14).

Frederiksen and Rosenow (18) consider that there are several resistant lines under field conditions in Texas, but that only a few of these are resistant under artificial conidial inoculations.

It is possible that certain types of resistance to SDM were circumvented by the artificial inoculation technique (10). Kenneth and Shahor (29) questioned the use of conidial inoculation test to screen hybrids for resistance to systemic SDM. Yeh (51) concludes that artificial inoculation techniques may by-pass natural resistance mechanisms and induce infection in cultivars which are not necessarily susceptible in the field. Nevertheless conidial infection in the field is always a potential threat and resistance to this inoculum per se should be sought. Craig (9, 10) found that the reactions of sorghum cultivars to conidial inoculation at the one to two leaf stage were very similar to their reactions to natural infection under conditions that favor high disease incidence in susceptible cultivars in the field. Schmitt and Freytag (41) reported obtaining best infection when seedlings were inoculated between emergence and the 3-leaf stage. Although the technique used by Schmitt and Freytag (41) differed from Craig's (9) in that conidia in a suspension of distilled water were sprayed with an atomizer, the principle was the same. Their results showed that this method of inoculation could be used to identify field resistance to SDM. Yeh and Frederiksen (51) warn that susceptibility lessens with plant age, suggesting a structural resistance in a developing seedling. Craig (9) defends the technique of artificial conidial inoculation because reactions of the sorghum genotype agreed with their field rating for resistance to SDM. Craig (9) even considers this technique good enough to select SDM resistant genotypes in a heterogeneous population. He does not know of any genotypes resistant to conidial inoculations that may be susceptible to oospore infection (8). Frederiksen (16) reported that to date, the conidial inoculation technique has proved more favorable

because of consistency in reproducing the inoculation conditions.

> Artificial conidial inoculation produces a local lesion symptom. Some sorghum genotypes vary in reaction to the development of these lesions. The reaction of sorghum lines to local lesions is closely related to their field reaction to SDM. There is an important relationship between the resistant lines to local lesions and their resistance to SDM in the field. Individual plants with local lesions reactions can be scored and the problem of escapes avoided. Not all SDM resistance in sorghum is associated with resistance to local lesions. The resistance level observed with the local lesion technique can be used to evaluate early heterogeneous generations (pp. 904-905).

Sorghum downy mildew can be effectively and practically controlled with the use of resistant or tolerant hybrids (3, 4, 8, 13, 16, 22, 24, 31, 33, 45, 51). Genetic resistance is considered the best method of control, since specific chemical or cultural methods for SDM are unknown (14, 31). Growing susceptible genotypes may cause the oospore population to build up (25). The widespread adoption of resistant hybrids has served to substantially reduce damage and curtail the spread of SDM (24, 33). Besides genetic resistance, there are some cultural practices that influence the severity of SDM in grain sorghum. However, cultural practices seem to be less effective than genetic resistance. Rotation with non-related crops is the most important cultural practice (3, 4, 14, 22, 45). Pratt's (32) data suggest that non-host plants might be grown in infested soil to stimulate germination of oospores of SDM and there-by provide biological control of SDM in sorghum. Destruction of sorghum crop stubble as soon after harvest as possible is another recommended practice (3, 4, 45). Deep plowing offers partial control in oospore infested soils (14, 22). Tarr (45) suggests roguing. Destruction of johnsongrass and sorghum regrowth or volunteer is helpful (26). Losses can be avoided by overplanting, since most plants systemically infected

as seedlings are poor competitors (15). Avoid planting sorghum after the more susceptible sudangrass or sorghum X sudangrass hybrids (4, 13). The use of systemic fungicides to control SDM has been attempted with no real success (17). Vitavax seed treatment was totally unsuccessful in controlling SDM (11, 21). KN_3 at very low rates appears to reduce oospore inoculum in the field effectively, but a concentration of 200 ppm results in almost a total loss of seed germination (20). Schwinn (43) considers that dithiocarbamate fungicides such as Zineb, Maneb, Mancozeb, and phthalamide derivatives such as Captam, Folpet, and Captafol have no major movement in the plant tissues, are purely residual fungicides, and are not active against sustemic downy mildews. Recently, Ridomil $^{(R)}$, a Ciba-Geigy systemic fungicide (Code Number CGA-48988) has been found to be very effective against SDM when applied as a seed treatment (29, 38, 43).

CHAPTER III

MATERIALS AND METHODS

Acquisition of the Pathogen

The pathogen was obtained by planting seeds of a susceptible genotype in downy mildew infested soil. This soil was obtained near College Station, Texas from fields having a history of SDM. Seeds were allowed to germinate and the seedlings to grow until symptoms of systemic infection appeared. Seedlings were grown to the four to six leaf stage before systemically infected leaves were cut and used for inoculation. Perpetuation of the pathogen was accomplished by utilizing conidia from these systemically infected leaf pieces 2-3 cm in length to inoculate 48 hr old seedings. Culture of the pathogen is easier by conidial inoculation of pre-germinated seeds than by relying on planting susceptible genotypes in SDM infested fields. A very high degree of infection is obtained by inoculating 48 hr old seedlings of any genotype. Depending on natural infection in sorghum fields in Oklahoma for screening would be very unreliable due to: the small acreage of sorghum in the state, the proximity of these areas to the experimental site, and principally to the extremely low occurance of SDM in these areas.

Maintenance of the Pathogen

The pathogen was maintained in infected plants throughout the

duration of this study. One hundred to 150, 48 hr old seedlings were placed in the center of a petri dish such that the plumule and coleorhiza were horizontal and faced upward. A 0.64 cm mesh wire screen was positioned 2 cm above the seeds. A single layer of cheesecloth (Curity cheesecloth grade 40) covered the wire screen. The cheesecloth and wire screen were sprayed with distilled water prior to placement on the bottom dish. The seedlings were sprayed as well so as to leave a thin film of water over the seeds and cover dish. Systemically infected leaf pieces, 2-3 cm long, from plants that had received at least 12 hr of natural or artificial light were placed, abaxial side down, on the cheesecloth over the seedlings. A petri dish cover enclosed the entire petri dish moist chamber. The chamber was then placed in a black plastic bag to ensure total darkness and placed inside a seed germinator or growth chamber at 21.1 C for 24 hr. After 24 hr the leaf pieces were viewed for evidence of sporulation. If evidence of sporulation was satisfactory for a majority of the leaf pieces within a petri dish then the seeds were individually transferred with a pair of tweezers to flats containing an unsterilized porous light potting soil. Vermicullite or sand was sprinkled over the seeds to cover them. The flats were then immediately placed on trays filled with water until the soil was saturated by capillary action.

Symptoms appeared in the first or second leaf of the emerging seedlings. The basal half of this leaf was mottled light green and the succesive leaves were completely mottled. Generally, a very high percentage of the seedlings were infected. Those seedlings that did not show early symptoms of systemic infection were removed from the flats. Seedlings were utilized for inoculum until initial symptoms of oospore

formation appeared. Systemically infected leaves from these plants were used for screening and for perpetuating the inoculum. Plants were watered and fertilized with a complete fertilizer as needed.

Planting of Genotypes

Selected genotypes to be screened were planted in 8.25 oz capacity waxed-paper drinking cups. The bottom of the cups were perforated twice with a standard lead pencil before being three-fourths filled with greenhouse potting soil. The soil was sieved and not autoclaved. Eight to ten seeds of each entry were treated with Vitavax 300 and distributed over the soil surface of each cup. Then soil was added to cover the seeds to a depth of about 2 cm. Seeds were allowed to germinate either in the greenhouse at ambient temperatures or in a growth chamber at 26.7 C for a 15 hr day and 21.1 C for a 9 hr night regime. Seedlings were allowed to grow until they reached the first to the second leaf stage for inoculation, because it is at this stage that artificial screening correlates best with field screening (10). Each entry was thinned to leave the five seedlings per cup that were most similar to the desired growth stage.

In both the greenhouse and growth chamber studies, the cups were placed in trays. Each tray held a maximum of 20 cups. The infection chamber utilized six trays at one time. A replication consisted of 20 entries randomized and repeated in each of six trays. Since five seedlings per entry were inoculated, a replication had a maximum of 30 seedlings per entry during one run or test. The experiment was repeated three times. Every entry was evaluated up to 21 days after inoculation and segregating entries were classified resistant or susceptible based

solely on a percentage of plants showing symptoms of systemic infection or local lesions. Genotypes segregating for resistance to SDM were classified as being resistant if they showed 25% or less infection. Pure lines were not classified and their reactions to inoculations were recorded and tabulated.

Inoculation Chamber

The inoculation chamber was the same for both the greenhouse and growth chamber studies. The inoculation chamber apparatus consisted of six plastic trays 34 x 24 x 16 cm. Each tray had a tight fitting plastic cover. A rectangular section with dimensions 24.1 x 20.3 cm was cut from the center of each cover. A 0.64 cm mesh wire screen was fitted over the missing protions of the cover. Two holes, 1.9 x 2.5 cm, about 5.1 to 6.4 cm apart were cut in the center of the screen to accommodate air hoses. A single layer of absorbent cheesecloth (Curity cheesecloth grade 40) was placed over the screen. Infected pieces of leaf tissue 2-3 cm long were placed with the abaxial side down so as to cover the entire surface of the cheesecloth. A paper diaper was saturated with tap water, lightly squeezed to remove excess water, and positioned over the infected leaf pieces after the plastic lining had been removed. The diaper was covered with a 2 ml thick plastic cover. Holes were made through the cheesecloth, diaper, and plastic cover to coincide with those in the screen for two air hoses.

The ends of two polyethylene air delivery hoses having a 1 cm inside diameter were plugged and six to eight holes 1-2 mm in diameter and 1-2 cm above the plugs were made around the hose. The two air delivery hoses rested on the 2 ml thick plastic cover and were adjusted to

keep the perforated holes 2.5 cm below the wire screen (Figure 1).

Each of the pair of air delivery hoses leading into each tray were connected to a Y-shaped glass connector. The opposite end of this tube was connected to the main air hose at a T-shaped aluminum connector by a 17.8-22.9 cm polyethylene hose with a 1 cm inside diameter. The main air delivery hose had a 1 cm inside diameter and was 66 cm above the bench floor. Three pairs of air delivery hoses were connected to each one of two main air delivery hoses. The two main air delivery hoses were connected to an aluminum T-connector. The opposite ends of these two main air delivery hoses were sealed air-tight. To the remaining end of the T-connector was connected an air feeder hose having a 1.9 cm inside diameter. This air feeder hose was connected to another T-connector. From one of the ends of this connector was connected a 1 cm inside diameter hose with a screw clamp which served as a manual adjusting pressure release valve. The third outlet of the fitting was connected to an air feeder hose 15.2 m in length. The center 12.2 m of this hose was coiled and submerged in a 26 gal capacity aluminum trash can that was three-fourths filled with tap water. The end 1.5 m of this hose led to the pressure outlet valve of a 1/3 hp vacuum pump (Gast Pump Model 0522-V4B-G180DK). A 2 gal humidifier (Northern Humidifier Model 77) was placed inside an inverted 26 gal capacity aluminum trash can and together comprised the humidifying chamber. A 1.8 m long hose with a 1.9 cm inside diameter lead into the top of the inverted can to a depth of 10 cm. The opposite end of this hose was connected to the vacuum inlet of the pump. The vacuum air filter of the pump was removed to prevent it from absorbing any moisture. The pressure-outlet air filter was kept to filter vaporized oil from being dispersed into the trays.

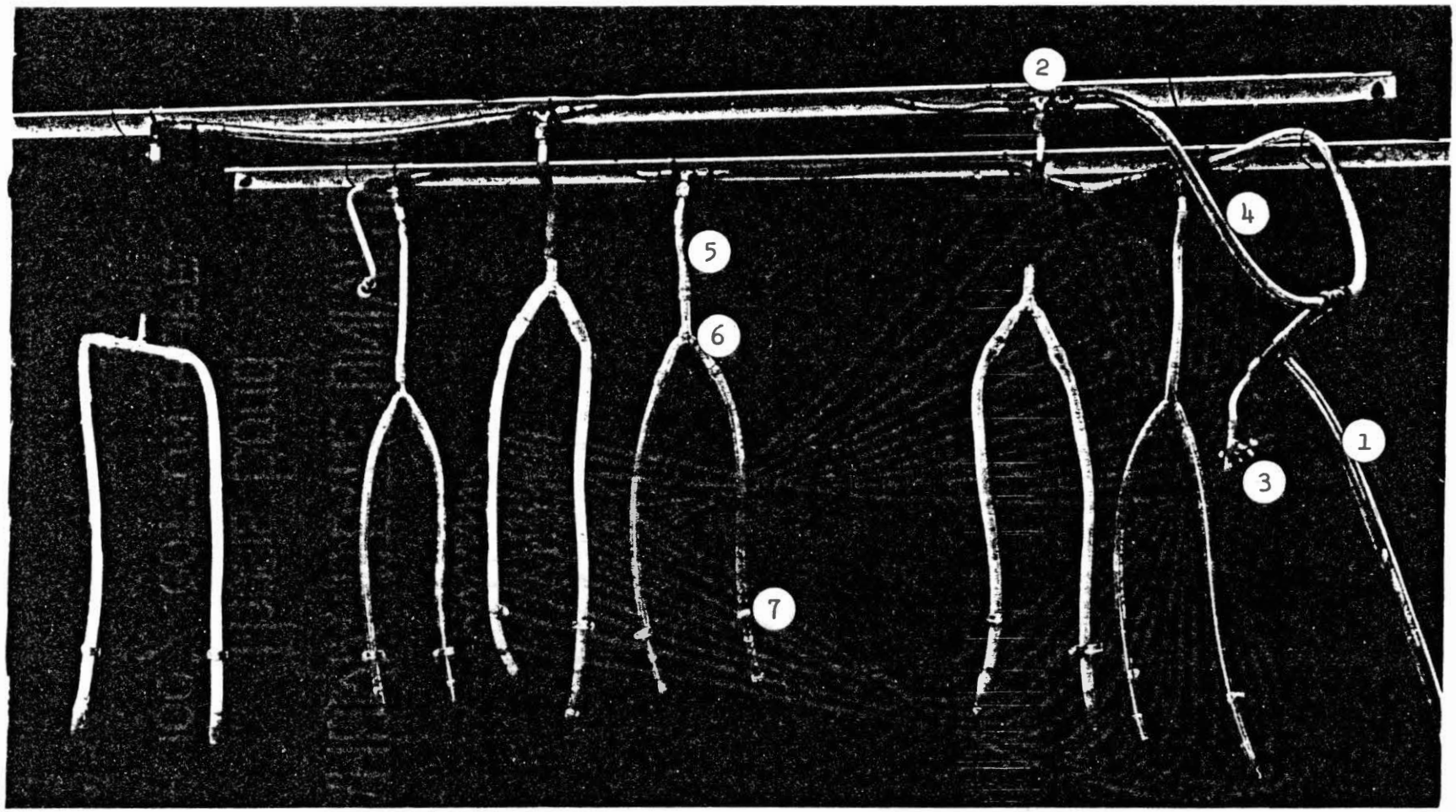

Figure 1. Air Distribution System of the SDM Artificial Conidial Inoculation Apparatus. 1) Air Feeder Hose (1.9 cm i.d.) from 1/3 hp Vacuum Pump Pressure Outlet Valve. 2) Aluminum T-Connector. 3) Screw Clamp Pressure Release Valve (1.0 cm i.d.). 4) Main Air Delivery Hose (1.0 cm i.d.). 5) Air Delivery Hose (1.0 cm i.d.). 6) Glass Y- or T-Connector. 7) Aluminum Hose Clamp.

Both the humidifier and vacuum pump were connected to an electric time switch. The time switch was set to turn on the pump six hours after the systemically infected leaf pieces had been placed on the trays and the lights turned off. The pump operated continuously at 1 psi and 25-30 mm of Hg vacuum for five hours. The humidifier operated continuously at a high setting and was kept filled with tap water.

Greenhouse Study

In the greenhouse study artificial inoculations were made at night during summer months. Sporulation of the pathogen occurs within a reported temperature range of 13 to 30 C. Two temperature recorders (Bacharach Instrument Co., Tempscribe Bimetal Temperature recorders, Code Number 14-0050) and two mercury thermometers were placed at bench level at opposite ends of the pair of three-tray sets. The thermometers were hung from the main air delivery hoses close to and above the temperature recorders. The artificial inoculation apparatus was set on a greenhouse bench at a height of 91 cm above the greenhouse floor. The bench lay 3-4 cm away from the cooling pads used to cool down the greenhouse in the summer. The cooling pads were operated whenever the artificial inoculation apparatus was utilized.

Seedlings were placed in the trays in the manner described previously in the late afternoon prior to sunset. Systemically infected leaf pieces were placed on the cheesecloth at this time also. Trays were half filled with tap water to saturate the soil with water, to reduce the air volume within the enclosed tray, and to help increase the relative humidity of the air. The high relative humidity required for sporulation of the fungus was obtained from evaporation of water from

the wet diaper, saturated soil, free water, and the incoming moist cool air from the air delivery hoses. Sporulation occurred at night and each tray was viewed for evidence of such the following morning. If from a visual check 80 percent or more of the leaf pieces had some degree of sporulation, inoculation of the seedlings was considered satisfactory. Evidence of satisfactory sporulation was a distinct visible white felt of conidia and conidiophores on the abaxial side of the leaf pieces. The leaf pieces were then discarded. Sporulation generally begins six hours after the leaf pieces are placed in the artificial inoculation chamber and proceeds for 2-4 hours. Night temperatures during which sporulation occurred were noted from the Tempscribe recorders. These temperatures were compared with the temperature range for sporulation. If the recorded night temperature did not fall within this temperature range then the set of trays was inoculated again the following evening. Only once did sporulation fail to occur due to night temperatures being in the lower scale of the temperature range for sporulation.

The following morning all of the water in each tray was siphoned out. Seedlings were kept in the greenhouse for 21 days and observed for symptoms of systemic infection. The seedlings that first showed symptoms of systemic infection or local lesions were removed from their cups and the data recorded. Seedlings were removed to prevent adjacent seedlings from possibly being inoculated. No seedlings were evaluated longer than 21 days after being inoculated.

Growth Chamber Study

A walk-in Sherer growth chamber, model CEL 512-37, was utilized to maintain the inoculum, to germinate seeds, and to make inoculations.

The entire artificial inoculation chamber was placed inside the growth chamber. In addition large flat shallow trays were placed on the growth chamber floor and were filled with tap water to help increase the humidity. A large bath towel resting on a wire screen was leaned against the growth chamber fan vents located near the floor. The bottom edge of the towel was submerged into one of the water trays. The towel absorbed water and the fans blowing air through the towel thus helped increase the humidity. Air exchange with outside atmosphere was kept at a minimum by closing all vents.

The growth chamber was set to operate continuously at 21.1 C without lights. The growth chamber was maintained at this temperature for two days to allow the water in the trays and water bath to attain this temperature. Although the night temperature was 21.1 C, in the day the heat from both flourescent and incandescent lights raised the temperature to 26.7 C. Temperature recordings were taken using two Tempscribe temperature recorders that were utilized in the greenhouse study. The two mercury thermometers, the same two utilized in the greenhouse study, rested on the bench and were used to check temperature readings on the recorders. In addition a Bendix Hygrothermograph was placed in the center of the bench to record both temperature and relative humidity.

Light readings were taken utilizing a quantum sensor (Li-Cor Quantum/Radiometer/Photometer/ Model Li-185A) at plant level, 91.4 cm below both incandescent and flourescent lights. A range of 1.60 to 1.65 uE m^{-2} sec^{-1} was recorded. The growth chamber was equipped with 30 flourescent and 12 incandescent lights. Even numbered flourescent lights were set to turn on first and the remaining odd numbered flourescent lights were set to turn on ½ hr later. All of the incandescent

lights were set to turn on 1 hr after the even numbered flourescent lights came on. Lights turned off in the same order.

The inoculum used in the growth chamber study was initiated and maintained in the growth chamber. Diseased plants received a minimum of 15 hr of artificial light. Systemically infected leaf pieces were then obtained and utilized in the artificial inoculation chamber. The artificial inoculation chamber was prepared similar to that in the greenhouse study. After the last tray had been prepared the lights were all manually turned off. The electric time switch was set to turn on the pump and humidifier after six hours and to operate them continuously for five hours. Twenty-four hours after the lights had been turned off, the water from each tray was siphoned out and the lights were set to resume their normal cycle. Flats were kept 1-2 days in the growth chamber before being transferred to the greenhouse. Seedlings were observed for 21 days and those showing symptoms of SDM were immediately removed to prevent adjacent seedlings from the possibility of being infected. Symptoms were recorded as either systemic infection or local lesions.

Screening Nurseries

Results of the conidial inoculation technique must correlate with results of field infection to oospores. The purpose of this inoculation technique is to give an indication of how the inoculated genotypes will respond to natural field inoculum. The reaction of the genotypes to field inoculum was indicated from field evaluations. A selected number of genotypes were screened in the summer of 1979 at Berclair, Texas. The following summer a larger number of genotypes were screened at Beeville, Texas. Both of these locations are utilized by Texas A&M

University as disease nurseries. The summer of 1980 was unusually dry and hot. Consequently the level of infection was very low and the data obtained should not be considered a reliable indicator of the field reaction of these genotypes to SDM. The number of plants showing symptoms of SDM for each entry were counted and recorded along with the number of plants per row.

CHAPTER IV

RESULTS AND DISCUSSION

It was intended to pattern a SDM artificial inoculation chamber after that designed by Craig (9). The artificial inoculation chamber was duplicated in principle but not in design. After obtaining seemingly successful greenhouse inoculations, ratings for similar genotypes were compared with those obtained by Craig. Data in Table I demonstrates that ratings were generally similar in percentage of SDM for similar genotypes. Similar ratings imply similar results from artificial inoculation methods, although the Texas A&M University data were obtained from a growth chamber with specific temperatures while the Oklahoma State University data were obtained from a greenhouse study with short night fluctuations. In addition, some of the ratings obtained were generally similar to those ratings reported in the literature for the same genotypes. For example, it is well known that TAM 430 is a highly resistant genotype and that Redlan and Wheatland are two susceptible genotypes to SDM. The reactions of these genotypes to SDM were corroborated by both TAMU and OSU inoculation data. It was considered that the artificial inoculation technique could be relied upon to determine the reaction of different genotypes to SDM. Genotypes from a heterogeneous population of Oklahoma State University sorghum germplasm resistant to conidial inoculations could now be identified with this technique, although genotypes resistant to field inoculum may not always

TABLE I

COMPARISON OF TEXAS A&M UNIVERSITY GROWTH CHAMBER AND OKLAHOMA STATE UNIVERSITY GREENHOUSE ARTIFICIAL CONIDIAL INOCULATIONS OF SELECTED SORGHUM GENOTYPES[1/]

Entry	Pedigree	TAMU[2]	OSU
1	IS 1143c	55/71	8/52
2	IS 2816c	0/67	0/90
3	IS 12610c	64/77	21/82
4	IS 12664c	52/50	35/72
5	IS 12683c	29/87	11/73
6	TAM 428	66/63	13/60
7	TAM 430	0/62	0/90
8	ROKY 34	3/67	4/73
9	ROKY 76	51/62	24/88
10	ROKY 78	1/79	0/60
11	Redlan	31/70	28/78
12	Wheatland	32/73	19/53
13	Redlan X Wiley (h_2)	68/118	40/88
14	RWD 3 X Weskan (bm_1)	50/45	53/85
15	Wheatland X Long Glume-33313	45/62	19/59
16	Cy 11-Korgi X Darset-Kaura	66/36	28/51
17	Cy 11-Korgi X Darset-Kaura	74/39	17/53
18	A Redlan X Cutchii-112112	46/59	16/49
19	A Redlan-ROKY 8 X Cy Wheatland-WBH	0/67	0/60
20	A Redlan X ROKY 34-1212212	2/70	6/67

[1/] Percent SDM/total number of plants evaluated for symptoms of SDM up to 21 days after inoculation.

[2/] Data provided by Dr. J. Craig, Dept. of Plant Sciences, Texas A&M University.

be identified with this technique.

The artificial inoculation apparatus was designed and used in both a greenhouse and a growth chamber study. In Table II ratings from the greenhouse study of 65 lines and hybrids are reported. The 20 OSU genotypes reported in Table I are included in Table II. A wide range of reactions to SDM from complete absence of infection as in IS 2816c, TAM 430, ROKY 78, and Tx 624 to a high degree of infection as in IS 12664c, B bm_1, and B bm_5 was observed. The apparatus utilized in the greenhouse study operated under less specific conditions than when used in the growth chamber study. The temperatures recorded were variable but still fell within an acceptable range for sporulation and germination of conidia.

Data in Table II show the growth chamber study ratings for 133 lines, hybrids, F_3, and F_4 segretating lines. As in Table II, a wide range of infection was observed. The range in Table III is wider than in Table II indicating either that genotypes with a greater degree of susceptibility were screened or that the growth chamber with inoculation conditions more specifically controlled increased the severity of infection. Several selections of crosses of susceptible X resistant genotypes were inoculated and evaluated. In comparing F_3 selections of crosses having a similar resistant male parent we note from Table III that 11 of 15 selections from Wheatland X IS 2816c had 20 percent or less infection while only 13 of 19 selections from Redlan X IS 2816c had 20 percent or less infection. The range of infection for the F_3 selections from Wheatland X IS 2816c was 0-39 percent while for Redlan X IS 2816c it was 3-43 percent. In the greenhouse study Wheatland had a 19 percent incidence of infection while Redlan had a 28 percent incidence of

TABLE II

SORGHUM DOWNY MILDEW RATINGS FOR SELECTED SORGHUM GENOTYPES FROM GREENHOUSE ARTIFICIAL CONIDIAL INOCULATIONS

Entry	Pedigree	Total Plants 1/			% SDM
		Inoc	Eval	Infec	Infection
1	IS 1143c	90	52	4	8
2	IS 2579c	90	74	25	34
3	IS 2801c	90	79	8	10
4	IS 2816c	90	90	0	0
5	IS 3063c	90	84	1	1
6	IS 3579c	90	83	1	1
7	IS 7384c	90	80	3	4
8	IS 12569c	90	86	3	4
9	IS 12610c	90	82	17	21
10	IS 12664c	90	72	25	35
11	IS 12666c	90	82	4	5
12	IS 12683c	90	73	8	11
13	IS 12684c	90	89	16	18
14	SC 108	90	90	10	11
15	SC 112	90	73	9	12
16	SC 120	90	84	3	4
17	SC 173	90	85	23	27
18	SC 175	90	83	6	7
19	SC 239	90	80	3	4
20	SC 414	90	90	6	7
21	TAM 428	90	60	8	13
22	TAM 430	90	90	0	0
23	TAM 2566	90	81	2	2
24	Tx 09	90	78	17	22
25	Tx 622	90	84	1	1
26	Tx 623	90	90	1	1
27	Tx 624	90	90	0	0
28	932127	90	90	9	10
29	945062	90	85	3	4
30	954114	90	74	4	5
31	ROKY 34	90	73	3	4
32	ROKY 47	90	85	14	17
33	ROKY 76	90	88	21	24
34	ROKY 78	90	60	0	0
35	A bm_1	90	83	13	16
36	B bm_1	90	38	15	39
37	B bm_3	90	89	9	10
38	B bm_5	90	51	20	40
39	A Redlan	90	90	17	19
40	B Redlan	90	90	9	10
41	A Wheatland	90	90	15	17
42	B Wheatland	90	90	11	12
43	A Dwarf Redlan	90	90	10	10

TABLE II (Continued)

Entry	Pedigree	Total Plants[1/] Inoc	Eval	Infec	% SDM Infection
44	B Dwarf Redlan	90	90	16	18
45	Redlan	90	78	22	28
46	Wheatland	90	53	10	19
47	Redlan X Wiley (h_2)	90	88	35	40
48	RWD 3 X Weskan (bm_1)	90	85	45	53
49	Wheatland X Long Glume-33313	90	59	11	19
50	Cy 11-Korgi X Darset-Kaura	90	51	14	28
51	Cy 11-Korgi X Darset-Kaura	90	53	9	17
52	A Redlan X Cutchii-112112	90	49	8	16
53	A Redlan-Y 8 X Cy Wheat-WBH	90	60	0	0
54	A Redlan X ROKY 34-1212212	90	67	4	6
55	Wheatland X Tx 2536	90	90	7	8
56	Wheatland X TAM 428	90	90	1	1
57	TAM 430 X TAM 428	90	90	0	0
58	ACCO GR-108	90	72	3	4
59	ACCO R-109A	90	80	22	27
60	ACCO R-1014	90	90	0	0
61	ACCO R-1019	90	86	3	3
62	ACCO R-1029A	90	77	15	20
63	Asgrow Dorado M	90	72	9	13
64	Conlee Tophand	90	83	4	5
65	Frontier 412R	90	68	0	0

[1/] Plants inoc - inoculated, eval - evaluated for symptoms of SDM up to 21 days after inoculation, infec - infected.

TABLE III

SORGHUM DOWNY MILDEW RATINGS FOR SELECTED SORGHUM GENOTYPES FROM GROWTH CHAMBER ARTIFICIAL CONIDIAL INOCULATIONS

Entry	Gen[1]	Pedigree	Total Plants[2] Inoc	Eval	Infec	% SDM Infection
1	F+	IS 2801c	90	86	4	5
2	F_3	A Redlan X IS 2801c-1-1	90	83	0	0
3	F_3	-1-4	90	69	23	33
4	F_3	-1-7	90	77	15	19
5	F_3	A Wheatland X IS3063c-1-6	90	86	15	17
6	F_3	-1-7	90	80	19	24
7	F_3	A Wheatland X IS 2816c-1-1	90	78	12	15
8	F_3	-1-2	90	86	26	30
9	F_3	-1-3	90	90	11	12
10	F_3	-1-4	90	84	12	14
11	F_3	-1-5	90	72	28	39
12	F_3	-1-6	90	83	19	23
13	F_3	-1-7	90	88	3	3
14	F_3	-1-8	90	88	3	3
15	F_3	-1-9	90	82	9	11
16	F_3	-1-10	90	83	6	7
17	F_3	-1-11	90	90	10	11
18	F_3	-1-12	90	62	20	32
19	F_3	-1-13	90	83	11	13
20	F_3	-1-14	90	90	10	11
21	F_3	-1-16	90	85	0	0
22	F+	IS 2816c	90	88	3	3
23	F_3	A Redlan X IS 2816c-1-1	90	77	19	25
24	F_3	-1-2	90	89	3	3
25	F_3	-1-3	90	90	13	14
26	F_3	-1-4	90	83	15	18
27	F_3	-1-5	90	80	12	15
28	F_3	-1-6	90	84	4	5
29	F_3	-1-7	90	64	12	19
30	F_3	-1-8	90	73	20	27
31	F_3	-1-9	90	72	31	43
32	F_3	-1-10	90	77	9	12
33	F_3	-1-11	90	85	24	28
34	F_3	-1-12	90	83	5	6
35	F_3	-1-13	90	77	20	26
36	F_3	-1-14	90	68	18	26
37	F_3	-1-15	90	78	7	9
38	F_3	-1-16	90	77	8	10
39	F_3	-1-17	90	90	9	10
40	F_3	-1-18	90	86	12	14
41	F_3	-1-19	90	80	15	19
42	F_3	A Wheatland X IS 12610c-1-1	90	76	21	28
43	F_3	-1-2	90	75	20	27

TABLE III (Continued)

Entry	Gen[1]	Pedigree	Total Plants[2] Inoc	Eval	Infec	% SDM Infection
44	F_3	A Wheatland X IS 12610c-1-2	90	80	10	13
45	F_3	-1-3	90	80	10	13
46	F_3	-1-5	90	83	16	19
47	F_3	-1-6	90	85	5	6
48	F_3	-1-7	90	66	11	17
49	F_3	-1-8	90	81	6	7
50	F_3	-1-9	90	73	14	19
51	F_3	-1-10	90	85	3	4
52	F_3	-1-11	90	90	7	8
53	F_3	-1-12	90	72	17	24
54	F_3	-1-13	90	86	8	9
55	F_3	-1-14	90	75	20	27
56	F_3	-1-15	90	87	13	15
57	F_3	-1-16	90	78	5	6
58	F+	IS 12610c	90	90	18	20
59	F_4	A Wheatland X IS 2801c-1-1-1	90	80	23	29
60	F_4	-1-2-1	90	75	13	17
61	F_4	-1-3-1	90	68	22	32
62	F_4	-1-3-2	90	72	26	36
63	F_4	-1-7-1	90	80	12	15
64	F_4	-1-7-2	90	90	11	12
65	F_4	-1-7-3	90	85	18	21
66	F_4	-1-7-4	90	83	17	20
67	F_4	-1-9-1	90	80	24	30
68	F_4	-1-9-2	90	83	21	25
69	F_4	-1-12-1	90	78	23	29
70	F_4	-1-12-2	90	72	38	53
71	F_4	A Redlan X IS 2801c-1-1-1	90	90	14	16
72	F_4	-1-2-1	90	63	20	32
73	F_4	-1-4-1	90	71	33	46
74	F_4	-1-4-2	90	62	17	27
75	F_4	-1-6-1	90	65	10	15
76	F_4	-1-9-1	90	82	56	68
77	F_4	-1-10-1	90	85	13	15
78	F_4	-1-12-1	90	81	35	43
79	F_4	-1-12-3	90	90	17	19
80	F_4	-1-13-1	90	62	13	21
81	F+	B Wheatland	90	88	11	13
82	F_4	A Wheatland X IS 2816c-1-1-1	90	90	6	7
83	F_4	-1-1-2	90	90	4	4
84	F_4	-1-1-3	90	75	17	23
85	F_4	-1-1-4	90	80	18	23
86	F_4	-1-2-1	90	90	5	6
87	F_4	-1-3-1	90	90	5	6
88	F_4	-1-3-2	90	88	4	5
89	F_4	-1-6-1	90	84	11	13
90	F_4	-1-7-1	90	77	14	18

TABLE III (Continued)

Entry	Gen[1]	Pedigree	Total Plants[2] Inoc	Eval	Infec	% SDM Infection
91	F	A Wheatland X IS 2816c-1-7-2	90	63	0	0
92	F	-1-9-1	90	81	0	0
93	F	-1-10-1	90	78	8	10
94	F	-1-11-1	90	78	12	15
95	F	-1-13-1	90	80	0	0
96	F	-1-14-1	90	81	28	35
97	F	-1-14-2	90	88	7	8
98	F	-1-14-3	90	79	21	27
99	F	-1-15-1	90	76	29	38
100	F	-1-15-2	90	90	14	16
101	F	-1-15-3	90	72	22	31
102	F	-1-15-4	90	75	20	27
103	F	-1-16-1	90	68	5	7
104	F	-1-17-1	90	83	16	19
105	F	-1-17-2	90	90	16	18
106	F	-1-17-3	90	82	9	11
107	F	-1-19-1	90	85	0	0
108	F	-1-19-2	90	69	0	0
109	F	-1-20-1	90	77	14	18
110	F	-1-20-2	90	90	0	10
111	F+	IS 1143c	60	43	6	14
112	F+	IS 2579c	60	53	6	11
113	F+	IS 3579c	60	56	13	23
114	F+	IS 12664c	60	60	18	30
115	F+	IS 12666c	60	54	1	2
116	F+	IS 12683c	60	55	9	16
117	F+	IS 12684c	60	57	8	14
118	F+	SC 108	60	52	1	2
119	F+	SC 112	60	58	8	14
120	F+	SC 120	60	53	0	0
121	F+	SC 173	60	57	20	35
122	F+	SC 175	60	46	0	0
123	F+	SC 239	60	56	15	27
124	F+	SC 414	60	48	10	21
125	F+	TAM 428	60	60	4	7
126	F+	TAM 430	60	58	5	9
127	F+	Tx 622	60	51	1	2
128	F+	Tx 623	60	53	2	4
129	F+	Tx 624	60	49	0	0
130	F+	Redlan	60	47	19	40
131	F_1	Wheatland X Tx 2536	60	57	15	26
132	F_1	Wheatland X TAM 428	60	60	7	12
133	F_1	TAM 430 X TAM 428	60	49	2	4

[1] Filial generation; + = Pure line.
[2] Plants inoc - inoculated, eval- evaluated for symptoms of SDM up to 21 days after inoculation, infec - infected.

infection. In the growth chamber study Wheatland had a 13 percent incidence of infection at 40%. Wheatland appeared to be a slighter better parent for developing resistance to SDM.

In comparing selections from crosses having a common susceptible female parent but different resistant male parents as in Wheatland X IS 2816c and Wheatland X IS 12610c we can observe from Table III that in selections from Wheatland X IS 2816c, 11 of 15 selections had 20 percent or less infection while in selections from Wheatland X IS 12610c, 11 of 16 selections had 20 percent or less infection. The range of infection for the F_3 selections from Wheatland X IS 2816c was 0-39 percent while for those selections from Wheatland X IS 12610c it was 4-47 percent. This may indicate that IS 2816c is a better male parent for contributing resistance in hybrid combinations with Wheatland than is IS 12610c. In the greenhouse study IS 2816c had a 0 percent infection while IS 12610c had 21 percent infection. In the growth chamber study IS 2816c had 3 percent infection while IS 12610c had 20 percent infection. From both these artificial conidial inoculation studies IS 2816c appears to possess a higher degree of resistance than does IS 12610c.

In general, when comparing ratings between greenhouse and growth chamber inoculations, growth chamber ratings were more severe. In Table IV only 11 of 26 entries have greenhouse ratings greater than growth chamber ratings. The growth chamber temperature during inoculations was a constant 21.1 C. The greenhouse temperature fluctuated and these temperature fluctuations may explain the difference in disease ratings compared to those obtained from the growth chamber. Nevertheless, one would not expect to obtain identical ratings even when all research variables are constant. One can expect that since the greenhouse

TABLE IV

COMPARISON OF GREENHOUSE AND GROWTH CHAMBER ARTIFICIAL CONIDIAL INOCULATIONS[1/]

Entry	Pedigree	Greenhouse[2/]	Growth Chamber[3/]
1	IS 1143c	8/52	14/43
2	IS 2579c	34/74	11/53
3	IS 2801c	10/79	5/86[2/]
4	IS 2816c	0/90	3/88[2/]
5	IS 3579c	1/83	23/56
6	IS 12610c	21/82	20/90[2/]
7	IS 12664c	35/72	30/60
8	IS 12666c	5/82	2/54
9	IS 12683c	11/73	16/55
10	IS 12684c	18/89	14/57
11	SC 108	11/90	2/52
12	SC 112	12/73	14/58
13	SC 120	4/84	0/53
14	SC 173	27/85	35/57
15	SC 175	7/83	0/46
16	SC 239	4/80	27/56
17	SC 414	7/90	21/48
18	TAM 428	13/60	7/60
19	TAM 430	0/90	9/58
20	Tx 622	1/84	2/51
21	Tx 623	1/90	4/53
22	Tx 624	0/90	0/49
23	Redlan	28/78	40/47
24	Wheatland	19/53	13/88[2/]
25	Wheatland X Tx 2536	8/90	26/57
26	TAM 430 X TAM 428	0/90	4/49

[1/] Percent SDM/total number of plants evaluated for symptoms of SDM up to 21 days after inoculation.
[2/] Total of 90 plants evaluated.
[3/] Total of 60 plants evaluated.

fluctuating night temperatures were still within the optimum temperature range for sporulation and for germination of conidia that subsequent infection should be unaffected and that disease ratings obtained should be reliable. This statement is supported by data in Table I where the greenhouse ratings were comparable to those disease ratings obtained by Craig (9). It is believed that genotype rating differences are influenced more by the degree of susceptibility than by the effects of fluctuating temperatures on sporulation, germination of conidia, and subsequent infection.

Field ratings to SDM were obtained from two different screening nurseries in South Texas in 1979 and 1980. Ratings in Table V indicate the disease count of 20 genotypes in Berclair, Texas in 1979. The stand count is variable for unknown reasons. The disease and stand counts were reported by the Texas A&M University, Department of Plant Sciences personnel who were experienced in SDM disease ratings. Regardless of the stand count, genotypes having a relatively high number of diseased plants in either or both replications were considered to be susceptible to field inoculum of SDM. IS 1143c, ROKY 76, and RWD 3 X Weskan (bm_1) showed resistant ratings in one replication but in the other replication indicated a strong degree of susceptibility to field inoculum. Conversely IS 12683c, TAM 430, ROKY 76, and A Redlan 34-1212212 showed resistant reactions to field inoculum of SDM in both replications. The remaining genotypes in Table V were considered susceptible or resistant in both replications.

Redlan and Wheatland, two known susceptible genotypes, gave moderately resistant reactions. The information provided in this table was used to compare genotypes to their respective artificial conidial

TABLE V

INCIDENCE OF SORGHUM DOWNY MILDEW IN OKLAHOMA STATE UNIVERSITY SORGHUM GERMPLASM AT BERCLAIR, TEXAS IN 1979

Entry	Pedigree	Rep I[1/]	Rep II[2/]
1	IS 1143c	1/15	10/15
2	IS 2816c	0/3	0/15
3	IS 12610c	10/38	8/43
4	IS 12664c	0/22	4/20
5	IS 12683c	2/10	1/29
6	TAM 428	0/17	[2/]
7	TAM 430	0/18	0/32
8	ROKY 34	1/25	0/48
9	ROKY 76	26/52	7/45
10	ROKY 78	0/32	1/44
11	Redlan	3/45	0/66
12	Wheatland	5/48	1/43
13	Redlan X Wiley (h_2)	12/20	10/35
14	RWD 3 X Weskan (bm_1)	7/12	2/26
15	Wheatland X Long Glume-33313	6/34	7/43
16	Cy 11-Korgi X Darset-Kaura	2/12	7/27
17	Cy 11-Korgi X Darset-Kaura	2/7	3/25
18	A Redlan X Cutchii-112112	1/21	5/27
19	A Redlan-ROKY 8 X Cy Wheatland-WBH	6/48	10/32
20	A Redlan X ROKY 34-1212212	2/43	2/40

[1/] Disease count/stand count, data provided by Texas A&M University.
[2/] No data recorded.

inoculation ratings. The disease nursery at Berclair, Texas is one that is used annually by the Department of Plant Sciences of Texas A&M University for field screening.

Table VI provides similar data as Table V but for different genotypes in a different nursery. The disease nursery at Beeville, Texas is one used annually as well by the Department of Plant Sciences of Texas A&M University. Both the stand and disease counts were taken by the author. It should be pointed out here that the summer of 1980 was unusually hot and dry. This may explain the extremely low ratings of SDM among genotypes reported from this nursery. The disease incidence was visibly greater in 1979 than in 1980. Unfortunately, the entire data reported should be viewed cautiously and not conisdered a reliable indicator of the genotypes reaction to SDM.

Seven distinct genotypes were screened in both disease nurseries. Table VII provides their disease ratings and compares the field ratings to greenhouse and growth chamber inoculations. TAM 428 which showed a susceptible reaction at Texas A&M University and a moderately resistant reaction in the greenhouse study was disease free in 1979 at Berclair, and in 1980 at Beeville, Texas. A Redlan-ROKY 8 X Cy Wheatland-WBH showed a resistant reaction to artificial screening both at Texas A&M University and in the greenhouse study but was susceptible to field inoculum in 1979 at Berclair, Texas. Both TAM 428 and A Redlan-ROKY 8 X Cy Wheatland-WBH are illustrations of a known drawback of the artificial conidial inoculation technique. Consequently, with certain genotypes resistance to conidial inoculations may not always indicate resistance to field inoculum. All other entries in Table VII correlate artificial conidial inoculations with field reactions to SDM. As

TABLE VI

INCIDENCE OF SORGHUM DOWNY MILDEW IN OKLAHOMA STATE UNIVERSITY SORGHUM GERMPLASM AT BEEVILLE, TEXAS IN 1980

Entry	Gen[1]	Pedigree	Rep I[2]	Rep II[2]
1	F+	TAM 430	0/40	0/25
2	F_4	A Wheatland X TAM 430-1-1-1	0/40	1/40
3	F_4	-1-2-1	0/40	0/20
4	F_4	-1-2-2	0/40	0/40
5	F_4	-1-3-1	0/40	0/40
6	F_4	-1-5-1	0/40	0/40
7	F_4	-1-5-2	0/40	0/40
8	F_4	-1-8-1	0/40	0/40
9	F_4	A Tx 2751 X TAM 430-1-1-3	0/40	0/40
10	F_4	-1-3-1	2/40	0/7
11	F_4	-1-4-1	0/40	0/ [3]
12	F_4	A Redlan X TAM 430-1-1-1	0/40	0/ [3]
13	F_4	-1-1-2	0/40	0/ [3]
14	F_4	-1-3-1	0/40	2/ [3]
15	F_4	-1-3-2	0/40	0/ [3]
16	F_4	-1-5-1	0/40	0/ [3]
17	F_4	-1-5-2	0/40	0/ [3]
18	F_4	-1-7-1	1/40	0/ [3]
19	F_4	-1-8-1	0/40	0/ [3]
20	F_4	-1-10-1	0/40	0/ [3]
21	F_4	-1-11-1	0/40	0/ [3]
22	F_4	-1-13-1	0/40	0/ [3]
23	F_4	-1-14-1	2/40	0/ [3]
24	F_4	-1-15-1	0/40	0/ [3]
25	F_4	-1-17-1	0/40	0/ [3]
26	F_4	A Tx 621 X TAM 430-1-1-1	0/40	0/ [3]
27	F_4	-1-1-2	0/40	0/ [3]
28	F_4	-1-6-1	0/40	0/ [3]
29	F+	Tx 622	0/40	0/ [3]
30	F_4	A Tx 622 X TAM 430-1-1-1	0/30	0/ [3]
31	F_4	-1-2-1	0/30	0/ [4]
32	F_4	-1-2-2	0/30	0/ [3]
33	F_4	-1-4-1	0/40	0/ [3]
34	F_4	-1-4-2	0/40	0/ [3]
35	F_4	-1-5-1	0/40	0/ [3]
36	F_4	-1-5-2	0/40	0/ [3]
37	F_4	-1-6-1	0/40	0/ [3]
38	F_4	-1-7-1	0/40	0/ [3]
39	F_4	-1-8-1	0/40	0/ [3]
40	F_4	-1-8-2	0/40	0/ [3]
41	F_4	A 7502 X TAM 430-1-1-1	0/40	0/ [3]
42	F_4	-1-1-2	0/30	0/ [3]
43	F_4	-1-3-1	0/30	0/ [4]
44	F_4	-1-4-1	0/40	0/

TABLE VI (Continued)

Entry	Gen[1]	Pedigree	Rep I[2]	Rep II[2]
45	F_4	A 7502 X TAM 430-1-5-1	0/40	0/ 3/
46	F_4	-1-6-1	0/40	0/ 3/
47	F_4	-1-7-1	0/40	0/ 3/
48	F_4	-1-8-1	0/40	0/ 3/
49	F_4	-1-9-1	0/40	0/ 3/
50	F_4	-1-9-2	0/40	0/ 3/
51	F_4	-1-10-1	0/40	0/ 3/
52	F_4	-1-12-1	0/40	0/ 3/
53	F_4	-1-13-1	0/25	0/ 4/
54	F_4	TAM 428	0/40	0/ 3/
55	F_4	ARK X TAM 428-1-2-1	0/40	0/ 3/
56	F_4	A Redlan X TAM 428-1-4-1	0/40	0/ 3/
57	F_4	-1-4-2	1/40	0/ 3/
58	F_4	-1-5-1	0/40	0/ 3/
59	F_4	-1-7-1	0/40	0/ 3/
60	F_4	-1-8-1	0/40	0/ 3/
61	F_4	-1-9-1	0/40	0/ 3/
62	F_4	-1-10-1	0/40	0/ 3/
63	F_6	A Redlan X IS 2801c-1-4-2-1-1	0/40	0/ 3/
64	F_6	-1-4-2-1-2	0/40	0/ 3/
65	F_6	-1-12-2-2-1	0/40	0/ 3/
66	F_6	-1-12-2-2-2	1/40	0/ 3/
67	F_6	-1-13-1-2-1	0/40	0/ 3/
68	F_6	-1-13-1-2-2	1/40	0/ 3/
69	F_5	A Wheatland X IS 2816c-1-5-1-1	0/40	-- 5/
70	F_5	-1-5-2-1	0/25	-- 5/
71	F_5	-1-5-2-2	0/40	0/ 3/
72	F_5	-1-5-2-3	0/40	0/ 3/
73	F_5	-1-6-2-1	0/10	0/ 3/
74	F_5	-1-6-2-2	0/15	0/ 3/
75	F_5	-1-6-4-1	1/20	0/ 3/
76	F_5	-1-6-4-2	0/40	0/ 3/
77	F_5	-1-6-6-1	0/20	0/ 3/
78	F_5	-1-6-6-2	0/40	0/ 3/
79	F_5	-1-7-1-1	0/15	0/ 3/
80	F_5	-1-7-1-2	1/40	0/ 3/
81	F_5	-1-7-1-3	0/20	0/ 3/
82	F_5	-1-12-2-1	0/25	0/ 3/
83	F_5	-1-12-2-2	0/40	0/ 3/
84	F_5	-1-12-2-3	0/40	0/ 3/
85	F_5	-1-14-7-1	0/40	0/ 3/
86	F_6	-1-1-2-2-1	0/15	0/ 3/
87	F_6	-1-1-2-2-2	0/20	0/ 3/
88	F_6	-1-1-2-4-1	0/25	0/ 3/
89	F_6	-1-1-2-4-2	0/40	2/ 3/
90	F_6	-1-1-3-3-1	0/40	0/ 3/
91	F_6	-1-8-1-1-1	0/40	0/

TABLE VI (Continued)

Entry	Gen[1]	Pedigree	Rep I[2]	Rep II[2]
92	F_6	A Wheatland X IS 2816c-1-8-1-1-2	0/40	0/ [3]
93	F_6	-1-13-1-1-1	0/40	0/ [3]
94	F_6	-1-13-1-1-2	0/40	0/ [3]
95	F_6	-1-14-1-1-1	0/40	0/ [3]
96	F_6	-1-14-1-1-2	1/40	0/ [3]
97	F_6	-1-14-1-2-1	0/40	0/ [3]
98	F_6	-1-14-1-2-2	0/40	0/ [3]
99	F_6	-1-14-1-2-3	0/40	0/ [3]
100	F_6	-1-14-2-2-1	3/40	0/ [3]
101	F_6	-1-14-2-2-2	0/40	0/ [3]
102	F_6	-1-14-2-2-3	1/40	0/ [3]
103	F_6	-1-14-3-2-1	0/40	0/ [4]
104	F_6	-1-14-3-2-2	0/40	0/ [3]
105	F_6	-1-17-2-2-1	0/20	0/ [3]
106	F_6	-1-17-2-2-2	0/15	0/ [3]
107	F_6	-1-17-2-2-3	0/25	0/ [3]
108	F_6	-1-19-1-1-1	0/20	0/ [3]
109	F_6	-1-19-1-2-1	0/25	0/ [3]
110	F_6	-1-19-1-2-2	0/25	0/ [3]
111	F_6	-1-19-1-2-3	0/30	0/ [5]
112	F+	ROKY 34	0/40	-- [3]
113	F+	ROKY 78	0/25	0/ [3]
114	F+	B bm_3	1/40	0/ [3]
115	F+	RWD 3 X Weskan (bm_1)	0/40	0/ [3]
116	F+	Cy 11-Korgi X Darset-Kaura	0/20	0/ [3]
117	F+	Cy 11-Korgi X Darset-Kaura	1/40	0/ [3]
118	F+	B Tr X S. splendidium-72112	0/40	0/ [3]
119	F+	A Redlan-ROKY 8 X Cy Wheatland-WBH	0/15	0/ [3]

[1] Filial generation; + = Pure line.
[2] Disease count/stand count; data provided by author.
[3] Stand count not recorded, but in the range of 25 to 40.
[4] Few plants.
[5] No plants.

TABLE VII

COMPARISON OF TEXAS A&M UNIVERSITY GROWTH CHAMBER AND OKLAHOMA STATE UNIVERSITY GREENHOUSE ARTIFICIAL CONIDIAL INOCULATIONS WITH 1979 BERCLAIR, AND 1980 BEEVILLE, TEXAS FIELD RATINGS FOR RESISTANCE TO SORGHUM DOWNY MILDEW

Entry	Pedigree	Artificial Conidial Inoculation[1]		Natural Field Infection	
		TAMU	OSU	1979 Berclair[2]	1980 Beeville[3]
1	TAM 428	66/63	13/60	0/17	0/40
2	TAM 430	0/62	0/90	0/50	0/65
3	ROKY 34	3/67	4/73	1/73	0/40
4	ROKY 78	1/79	0/60	1/76	0/25
5	RWD 3 X Weskan (bm_1)	50/45	53/85	9/38	0/40
6	Cy 11-Korgi X Darset-Kaura	66/36	28/51	9/39	0/20
7	Cy 11-Korgi X Darset-Kaura	74/39	17/53	5/32	1/40
8	A Redlan-ROKY 8 X Cy Wheatland-WBH	0/67	0/60	16/80	0/15

[1] % SDM/total number of plants evaluated for symptoms of SDM up to 21 days after inoculation.
[2] Disease count/stand count for sum of two replications.
[3] Disease count/stand count for one replication.

indicated above the 1980 disease nursery ratings do not complement the 1979 ratings. It may be considered that seven different genotypes is not a sufficiently large number of genotypes to compare ratings from two consecutive years as two different nurseries with conidial inoculations.

The purpose of SDM disease nurseries like those at Berclair, and Beeville, Texas are to obtain natural genotypic reactions to field inoculum of SDM. This natural reaction is obtained after repeated exposure of the genotype to field inoculum from time to time and from place to place. The genotypes screened at Berclair, Texas were replicated while those at Beeville, Texas were not. Generally, one does not doubt a susceptible reaction of a genotype from a single screening in a disease nursery, but one could question a resistant reaction. Resistant genotypes and escapes are indistinguishable and phenotypically identical. Thus, the authenticity of the resistant field ratings from Table V and VI can be questioned. More consistent data can be obtained from the artificial inoculation chamber, if it is carried out properly. In general, artificial inoculation data are expected to represent field reactions but an exact correlation is not possible. The correlation improves when screening of genotypes is done at the one to two leaf stage.

Data in Table VIII compares greenhouse and growth chamber conidial inoculations with 1979 and 1980 field ratings. Not all genotypes were screened in both greenhouse or growth chamber studies and neither were all genotypes screened in Berclair or Beeville, Texas. The purpose of Table VIII is to demonstrate that reactions to artificial conidial inoculations agree with reactions to field inoculum of SDM. This agreement is not clearly visible in the majority of genotypes screened. The

TABLE VIII

COMPARISON OF BEEVILLE AND BERCLAIR, TEXAS FIELD RATINGS FOR RESISTANCE TO SORGHUM DOWNY MILDEW AND REACTIONS TO ARTIFICIAL CONIDIAL INOCULATIONS AMONG SELECTED OKLAHOMA STATE UNIVERSITY GENOTYPES

Entry	Pedigree	Greenhouse[1]	Growth Chamber[1]	1979[2] Berclair, Texas Rep I	1979[2] Berclair, Texas Rep II	1980[2] Beeville, Texas Rep I	1980[2] Beeville, Texas Rep II
1	IS 1143c	8/52	14/43	1/15	10/15		
2	IS 2816c	0/90	3/88	0/3	0/15		
3	IS 12610c	21/82	20/90	10/38	8/43		
4	IS 12664c	35/72	30/60	0/22	4/22		
5	IS 12683c	11/73	16/55	2/10	1/29		
6	TAM 428	13/60	7/60	0/17	-- [3]	0/40	0/ [4]
7	TAM 430	0/90	9/58	0/18	0/32	0/40	0/25
8	Tx 622	1/84				0/40	0/ [4]
9	ROKY 34	4/73		1/25	0/48	0/40	
10	ROKY 76	24/88		26/52	7/45		
11	ROKY 78	0/60		0/32	1/44	0/25	0/ [4]
12	B bm_3	10/89				1/40	0/ [4]
13	Redlan	28/78	40/45	3/45	0/66		
14	Wheatland	19/53	13/88	5/48	1/43		
15	Redlan X Wiley (h_1)	40/88		12/20	10/35		
16	RWD X Weskan (bm_3)	53/85		7/12	2/26	0/40	0/ [4]
17	Wheatland X Long Glume-33313	19/59	6/34	7/43			
18	Cy 11-Korgi X Darset-Kaura	28/51		2/12	7/27	0/20	0/ [4]
19	Cy 11-Korgi X Darset-Kaura	17/53		2/7	3/25	1/40	0/ [4]
20	A Redlan X Cutchii-112112	16/49		1/21	5/27		
21	A Redlan-ROKY 8 X Cy Wheatland-WBH	0/60		6/48	10/32	0/40	0/ [4]
22	A Redlan-ROKY 34-1212212	6/67		2/43	2/40		

1/% SDM/total number of plants evaluated up to 21 days after inoculation.
2/Disease count/stand count. 3/No data recorded. 4/Stand count not recorded but in the range of 25-40.

agreement is very pronounced for IS 12610c in comparison of greenhouse or growth chamber with field reactions in Berclair, Texas. IS 2816c, ROKY 34, ROKY 76, ROKY 78, Redlan X Wiley, RWD 3 X Weskan, and Cy 11-Korgi X Darset-Kaura all show an agreement between their artificial conidial inoculation reactions and field reactions.

IS 12664c which showed a susceptible reaction to artificial conidial inoculations in both greenhouse and growth chamber studies had a resistant reaction in Replication I and a moderately resistant reaction in Replication II in Berclair, Texas. A Redlan-ROKY 8 X Cy Wheatland-WBH had 0% infection in the greenhouse but in Replication II in Berclair, Texas it had 1/3 systemically infected plants, an incidence high enough to consider this genotype susceptible. As mentioned before genotypes resistant to field inoculum may not always be identified with this technique, though this the exception and not the rule.

Data in Table IX provides an indication of the inheritance of resistance to SDM. No data were obtained from screening F_1 or F_2 in this study. Assuming all the F_3 genotypes screened originated from a single cross between a resistant and a susceptible parent, then it can be assumed from F_3 segregation data that resistance is dominant to susceptibility. Actually, the F_3 genotypes in Table IX originated from five different crosses involving four different resistant male parents and two different susceptible female parents. Data in Table IX clearly indicate that resistance is dominant to susceptibility and that from the ratio of resistant to susceptible F_3 genotypes resistance appears to be controlled by one major gene. This statement is in agreement with that reported on the mode of inheritance of resistance to SDM in the current literature.

TABLE IX

RATIO OF RESISTANT TO SUSCEPTIBLE F_3 SORGHUM GENOTYPES FROM GROWTH CHAMBER ARTIFICIAL CONIDIAL INOCULATIONS

Study	Total Number of Entries Inoculated	Resistant[1] Entries	Susceptible[2] Entries	R:S[3]
Growth Chamber	55	42	13	3.23

[1] Segregating lines with 25% or less conidial infection up to 21 days after inoculation.

[2] Segregating lines with more than 25% conidial infection up to 21 days after inoculation.

[3] Ratio of resistant to susceptible segregating lines.

In Table X F_4 selections were compared with their F_3 parents. It was of interest to find out if F_4 genotypes behaved as true resistant or susceptible lines or if they appeared to be segregating for resistance. Susceptible F_3 lines are expected to produce susceptible F_4 lines and can be identified as such with the artificial conidial inoculation technique. Susceptible F_4 lines can arise from either a susceptible F_3 line or a heterozygous F_3 line. It is important to identify heterozygous F_3 genotypes, as these will engender resistant as well as undesirable susceptible progenies. Resistant and susceptible F_4 genotypes from a single resistant F_3 genotype indicate that the F_3 genotype was apparently heterozygous for resistance to SDM. Genotypes having 25 percent or less infection were considered resistant while those selections with more than 25 percent infection were considered susceptible genotypes.

Redlan X IS 2801c-1-1-1 with 16 percent infection originated from an apparently homozygous resistant F_3 genotype having 0 percent infection. It would then be expected that the F_4 selections from Redlan X IS 2801c-1-4 with 33 percent infection would be susceptible, and indeed, two progenies had 46 and 27 percent infection.

Wheatland X IS 2816c-1-3-1, -1-3-2, -1-8-1, -1-10-1, and -1-16-1 all have relatively low infections indicating that they originated from apparently homozygous resistant F_3 genotypes. The F_3 genotypes from which the above F_4 selections were obtained had 12, 12, 3, 7, and 0 percent infection, respectively.

Wheatland X IS 2816c-1-1, -1-2, and -1-14 are clearly shown to be heterozygous from their respective F_4 selections. Wheatland X IS 2816c-1-1 with 15 percent infection is the source of two selections with

TABLE X

RELATIONSHIP OF F_3 PARENTS AND F_4 PROGENIES REACTIONS TO SDM ARTIFICIAL CONIDIAL INOCULATIONS

Cross			Generation	
Susceptible Parent	X	Resistant Parent	F_3	F_4
Redlan 40/47	X	IS 2801c 5/86	-1-1 0/83	-1-1-1 16/90
"		"	-1-4 33/69	-1-4-1 46/71
"		"	"	-1-4-2 27/62
Wheatland 13/88	X	IS 2816c 3/88	-1-1 15/78	-1-1-1 7/90
"		"	"	-1-1-2 4/90
"		"	"	-1-1-3 23/75
"		"	"	-1-1-4 23/80
"		"	-1-2 30/86	-1-2-1 6/90
"		"	-1-3 12/90	-1-3-1 6/90
"		"	"	-1-3-2 5/88
"		"	-1-6 23/83	-1-6-1 13/84
"		"	-1-7 13/79	-1-7-1 18/77
"		"	"	-1-7-2 0/63
"		"	-1-8 3/88	-1-8-1 0/81
"		"	-1-10 7/83	-1-10-1 10/78

TABLE X (Continued)

Susceptible Parent X Resistant Parent			Generation F_3	Generation F_4
Wheatland 13/88	X	IS 2816c 3/88	-1-11 11/90	-1-11-1 15/78
"		"	-1-14 11/90	-1-14-1 35/81
"		"	"	-1-14-2 8/88
"		"	"	-1-14-3 27/79
"		"	-1-16 0/85	-1-16-1 7/68

1/ % SDM/total number of plants evaluated for symptoms of SDM up to 21 days after inoculation.

relatively low infections, and two selections with relatively high infections but not high enough to classify them as susceptible genotypes. The infection percentage of these four F_4 selections derived from Wheatland X IS 2816c-1-1 are 7, 4, 23 and 23 percent. Wheatland X IS 2816c-1-2 although having 30 percent infection and therefore classified as a susceptible genotype had a selection derived from it with 6 percent infection. This indicated that although it was classified as a susceptible genotype it must have been a heterozygous genotype segregating in the F_4 generation for resistance to SDM. Wheatland X IS 2816c-1-14 with 11 percent infection engendered one F_4 selection with 8 percent infection but two susceptible F_4 selections with 27 and 35 percent infection. The remaining F_3 selections of Wheatland X IS 2816c-1-1, -1-7, and -1-11 were not as readily demonstrated to be resistant from their respective F_4 selections. The F_4 selections were either resistant or moderately resistant while the F_3 genotypes from which they were selected appeared to be resistant. An exception was of Wheatland X IS2816c-1-6 which had a relatively high infection of 23 percent but remained in the resistant percentage range of infection.

To satisfactorily determine the heterozygosity of an F_3 genotype a number of selections from the F_3 genotype should be screened. Especially, F_3 genotypes with relatively high infections but remaining in the resistant percentage range of classification require several of its F_4 selections to be screened. This may ensure proper identification of the reaction of F_3 genotype to artificial conidial inoculation. Genotypes with high infections may be concluded to be definitely susceptible and selections derived from these genotypes can be assummed to be

susceptible. Also those genotypes with very low infections may be concluded to be resistant and selections derived from these genotypes can be assumed to be resistant.

CHAPTER V

SUMMARY AND CONCLUSIONS

The purpose of this study was threefold. Primarily, it was desired to pattern an artificial conidial inoculation technique after that developed by Craig (9) at the Department of Plant Sciences, Texas A&M University for screening sorghum genotypes for resistance to SDM. In addition, the study was conducted to identify resistant sorghum genotypes from segregating sorghum populations and to get an indication of the mode of inheritance of resistance to SDM.

A SDM artificial conidial inoculation apparatus was devised to inoculate seedlings in the 1-2 leaf stage with conidia from sporulating 2-3 cm sections of systemically infected sorghum leaves. These conidia were distributed within inoculation chamber trays onto the seedlings by incoming moist cooled air from a network of air delivery hoses at 1 psi. The seedlings were observed for symptoms of SDM up to 21 days after inoculation.

Ratings from artificial conidial inoculations of selected sorghum genotypes from a greenhouse study were compared with those ratings obtained by Craig (9) and those reported in the current literature for similar genotypes. It was concluded that the ratings obtained from the artificial conidial inoculation technique in the greenhouse study were reliable. It was then considered that the inoculation technique was performing as desired. Once this objective had been accomplished

fulfilling the remaining two objectives was possible.

Both a greenhouse study and a growth chamber study were conducted to determine the reaction of several sorghum lines, hybrids, and segregating F_3 and F_4 genotypes to artificial conidial inoculations of SDM. Some F_3 genotypes were found to be apparently segregating for resistance to SDM based on the reaction of F_4 selection groups. Some resistant and susceptible sorghum lines and hybrids were identified.

From the growth chamber study resistance to SDM was found to be dominant to susceptibility based on F_3 segregation patterns. Resistance is apparently controlled by a single dominant major gene, fitting a 3:1 ratio of resistant to susceptible lines. This finding is in agreement with that reported in the current literature.

LITERATURE CITED

1. Anahosur, K. H. 1980. Current sorghum downy mildew research in All India Sorghum Project. p. 200-206. ICRISAT. Proc. Int. Workshop on Sorghum Diseases, 11-15 December 1978, Hyderabad, India.

2. Anonymous. 1973. A compendium of corn diseases. American Phytopathological Society, St. Paul, Minnesota. 64 p.

3. Amador, J., R. W. Berry, R. A. Frederiksen, C. W. Horne, W. H. Thomas, and R. W. Toler. 1974. Sorghum diseases. Texas Agric. Ext. Service B-1085.

4. Asgrow Seed Company. 1974. Troublesome grain sorghum diseases - a management manual. Asgrow Seed Company. Des Moines, Iowa.

5. Bain, D. C., and W. W. Alford. 1969. Evidence that downy mildew (Sclerospora sorghi) or sorghum is seedborne. Plant Dis. Reptr. 53:802-803.

6. Bonde, M. R., C. G. Schmitt, and R. W. Dapper. 1978. Effect of dew-period temperature on germination of conidia and systemic infection of maize by Sclerospora sorghi. Phytopathology 68: 219-222.

7. Cosper, J. W. 1969. Inoculation, infection, and reproduction of Sclerospora sorghi (Kulk.) Weston & Uppal in sorghum. M.S. Thesis, Texas A&M University, College Station.

8. Craig, J. 1975. Inoculation techniques for testing resistance to sorghum downy mildew. p. 243-254. Proc. Int. Sorghum Workshop, January 7-11, 1975. Univ. Puerto Rico, Mayaguez.

9. Craig, J. 1976. An inoculation technique for identifying resistance to sorghum downy mildew. Plant Dis. Reptr. 60:350-352.

10. Craig, J. 1980. Sorghum downy mildew research at Texas A&M University. p. 195-199. ICRISAT. Proc. Int. Workshop on Sorghum Diseases, 11-15 December 1978, Hyderabad, India.

11. Dange, S. R. S. 1976. Sorghum downy mildew (Sclerospora sorghi) on maize in Rajasthan, India. Kasetsart J. 10:121-127.

12. Dange, S. R. S., and R. J. Williams. 1980. The ICRISAT sorghum downy mildew program. p. 209-212. ICRISAT. Proc. Int. Workshop on Sorghum Diseases, 11-15 December 1978, Hyderabad, India.

13. Edmunds, L. K., and N. Zummo. 1975. Sorghum diseases in the United States and their control. USDA Handb. No. 468, Government Printing Office, Washington, D.C.

14. Frederiksen, R. A. 1978. Sorghum diseases in South Texas: the potential for control. p. 9-18. In Proceedings of the South Texas Grain Sorghum Symposium, 31 October to 1 November 1978. Victoria and Robstown, Texas.

15. Frederiksen, R. A. 1980. Seed Transmission of Peronsclerospora sorghi in grain sorghum: how can it be avoided? Texas Agric. Exp. Stn. MP-1453.

16. Frederiksen, R. A. 1980. Sorghum downy mildew in the United States: Overview and Outlook. Plant Disease 64:903-908.

17. Frederiksen, R. A., and B. L. Renfro. 1977. Global status of maize downy mildew. Ann. Rev. Phytopathology 15:249-275.

18. Frederiksen, R. A., and D. T. Rosenow. 1980. Breeding for disease resistance in sorghum. p. 137-167. In: M. K. Harris (ed.) Biology and breeding for resistance to arthropods and pathogens in agricultural plants. Texas Agric. Exp. Stn. MP-1451.

19. Frederiksen, R. A., A. J. Bockholt, D. T. Rosenow, and L. Reyes. 1970. Problems and progress of sorghum downy mildew in the United States. Indian Phytopathology 23:321-338.

20. Frederiksen, R. A., A. J. Bockholt, L. E. Clark, J. W. Cosper, J. Craig, J. W. Johnson, B. L. Jones, P. Matocha, F. R. Miller, L. Reyes, D. T. Rosenow, D. Tuleen, and H. J. Walker. 1973. Sorghum downy mildew: a disease of maize and sorghum. Texas Agric. Exp. Stn. Res. Monogr. 2:1-32.

21. Frezzi, M., and E. E. Teyssandier. 1980. Summary and historical review of sorghum diseases in Argentina. p. 11-14. ICRISAT. Proc. Int. Workshop on Sorghum Diseases, Hyderabad, India.

22. Futrell, M. C. 1972. The potential danger of downy mildew and fusarium to sorghum and corn. Proc. of the Twenty-seventh Annual Corn and Sorghum Research Conf. 27:36-43.

23. Futrell, M. C., and O. J. Webster. 1966. New sources of resistance to the downy mildew diseases of sorghum. Plant Dis. Reptr. 50:641-644.

24. Horne, C. W., and R. W. Berry. 1980. Sorghum disease atlas. Texas Agric. Ext. Service B-1323.

25. Horne, C. W., R. A. Frederiksen, R. W. Toler, and J. D. Trampota. 1978. Disease ratings of commercial grain sorghum and corn hybrids. Texas Agric. Ext. Service MP-1352.

26. Jones, B. L. 1971. Techniques for artificially inoculating sorghum with Sclerospora sorghi. p. 3-5. In Seventh Biennial Grain Sorghum Research and Utilization Conference. Lubbock, Texas.

27. Jones, B. L. 1971. The mode of Sclerospora sorghi infection of Sorghum bicolor leaves. Phytopathology 61: 406-408.

28. Jones, B. L. 1978. The mode of systemic infection of sorghum and sudangrass by conidia of Sclerospora sorghi. Phytopathology 68:732-735.

29. Kenneth, R., and G. Shahor. 1973. Systemic infection of sorghum and corn by conidia of Sclerospora sorghi. Phytoparasitica 1:13-21.

30. Malaguti, G. 1980. Sorghum downy mildew in the Americas. P. 184-194. ICRISAT. Proc. Int. Workshop on Sorghum Diseases, 11-15 December 1978. Hyderabad, India.

31. Miller, F. R., R. A. Frederiksen, S. T. Ali Khan, and D. T. Rosenow. 1968. Reaction of selected sorghum varieties and lines to downy mildew. Texas Agric. Exp. Stn. MP-890.

32. Pratt, R. G. 1978. Germination of oospores of Sclerospora sorghi in the presence of growing roots of host and nonhost plants. Phytopathology 68:1606-1613.

33. Pratt, R. G., and G. D. Janke. 1978. Oospores of Sclerospora sorghi in soils of South Texas and their relationship to the incidence of downy mildew in grain sorghum. Phytopathology 68:1600-1605.

34. Rao, N. G. P., R. V. Vidyabhushanam, B. S. Rana, V. Jaya Mohan Rao, and M. J. Vasudeva Rao. 1980. Breeding sorghums for disease resistance in India. p. 430-433. ICRISAT. Proc. Int. Workshop on Sorghum Diseases, 11-15 Decmeber 1978. Hyderabad, India.

35. Reyes, L., D. T. Rosenow, R. W. Berry, and M. C. Futrell. 1964. Downy mildew and head smut diseases of sorghum in Texas. Plant Dis. Reptr. 48:249-253.

36. Riccelli, M. 1980. Current strategies and progress in breeding disease resistant sorghums in Venezuela. p. 434-453. ICRISAT. Proc. Int. Workshop on Sorghum Diseases, 11-15 December 1978, Hyderabad, India.

37. Rosenow, D. T., A. J. Bockholt, and R. A. Frederiksen. 1968. Inheritance of resistance to downy mildew, Sclerospora sorghi. Sorghum Newsletter 11:101.

38. Safeeulla, K. M., and H. S. Shetty. 1980. Sorghum downy mildew in Asia: assessment of present knowledge and future research needs. p. 173-183. ICRISAT. Proc. Int. Workshop on Sorghum Diseases, 11-15 December 1978. Hyderabad, India.

39. Safeeulla, K. M., and M. J. Thirumalachar. 1955. Resistance to infection by Sclerospora sorghi of sorghum and maize varieties in Mysore, India. Phytopathology 45:128-131.

40. Schmitt, C. G., and R. E. Freytag. 1973. Studies on conidial release by Sclerospora sorghi. p. 26-31. Report of a Workship on the downy mildews of sorghum and corn. Texas Agric. Exp. Stn. Tech. Report No. 74-1.

41. Schmitt, C. G., and R. E. Freytag. 1974. A quantitative technique for inoculating corn and sorghum with conidia of Sclerospora sorghi. Plant Dis. Reptr. 58:825-829.

42. Schmitt, C. G., C. E. Scott, and R. E. Freytag. 1977. Response of maize diallel cross to Sclerospora sorghi, cause of sorghum downy mildew. Plant Dis. Reptr. 61:607-608.

43. Schwinn, F. J. 1980. Prospects for chemical control of the cereal downy mildews. p. 220-222. ICRISAT. Proc. Int. Workshop on Sorghum Diseases, 11-15 December 1978. Hyderabad, India.

44. Shaw, C. G. 1978. Peronosclerospora species and other downy mildews of the gramineae. Mycologia 70:594-604.

45. Tarr, S. A. J. 1962. Diseases of Sorghum, Sudan Grass and Broom Corn, University Press, Oxford, England.

46. Toler, R. W., and R. A. Frederiksen. 1971. Sorghum Diseases. p. 15-28. In: Grain Sorghum Research in Texas . . . 1970. Texas Agric. Exp. Stn. Consolidated PR-2938-2949.

47. Tontyaporn, S. 1976. Cytology of Sclerospora sorghi and an attempt to culture on maize tissue. Kasetsart J. 10:116-117.

48. Ullstrup, A. J. 1974. Corn Diseases in the United States and their control. USDA Agric. Handb. No. 199, Government Printing Office, Washington, D.C.

49. Weston, W. H., Jr., and B. N. Uppal. 1932. The basis for Sclerospora sorghi as a species. Phytopathology 22:573-586.

50. Williams, R. T., R. A. Frederiksen, and T. C. Girard. 1978. Sorghum and Pearl Millet disease identification handbook. ICRISAT Information Bul. No. 2.

51. Yeh, T., and R. A. Frederiksen. 1980. Sorghum downy mildew: biology of systemic infection by conidia and of a resistant response in sorghum. Phytopathology 70:372-376.

VITA 2

Quentin B. Kubicek

Candidate for the Degree of

Master of Science

Thesis: AN INOCULATION TECHNIQUE USED TO IDENTIFY RESISTANCE TO SORGHUM DOWNY MILDEW

Major Field: Agronomy

Biographical:

Personal Data: Born in Maracaibo, Venezuela, July 21, 1954, the son of Quentin and Olga Kubicek.

Education: Attended Colegio Americano, Altos de la Trinidad, Caracas, Venezuela, 1968-1971 and Escuela Americana, Tegucigalpa, Honduras, 1972; received the Bachelor of Science degree in Agriculture in Agronomy from Louisiana State University and Agricultural and Mechanical College, December, 1976; completed the requirements for the Master of Science degree at Oklahoma State University in December, 1981.

Professional Experience: Part time and full time graduate research assistant for Dr. Dale E. Weibel, Oklahoma State University, 1980-1981.

Professional Organizations: American Phytopathological Society.