EFFECT OF FUSARIUM VIRGULIFORME AND HETERODERA GLYCINES ON SOYBEAN

by

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B.S., Kansas State University, 2007

A THESIS

submitted in partial fulfillment of the requirements for the degree

MASTER OF SCIENCE

Department of Agronomy
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KANSAS STATE UNIVERSITY
Manhattan, Kansas

2010

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Abstract

Fusarium virguliforme, the soilborne fungus which causes sudden death syndrome (SDS) of soybean, and *Heterodera glycines* Ichinohe, soybean cyst nematode (SCN), are two economically important pathogens in the Midwest. The pathogens are often found together in soybean (Glycine max (L.) Merr.) fields. This study was conducted to determine the effect of soybean genotype, *F. virguliforme* populations, and *H. glycines* populations upon yield and to examine the interaction between the two pathogens. In 2008 and 2009, four genotypes with different levels of resistance to SDS and *H. glycines* were planted at seven environments. *F. virguliforme* and *H. glycines* soil populations were quantified at planting, midseason, and harvest. At the end of the growing season, area under the disease progress curves of SDS, *F. virguliforme* root populations, and *H. glycines* reproductive indices were determined and plots harvested for seed yield. Soil populations of *F. virguliforme* and *H. glycines* at planting, midseason, and harvest varied across environments. Within environments, generally, they were not significantly different. Seed yield varied within and across environments. As disease pressure increased, the performance of resistant genotypes increased compared to susceptible genotypes. Genotypes resistant to SDS yielded higher than susceptible genotypes. There were negative correlations between yield and disease rating and *F. virguliforme* root populations. *F. virguliforme* soil populations and *H. glycines* populations at planting were positively correlated. It is important to manage both SDS and *H. glycines* in fields with a history of the two diseases. This can be achieved through genetic resistance. Information in this study will improve decisions regarding genotype selection to minimize losses to SDS and *H. glycines*.

Abbreviations: AUDPC, area under the disease progress curve of sudden death syndrome; DI, disease incidence; DS, disease severity; DX, disease index; FI, female index; FVHv, *F. virguliforme* soil population at harvest; FVMd, *F. virguliforme* soil population at midseason; FVPl, *F. virguliforme* soil population at planting; FVRt, *F. virguliforme* root population; J2, stage two juvenile; PDA, potato dextrose agar; MNS, modified Nash-Snyder media; SCN, soybean cyst nematode; SCN Pi, soybean cyst nematode soil population at planting; SCN Pf, soybean cyst nematode population at harvest; SCN Pm, soybean cyst nematode population at midseason; SCN Rf, soybean cyst nematode reproductive factor; SDS, sudden death syndrome.

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Acknowledgements

I would like to thank my advisor, Dr. William Schapaugh, for all of his support, encouragement, and advice throughout the years and for being a cornerstone in the development of my knowledge about plant breeding and soybean. I would also like to thank my committee members Timothy Todd and Dr. Christopher Little for all of their time spent helping me, answering my questions, and feedback. I am especially grateful to Tom Oakley and his crew in the Nematology Lab for their work with my soil samples and to Lance Noll in the Row Crops Pathology Lab for his help with mycology. Special thanks also goes out to Russell Dille for my personalized soil probe and being willing to lend a helping hand to my research when necessary. I would also like to thank my undergraduate worker Cale Miller for making all of the hours spent collecting soil samples in the field and working in the lab fun. Thanks to the rest of the Soybean Project crew, graduate students Jianye Meng and Pam Rzodkiewicz and the undergraduates, for their help with miscellaneous tasks.

Thanks to my family for all of their unwavering support throughout my entire life, especially my mom and dad, John and Joe, Mumzy and Pop Pop, and Mom Mom. Finally, thanks to my friends for becoming my family when I was a thousand miles from home. My K-State friends, Jessie, Emily, Duncan, Julie, Michaela, Jenny, Aly, Katie, Diana, Dena, Mark, and others, have truly made Manhattan a home away from home and have made my time spent here some of the best years of my life.

Dedication

To Mumzy, for always encouraging me to follow my dreams no matter where they might take me.

CHAPTER 1 - Literature Review

Introduction

Soybean (*Glycine max* (L.) Merr.) is an important agronomic crop both in the United States and around the world with several uses in industry along with uses in foodstuffs for humans and livestock. Processed soybeans are the largest source of protein feed and the second largest source of vegetable oil in the world. They account for 90 percent of total oilseed production in the United States. Over 43 percent of United States' soybean and soybean products were exported in 2007/2008 (USDA-ERS, 2010). In the United States in 2009, yields averaged approximately 44 bushels per acre and over 3.36 billion bushels from over 76.4 million acres were harvested making it the largest soybean harvest on record. Prices remained high as they have since 2007 at around ten dollars a bushel. In Kansas, soybean is the third largest crop behind wheat and corn. Over 3.65 million acres were harvested in the state in 2009 at a value of approximately 1.5 billion dollars (USDA-NASS, 2010). With high prices and increased yields, soybean will continue to be major crop for producers (USDA-ERS, 2010),

Significant threats to soybean yield and producer revenue include plant pathogens which interfere with normal plant growth and development often resulting in decreased seed set and quality. *Fusarium virguliforme*, the fungal agent of sudden death syndrome (SDS) of soybean, and soybean cyst nematode (SCN), *Heterodera glycines* Ichinohe, are two of these economically important pathogens. Both pathogens are soilborne and are present in many of the top producing soybean areas in the United States including Kansas. Often, their presence leads to a negative impact on yield. With similar environmental conditions necessary for development, SDS and

SCN often occur in association with each other in a field. Many times, the appearance of SDS in a field is the first indication a producer's field also has SCN.

Sudden Death Syndrome

Sudden death syndrome was first observed in Arkansas in 1972 by H.J. Walters and has become widespread across soybean growing regions in the United States, Argentina and Brazil (Rupe et al., 2001; Ploper, 1993; Nakajima et al., 1993). Symptoms were first reported in Kansas in 1993 (Jardine and Rupe, 1993). From 1996 to 2005, SDS was listed by Wrather et al. (2006) as one of the most important soybean diseases in the United States (Wrather et al., 2006). Losses up to 80 percent have been attributed to the disease with yield losses between 5 percent and 15 percent more common (Roy et al., 1997). In 2005, it was estimated yield loss due to SDS was valued at $118.8 million ($236/t) across the United States (Wrather et al., 2006). Although found across most soybean growing regions, SDS occurrence and yield loss is most common in the Ohio River Valley and upper Mississippi River Delta (Hershman et al., 1990; Melgar et al., 1994).

The fungal causal agent of the disease was originally referred to as *Fusarium solani* (Mart.) Sacc. (Roy et al., 1989; Rupe, 1989) but in subsequent years was specified as *F. solani* f. sp. *glycines* to designate its soybean host (Roy et al., 1997). In 2003, Aoki et al. (2003) observed two morphologically and phylogenetically distinct species classified as *Fusarium solani* which caused sudden death syndrome, *F. tucumaniae* in South America and *F. virguliforme* in North America. *F. tucumaniae* differed from other species of *F. solani* in that it had septate, falcate, footed conidia and tall and slender aerial condiophores in addition to long slender, sporodochial conidiophores with pointed apices. In comparison to *F. virguliforme*, a second type of sporodochial conidia was never formed. *F. virguliforme* produced comma-shaped sporodochial

conidia in addition to septate, falcate, footed aerial conidia. Short conidophores also produced oblong-ellipsoid to short clavate conidia. Most of the current literature in the United States now refers to the causal agent of sudden death syndrome as *F. virguliforme* (Aoki et al., 2003).

The pathogen overwinters freely in the soil and in soybean residue. It infects roots early in the growing season with symptoms commonly developing after flowering and before pod-fill. Early symptoms tend to appear on the uppermost leaves as small scattered, interveinal light green or chlorotic spots giving a mottled appearance. The spots then enlarge and can become necrotic or may coalesce to form larger areas of interveinal leaf chlorosis. Eventually, most of the affected tissue will become necrotic with only minimal green tissue remaining. In severe cases, defoliation occurs with petioles remaining attached to the stem. Flowers and pods can also be aborted with the younger tissues aborted first leading to decreased seed and pod-fill. Root symptoms become more pronounced with increased severity of foliar symptoms. Roots of infected plants tend to exhibit crown necrosis and lateral root rots exhibiting grayish to reddish brown discoloration of the xylem. Blue-green sporulation may be seen on the taproot and lower stem. Plants are easily pulled from the soil (Roy et al., 1997).

In 1989, Roy et al. (1989) and Rupe (1989) completed Koch's postulates for SDS in two separate experiments. The isolates from plants displaying symptoms of SDS produced slow growing blue masses of macroconidia on PDA. Few microconidia were produced, and the isolates stained PDA a dark maroon. (Roy et al., 1989; Rupe, 1989). *F. virguliforme* was only isolated from the roots and lower stems but not from the leaves (Rupe 1989). Jin et al. (1996) proposed fungal toxins produced in or on the roots were translocated to the leaves (Jin et al., 1996). In the mid 1990s, a modified Nash-Snyder's medium (MNS) began to be used to isolate and identify colonies of *F. virguliforme* in addition to PDA. Cho et al. (2001) completed Koch's

postulates using this media type. Two hundred and eighty two isolates resembling *F. virguliforme* were collected from soil in two fields with a history of SDS. They were plated on MNS and 112 isolates were tested for pathogenicity in the greenhouse (Cho et al., 2001).

Sudden death syndrome has been associated with high soil moisture and cool temperatures. Irrigated fields with high fertility and yield potential tend to exhibit the worst symptoms. Scherm and Yang (1996) conducted greenhouse and field experiments examining the effect of temperature and moisture on development of SDS symptoms. In the greenhouse, root symptoms were most severe at 18°C while foliar symptoms were most severe at temperatures between 22 and 24°C. Symptoms were light at temperatures over 30°C. Favorable conditions for disease development in the roots followed by favorable conditions for plant development leads to increased disease because increased levels of the toxin in the roots are better able to be translocated to the leaves (Li et al., 2009). The wettest treatments in both greenhouse and field produce the worst SDS symptoms. Compacted field areas maintain more moisture for longer period of time also positively influencing symptoms (Scherm and Yang, 1996). Field irrigation during late to mid reproductive stages increases SDS development (Neto et al., 2007). Soils amended with calcium phosphate, potassium phosphate, potassium sulfate, sodium phosphate, or potassium nitrate result in a 21% to 45% increase in SDS severity (Sanogo and Yang, 2000).

The options for management of SDS are limited. Fungicides have limited effects on the control of *F. virguliforme* as root infection takes place underground in the root early in the growing season (Henricksen and Elen, 2005). Data obtained from studies on tillage practices has been contradictory. No-till, disk till, and chisel till decreased and increased area under the disease progress curve (AUFDPC) in foliage depending on the year (Vick et al., 2005). Although there are rotations that decrease the presence of other soybean pathogens, there does not appear

to be a rotation that significantly lowers the level of *F. virguliforme* in the soil (Rupe et al., 1997).

Resistant cultivars are the best method for SDS control. Field resistance is classified as horizontal, rate reducing and partial. It is controlled by many genes and is highly heritable (Njiti et al., 1996). Several resistant genotypes have been identified and have been made available to producers; however, none of the current packages resistance are complete. All genotypes will display some SDS symptoms if conditions are favorable (Hershman, 1990).

Soybean Cyst Nematode

Heterodera glycines, soybean cyst nematode (SCN), is the most important pathogen of soybean in the United States. It was first reported in 1954 in North Carolina and has spread to most soybean producing areas. It was first noted in Kansas in Doniphan County in 1985 and is now found in several eastern Kansas counties with losses estimated at approximately 19,530 tonnes in 2005 (Sim and Todd, 1986; Wrather et al., 2006). Countrywide losses were estimated at 1.9 million tonnes (Wrather et al., 2006). Yield losses can reach up to 40 percent in a field.

SCN have an egg and four juvenile stages. The eggs can overwinter within a female cyst under harsh environmental conditions (Alston and Schmitt, 1988). The first stage juvenile develops within an egg and molts to become a second stage juvenile (J2). The second stage is the infective stage as the J2 hatches from the egg and moves a short distance through the soil to the root tips. It penetrates the root and establishes a feeding site where it engorges. The juvenile will molt three more times before becoming an adult. Females become immobile and continue to feed on the root. Their bodies swell and become yellow, lemon-shaped cysts containing approximately 100 to 200 eggs. Eggs only develop if they are fertilized by a male. At death, the cysts are brown and dislodge from the root. Males remain vermiform and mobile. They do not

feed on roots but will enter the root to fertilize females. After mating, males exit the root and die. The life cycle takes about 25 to 40 days with several generations occurring in a single growing season (Triantaphyllou and Hirshmann, 1962; Jardine and Todd, 2001).

Since the most common symptom is yield loss, producers often are not aware of the presence of SCN until the end of a growing season. Visible symptoms include stunting and/or chlorosis. Root mass and nodulation may be decreased. In severe cases, premature plant death can occur. Above-ground symptoms can be confused with nutrient deficiencies, herbicide injury, and other disease. The distribution of nematodes in the soil is variable. Diseased areas are usually scattered throughout a field and can appear oval shaped. SCN moves with soil, therefore field-scale lesions expand in the direction of tillage (Jardine and Todd, 2001).

SCN is heavily influenced by environmental conditions including soil type, moisture levels, and temperature. Higher soil and root populations are observed in sandy soils (Todd and Pearson, 1988). SCN can increase to damaging levels in fine textured soils but at a lower rate due to decreased reproductive levels. Several stages of the nematode life cycle, including hatch, movement, and development, require aerobic respiration. Fine textured soils retain water for longer periods of time creating anaerobic conditions unfavorable to the life cycle. Heavier textured soils can allow for easier nematode movement as there is more space between soil particles (Koenning and Barker, 1995). SCN survival is also affected by temperature. Slack et al. (1972) noted larvae survived for over 630 days in water at temperatures between 0°C and 12°C but died when ice crystals formed or after a day at 40°C. In natural soils, eggs survived 6 to 8 years between 0°C and 20°C and were not immediately killed by extreme high and low temperatures (below freezing and above 40°C). At temperatures over 20°C, larvae survival time decreased with increased temperatures (Slack et al., 1972a). Optimum temperature for an egg to

hatch is 24°C where there is low mortality and development is rapid. Hatch has been observed between 20°C and 30°C (Alston and Schmitt., 1988). Penetration, development, and reproduction of SCN are negatively affected at temperatures below 14°C and above 33°C (Slack et al., 1972b).

There are a variety of control methods for SCN, but none completely eliminate the pest from the soil. Crop rotation to a non-host such as corn, grain sorghum, or wheat can cause a decline in SCN numbers (Long and Todd, 2001). The rate of decline is dependent on initial SCN levels and environment. Eggs survive better in the north central United States than they do in the south. Nematicides are available but are not cost effective. Resistant genotypes are the best form of control. They limit the reproductive capacity of the nematode resulting in a population decline over the growing season. Most resistance in commercial genotypes is derived from Peking or PI88788. SCN races are diverse and can adapt to resistance (Jardine and Todd, 2001).

The SCN HG type system is a relatively new way of classifying SCN resistance and SCN genetic diversity. Prior to this system, resistance was classified using the SCN race system. This method incorporated four sources of resistance: Pickett, Peking, PI88788, and PI90763. If a SCN race had a female index (FI) above 10 percent it was designated with a "+" while a female index at or below 10 percent was considered resistant and designated with a "-" (Schmitt and Shannon, 1992). Female index is calculated by dividing the mean number females on a test soybean line by the mean number of females on the standard susceptible and multiplying by 10. The problem with the race classification system was that it only incorporated four sources of resistance and was not quantitative. The HG type system addresses some of these problems by allowing for the addition of several sources of resistance. A standard susceptible, Lee, is used, and the number of females found on it are reported. SCN populations are collected by environment and are tested

on several differentials with each female index noted. Anything with a female index above ten is considered susceptible, and the HG type corresponds to the differentials a population reproduces on (Niblack et al., 2002).

Relationship between Sudden Death Syndrome and Soybean Cyst Nematode

In his early observations of sudden death syndrome, Hirrel (1983) noted the dual presence of SDS symptoms and soybean cyst nematode in affected fields. He noted SCN was associated with 70 to 80% of plants displaying SDS symptoms in thirty fields across four states (Hirrel, 1983). Population levels of SCN were reduced by 47% by the presence of *F. virguliforme* in a coinoculation study by McLean and Lawrence (1995). The lifestage development of the nematode increased by 3% over a period of 30 days. After 40 days, the fungus was found in 37% of examined cysts. It was also isolated from the cortex and syncytia in plant tissue near developing juveniles (McLean and Lawrence, 1995). Roy et al. (1998) isolated *F. virguliforme* in cysts from a majority of fields in the Midwest and South affected with both pathogens at a slightly lower rate. The fungus survived at 10°C for length of time equivalent to overwintering (Roy et al., 1998). The ability of *F. virguliforme* to survive in SCN cysts can possibly influence the severity of SDS and its dispersal. The cysts provide an environment that is sheltered from other soil microorganisms than in soil or soybean residue alone. In an isolated environment, there is reduced competition with other organisms leading to enhanced survival. Cysts could also possibly be a food source to spur the growth of chlamydospores in the spring. The spread of SDS from its original place of detection in Arkansas could be in part due to *F. virguliforme* colonized cysts (McLean and Lawrence, 1995). SCN may also result in increased *F. virguliforme* levels by providing wounds for *F. virguliforme* to enter the plant and reproduce (Roy et al., 1989; Scherm et al., 1998). The possible decreased levels of SCN in SDS affected

fields at flowering and harvest can be attributed to the root necrosis, which limits the food supply for the nematode. Also, the parasitic nature of *F. virguliforme* hinders normal nematode growth and development leading to decreased survival (Rupe et al., 1993).

In early greenhouse studies where soil was inoculated with *F. virguliforme* and SCN, SCN caused more severe foliar symptoms but was not necessary for disease infection (Roy et al., 1989). A field microplot study produced similar results. Foliar disease symptoms appeared three to seven days earlier and were more severe in plots with *F. virguliforme* and SCN than those with only *F. virguliforme* (McLean and Lawrence, 1993). In a later microplot study, *F. virguliforme* and SCN also damaged plants synergistically in coinoculated plots. Plots with natural levels of SCN also had high levels of SDS. In one season, only coinoculated plots displayed SDS symptoms. All results pointed to a positive correlation between the pathogen (Xing and Westphal, 2006). Enhanced symptoms of SDS by high populations of SCN may explain the clustered pattern of diseased plants in a field. SCN is usually not distributed evenly through the field, so "hot spots" of SDS may occur where SCN populations are particularly high (Scherm and Yang, 1996).

While studies have supported the association between SCN and SDS, others looking for a positive correlation between the two pathogens have not been as strong. A correlation between cyst counts and disease severity in a Scherm et al. (1998) study on soil variables in SDS fields in Iowa was visible but weak, and the cross-correlation coefficients for SCN cysts were not always significant (Scherm et al., 1998). Gao et al. (2006) inoculated a susceptible genotype with different levels of *F. virguliforme* and SCN and conducted real-time polymerase chain reaction analysis. The infection of soybean roots by SCN did not affect colonization by the fungus. The only significant main effect was fungal population. While both pathogens reduced plant growth,

SCN did not increase foliar symptoms of SDS. Overall, statistical interactions between SCN and *F. virguliforme* were rarely significant (Gao et al., 2006). Inconsistencies in the relationship between SDS and SCN demonstrate the need for continued research.

Research Objectives

It has been suggested the presence of SCN increases the severity of SDS symptoms and therefore, decreases yields. The objectives of this research were to examine the effect of *Fusarium virguliforme* and *Heterodera glycines* upon yield of soybean genotypes with different resistance to the two pathogens and to examine the possible interaction between the two pathogens.

References

Alston, D. G. and Schmitt, D. P. Development of *Heterodera glycines* as influence by temperature. 1988. Journal of Nematology 20(3):366-372.

Aoki, T., O'Donnell, K., Homma, Y., and Lattanzi, A. R. 2003. Sudden-death syndrome is caused by two morphologically and phylogenetically distinct species within a *Fusarium solani* species complex—*F. virguliforme* in North America and *F. tucumaniae* in South America. Mycologia 95(4):660-684.

Cho, J. H., Rupe, J. C., Cummings, M. S., and Gbur, E. E., Jr. 2001. Isolation and identification of *Fusarium solani* f. sp. *glycines* from soil on modified Nash and Snyder's medium. Plant Disease 85:256-260.

Gao, X., Jackson, T. A., Hartman, G. L., and Niblack, T. L. 2006. Interactions between the soybean cyst nematode and *Fusarium solani* f. sp. *glycines* based on greenhouse factorial experiments. Phytopathology 96:1409-1415.

Hartman, G. L., Huang, Y. H., Nelson, R. I., and Noel, G. R. 1997. Germplasm evaluation of *Glycine max* for resistance to *Fusarium solani*, the causal organism of soybean sudden death syndrome. Plant Disease 81:515-518.

Henriksen, B, and Elen, O. 2005. Grain infection level in wheat, barley, and oat after early level applications of fungicides and herbicides. Journal of Phytopathology 153: 214-220.

Hershman, D. E., Hendrix, J. W., Stuckey, R. E., Bachi, P. R., and Henson, G. 1990. Influence of planting date and cultivar on soybean sudden-death syndrome in Kentucky. Plant Disease 74:761-766.

Hirrel, M. C. 1983. Sudden death syndrome of soybean: A disease of unknown etiology. Phytopathology 73:501-502.

Jardine, D. J. and Rupe, J. C. 1993. First report of sudden-death syndrome of soybeans caused by *Fusarium solani* in Kansas. Plant Disease 77:1264.

Jardine, D. J. and Todd, T. C. 2001. Soybean cyst nematode. Kansas State University, Manhattan, Kansas.

Jin, H., Hartman, G. L., Nickell, C. D., and Widholm, J. M. 1996. Characterization and purification of a phytotoxin produced by *Fusarium solani*, the causal agent of soybean sudden death syndrome. Phytopathology 86:277-282.

Koenning, S. R. and Barker, K. R. 1995. Soybean photosynthesis and yield as influenced by *Heterodera glycines*, soil type, and irrigation. Journal of Nematology 27(1): 51-62.

Li, S., Hartman, G. L., and Chen, Y. 2009. Evaluation of aggressiveness of *Fusarium virguliforme* isolates that cause sudden death syndrome. Journal of Plant Pathology 91(1):77-86.

Long, J. H., Jr., and Todd, T. C. 2001. Effect of crop rotation and cultivar resistance on seed yield
and the soybean cyst nematode in full-season and double-cropped soybean. Crop Science 41:1137-1143.

McLean, K. S. and Lawrence, G. W. 1993. Interrelationship of *Heterodera glycines* and *Fusarium solani* in sudden death syndrome of soybean. Journal of Nematology 25:434-439.

McLean, K. S. and Lawrence, G. W. 1995. Development of *Heterodera glycines* as as affected by *Fusarium solani* the causal agent of sudden death syndrome of soybean. Journal of Nematology. 27:70-77.

Melgar, J. and Roy, K. W. 1994. Soybean sudden death syndrome: Cultivar reactions to inoculation in a controlled environment and host range and virulence of the causal agent. Plant Disease 78:265-268.

Nakajima, T. Mitsueda, T. Charchar, M. J. 1993. Occurrence of soybean sudden death syndrome caused by *Fusarium solani* in Brazil. Abstract, 7th Int *Fusarium* Workshop, Pennsylvania State University, University Park. p 79.

Neto, A. L., Hashmi, R., Schmidt, M., Carlson, S. R., Hartman, G. L., Li, S., Nelson, R. L., and Diers, R. W. 2006. Mapping and confirmation of a new sudden death syndrome resistance QTL on linkage group D2 from the soybean genotypes PI 567374 and 'Ripley.' Molecular Breeding 20:53-62.

Niblack, T. L., Arelli, P. R., Noel, G. R., Opperman, G. H., Orf, J. H., Schmitt, D. P.,

Njiti, V. N., Shenaut, M. A., Suttner, R. J., Schmidt, M. E., and Gibson, P. T. 1996. Soybean response to sudden death syndrome: Inheritance influenced by cyst nematode resistance in Pyramid x Douglas progenies. Crop Science 36:1165-1170.

Ploper, D. 1993. Síndrome de la muerte súbita: Nueva enfermedad de la soja en el noroeste argentine. Avance Agroindustrial. Año 13. No. 54:5-9.

Roy, K. W., Hershman, D. E., Rupe, J. C, and Abney, T. S. 1997. Sudden death syndrome of soybean. Plant Disease 81(10):1100-1111.

Roy, K. W., Patel, M. V., and Ratnayake, S. 1998. Internal colonization of *Heterodera glycines* cysts by *Fusarium solani* f. sp. *glycines*, the cause of sudden death syndrome, and other fusaria in soybean fields in the Midwestern and southern United States. Mycologia.

Roy, K. W., Lawrence, G. W., Hodges, H. H., and Killebrew, J. F. 1989. Sudden death syndrome of soybean: *Fusarium solani* as incitant and relation of *Heterodera glycines* to disease severity. Phytopathology 79:191-197.

Rupe, J. C. 1989. Frequency and pathogenicity of *Fusarium solani* recovered from soybeans with sudden death syndrome. Plant Disease 73:581-584.

Rupe, J. C., Correll, J. C., Guerber, J. C., Becton, C. M., Cummings, M. S., Gbur, J. J, Yount, P.A. 2001. Differentiation of the sudden-death syndrome pathogen of soybean *Fusarium solani* f. sp. *glycines*, from other isolates of *F. solani* based on cultural morphology, pathogenicity, and mitochondrial DNA restriction fragment length polymorphisms. Canadian Journal of Botany 79:829-855.

Rupe, J. C., Robbins, R. T., and Gbur, E. E., Jr. 1997. Effect of crop rotation on soil population densities of *Fusarium solani* and *Heterodera glycines* and on the development of sudden death syndrome of soybean. Crop Protection 16(6): 575-580.

Rupe, J. C., Sabbe, W. F., Robbins, R.T, and Gbur, E. E., Jr. 1993. Soil and plant factors associated with sudden death syndrome of soybean. Journal of Production Agriculture. 6:218-221.

Sanogo, S. and Yang, X. B. 2000. Relation of sand content, pH, and potassium and phosphorous nutrition to the development of sudden death syndrome in soybean. Canadian Journal of Plant Pathology 23:174-180.

Scherm, H., and Yang, X. B. 1996. Development of sudden death syndrome of soybean in relation to soil temperature and soil water matric potential. Phytopathology 86:642-649.

Scherm, H., Yang, X. B., and Lundeen, P. 1998. Soil variables associated with sudden death syndrome in soybean fields in Iowa. Plant Disease 82:1152-1157.

Schmitt, D. P and Shannon J. G. 1992. Differentiating soybean responses to *Heterodera glycines* races. Crop Science 32:275-277.

Shannon, J. G., and Tylka, G. L. 2002. A revised classification scheme for genetically diverse populations of *Heterodera glycines*. Journal of Nematology 34(4):279-288.

Sim, T., IV and Todd, T. C. 1986. First field observation of the soybean cyst nematode in Kansas. Plant Disease 70: 603.

Slack, D.A., Riggs, R. D, and Hamblen, M.L. 1972. The effect of temperature and moisture the survival of *Heterodera glycines* in the absence of a host. Journal of Nematology 4(4):263-266.

Slack, D.A., Riggs, R. D, and Hamblen, M.L. 1972. Temperature effects on penetration and reproduction of soybean cyst nematode. Phytopathology 62:762.

Todd, T. C. and Pearson, A. S. 1988. Establishment of *Heterodera glycines* in three soil types. Annals of Applied Nematology 2:57-60.

Triantaphyllou, A. C. and Hirshmann, K. 1962. Oogenesis and mode of reproduction in the soybean cyst nematode, *Heterodera glycines*. Nematologica 7:235.

USDA-Economic Research Services. 2010. Soybeans and Oil Crops, available at <http://www.ers.usda.gov/briefing/Baseline/crops.htm>. Accessed March 9, 2010.

USDA-National Agricultural Statistics Service. 2010. Quick Stats, available at <http://www.nass.usda.gov/QuickStats/index2.jsp>. Accessed January 17, 2010.

Vick, C. M., Bond, J. P, Chong, S. K., and Russin, J. S. 2006. Response of sudden death syndrome to tillage and cultivar. Canadian Journal of Plant Pathology 28:77-83.

Wrather, J. A. and Koenning, S. R. 2006. Estimates of disease effects on soybean yields in the United states 2003 to 2005. Journal of Nematology 38(2):173-180.

Xing, L. J. and Westphal A. 2006. Interaction of *Fusarium solani* f. sp. *glycines* and *Heterodera glycines* in sudden death syndrome of soybean. Phytopathology 96:763-770.

CHAPTER 2 - Performance of Genotypes Differing in Resistance to SDS and SCN and Pathogen Interaction

Introduction

Sudden death syndrome (SDS) is an economically important disease of soybean (*Glycine max* (L.) Merr.) across soybean growing regions in the United States (Rupe et al., 2001). Losses close to 100 percent have been attributed to the disease with yield losses between 5 and 15 percent more common (Rupe and Hartman, 1999). From is earliest detection, SDS has been commonly associated with high soil moisture and cool temperatures. Irrigated fields with high fertility and yield potential tend to exhibit the most severe symptoms.

The causal agent of SDS is *Fusarium virguliforme*, formerly *F. solani* f. sp. *glycines* (Roy et al., 1997; Aoki et al., 2001). The pathogen overwinters in soil and soybean residue. It infects roots early in the growing season with symptoms commonly developing after flowering and before pod-fill. Foliar symptoms include interveinal chlorosis and necrosis. In severe cases, premature defoliation, pod and seed abortion, and death occur. Roots of infected plants tend to exhibit crown necrosis and lateral root rot with gray to red-brown discoloration of the xylem. Blue-green sporulation may be seen on the taproot and lower stem (Roy et al., 1997). Resistant genotypes are the best method of SDS control. Several resistant genotypes have been identified and have been made available to producers; however, none of the current resistance packages are complete. All genotypes will display some SDS symptoms if conditions are favorable (Hershman, 1990).

Early observations of SDS noted the dual presence of *Heterodera glycines* Ichinohe (SCN), and since this time, the two have been associated (Hirrel, 1983). While not necessary for

infection, SCN causes earlier and more severe foliar SDS symptoms. Foliar disease symptoms appeared three to seven days earlier and were more severe in plots with *F. virguliforme* and SCN than those with only *F. virguliforme* (McLean and Lawrence, 1993; Roy et al., 1989; Xing and Westphal, 2006). Plots with natural levels of SCN also have displayed high levels of SDS with a positive correlation between the two diseases (Scherm et al., 1998).

While some reports have noted a positive correlation between SCN population and SDS symptoms, other reports have not (Gao et al., 2006; Hershman et al., 1990; Roy et al., 1997.; Scherm et al., 1998; Xing and Westphal, 2006). Correlation between cyst counts and disease severity was visible but weak, and the cross-correlation coefficients for SCN cysts were not always significant (Scherm et al., 1998). Gao et al. (2006) demonstrated while both pathogens reduced plant growth, SCN population did not increase foliar symptoms of SDS. Statistical interactions between SCN and *F. virguliforme* were rarely significant (Gao et al., 2006). Inconsistencies in the relationship between SDS and SCN demonstrate the need to continued research on the matter.

The objectives of this research were to: 1) characterize the performance of soybean genotypes to SDS and SCN; and 2) evaluate relationships between populations of SCN and *F. virguliforme* populations and the development of SDS symptoms.

Materials and Methods

Field Study

In 2008, four soybean genotypes were planted at three environments at Manhattan, Rossville, and Topeka, Kansas. The 2008 genotypes were selected based on SDS and SCN ratings in the 2007 Kansas Soybean Performance Tests to encompass resistance and susceptibility to both diseases (Schapaugh and Lingenfelser, 2007) (Table 2.1). In 2009, the four soybean genotypes were planted at five environments including Manhattan, Morganville, Ottawa, Rossville, and Topeka, Kansas. All SCN-resistant genotypes carried resistance derived from PI88788. Additionally, all genotypes used in this experiment were glyphosate-resistant.

The genotypes were planted in a randomized completed block design with four replications and in eight, 3.4-meter rows per plot using a four-row ALMACO plot planter (ALMACO, Nevada, IA). Row spacing was 76 cm and seeding rate was 30 seeds per meter. Planting dates in 2008 for Rossville and Topeka were May 16 and for Manhattan, June 10. In 2009, Rossville and Topeka were planted on May 8. Morganville 2009 was planted on May 11, Manhattan on May 13, and Ottawa on May 19. Weeds were controlled with post-emergence herbicide applications and by hand. Soil samples were taken on a per plot basis at planting, midseason, and harvest using a soil probe. Each soil core was 15.2 cm deep with a circumference of 15.2 cm. At each sampling time, eight cores were taken in a zig-zag pattern per plot and bulked together in a plastic soil sample bag for enumeration of *F. virguliforme* and SCN. At harvest, a large bulk soil sample per environment was also collected. Approximately, 15 to 20 soil cores (25 cm deep with a circumference of 15.7 cm) were collected randomly across the

field and placed in a 19-liter bucket for SCN characterization. The samples were homogenized over a 6.35-mm metal mesh. Soils were stored at 6°C until processing.

As SDS symptoms appeared, disease incidence and severity readings were taken every two weeks and corresponded to growth stages R5, R6, and R7 (Table A.1)(Fehr et al., 1971). Disease incidence (DI) was measured on each plot as a percentage of plants displaying SDS symptoms. Disease severity (DS) was rated on each plot on a scale from 1 to 9 according to Njiti et al. (2003). Disease index (DX) was calculated as a percent with a range of 0 to 100 using the following equation:

Equation 2.1

$$DX = \frac{DS \times DI}{9}$$

Using the disease index, area under the disease progress curve of SDS (AUDPC) was calculated according to Shaner and Finney (1977) (Table A.2).

Equation 2.2

$$\sum_{i=1}^{n} [(Y_{i+n1} + Y_i)/2][X_{i+1} - X_i]$$

Where:

Y_i = disease index (per unit) at the *i*th observation

X_i = time (days after planting) at the *i*th observation

n = total number of observations

Prior to harvest, 20 tap root samples per plot were collected for quantification of *F. virguliforme* per gram of root tissue. The center four rows of each plot were evaluated for plant

height, maturity, and lodging (Table A.3). Plant height was measured in centimeters as distance between the soil line and the top of the main stem. Maturity was recorded as the date where 95% of the pods reached mature color. Lodging ratings were between 1 and 5, with one equaling all plants erect and 5 equaling all plants prostrate. Immediately before harvest, 0.5 meters from the ends of the center four rows of each plot were trimmed. The center four rows, 2.5 meters long, were harvested for seed yield using a plot combine and yield reported as kilograms per hectare.

Quantification of *F. virguliforme* Soil Populations

Portions of planting, midseason, and harvest soil samples were air dried and homogenized to determine total soil populations, total *Fusarium* populations, and soil population of *Fusarium virguliforme* (Tables A.2 and A.4). A 1-g subsample of each homogenized sample was placed in 10 ml of sterile distilled water (dilution 1). One ml of dilution 1 was placed in 10 ml of sterile distilled water (dilution 2). Two hundred fifty µl of dilution 1 was spread over four plates of modified Nash-Snyder's (MNS) medium for the enumeration of *F. virguliforme* colony forming units (cfu) (Nash and Snyder, 1962). The medium consisted of 20 g agar, 15 g peptone, 1 g KH_2PO_4, 0.5 g $MgSO_4 \cdot H_2O$, and 1 L deionized water. After autoclaving and cooling, 0.3 g of streptomycin, 0.1 g of neomycin, 0.1 g chlortetracycline, 0.05 g rifampicin, and 0.24 g pentachloronitrobenzene (PCNB) (Terrachlor, Uniroyal Chemical CO., Vaugntuk, CT) were added to the MNS. Plates were incubated at room temperature for 10 to 14 days. Total *Fusarium* colonies on MNS and total *Fusarium virguliforme* on MNS were reported as cfu per gram of soil.

Two hundred fifty µl of dilution 2 was spread over four plates of half strength potato dextrose agar (PDA)(Difco Laboratories, Detroit, MI) to determine total number of colony forming units per gram of soil. Once autoclaved and cooled, the half strength PDA was amended

with antibiotic stock solutions. One thousand µl streptomycin, 2000 µl tetracycline, and 200 µl penicillin were added per 1 L of media. The stock solutions were as follows: 0.1 g of streptomycin per 10 ml sterile distilled water, 0.5 g of penicillin per 10 ml sterile distilled water, and 0.05 g tetracycline per 10 ml ethanol. Plates were incubated at room temperature. Total fungal population on PDA was reported as cfu per gram of soil.

Quantification of Soybean Cyst Nematode Soil Populations

SCN population densities were determined at planting, midseason, and harvest (Table A.5). One hundred ml of soil from each sample were placed in a 3.8-L jug and filled with water. The solution was poured over a tea strainer and 100-mesh sieve and contents on the 100-mesh sieve washed into a 50-ml beaker. The sample was washed into a 400-ml centrifuge tube and injected with 15 ml of 65% sucrose solution. It was placed in a centrifuge for 30 seconds at 800 r.p.m. The sample was then collected on a 100-mesh sieve and washed back into a 50-ml beaker for cysts to be viewed under a dissecting microscope. The sample was washed into a 700-ml centrifuge tube so that it was half full. It was ground for five minutes using a drill bit to release eggs and J2 from cysts and poured over stacked 200-mesh and 500-mesh sieves. Contents of the 500-mesh sieve were washed back into a 50-ml beaker ensuring eggs, J2, and water were at 20 ml. Eggs and juveniles were counted under a compound microscope on a 1-ml counting slide at 40X and reported as eggs and J2 per 100 cc soil.

SCN reproductive factor (Rf) was calculated using the following formula:

Equation 2.3

$$Rf = \frac{Pf}{Pi}$$

Where:

Pf = Number of eggs and juveniles per 100 cc soil at harvest

Pi = Number of eggs and juveniles per 100 cc soil at planting

Quantification of *F. virguliforme* Root Populations

Ten tap roots per plot were treated as one sample, washed, and surface sterilize with a 10% household bleach solution. They were rinsed with distilled water and air dried overnight. The samples were ground using a UDY cyclone sample mill (UDY Corporation, Fort Collins, CO), passed through a 0.5-mm screen, and 0.5 g of root tissue per sample was added to 10 ml of sterile distilled water. Two hundred fifty µl of the solution were plated on four plates of the MNS medium. Plates were incubated at root temperature for 10 to 14 days and *F. virguliforme* populations reported as cfu per gram of root tissue (Table A.2).

SCN Characterization

Large soil samples collected in 2009 at Manhattan, Morganville, Topeka, and Rossville were each mixed with steamed sand resulting in a 50% field sample/50% steamed sand mixture, to enhance SCN reproduction and growth. The test was set up in the greenhouse using a randomized complete block design. Plastic containers measuring 4.0 cm in diameter and 13.5 cm in length with 2.0 to 3.0 g of cotton placed in the bottom were filled with 450 to 500 g of the sand/soil mixture. Six replications of one seed per differential were planted under 30 to 40 g of

soil. The following differential lines were used: PI88788, COM1, COM2, and COM3, with KS3406RR as the standard susceptible. All genotypes had resistance derived from PI88788.

Greenhouse conditions were maintained at an ambient air temperature of 27°C to maintain soil temperature at a constant range of 27 to 28°C under 16-hour days. Artificial lighting was used to maintain a minimum 12-hour photoperiod year-round for proper growth and development. The plants were watered once a day using a standard spray wand attached to a garden hose.

The test was maintained for 35 days. At the end of this time period, plant shoots and leaves were removed and discarded. Each root system was soaked in a 4.0-L plastic bucket of water to loosen soil. Roots were then placed on a tea strainer placed on top of a 100-mesh sieve. SCN females were dislodged from the roots by spraying water. Contents on the 100-mesh sieve were then washed onto a 20-mesh sieve stacked on a 60-mesh sieve with the contents remaining on the 60-mesh sieve being washed into a 50-ml beaker. The females were quantified under a dissecting microscope. The FI was calculated for each soybean differential as follows:

Equation 2.4

$$FI = \left(\frac{mean\ number\ of\ females\ on\ test\ indicator\ soybean\ genotype}{mean\ number\ of\ females\ on\ susceptible\ check} \right) \times 100$$

Statistical Analysis

Prior to statistical analysis, nematode and fungal quantification data were transformed to $\log_{10}(x + 1)$ values to reduce heterogeneity of variances. All data was subjected to two-way analysis of variance using SAS (SAS Institute Inc., Cary, NC) PROC GLM and PROC MIXED to determine genotype, environment, and genotype by environment interactions. Genotype and environment were considered fixed factors. Means were separated using Fisher's protected least

significant different values (LSD). Orthogonal contrasts were also used to compare genotypes resistant to SDS (COM1 and COM2) to genotypes susceptible to SDS (COM3 and KS3406RR) and also the genotype susceptible to SDS but with some moderate resistance to SCN (COM3) to the genotype susceptible to SDS and SCN (KS3406RR). A $p \leq 0.05$ was considered significant except where noted. PROC CORR was used to generate Pearson's correlation coefficients to determine the correlations between SCN, SDS, and yield measurements using the average value for each genotype at each environment (n=28).

Results

Data collected from the Ottawa 2009 environment was not presented in this research due to severe foliar damage caused by a microburst in June 2009.

SCN Female Index

HG types were different at all environments (Table 2.2). None of the genotypes had a female index ≤10 at any environment from soil samples collected from the four 2009 environments (Table 2.3). Genotype KS3406RR was used as the standard susceptible and had a female index of approximately 100% at each location. At Manhattan, Morganville, Rossville, and Topeka, cyst counts on KS3406RR were 251, 111, 47, and 191, respectively (Data not shown). At Manhattan and Morganville, female indices of COM1 and COM2 were between 10 and 30%. COM3 had a female index between 31 and 60%. At Rossville, COM1 and COM2 had female indices >60% and COM3 had a female index between 31 and 60%. All female indices on COM1, COM2, and COM3 fell between 10 and 30% at Topeka.

Soybean Cyst Nematode Populations

SCN populations were significantly different across environments at planting, midseason, and harvest (Table 2.4). The highest populations at each sampling time were observed at Rossville in 2008 where populations across all plots averaged 2.59 eggs and J2/100 cc at planting, 3.00 eggs and J2/100 cc at midseason, and 3.05 eggs and J2/100 cc soil at harvest (Table 2.5). The lowest SCN populations at planting were seen at Manhattan 2008 where populations were 1.78 eggs and J2/100 cc soil. SCN populations at midseason and harvest were

lowest at Morganville 2009 with a midseason population of 2.33 eggs and J2/100 cc and a final population of 1.87 eggs and J2/100 cc soil.

There were no significant differences seen among genotypes and there was no genotype by environment interaction for SCN soil populations at planting and midseason (Table 2.4). Averaged across environments at harvest, KS3406RR and COM2 had the highest SCN soil populations while COM1 and COM3 had the lowest populations (Table 2.6). Additionally, there was a significant genotype by environment interaction seen with SCN populations at harvest (Table 2.4). Populations were similar among genotypes with the exception of Manhattan, Morganville, and Topeka 2009 (Fig. 2.1A). At Morganville and Topeka in 2009, genotypes susceptible SDS had higher final populations at planting. At Manhattan and Morganville 2009, KS3406RR had higher populations than COM3.

The SCN reproductive factor was significantly affected by environment (Table 2.4). The highest reproductive factor of 8.3 at Manhattan 2009 was not different from Manhattan 2008 with a value of 7.8 (Table 2.5). Topeka 2008 had a factor of 5.8. It was also not different from Manhattan 2008 or 2009 and additionally was not different from Rossville 2008 and Topeka 2009 with factors of 3.7 and 3.9, respectively. Rossville 2008 and Topeka 2009 were not different from Manhattan 2009 with a factor of 2.3 and Morganville 2009 with a factor of 0.9. Reproduction was favored at environments such as Manhattan 2008 and Rossville 2009, while it was limited at environments such as Morganville where the reproduction factor was low. A higher final SCN population did not necessarily translate into a higher reproductive factor as this measure also took into account initial SCN population. SCN reproductive factors did not differ among genotypes and the genotype by environment interaction was not significant (Tables 2.4 and 2.6).

Fungal Soil Populations

F. virguliforme represented between >1 and 6 percent of the total fungal population at planting, between >1 and 4 percent at midseason, and between 1 and 6 percent at harvest. *F. virguliforme* represented between 3 and 25% of the total *Fusarium* population at planting, between <1 and 21 percent at midseason, and between 3 and 33 percent at harvest. For the most part, proportions of *F. virguliforme* decreased from planting to midseason and increased from midseason to harvest. Proportions of *F. virguliforme* tended to be highest were SDS symptoms were observed (Table 2.7).

F. virguliforme Populations

F. virguliforme soil populations were significantly different across environments at planting, midseason, and harvest (Table 2.4). Rossville 2008 consistently had the highest populations at planting, midseason, and harvest with 3.00, 3.32, and 3.50 cfu/g, respectively (Table 2.8). Rossville 2009 had the lowest populations at planting, midseason, and harvest. This environment averaged 2.22 cfu/g at planting, 0.78 cfu/g at midseason, and 2.40 cfu/g at harvest.

While a significant genotype effect and genotype by environment interaction was observed with *F. virguliforme* soil populations at planting, this just represents certain genotypes being planted into field areas with higher populations more than others rather than a true genotype differences (Tables 2.4 and 2.9). However, another genotype by environment interaction was observed with *F. virguliforme* populations at midseason (Fig. 2.4). Populations at all environments except for Rossville and Topeka 2009 were not statistically different (Fig. 2.1B). At Rossville and Topeka 2009, genotypes susceptible to SDS had higher levels of *F. virguliforme* than those resistant. Also, at Rossville 2009, KS3406RR had higher soil levels than COM3.

Significant differences were observed between *F. virguliforme* root populations by environment (Table 2.4). Root populations at Manhattan 2009, Morganville 2009, and Rossville 2009 were significantly greater than the other four environments (Table 2.8). Root populations were lowest at Topeka 2008 with 2.19 cfu/g of root tissue. A significant genotype effect or genotype by environment interaction was not observed with root population (Tables 2.4 and 2.9).

Soybean Cyst Nematode and *F. virguliforme* Populations

Significant correlations were observed between SCN and *F. virguliforme* populations at planting (Table 2.10). The higher the levels of SCN the higher the levels of *F. virguliforme* and vice versa. SCN reproductive factor was negatively correlated to *F. virguliforme* populations at planting and midseason. *F. virguliforme* root populations at harvest and SCN soil populations at midseason and harvest were also negatively correlated.

Disease Development

Foliar symptoms of SDS were observed at all environments except at Topeka in 2008 (Fig. 2.1C). Severity of foliar symptoms is linked to the value of AUDPC with more severe foliar symptoms resulting in a higher AUDPC. It was highly significant across environments. Symptoms were least severe at Manhattan 2008, Manhattan 2009, and Rossville 2009 and most severe at Rossville 2008, Morganville 2009, and Topeka 2009 (Table 2.8). With the exception of Manhattan 2008, where symptoms first occurred later in the growing season at the R7 growth stage, and Rossville 2009, where symptoms occurred at the R6 growth stage, symptoms first occurred at approximately the R5 growth stage. As time progressed, the severity of foliar symptoms and the disease index increased.

Genotypes differed significantly in AUDPC (Table 2.9). KS3406RR had the highest AUDPC while COM3 had the second highest AUDPC. Genotypes COM1 and COM2 had the lowest AUDPC and were statistically similar.

There was also a significant genotype by environment interaction with AUDPC (Fig. 2.4). At Rossville 2008, Manhattan 2009, Morganville 2009, and Topeka 2009, genotypes susceptible to SDS had a higher AUDPC than those resistant (Fig. 2.1C). Furthermore, at Rossville 2008, Morganville 2009, and Topeka 2009, KS3406RR had a higher AUDPC than COM3.

AUDPC and Disease Variables

There were significant positive correlations between AUDPC and *F. virguliforme* soil population at harvest and SCN soil population at planting (Table 2.10). The correlation coefficient between AUDPC and *F. virguliforme* population at harvest was 0.34 ($p \leq 0.05$). AUDPC and SCN population at planting had a also had a positive correlation coefficient of 0.45 ($p \leq 0.01$).

Yield

Seed yield was highly significant across environments (Table 2.4). Yields ranged from 4811 kg ha^{-1} at Topeka 2008 to 2513 kg ha^{-1} at Morganville 2008 (Fig. 2.2). Genotypes also differed significantly in seed yield (Table 2.4). The highest yielding entry, COM2, had an average yield of 3951 kg ha^{-1} across environments. Genotype KS3406RR had the lowest average yield of 2614 kg ha^{-1}. Genotypes COM1 and COM3 yielded 3623 kg ha^{-1} yield and 3003 kg ha^{-1}, respectively

The performance among the genotypes varied across environments (Table 2.4)

Genotype COM1 was only significantly different from the highest yielding entry at Rossville 2008 and 2009 (Fig. 2.1D). It yielded lowest at Rossville 2009 with 2903 kg ha $^{-1}$ and highest at Topeka 2008 with 4952 kg ha $^{-1}$. Genotype COM2 was either the highest yielding or not significantly different from the highest yielding genotype(s) at each environment. Its lowest seed yield was at Topeka 2009 with 3407 kg ha $^{-1}$ and its highest at Topeka 2008 with 5214 kg ha $^{-1}$. Genotype COM3 was not significantly different from the lowest yielding genotype(s) at Manhattan 2008, Topeka 2008, Morganville 2008, and Topeka 2008. It was not significantly different from the highest yielding entries at Rossville 2009. The genotype's lowest performance was 1781 kg ha $^{-1}$ at Morganville 2008, while its highest was 4623 kg ha $^{-1}$ at Topeka 2008. KS3406RR was the lowest yielding or not significantly different from the lowest yielding genotype(s) at each environment. The lowest yield of KS3406RR was 1519 kg ha $^{-1}$ at Rossville 2008. The highest yield was 4448 kg ha $^{-1}$ at Topeka 2008. Genotypes resistant to SDS yielded higher than those susceptible at all environments except for Rossville 2009 (Fig. 2.1E). Additionally, at Rossville 2008 and Manhattan 2009 there were significant differences between the yields of KS3406RR and COM3.

Impact of Disease Variables on Yield

There was a significant negative correlation between yield and *F. virguliforme* root populations n= -0.48 ($p \leq 0.01$) (Table 2.10). A larger negative correlation was observed between yield and AUDPC with a correlation coefficient of -0.76 ($p \leq 0.01$). This correlation is higher with genotypes susceptible to SDS and lower with genotypes resistant to SDS (Table A.6). At locations where SDS symptoms were observed, a downward trend can be observed when yield is plotted against AUDPC (Fig. 2.3). A high AUDPC led to decreased yields.

Discussion

Genotypes in this study were selected to encompass different combinations of resistance and susceptibility to SDS and SCN using disease ratings and female indices determined by the Department of Agronomy at Kansas State University in the 2007 Kansas Soybean Performance Test (Table 2.1). Genotype COM1 was initially classified as resistant to both SDS and SCN while COM2 was resistant to SDS and displayed moderate resistance to SCN. Genotype COM3 was susceptible to SDS and displayed moderate resistance to SCN, and KS3406RR was susceptible to both diseases. When selecting genotypes for the experiment, SDS field ratings and SCN female indices based on race 3 and race 4 were known. Female indices based on genotype reaction to specific SCN populations at the environments used in this study were not. Genotypes were similar in maturity and there were no problems with lodging at any location (Table 2.11). However, there were some height differences among genotypes.

Symptoms of SDS occurred at all environments on all genotypes except at Topeka 2008 and Rossville 2009 (Fig. 2.1D). *F. virguliforme* and SCN soil populations were observed at both of these environments (Tables 2.5 and 2.8). At these environments, warm and dry weather conditions prevailed at mid pod-fill, and disease did not develop. Symptoms were observed in the fields surrounding both experiments. These fields were not irrigated, and it is possible that irrigation would have enhanced the development of symptoms at these environments.

Resistance to SDS has been classified as partial, polygenic, and environmentally dependent (Njiti et al., 1996). Since SDS resistance is not complete, any genotype can display symptoms if disease conditions are optimum (Hershman et al., 1990; Njiti et al., 1996). At environments where SDS occurred, symptoms were also observed on SDS resistant genotypes;

however, they had much lower AUDPC than susceptible genotypes (Fig. 2.1C). The highest yielding genotypes were resistant to SDS and grown in environments where AUDPC was low.

Genotypes were selected prior to calculating their specific female indices based on reactions to the SCN populations at the environments used in this study. Once these specific indices were determined, some differences were observed between the general indices and those based on an environment's specific SCN population. Initially, COM1 was classified as resistant, COM2 as moderately resistant, COM3 as moderately resistant, and KS3406RR as susceptible to SCN (Table 2.1) (Schmitt and Shannon, 1992). At Manhattan 2009 and Morganville 2009, COM1 was moderately resistant and COM3, moderately susceptible (Table 2.3). At Rossville 2009, COM1 and COM2 were susceptible. This could possibly account for the low yield of COM1 at Rossville 2009 where it was statistically similar to KS3406RR, the lowest yielding genotype. Genotype COM3 was moderately susceptible based on the data obtained from the SCN population there. At Topeka 2009, COM1 was moderately resistant. Genotype KS3406RR continued to be susceptible at all environments. The female indices calculated for each genotype at each 2009 environment can be applied to the corresponding 2008 environment as they were in close proximity to each other increasing the likelihood that SCN populations were similar.

Generally, SDS-resistant and -susceptible genotypes were the highest and lowest yielding genotypes regardless of their resistance or susceptibility to SCN, respectively (Figs. 2.1D and 2.1E). Genotype COM2 yielded the highest or was not significantly different from the highest yielding genotype at all locations (Fig. 2.1D). Genotype COM1 significantly differed from the highest yielding entry at only two environments. Both COM1 and COM2 had low SDS ratings and a SCN resistance classification of moderately resistant according to calculated female index across all environments with the exception of Rossville where they were susceptible (Figure

2.1C and Table 2.3). This can help explain their relatively similar performances. The two genotypes susceptible to SDS, COM3 and KS3406RR were the lowest yielding genotypes at all environments except for Rossville 2009 (Fig. 2.1D). However, COM3 was sometimes not statistically different from one of the higher yielding genotypes. Genotype KS3406RR was either the lowest performing genotype or was statistically similar to COM3. However, while KS3406RR was susceptible to SDS and SCN across environments, the female index of COM3 fluctuated across environments (Table 2.3). The genotype was classified as either moderately resistant or moderately susceptible to SCN depending on environment.

When SDS symptoms were present, there was an increase in performance of SDS-resistant genotypes compared to SDS-susceptible genotypes (Fig. 2.1E). At Rossville 2008, where SDS symptoms were present, COM2, the highest yielding genotype, yielded 140 percent more than the lowest yielding genotype, KS3406RR. At Topeka 2008, where SDS symptoms were not present, COM2 yielded 15 percent more than KS3406RR.

Similar to previous studies, soil populations of *F. virguliforme* remained relatively constant at planting and midseason and increased at harvest (Table 2.8) (Rupe et al., 1997). At each sampling time, populations were slightly lower than previous observations of *F. virguliforme* soil levels (Roy et al., 1997, Rupe et al., 1997). While SCN final populations were over the damage threshold of 300 eggs and J2/100 cc soil for sandy soils, they were not high enough to warrant switching to non-host crops as a method of control (Jardine and Todd, 2001).

Yield was not negatively affected by *F. virguliforme* or SCN populations with the exception of *F. virguliforme* root population (Table 2.10). While some isolates are better root colonizers others are better at translocating toxins. Higher levels of *F. virguliforme* in the root increase the probability that more aggressive isolates with a high level of toxin translocation

capability are present, possibly leading to more disease and decreased yields (Li et al., 2009). As expected, yield was also negatively correlated to AUDPC. Thus, the greater the disease severity, the lower the yield.

Previous research has suggested both the presence and absence of a relationship between SDS and SCN (Gao et al., 2006; Hershman et al., 1990; Roy et al., 1997., Scherm and Yang, 1998; Xing and Westphal, 2006). In this study, there was a correlation observed between *F. virguliforme* and SCN soil populations at planting (Table 2.10). *F. virguliforme* has the ability to overwinter within SCN cysts, so elevated cyst levels could result in higher fungal levels at planting (McLean and Lawrence, 1993; Rupe et al., 1993). SCN may also increase fungal levels by providing wounds for *F. virguliforme* to enter the plant and reproduce (Roy et al., 1989; Scherm et al., 1998). Additionally, SCN populations at midseason and planting were negatively affected by *F. virguliforme* root populations. The possible decreased levels of SCN these times can be attributed to the root necrosis caused by SDS, which limits the food supply for the nematode (Rupe et al., 1993; Scherm et al., 1998)

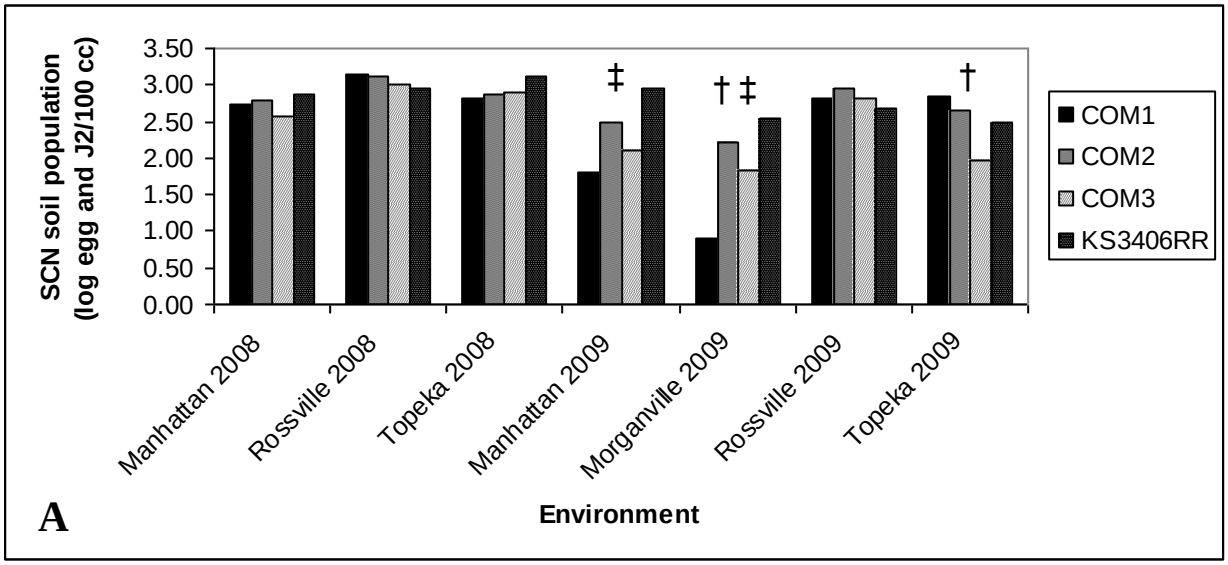
SCN levels at planting levels were also positively correlated with AUDPC of SDS suggesting the initial presence of SCN may enhance SDS disease development with the possibility of further lowering yields. However, in a previous study concerning the occurrence of SDS in East-Central Illinois, SCN was detected at every environment, but the soil populations of SCN did not differ from unaffected and diseased areas of the fields (Hartman et al., 1995) *F. virguliforme* soil levels at harvest were also positively correlated to AUDPC. High soil levels of *F. virguliforme* lead to increased SDS symptoms (Scherm et al., 1998).

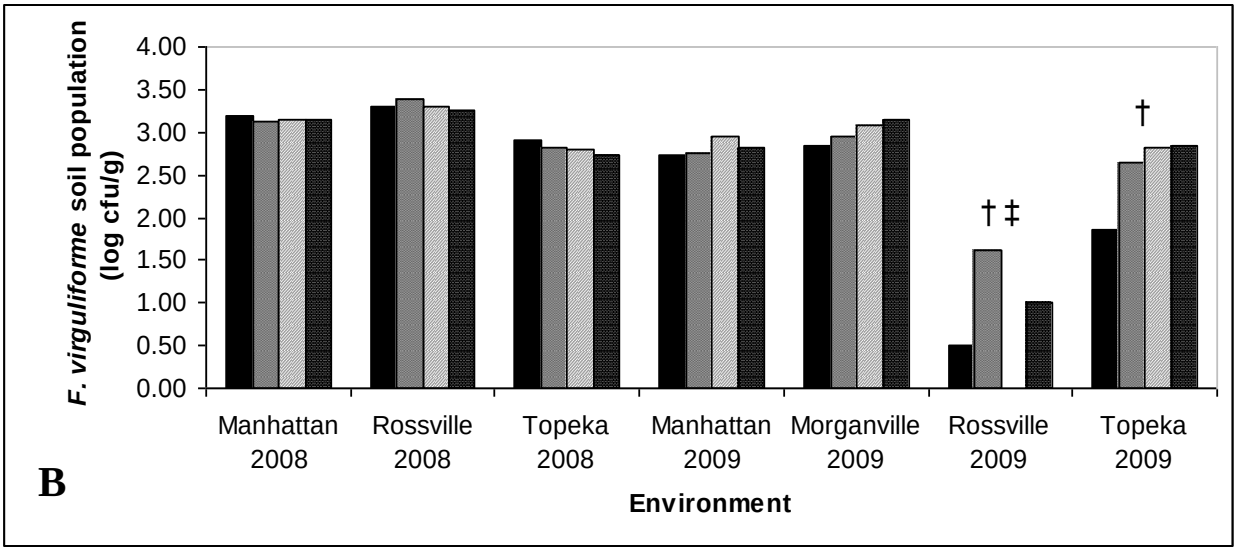
Interactions between SDS and SCN, differences in genotype performance, and yield losses due to disease were observed across environments. It is important to continue research on

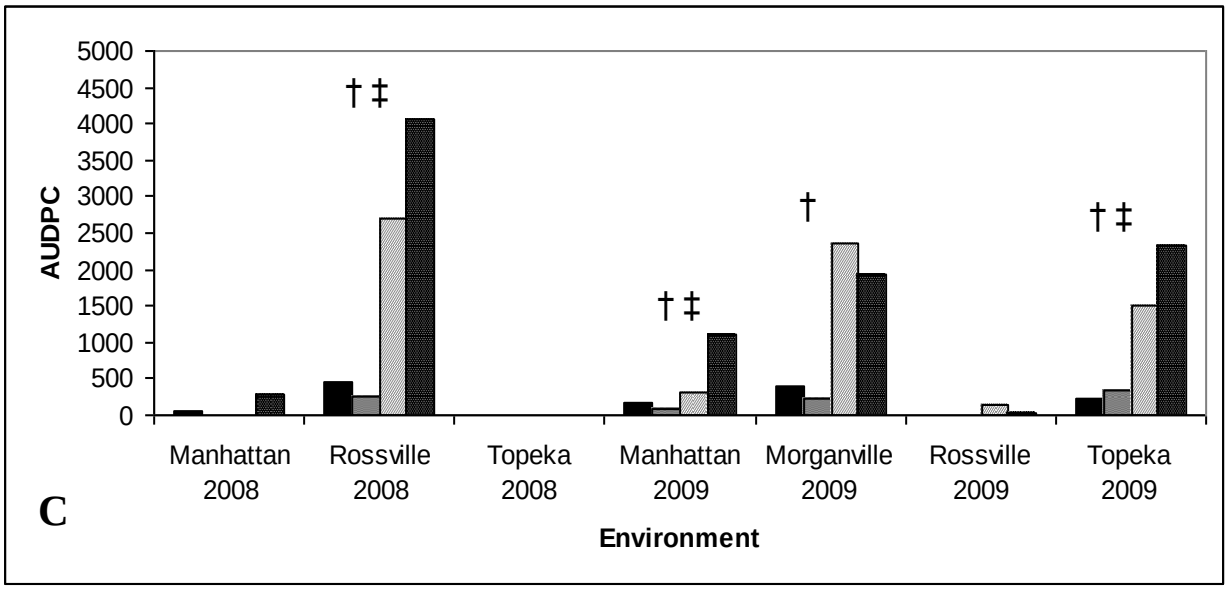
SDS and SCN and continue to develop resistant varieties. In fields with a history of SDS, it is important for producers to manage both SDS and SCN in order to maximize yields and limit losses. This can be achieved through genetic resistance.

Figures

Fig. 2.1. SCN soil populations at harvest (A) *F. virguliforme* soil populations at midseason (B) AUDPC (C), seed yield (D), and seed yield relative to KS3406RR (E) for four soybean genotypes evaluated across seven Kansas environments in 2008 and 2009.



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Morganville 2009
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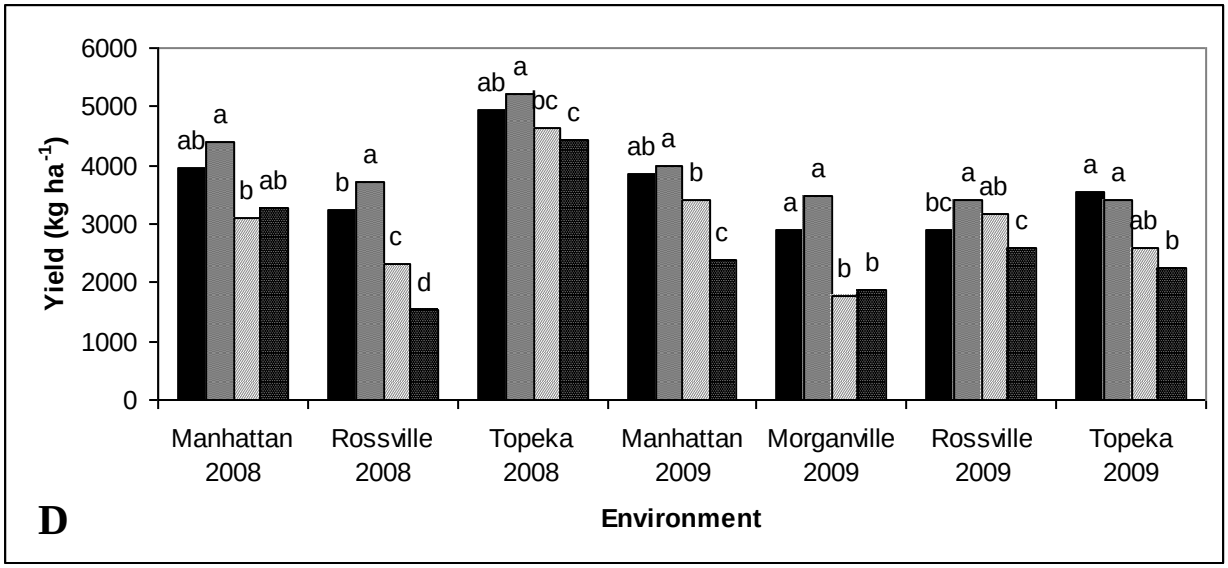
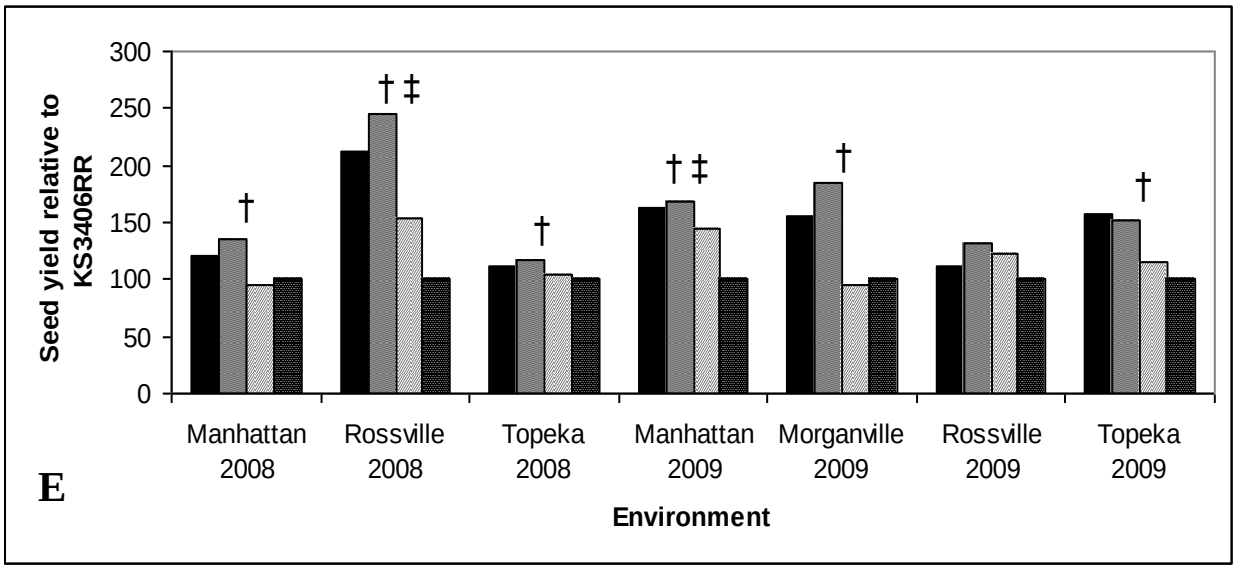
Topeka 2009
Environment
C


6000
5000
4000
3000
2000
1000
0
Yield (kg ha-1)
ab a b ab
b a c d
ab a bc c
ab a b c
a a b b
bc a ab c
a a ab b
Manhattan 2008
Rossville 2008
Topeka 2008
Manhattan 2009
Morganville 2009
Rossville 2009
Topeka 2009
Environment
D

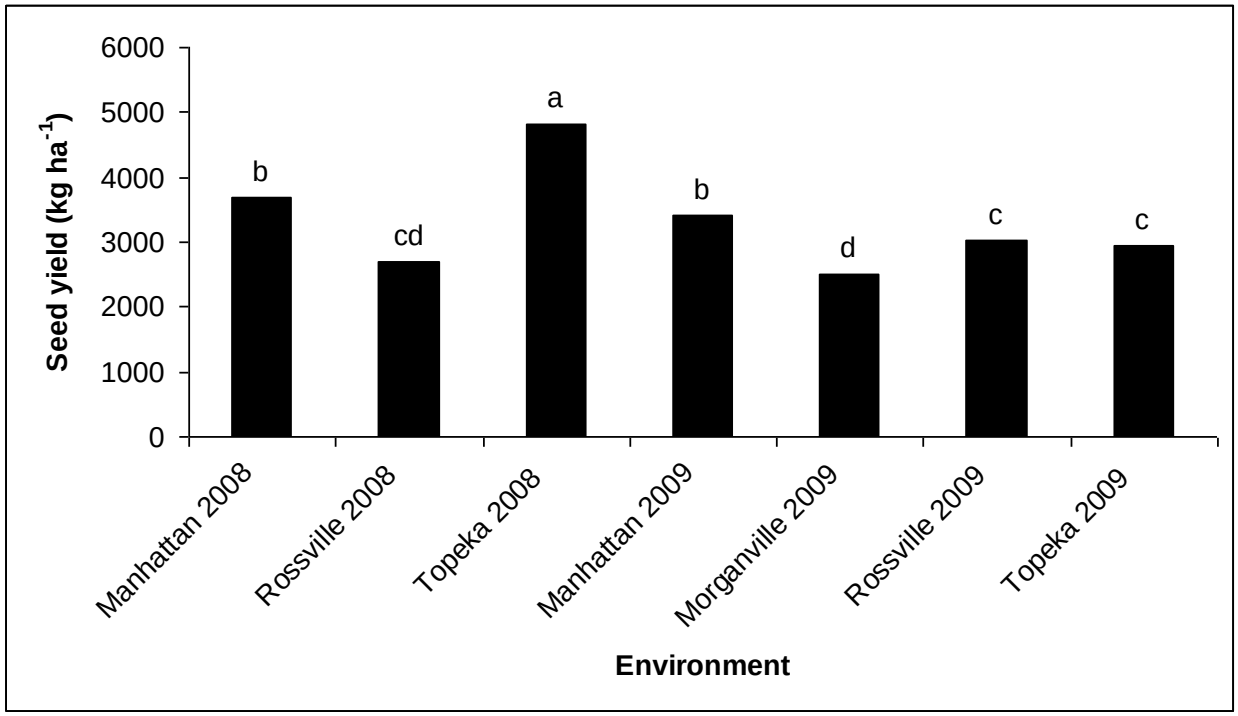


†Within an environment, genotypes resistant to SDS (COM1 and COM2) are significantly different from genotypes susceptible to SDS (COM3 and KS3406RR) at $p \leq 0.05$.

‡Within an environment, the susceptible to SDS and displaying moderate resistance to SCN (COM3) is significantly different from the genotype susceptible to SDS and SCN (KS3406RR) at $p \leq 0.05$.

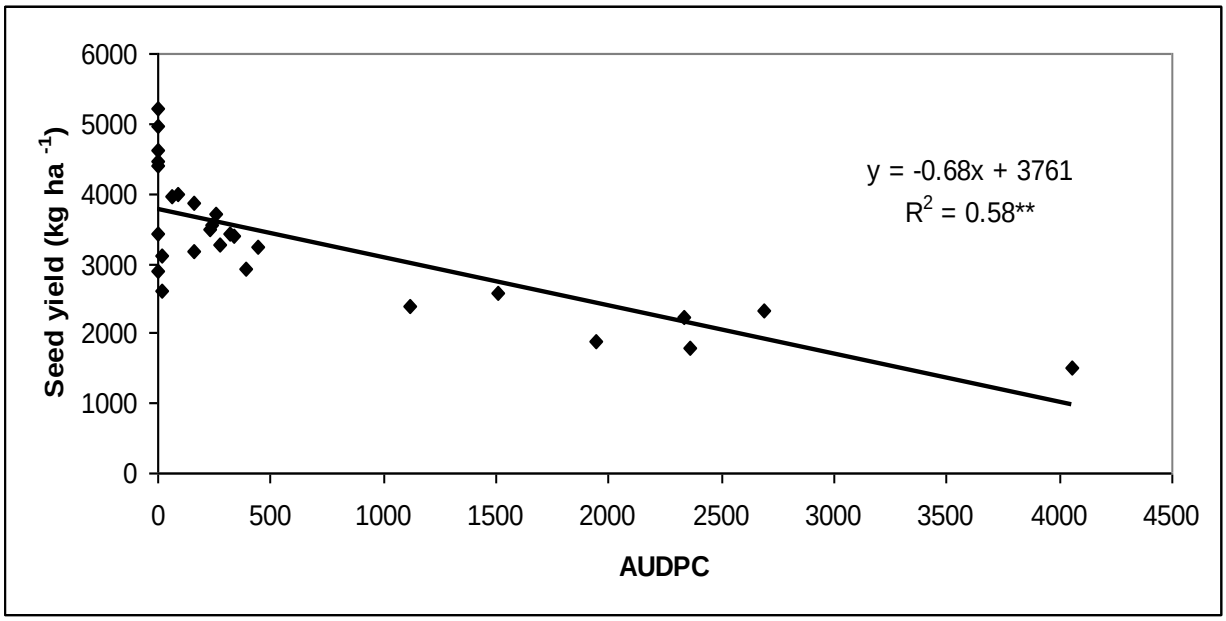
†Bars within environments with the same letter are not significantly different at $p \leq 0.05$.

Fig. 2.2. Total average seed yield of all four genotypes at seven Kansas environments in 2008 and 2009.



†Bars with the same letter are not significantly different at $p \leq 0.05$.

Fig. 2.3. Seed yield versus AUDPC. (♦) represents the average values of one of four genotypes at one of seven Kansas environments.



**, significant at $p \leq 0.01$.

Tables

Table 2.1. SDS and SCN resistance for four soybean genotypes from the 2007 Kansas Soybean Performance test. Performance test data can be found at http://www.agronomy.ksu.edu/extension/DesktopDefault.aspx?tabid=94.

		SCN Resistance	
Genotype	SDS Resistance	Race 3	Race 4
COM1	R†	R	MR
COM2	MR	MR	S
COM3	S	MR	MS
KS3406RR	S	S	S

†R=resistant, MR=moderately resistant, MS=moderately susceptible, S=susceptible

Table 2.2. Types of SCN on PI lines at four Kansas locations in 2009 (Rzodkiewicz, 2010).

Location	No. females on Lee 74	Female index							HG type
		1 PI 548402	2 PI 88788	3 PI 90763	4 PI 437654	5 PI 209332	6 PI 89772	7 PI 548316	
Manhattan	131	27.2	8.0	13.8	5.0	6.3	18.2	12.7	1.3.6.7
Morganville	40	4.2	31.9	0.0	1.1	19.6	2.5	26.5	2.5.7
Rossville	173	14.5	28.3	4.2	0.1	56.0	5.9	37.9	1.2.5.7
Topeka	78	8.8	18.6	4.2	0.2	4.8	3.2	50.3	2.7

Table 2.3. SCN female indices of four soybean genotype at four Kansas locations in 2009 (mean ± standard error).

	Female index			
Genotype	Manhattan	Morganville	Rossville	Topeka
COM1	29.5 ± 17.0	15.2 ± 17.4	101.1 ± 90.0	14.7 ± 11.5
COM2	23.2 ± 12.2	16.8 ± 10.1	81.5 ± 49.7	11.8 ± 12.5
COM3	31.2 ± 16.8	39.2 ± 53.1	55.8 ± 43.1	24.9 ± 15.7
KS3406RR	99.9 ± 32.2	99.8 ± 71.0	99.3 ± 60.4	99.9 ± 46.5
PI88788	1.9 ± 1.5	0.5 ± 0.8	1.3 ± 2.9	0.3 ± 0.5
LSD (0.05)	29.3	44.5	74.8	29.7

Table 2.4. *P* values for SCN soil populations at planting (SCN Pi), midseason (SCN Pm), and harvest (SCN Pf), SCN reproductive factor (SCN Rf), *F. virguliforme* populations at planting (FVPl), midseason (FVMd), and harvest (FVHv), *F. virguliforme* root populations (FVRt), AUDPC, and seed yield from analyses of variance.

		Variable									
Source	d.f.	SCN Pi	SCN Pm	SCN Pf	SCN Rf	FVPl	FVMd	FVHv	FVRt	AUDPC	Yield
Environment	6	<.0001**	<.0001**	<.0001**	<.0001**	<.0001**	<.0001**	<.0001**	<.0001**	<.0001**	<.0001**
Genotype	3	0.2895	0.1489	0.0106*	0.9609	0.0238*	0.0767	0.9148	0.1419	<.0001**	<.0001**
Gen x Env	18	0.4067	0.2414	0.0252*	0.1372	0.0009**	0.0127*	0.996	0.7349	<.0001**	0.0183*

*, ** indicates significant difference at $p \leq 0.05$ and $p \leq 0.01$, respectively.

Table 2.5. SCN populations at planting, midseason, and harvest and reproductive factor for seven Kansas environments in 2008 and 2009.

Year	Environment	Planting	Midseason	Harvest	SCN Rf
		-----log eggs and J2/100 cc-----			
2008	Manhattan	1.78 d†	2.55 cd	2.75 ab	7.6a
	Rossville	2.59 a	3.00 a	3.05 a	3.7bc
	Topeka	2.28 bc	2.82 ab	2.82 a	5.8ab
2009	Manhattan	2.31 b	2.44 de	2.33 c	2.3c
	Morganville	2.34 ab	2.33 e	1.87 d	0.9c
	Rossville	2.05 c	2.70 bc	2.82 a	8.3a
	Topeka	2.41 ab	2.63 cd	2.42 cb	3.9bc
LSD(0.05)		0.25	0.20	0.34	3.2

†Means followed by the same letter, within the same column, are not significantly different at $p \leq 0.05$.

Table 2.6. SCN populations at planting, midseason, and harvest and reproductive factor across all environments separated by four soybean genotypes.

	Soybean cyst nematode measurements			
	Soil population			
Genotype	Planting	Midseason	Harvest	SCN Rf
	-----log eggs and J2/100 cc-----			
COM1	2.26 a	2.71 a	2.40 b	4.3a
COM2	2.29 a	2.65 a	2.72 a	4.8a
COM3	2.14 a	2.54 a	2.46 b	4.7a
KS3406RR	2.31 a	2.66 a	2.80 a	4.6a
LSD(0.05)	0.19	0.18	0.26	2.4

†Means followed by the same letter, within the same column are not significantly at $p \leq 0.05$.

Table 2.7. Planting, midseason, and harvest proportions of *F. virguliforme* to total fungal and *F. virguliforme* to total *Fusarium* soil populations of four soybean genotypes at seven Kansas environments in 2008 and 2009.

			F. virguliforme cfu/g soil/ total fungal cfu/g soil			*F. virguliforme* cfu/g soil/ total *Fusarium* cfu/g soil		
Year	Environment	Genotype	Planting	Midseason	Harvest	Planting	Midseason	Harvest
2008	Ashland	COM1	0.02	0.01	0.03	0.12	0.10	0.18
		COM2	0.02	0.02	0.03	0.15	0.08	0.15
		COM3	0.02	0.01	0.03	0.20	0.10	0.21
		KS3406RR	0.02	0.01	0.02	0.13	0.09	0.15
	Rossville	COM1	0.06	0.03	0.05	0.26	0.21	0.33
		COM2	0.06	0.03	0.03	0.23	0.14	0.18
		COM3	0.05	0.04	0.06	0.23	0.17	0.25
		KS3406RR	0.05	0.03	0.06	0.25	0.14	0.23
	Topeka	COM1	0.03	0.01	0.01	0.16	0.05	0.07
		COM2	0.03	0.01	0.01	0.15	0.03	0.05
		COM3	0.02	0.01	0.02	0.10	0.04	0.09
		KS3406RR	0.02	0.01	0.01	0.10	0.03	0.06
2009	Ashland	COM1	0.01	0.01	0.03	0.06	0.04	0.14
		COM2	0.01	0.02	0.03	0.06	0.06	0.13
		COM3	<.01	0.01	0.04	0.02	0.03	0.14
		KS3406RR	0.01	0.01	0.03	0.05	0.05	0.13
	Morganville	COM1	0.02	0.02	0.06	0.05	0.05	0.12
		COM2	0.02	0.03	0.05	0.08	0.06	0.11
		COM3	0.02	0.02	0.05	0.05	0.06	0.13
		KS3406RR	0.02	0.03	0.05	0.10	0.08	0.14
	Rossville	COM1	0.01	<.01	0.01	0.03	<.01	0.04
		COM2	0.01	<.01	0.04	0.04	<.01	0.10
		COM3	0.01	0.01	0.02	0.06	0.01	0.04
		KS3406RR	0.03	<.01	0.01	0.09	<.01	0.03
	Topeka	COM1	<.01	0.01	0.03	0.06	0.02	0.05
		COM2	0.01	0.03	0.03	0.07	0.06	0.07
		COM3	0.01	0.01	0.04	0.03	0.04	0.10
		KS3406RR	0.04	0.02	0.06	0.19	0.04	0.14

Table 2.8. *F. virguliforme* soil populations at planting, midseason, and harvest, root population at harvest, and AUDPC for seven Kansas environments in 2008 and 2009.

		F. virguliforme variables				
		Soil population			Root population	
Year	Environment	Planting	Midseason	Harvest	Harvest	AUDPC
		----------LOG cfu/g----------				
	Manhattan	2.71 b	3.15 ab	3.44 ab	3.51 bc	88.3 d
2008	Rossville	3.00 a	3.32 a	3.50 a	3.19 bc	1863.6 a
	Topeka	2.71 b	2.82 cd	3.04 c	2.19 d	0.0 d
	Manhattan	2.71 b	2.81 cd	3.24 bc	4.07 a	421.1 c
2009	Morganville	2.84 ab	3.00 bc	3.22 bc	3.67 ab	1230.9 b
	Rossville	2.22 c	0.78 e	2.40 d	3.77 ab	43.1 d
	Topeka	2.78 ab	2.54 d	3.11 c	3.57 bc	1106.6 b
LSD(0.05)‡		0.24	0.31	0.23	0.43	242.7

†Means followed by the same letter, within the same column, are not significantly different at $p \leq 0.05$.

Table 2.9. *F. virguliforme* soil populations at planting, midseason, and harvest, *F. virguliforme* root population, and AUDPC of SDS for each of four soybean genotypes over all environments.

	Fusarium virguliforme variables				
	Soil population			Root population	
Genotype	Planting	Midseason	Harvest	Harvest	AUDPC
	----------log cfu/g----------				
COM1	2.57 b	2.45 b	3.14 a	3.47 a	184 c
COM2	2.66 ab	2.59 ab	3.17 a	3.21 a	131 c
COM3	2.81 a	2.76 a	3.10 a	3.46 a	1007 b
KS3406RR	2.80 a	2.71 a	3.13 a	3.57 a	1393 a
LSD(0.05)	0.18	0.23	0.18	0.37	206

†Means followed by the same letter, within the same column, are not significantly different at $p \leq 0.05$.

Table 2.10. Pearson's correlation coefficients for yield, *F. virguliforme* soil populations at planting (FVPl), midseason (FVMd), and harvest (FVHv), *F. virguliforme* root population (FVRt), SCN soil populations at planting (SCN Pi), midseason (SCN Pm), and harvest (SCN Pf), SCN reproductive factor (SCN Rf), and AUDPC (n=28).

	Variables								
Variables	FVPl	FVMd	FVHv	FVRt	SCN Pi	SCN Pm	SCN Pf	SCN Rf	AUDPC
Yield	0.03	0.03	-0.06	-0.48**	-0.32	0.12	0.07	-0.09	-0.76**
FVPl		0.69**	0.56*	-0.15	0.35*	0.06	-0.08	-0.40*	0.25
FVMd			0.91**	-0.20	0.25	-0.05	-0.10	-0.46*	0.32
FVHv				-0.11	0.18	0.01	-0.06	-0.25	0.34*
FVRt					-0.12	-0.53**	-0.37*	0.21	0.09
SCN Pi						0.30	-0.12	-0.57**	0.45**
SCN Pm							0.72**	0.18	-0.01
SCN Pf								0.35	0.08
SCN Rf									-0.19

*, ** indicates significant difference at $p \leq 0.05$ and $p \leq 0.01$, respectively.

Table 2.11. Height, maturity, and lodging of four soybean genotypes at seven Kansas environments in 2008 and 2009.

Year	Environment	Entry	Height†	Maturity‡	Lodging§
			---cm---		
2008	Ashland	COM1	81.3	34.0	1.0
		COM2	99.8	39.0	1.8
		COM3	81.3	36.0	1.0
		KS3406RR	82.6	31.0	1.0
		LSD(0.05)	5.8	1.1	0.4
	Rossville	COM1	79.5	18.0	2.0
		COM2	98.6	25.0	2.8
		COM3	88.4	23.0	1.5
		KS3406RR	72.4	16.0	1.5
		LSD(0.05)	16.0	0.9	0.9
	Topeka	COM1	105.4	26.0	2.0
		COM2	113.8	34.0	2.0
		COM3	98.6	35.0	1.0
		KS3406RR	92.7	26.0	2.0
		LSD(0.05)	2.8	2.1	0.0
2009	Ashland	COM1	97.3	18.0	1.0
		COM2	113.8	27.0	1.3
		COM3	102.4	23.0	1.3
		KS3406RR	88.9	11.0	2.0
		LSD(0.05)	8.6	6.2	0.5
	Morganville	COM1	88.4	19.0	1.3
		COM2	109.2	22.0	2.8
		COM3	88.9	8.0	1.0
		KS3406RR	83.3	8.0	1.8
		LSD(0.05)	9.9	1.7	0.8
	Rossville	COM1	96.5	16.0	1.3
		COM2	116.8	28.0	2.5
		COM3	103.6	18.0	1.5
		KS3406RR	88.9	15.0	2.0
		LSD(0.05)	14.2	3.8	0.9
	Topeka	COM1	105.4	17.0	1.5
		COM2	113.8	18.0	1.8
		COM3	102.4	13.0	1.8
		KS3406RR	92.7	11.0	3.3
		LSD(0.05)	9.1	4.0	0.8

†Height is measured as distance in centimeters from soil line to top of main stem.
‡Maturity is measured as days after September 1st.
§Lodging is measured on a scaled from 1 to 5 with 1 = all plants erect, and 5 = all plants prostrate.

References

Aoki, T., O'Donnell, K., Homma, Y., and Lattanzi, A. R. 2003. Sudden-death syndrome is caused by two morphologically and phylogenetically distinct species within a *Fusarium solani* species complex—*F. virguliforme* in North America and *F. tucumaniae* in South America. Mycologia 95(4):660-684.

Fehr, W. R., Cavines, C. E., Burmood, D. T., and Pennington, J. S. 1971. Stages of development descriptions for soybeans, *Glycine max* (L.) Merrill. Crop Science 11: 929-931.

Gao, X., Jackson, T. A., Hartman, G. L., and Niblack, T. L. 2006. Interactions between the soybean cyst nematode and *Fusarium solani* f. sp. *glycines* based on greenhouse factorial experiments. Phytopathology 96:1409-1415.

Hershman, D. E., Hendrix, J. W., Stuckey, R. E., Bachi, P. R., and Henson, G. 1990. Influence of planting date and cultivar on soybean sudden-death syndrome in Kentucky. Plant Disease 74:761-766.

Hirrel, M. C. 1983. Sudden death syndrome of soybean: A disease of unknown etiology. Phytopathology 73:501-502.

Jardine, D. J. and Todd, T. C. 2001. Soybean cyst nematode. Kansas State University, Manhattan, KS.

Li, S., Hartman, G. L., and Chen, Y. 2009. Evaluation of aggressiveness of *Fusarium virguliforme* isolates that cause soybean sudden death syndrome. Journal of Plant Pathology. 91:77-86.

McLean, K. S. and Lawrence, G. W. 1993. Interrelationship of *Heterodera glycines* and *Fusarium solani* in sudden death syndrome of soybean. Journal of Nematology 25:434-439.

McLean, K. S. and Lawrence, G. W. 1995. Development of *Heterodera glycines* as as affected by *Fusarium solani* the causal agent of sudden death syndrome of soybean. Journal of Nematology. 27:70-77.

Nash, S. M. and Snyder W. C. 1962. Quantitative estimations by plate counts of propagules of the bean root rot *Fusarium* in field soils. Phytopathology 52:567-572.

Njiti, V. N., Myers, O.M., Jr., Schroeder, D., and Lightfoot, D. A. 2003. Roundup Ready soybean: glyphosate effects on *Fusarium solani* root colonization and sudden death syndrome. Agronomy Journal 95:1140-1145.

Njiti, V. N., Shenaut, M. A., Suttner, R. J., Schmidt, M. E., and Gibson, P. T. 1996. Soybean response to sudden death syndrome: Inheritance influenced by cyst nematode resistance in Pyramid x Douglas progenies. Crop Science 36:1165-1170.

Roy, K. W., Hershman, D. E., Rupe, J. C, and Abney, T. S. 1997. Sudden death syndrome of soybean. Plant Disease 81(10):1100-1111.

Roy, K. W., Lawrence, G. W., Hodges, H. H., and Killebrew, J. F. 1989. Sudden death syndrome of soybean: *Fusarium solani* as incitant and relation of *Heterodera glycines* to disease severity. Phytopathology 79:191-197.

Rupe, J. C. 1989. Frequency and pathogenicity of *Fusarium solani* recovered from soybeans with sudden death syndrome. Plant Disease 73:581-584.

Rupe, J. C., Correll, J. C., Guerber, J. C., Becton, C. M., Cummings, M. S., Gbur, J. J, Yount, P.A. 2001. Differentiation of the sudden-death syndrome pathogen of soybean *Fusarium solani* f. sp. *glycines*, from other isolates of *F. solani* based on cultural morphology, pathogenicity, and mitochondrial DNA restriction fragment length polymorphisms. Canadian Journal of Botany 79:829-855.

Rupe, J. C, and Hartman, G. L. 1999. Sudden death syndrome. In: Hartman GL, Sinclair JB, and Rupe JC (eds) Compendium of soybean disease. APS Press, St Paul pp 37-39.

Rupe, J. C., Robbins, R. T., and Gbur, E. E., Jr. 1997. Effect of crop rotation on soil population densities of *Fusarium solani* and *Heterodera glycines* and on the development of sudden death syndrome of soybean. Crop Protection 16(6):575-580.

Rupe, J. C., Sabbe, W. F., Robbins, R.T, and Gbur, E. E., Jr. 1993. Soil and plant factors associated with sudden death syndrome of soybean. Journal of Production Agriculture. 6:218-221.

Rzodkiewicz, P. 2010. Characterization of soybean cyst nematode diversity in Kansas. M.S. thesis. Kansas State University, Manhattan.

Schapaugh, W. T., Jr., and Lingenfelser, J. 2007. 2007 Kansas soybean performance tests with soybean varieties. Kansas State University, Manhattan, KS.

Scherm, H., Yang, X. B., and Lundeen, P. 1998. Soil variables associated with sudden death syndrome in soybean fields in Iowa. Plant Disease 82:1152-1157.

Schmitt, D. P and Shannon J. G. 1992. Differentiating soybean responses to *Heterodera glycines* races. Crop Science 32:275-277.

Shaner, G. and Finney, R. E. 1977. The effect of nitrogen fertilization on the expression of slow-mildewing resistance in Knox wheat. Phytopathology 67:1183-1186.

Xing, L. J. and Westphal A. 2006. Interaction of *Fusarium solani* f. sp. *glycines* and *Heterodera glycines* in sudden death syndrome of soybean. Phytopathology 96:763-77.

Appendix A - Supplementary Data

Table A.1. Disease severities (SEV), incidences (INC), and indices (IND) according to environments, genotypes, and replications (Rep) used in this study.

Environment	Genotype	Rep	SEV R5†	INC R5	IND R5	SEV R6	INC R6	IND R6	SEV R6	INC R7	IND R7
Manhattan 2008	COM1	1	0	0	0	0	0	0	0	0	0
Manhattan 2008	COM1	2	0	0	0	0	0	0	3	50	17
Manhattan 2008	COM1	3	0	0	0	0	0	0	0	0	0
Manhattan 2008	COM1	4	0	0	0	0	0	0	3	20	7
Manhattan 2008	COM2	1	0	0	0	0	0	0	0	0	0
Manhattan 2008	COM2	2	0	0	0	0	0	0	0	0	0
Manhattan 2008	COM2	3	0	0	0	0	0	0	0	0	0
Manhattan 2008	COM2	4	0	0	0	0	0	0	2	5	1
Manhattan 2008	COM3	1	0	0	0	0	0	0	0	0	0
Manhattan 2008	COM3	2	0	0	0	0	0	0	2	5	1
Manhattan 2008	COM3	3	0	0	0	0	0	0	2	10	2
Manhattan 2008	COM3	4	0	0	0	0	0	0	2	10	2
Manhattan 2008	KS3406RR	1	0	0	0	0	0	0	4	60	27
Manhattan 2008	KS3406RR	2	0	0	0	0	0	0	4	70	31
Manhattan 2008	KS3406RR	3	0	0	0	0	0	0	4	60	27
Manhattan 2008	KS3406RR	4	0	0	0	0	0	0	4	60	27

Table A.1. Continued.

Environment	Genotype	Rep	SEV R5†	INC R5	IND R5	SEV R6	INC R6	IND R6	SEV R6	INC R7	IND R7
Manhattan 2009	COM1	1	1	1	0	0	0	0	6	30	20
Manhattan 2009	COM1	2	1	5	1	1	5	1	6	40	27
Manhattan 2009	COM1	3	1	10	1	1	10	1	1	10	1
Manhattan 2009	COM1	4	1	1	2	1	15	2	5	15	8
Manhattan 2009	COM2	1	0	0	0	0	0	0	1	5	1
Manhattan 2009	COM2	2	0	0	0	2	5	1	6	20	13
Manhattan 2009	COM2	3	0	0	0	2	1	0	6	25	17
Manhattan 2009	COM2	4	1	15	2	1	15	2	1	15	2
Manhattan 2009	COM3	1	0	0	0	4	5	2	4	30	13
Manhattan 2009	COM3	2	2	5	1	4	30	13	7	40	31
Manhattan 2009	COM3	3	0	0	0	2	15	3	6	65	43
Manhattan 2009	COM3	4	1	15	2	4	15	7	6	25	17
Manhattan 2009	KS3406RR	1	2	15	3	4	70	31	8	60	53
Manhattan 2009	KS3406RR	2	2	5	1	5	30	17	8	70	62
Manhattan 2009	KS3406RR	3	2	15	3	5	50	28	8	75	67
Manhattan 2009	KS3406RR	4	2	50	11	6	75	50	8	75	67
Morganville 2009	COM1	1	0	0	0	2	10	2	2	10	2
Morganville 2009	COM1	2	3	5	2	4	30	13	7	70	54
Morganville 2009	COM1	3	3	1	0	4	40	18	8	40	36
Morganville 2009	COM1	4	1	1	0	2	10	2	6	60	40
Morganville 2009	COM2	1	0	0	0	4	20	9	4	20	9
Morganville 2009	COM2	2	2	1	0	3	15	5	3	25	8
Morganville 2009	COM2	3	0	0	0	2	10	2	4	40	18
Morganville 2009	COM2	4	2	1	0	3	30	10	6	60	40
Morganville 2009	COM3	1	5	70	39	7	90	70	9	90	90
Morganville 2009	COM3	2	5	40	22	7	50	39	9	75	75
Morganville 2009	COM3	3	3	40	13	7	55	43	8	60	53
Morganville 2009	COM3	4	4	40	18	6	80	53	9	70	70
Morganville 2009	KS3406RR	1	4	60	27	8	50	44	9	85	85
Morganville 2009	KS3406RR	2	4	60	27	6	60	40	9	95	95
Morganville 2009	KS3406RR	3	3	25	8	5	20	11	9	90	90
Morganville 2009	KS3406RR	4	3	25	8	6	70	47	8	60	53

Table A.1. Continued.

Environment	Genotype	Rep	SEV R5†	INC R5	IND R5	SEV R6	INC R6	IND R6	SEV R6	INC R7	IND R7
Rossville 2008	COM1	2	2	5	1	2	20	4	2	30	7
Rossville 2008	COM1	3	2	25	6	2	25	6	3	25	8
Rossville 2008	COM1	4	2	35	8	3	35	12	3	35	12
Rossville 2008	COM2	1	3	5	2	3	15	5	3	20	7
Rossville 2008	COM2	2	2	10	2	3	10	3	3	15	5
Rossville 2008	COM2	3	2	10	2	3	25	8	3	25	8
Rossville 2008	COM2	4	2	15	3	4	15	7	4	15	7
Rossville 2008	COM3	1	4	80	36	5	80	44	6	90	60
Rossville 2008	COM3	2	4	60	27	5	70	39	6	75	50
Rossville 2008	COM3	3	4	70	31	5	80	44	6	70	47
Rossville 2008	COM3	4	4	75	33	5	75	42	6	80	53
Rossville 2008	KS3406RR	1	5	95	53	6	95	63	7	95	74
Rossville 2008	KS3406RR	2	5	95	53	6	95	63	7	95	74
Rossville 2008	KS3406RR	3	5	85	47	6	85	57	7	90	70
Rossville 2008	KS3406RR	4	5	85	47	6	85	57	7	85	66
Rossville 2009	COM1	1	0	0	0	0	0	0	0	0	0
Rossville 2009	COM1	2	0	0	0	0	0	0	0	0	0
Rossville 2009	COM1	3	0	0	0	0	0	0	0	0	0
Rossville 2009	COM1	4	0	0	0	0	0	0	0	0	0
Rossville 2009	COM2	1	0	0	0	0	0	0	0	0	0
Rossville 2009	COM2	2	0	0	0	0	0	0	0	0	0
Rossville 2009	COM2	3	0	0	0	0	0	0	0	0	0
Rossville 2009	COM2	4	0	0	0	0	0	0	0	0	0
Rossville 2009	COM3	1	0	0	0	6	30	20	6	30	20
Rossville 2009	COM3	2	0	0	0	0	0	0	0	0	0
Rossville 2009	COM3	3	0	0	0	4	25	11	4	25	11
Rossville 2009	COM3	4	0	0	0	0	0	0	0	0	0
Rossville 2009	KS3406RR	1	0	0	0	0	0	0	0	0	0
Rossville 2009	KS3406RR	2	0	0	0	3	10	3	3	10	3
Rossville 2009	KS3406RR	3	0	0	0	0	0	0	0	0	0
Rossville 2009	KS3406RR	4	0	0	0	0	0	0	0	0	0

Table A.1. Continued.

Environment	Genotype	Rep	SEV R5†	INC R5	IND R5	SEV R6	INC R6	IND R6	SEV R6	INC R7	IND R7
Topeka 2008	COM1	2	0	0	0	0	0	0	0	0	0
Topeka 2008	COM1	3	0	0	0	0	0	0	0	0	0
Topeka 2008	COM1	4	0	0	0	0	0	0	0	0	0
Topeka 2008	COM2	1	0	0	0	0	0	0	0	0	0
Topeka 2008	COM2	2	0	0	0	0	0	0	0	0	0
Topeka 2008	COM2	3	0	0	0	0	0	0	0	0	0
Topeka 2008	COM2	4	0	0	0	0	0	0	0	0	0
Topeka 2008	COM3	1	0	0	0	0	0	0	0	0	0
Topeka 2008	COM3	2	0	0	0	0	0	0	0	0	0
Topeka 2008	COM3	3	0	0	0	0	0	0	0	0	0
Topeka 2008	COM3	4	0	0	0	0	0	0	0	0	0
Topeka 2008	KS3406RR	1	0	0	0	0	0	0	0	0	0
Topeka 2008	KS3406RR	2	0	0	0	0	0	0	0	0	0
Topeka 2008	KS3406RR	3	0	0	0	0	0	0	0	0	0
Topeka 2008	KS3406RR	4	0	0	0	0	0	0	0	0	0
Topeka 2009	COM1	1	0	0	0	2	40	9	2	40	9
Topeka 2009	COM1	2	1	1	0	2	25	6	6	70	47
Topeka 2009	COM1	3	2	5	1	3	35	12	7	30	23
Topeka 2009	COM1	4	1	1	0	2	10	2	2	10	2
Topeka 2009	COM2	1	2	5	1	3	15	5	6	60	40
Topeka 2009	COM2	2	1	20	2	2	15	3	6	40	27
Topeka 2009	COM2	3	3	10	3	3	20	7	6	40	27
Topeka 2009	COM2	4	2	10	2	3	5	2	6	20	13
Topeka 2009	COM3	1	2	5	1	4	30	13	6	40	27
Topeka 2009	COM3	2	4	30	13	4	50	22	9	50	50
Topeka 2009	COM3	3	5	50	28	7	65	51	9	65	65
Topeka 2009	COM3	4	4	35	16	7	50	39	9	60	60
Topeka 2009	KS3406RR	1	1	25	3	3	25	8	9	40	40
Topeka 2009	KS3406RR	2	5	50	28	8	65	58	9	90	90
Topeka 2009	KS3406RR	3	5	65	36	8	85	76	9	90	90
Topeka 2009	KS3406RR	4	4	45	20	8	70	62	9	90	90

Table A.2. *F. virguliforme* populations at planting (FVPl), midseason (FVMd), harvest (FVHv), *F. virguliforme* root populations (FVRt), and AUDPC of SDS according to environments, genotypes, and replications (Rep) used in this study.

Environment	Genotype	Rep	FVPl	FVMd	FVHv	FVRt	AUDPC
Manhattan 2008	COM1	1	400	1200	3400	6000	0
Manhattan 2008	COM1	2	400	2200	3200	4800	167
Manhattan 2008	COM1	3	800	1500	1900	4000	0
Manhattan 2008	COM1	4	500	1400	3400	3400	67
Manhattan 2008	COM2	1	400	900	3000	3800	0
Manhattan 2008	COM2	2	700	2100	3300	800	0
Manhattan 2008	COM2	3	800	2000	2700	4400	0
Manhattan 2008	COM2	4	700	800	3200	3400	11
Manhattan 2008	COM3	1	400	1000	2600	600	0
Manhattan 2008	COM3	2	400	1900	3000	2400	11
Manhattan 2008	COM3	3	1100	1700	3200	7600	22
Manhattan 2008	COM3	4	600	1200	2900	5000	22
Manhattan 2008	KS3406RR	1	300	1000	2400	4000	267
Manhattan 2008	KS3406RR	2	100	1500	2600	1800	311
Manhattan 2008	KS3406RR	3	800	1300	1900	3600	267
Manhattan 2008	KS3406RR	4	800	1800	2000	5400	267
Manhattan 2009	COM1	1	1100	400	1400	5400	155
Manhattan 2009	COM1	2	300	600	2600	13400	234
Manhattan 2009	COM1	3	300	400	1500	10600	77
Manhattan 2009	COM1	4	1400	900	2200	20200	166
Manhattan 2009	COM2	1	400	600	2400	2400	4
Manhattan 2009	COM2	2	300	500	1800	14800	115
Manhattan 2009	COM2	3	300	500	1700	14000	128
Manhattan 2009	COM2	4	100	700	1600	11600	116
Manhattan 2009	COM3	1	700	700	1800	16400	130
Manhattan 2009	COM3	2	500	800	1600	19000	467
Manhattan 2009	COM3	3	600	900	1900	21800	370
Manhattan 2009	COM3	4	1100	1200	1800	15800	296
Manhattan 2009	KS3406RR	1	900	500	1200	9000	982
Manhattan 2009	KS3406RR	2	900	500	1200	6200	746
Manhattan 2009	KS3406RR	3	400	600	1100	19400	1037
Manhattan 2009	KS3406RR	4	700	1300	3300	11400	1714

Table A.2. Continued.

Environment	Genotype	Rep	FVPl	FVMd	FVHv	FVRt	AUDPC
Morganville 2009	COM1	1	700	800	1400	7200	46
Morganville 2009	COM1	2	400	1500	2600	9000	665
Morganville 2009	COM1	3	800	300	2000	6000	514
Morganville 2009	COM1	4	700	600	800	1400	334
Morganville 2009	COM2	1	1100	900	1700	6800	182
Morganville 2009	COM2	2	500	900	1100	800	139
Morganville 2009	COM2	3	100	700	2200	1800	162
Morganville 2009	COM2	4	600	1200	1600	3200	441
Morganville 2009	COM3	1	1500	1300	1300	10600	3529
Morganville 2009	COM3	2	700	1400	2200	15200	2179
Morganville 2009	COM3	3	1000	1200	1800	7000	1623
Morganville 2009	COM3	4	800	1000	1000	4600	2107
Morganville 2009	KS3406RR	1	800	2400	3000	2800	2549
Morganville 2009	KS3406RR	2	1600	1300	1500	8800	2566
Morganville 2009	KS3406RR	3	700	1300	1800	5800	1236
Morganville 2009	KS3406RR	4	600	1000	2100	4600	1423
Rossville 2008	COM1	1	1200	3000	3400	3200	492
Rossville 2008	COM1	2	1100	1500	3500	800	175
Rossville 2008	COM1	3	1000	1400	3100	1200	438
Rossville 2008	COM1	4	800	2700	3400	800	671
Rossville 2008	COM2	1	1000	3100	3800	1600	213
Rossville 2008	COM2	2	1300	2700	3600	600	204
Rossville 2008	COM2	3	1200	1800	2800	200	304
Rossville 2008	COM2	4	700	2500	4600	3000	325
Rossville 2008	COM3	1	1000	1500	3100	1600	2983
Rossville 2008	COM3	2	1200	3300	2100	3800	2358
Rossville 2008	COM3	3	1600	2100	3200	2200	2650
Rossville 2008	COM3	4	700	1600	2400	6200	2775
Rossville 2008	KS3406RR	1	1300	1500	3200	1400	4275
Rossville 2008	KS3406RR	2	800	1900	3100	2200	4275
Rossville 2008	KS3406RR	3	500	1800	2500	2800	3854
Rossville 2008	KS3406RR	4	1100	2200	3600	1200	3825

Table A.2. Continued.

Environment	Genotype	Rep	FVPl	FVMd	FVHv	FVRt	AUDPC
Rossville 2009	COM1	1	200	100	700	8000	0
Rossville 2009	COM1	2	0	0	300	3200	0
Rossville 2009	COM1	3	0	0	200	6000	0
Rossville 2009	COM1	4	400	0	200	7400	0
Rossville 2009	COM2	1	300	100	500	3200	0
Rossville 2009	COM2	2	300	300	300	1200	0
Rossville 2009	COM2	3	400	100	800	3800	0
Rossville 2009	COM2	4	400	0	100	6200	0
Rossville 2009	COM3	1	300	0	2100	12200	400
Rossville 2009	COM3	2	100	0	0	2000	0
Rossville 2009	COM3	3	300	0	600	9800	222
Rossville 2009	COM3	4	300	0	1000	12600	0
Rossville 2009	KS3406RR	1	2400	0	100	14200	0
Rossville 2009	KS3406RR	2	500	100	100	10400	67
Rossville 2009	KS3406RR	3	200	100	500	8200	0
Rossville 2009	KS3406RR	4	500	0	400	4600	0
Topeka 2008	COM1	1	800	900	1600	200	0
Topeka 2008	COM1	2	600	900	300	1400	0
Topeka 2008	COM1	3	700	700	1500	1400	0
Topeka 2008	COM1	4	1000	700	1000	600	0
Topeka 2008	COM2	1	700	800	1200	200	0
Topeka 2008	COM2	2	400	400	1100	0	0
Topeka 2008	COM2	3	500	800	2700	1200	0
Topeka 2008	COM2	4	400	800	1500	0	0
Topeka 2008	COM3	1	700	600	1300	1200	0
Topeka 2008	COM3	2	700	900	500	1400	0
Topeka 2008	COM3	3	600	600	1400	200	0
Topeka 2008	COM3	4	800	500	1100	0	0
Topeka 2008	KS3406RR	1	400	500	1200	800	0
Topeka 2008	KS3406RR	2	500	600	800	200	0
Topeka 2008	KS3406RR	3	600	600	700	200	0
Topeka 2008	KS3406RR	4	600	500	1400	200	0

Table A.2. Continued.

Environment	Genotype	Rep	FVPl	FVMd	FVHv	FVRt	AUDPC
Topeka 2009	COM1	1	400	200	400	0	178
Topeka 2009	COM1	2	900	0	1700	54200	364
Topeka 2009	COM1	3	200	700	1800	17800	363
Topeka 2009	COM1	4	300	200	1600	6200	50
Topeka 2009	COM2	1	400	400	100	1200	369
Topeka 2009	COM2	2	300	500	2000	14400	326
Topeka 2009	COM2	3	400	900	1600	10800	432
Topeka 2009	COM2	4	300	200	2000	25200	222
Topeka 2009	COM3	1	900	800	800	0	406
Topeka 2009	COM3	2	1200	900	2300	28400	1324
Topeka 2009	COM3	3	500	1300	1700	14400	2584
Topeka 2009	COM3	4	400	200	1100	6800	1737
Topeka 2009	KS3406RR	1	200	800	1000	6400	505
Topeka 2009	KS3406RR	2	800	700	3600	26000	2835
Topeka 2009	KS3406RR	3	400	900	1400	12400	3530
Topeka 2009	KS3406RR	4	4800	500	2700	10600	2481

Table A.3. Yield, lodging, height, and maturity data according to environment, genotypes, and replications (Rep) used in this study.

Environment	Genotype	Rep	Yield	Lodging	Height	Maturity
Manhattan 2008	COM1	1	4038.1	1	88.9	34
Manhattan 2008	COM1	2	4118.7	1	83.8	34
Manhattan 2008	COM1	3	3997.8	1	81.3	33
Manhattan 2008	COM1	4	3668.6	1	71.1	33
Manhattan 2008	COM2	1	4098.6	2	101.6	39
Manhattan 2008	COM2	2	4508.4	2	101.6	40
Manhattan 2008	COM2	3	4595.8	2	96.5	39
Manhattan 2008	COM2	4	4414.4	1	99.1	39
Manhattan 2008	COM3	1	4555.5	1	91.4	36
Manhattan 2008	COM3	2	4011.2	1	86.4	36
Manhattan 2008	COM3	3	1921.6	1	83.8	37
Manhattan 2008	COM3	4	1975.4	1	83.8	35
Manhattan 2008	KS3406RR	1	2808.5	1	81.3	32
Manhattan 2008	KS3406RR	2	3346.1	1	83.8	32
Manhattan 2008	KS3406RR	3	3540.9	1	83.8	30
Manhattan 2008	KS3406RR	4	3346.1	1	81.3	30
Manhattan 2009	COM1	1	3964.2	1	101.6	20
Manhattan 2009	COM1	2	3554.4	1	99.1	13
Manhattan 2009	COM1	3	3964.2	1	96.5	23
Manhattan 2009	COM1	4	3950.8	1	91.4	17
Manhattan 2009	COM2	1	4716.7	2	114.3	27
Manhattan 2009	COM2	2	3641.7	1	109.2	24
Manhattan 2009	COM2	3	3493.9	1	111.8	28
Manhattan 2009	COM2	4	4118.7	1	119.4	29
Manhattan 2009	COM3	1	3708.9	2	111.8	26
Manhattan 2009	COM3	2	3225.1	1	99.1	25
Manhattan 2009	COM3	3	3023.6	1	96.5	15
Manhattan 2009	COM3	4	3715.6	1	101.6	27
Manhattan 2009	KS3406RR	1	2109.8	2	83.8	11
Manhattan 2009	KS3406RR	2	2714.5	2	94.0	11
Manhattan 2009	KS3406RR	3	2082.9	2	88.9	11
Manhattan 2009	KS3406RR	4	2600.3	2	88.9	11

Table A.3. Continued.

Environment	Genotype	Rep	Yield	Lodging	Height	Maturity
Morganville 2009	COM1	1	3265.4	2	86.4	20
Morganville 2009	COM1	2	2727.9	1	86.4	19
Morganville 2009	COM1	3	2593.5	1	91.4	19
Morganville 2009	COM1	4	3037.0	1	88.9	19
Morganville 2009	COM2	1	3446.8	2	119.4	23
Morganville 2009	COM2	2	3977.6	3	104.1	23
Morganville 2009	COM2	3	3426.7	3	96.5	23
Morganville 2009	COM2	4	3090.7	3	116.8	19
Morganville 2009	COM3	1	1290.0	1	91.4	8
Morganville 2009	COM3	2	2136.6	1	78.7	8
Morganville 2009	COM3	3	1619.3	1	88.9	8
Morganville 2009	COM3	4	2069.5	1	96.5	8
Morganville 2009	KS3406RR	1	1632.7	2	83.8	7
Morganville 2009	KS3406RR	2	1209.4	1	78.7	8
Morganville 2009	KS3406RR	3	2788.4	2	86.4	8
Morganville 2009	KS3406RR	4	1888.0	2	83.8	8
Rossville 2008	COM1	1	3487.2	2	83.8	18
Rossville 2008	COM1	2	3245.3	2	76.2	18
Rossville 2008	COM1	3	2862.3	2	81.3	19
Rossville 2008	COM1	4	3299.0	2	76.2	18
Rossville 2008	COM2	1	3876.9	3	104.1	25
Rossville 2008	COM2	2	3601.4	3	96.5	24
Rossville 2008	COM2	3	3682.0	3	101.6	25
Rossville 2008	COM2	4	3722.3	2	91.4	25
Rossville 2008	COM3	1	2156.8	2	91.4	22
Rossville 2008	COM3	2	2210.6	1	76.2	23
Rossville 2008	COM3	3	2217.3	2	96.5	23
Rossville 2008	COM3	4	2667.4	1	88.9	22
Rossville 2008	KS3406RR	1	1579.0	1	71.1	16
Rossville 2008	KS3406RR	2	1397.6	2	86.4	17
Rossville 2008	KS3406RR	3	1390.8	1	53.3	16
Rossville 2008	KS3406RR	4	1706.6	2	78.7	16

Table A.3. Continued.

Environment	Genotype	Rep	Yield	Lodging	Height	Maturity
Rossville 2009	COM1	1	3131.1	2	96.5	19
Rossville 2009	COM1	2	3386.4	1	104.1	14
Rossville 2009	COM1	3	3413.3	1	106.7	16
Rossville 2009	COM1	4	1673.0	1	78.7	15
Rossville 2009	COM2	1	3500.6	3	124.5	27
Rossville 2009	COM2	2	3480.4	2	111.8	28
Rossville 2009	COM2	3	3507.3	3	116.8	29
Rossville 2009	COM2	4	3151.2	2	114.3	27
Rossville 2009	COM3	1	3420.0	1	99.1	14
Rossville 2009	COM3	2	3406.5	2	96.5	19
Rossville 2009	COM3	3	3252.0	1	116.8	20
Rossville 2009	COM3	4	2667.4	2	101.6	20
Rossville 2009	KS3406RR	1	2748.1	2	91.4	18
Rossville 2009	KS3406RR	2	3016.8	2	96.5	14
Rossville 2009	KS3406RR	3	2942.9	2	81.3	14
Rossville 2009	KS3406RR	4	1652.9	2	86.4	14
Topeka 2008	COM1	1	4965.3	2	101.6	26
Topeka 2008	COM1	2	5153.5	2	104.1	26
Topeka 2008	COM1	3	4831.0	2	104.1	26
Topeka 2008	COM1	4	4851.1	2	109.2	26
Topeka 2008	COM2	1	5019.1	2	114.3	34
Topeka 2008	COM2	2	4938.5	2	114.3	35
Topeka 2008	COM2	3	5200.5	2	111.8	34
Topeka 2008	COM2	4	5697.7	2	121.9	33
Topeka 2008	COM3	1	4562.2	1	94.0	33
Topeka 2008	COM3	2	4454.7	1	96.5	36
Topeka 2008	COM3	3	4649.5	1	99.1	35
Topeka 2008	COM3	4	4817.5	1	104.1	35
Topeka 2008	KS3406RR	1	4320.3	2	96.5	29
Topeka 2008	KS3406RR	2	4481.6	2	99.1	26
Topeka 2008	KS3406RR	3	4199.4	2	96.5	26
Topeka 2008	KS3406RR	4	4790.6	2	101.6	24

Table A.3. Continued.

Environment	Genotype	Rep	Yield	Lodging	Height	Maturity
Topeka 2009	COM1	1	2754.8	2	99.1	15
Topeka 2009	COM1	2	3547.6	2	111.8	16
Topeka 2009	COM1	3	3635.0	1	104.1	18
Topeka 2009	COM1	4	4192.7	1	106.7	20
Topeka 2009	COM2	1	2607.0	3	109.2	14
Topeka 2009	COM2	2	3339.3	4	111.8	16
Topeka 2009	COM2	3	3527.5	3	106.7	19
Topeka 2009	COM2	4	4152.3	3	127.0	24
Topeka 2009	COM3	1	2828.7	1	91.4	14
Topeka 2009	COM3	2	2963.1	3	106.7	14
Topeka 2009	COM3	3	1921.6	1	106.7	12
Topeka 2009	COM3	4	2607.0	2	104.1	12
Topeka 2009	KS3406RR	1	3171.4	2	94.0	12
Topeka 2009	KS3406RR	2	2136.6	2	94.0	11
Topeka 2009	KS3406RR	3	1558.8	1	88.9	11
Topeka 2009	KS3406RR	4	2109.8	2	94.0	11

Table A.4. Total fungal colonies on modified Nash-Snyder at planting (MNSPl), midseason (MNSMd), and harvest (MNSHv) and total fungal colonies on potato dextrose agar at planting (PDAPl), midseason (PDAMd), and harvest (PDAHv) according to environments, genotypes, and replications (Rep) used in this study.

Environment	Genotype	Rep	MNSPl	PDAPl	MNSMd	PDAMd	MNSHv	PDAHv
Manhattan 2008	COM3	1	2530	30700	6600	47000	6600	47000
Manhattan 2008	COM3	2	3780	21500	20100	153000	20100	153000
Manhattan 2008	COM3	3	2780	24100	17000	87000	17000	87000
Manhattan 2008	COM3	4	3640	27900	13800	159000	13800	159000
Manhattan 2008	KS3406RR	1	3620	17200	11300	81000	11300	81000
Manhattan 2008	KS3406RR	2	3880	24000	13200	107000	13200	107000
Manhattan 2008	KS3406RR	3	4630	21800	22800	96000	22800	96000
Manhattan 2008	KS3406RR	4	2800	29100	13900	124000	13900	124000
Manhattan 2008	COM1	1	3560	27200	10100	114000	10100	114000
Manhattan 2008	COM1	2	4620	28700	23300	122000	23300	122000
Manhattan 2008	COM1	3	4110	34300	19000	136000	19000	136000
Manhattan 2008	COM1	4	4920	35400	12400	86000	12400	86000
Manhattan 2008	COM2	1	3130	23900	16000	77000	16000	77000
Manhattan 2008	COM2	2	3680	26100	24500	124000	24500	124000
Manhattan 2008	COM2	3	4750	25500	21800	84000	21800	84000
Manhattan 2008	COM2	4	4580	25100	13600	95000	13600	95000
Manhattan 2009	COM3	1	14200	60000	11600	36000	14000	41000
Manhattan 2009	COM3	2	13600	108000	31700	185000	13400	53000
Manhattan 2009	COM3	3	14900	79000	12100	57000	13500	42000
Manhattan 2009	COM3	4	16400	33000	18000	74000	13000	58000
Manhattan 2009	KS3406RR	1	14400	33000	15100	77000	11500	33000
Manhattan 2009	KS3406RR	2	19800	82000	14000	53000	13800	56000
Manhattan 2009	KS3406RR	3	8600	53000	15100	81000	12300	51000
Manhattan 2009	KS3406RR	4	13200	53000	19000	63000	13700	65000
Manhattan 2009	COM1	1	14400	54000	20500	53000	13800	50000
Manhattan 2009	COM1	2	10800	54000	13200	92000	14900	66000
Manhattan 2009	COM1	3	11700	60000	13700	46000	13600	70000
Manhattan 2009	COM1	4	14700	56000	12000	64000	13500	55000
Manhattan 2009	COM2	4	12700	75000	16800	55000	12100	49000
Manhattan 2009	COM2	1	16100	67000	15700	53000	16100	48000
Manhattan 2009	COM2	2	10500	60000	11800	66000	14800	52000
Manhattan 2009	COM2	3	11200	58000	16500	46000	12400	60000

Table A.4. Continued.

Environment	Genotype	Rep	MNSPl	PDAPl	MNSMd	PDAMd	MNSHv	PDAHv
Morganville 2009	COM3	1	8900	34000	12000	60000	11300	30000
Morganville 2009	COM3	2	10600	38000	16500	43000	16000	33000
Morganville 2009	COM3	3	18800	40000	17100	41000	10800	34000
Morganville 2009	COM3	4	7200	27000	15800	27000	11100	28000
Morganville 2009	KS3406RR	1	6100	31000	17400	55000	14100	37000
Morganville 2009	KS3406RR	2	11700	54000	15900	51000	12900	51000
Morganville 2009	KS3406RR	3	10800	38000	20400	73000	13800	38000
Morganville 2009	KS3406RR	4	10300	28000	19800	51000	18200	33000
Morganville 2009	COM1	1	18300	52000	14700	52000	16200	31000
Morganville 2009	COM1	2	8100	39000	17300	32000	17500	27000
Morganville 2009	COM1	3	15400	42000	16200	49000	14500	27000
Morganville 2009	COM1	4	9100	29000	17400	41000	10800	30000
Morganville 2009	COM2	1	13700	49000	20400	41000	12800	35000
Morganville 2009	COM2	2	12600	88000	20900	29000	16100	30000
Morganville 2009	COM2	3	12400	59000	17500	46000	15700	24000
Morganville 2009	COM2	4	10200	51000	19700	39000	13700	37000
Rossville 2008	COM3	1	4950	18500	11800	62000	11800	62000
Rossville 2008	COM3	2	3910	22700	15300	92000	15300	92000
Rossville 2008	COM3	3	7060	26800	23700	53000	23700	53000
Rossville 2008	COM3	4	2580	21100	8200	38000	8200	38000
Rossville 2008	KS3406RR	1	3850	24400	9100	32000	9100	32000
Rossville 2008	KS3406RR	2	4000	17500	24300	82000	24300	82000
Rossville 2008	KS3406RR	3	4130	11100	11700	48000	11700	48000
Rossville 2008	KS3406RR	4	2650	20800	8400	57000	8400	57000
Rossville 2008	COM1	1	5240	21300	14400	105000	14400	105000
Rossville 2008	COM1	2	3660	14600	6400	50000	6400	50000
Rossville 2008	COM1	3	4020	14800	6400	42000	6400	42000
Rossville 2008	COM1	4	2940	19800	13500	69000	13500	69000
Rossville 2008	COM2	1	5670	23200	20800	121000	20800	121000
Rossville 2008	COM2	2	4760	14900	9100	59000	9100	59000
Rossville 2008	COM2	3	6300	19000	16600	79000	16600	79000
Rossville 2008	COM2	4	2950	17000	14300	62000	14300	62000

Table A.4. Continued.

Environment	Genotype	Rep	MNSPl	PDAPl	MNSMd	PDAMd	MNSHv	PDAHv
Rossville 2009	COM3	1	2900	30000	9600	13000	9100	24000
Rossville 2009	COM3	2	7000	30000	11800	20000	7400	22000
Rossville 2009	COM3	3	6400	34000	10000	19000	11600	33000
Rossville 2009	COM3	4	7900	50000	10700	17000	12800	19000
Rossville 2009	KS3406RR	1	18600	41000	8500	22000	6100	24000
Rossville 2009	KS3406RR	2	6600	23000	14300	21000	7200	28000
Rossville 2009	KS3406RR	3	6800	28000	11600	32000	14100	32000
Rossville 2009	KS3406RR	4	6400	50000	12800	31000	8000	19000
Rossville 2009	COM1	1	7500	32000	6300	21000	9200	17000
Rossville 2009	COM1	2	5200	19000	13600	33000	9200	33000
Rossville 2009	COM1	3	4700	24000	13200	19000	11000	25000
Rossville 2009	COM1	4	6200	41000	8200	18000	6500	29000
Rossville 2009	COM2	1	8300	35000	7300	23000	8700	31000
Rossville 2009	COM2	2	5700	25000	11000	20000	8000	27000
Rossville 2009	COM2	3	6500	22000	8700	18000	12400	27000
Rossville 2009	COM2	4	5800	22000	13900	42000	7300	19000
Topeka 2008	COM3	1	3710	35000	16900	92000	16900	92000
Topeka 2008	COM3	2	5580	28000	15200	91000	15200	91000
Topeka 2008	COM3	3	4560	28200	17900	63000	17900	63000
Topeka 2008	COM3	4	5610	31200	20300	75000	20300	75000
Topeka 2008	KS3406RR	1	4890	27400	12300	97000	12300	97000
Topeka 2008	KS3406RR	2	5650	24300	15800	47000	15800	47000
Topeka 2008	KS3406RR	3	4750	26400	14200	67000	14200	67000
Topeka 2008	KS3406RR	4	5130	33000	23900	83000	23900	83000
Topeka 2008	COM1	1	4620	23600	13100	76000	13100	76000
Topeka 2008	COM1	2	5290	26900	15700	68000	15700	68000
Topeka 2008	COM1	3	3690	28800	14400	71000	14400	71000
Topeka 2008	COM1	4	5720	44400	17100	84000	17100	84000
Topeka 2008	COM2	1	3990	23800	40900	110000	40900	110000
Topeka 2008	COM2	2	4820	26800	17300	76000	17300	76000
Topeka 2008	COM2	3	5880	27100	16400	80000	16400	80000
Topeka 2008	COM2	4	4590	27600	17400	94000	17400	94000

Table A.4. Continued.

Environment	Genotype	Rep	MNSPl	PDAPl	MNSMd	PDAMd	MNSHv	PDAHv
Topeka 2009	COM3	1	9600	62000	16700	32000	12700	35000
Topeka 2009	COM3	2	13200	55000	14100	35000	21500	49000
Topeka 2009	COM3	3	11300	27000	15300	25000	11500	39000
Topeka 2009	COM3	4	8300	52000	9700	42000	12200	22000
Topeka 2009	KS3406RR	1	7200	29000	16800	9000	16200	19000
Topeka 2009	KS3406RR	2	12100	38000	23100	50000	18200	39000
Topeka 2009	KS3406RR	3	5600	45000	11700	40000	14400	29000
Topeka 2009	KS3406RR	4	8100	48000	14100	29000	14900	48000
Topeka 2009	COM1	1	9500	78000	16000	34000	21900	38000
Topeka 2009	COM1	2	7900	61000	16800	42000	46900	71000
Topeka 2009	COM1	3	6800	51000	13200	25000	21000	29000
Topeka 2009	COM1	4	7000	341000	14000	24000	17200	53000
Topeka 2009	COM2	1	11800	73000	14500	32000	14400	55000
Topeka 2009	COM2	2	10900	44000	16800	41000	19500	39000
Topeka 2009	COM2	3	7700	44000	13200	14000	29300	56000
Topeka 2009	COM2	4	13900	61000	13200	22000	26300	55000

Table A.5. SCN soil populations at planting (SCN Pi), midseason (SCN Pm), and harvest (SCN Pf), and SCN reproductive factors (SCN Rf) according to environments, genotypes, and replications (Rep) used in this study.

Environment	Genotype	Rep	SCN Pi	SCN Pm	SCN Pf	SCN Rf
Manhattan 2008	COM1	1	160	300	500	3.1
Manhattan 2008	COM1	2	220	620	940	4.3
Manhattan 2008	COM1	3	340	220	260	0.8
Manhattan 2008	COM1	4	0	340	720	2.1
Manhattan 2008	COM2	1	20	840	620	31.0
Manhattan 2008	COM2	2	180	440	560	3.1
Manhattan 2008	COM2	3	80	440	1000	12.5
Manhattan 2008	COM2	4	240	360	440	1.8
Manhattan 2008	COM3	1	20	620	220	11.0
Manhattan 2008	COM3	2	100	200	440	4.4
Manhattan 2008	COM3	3	40	200	560	14.0
Manhattan 2008	COM3	4	0	140	380	2.7
Manhattan 2008	KS3406RR	1	60	380	1000	16.7
Manhattan 2008	KS3406RR	2	180	280	380	2.1
Manhattan 2008	KS3406RR	3	180	300	860	4.8
Manhattan 2008	KS3406RR	4	140	980	1000	7.1
Manhattan 2009	COM1	1	480	400	220	0.5
Manhattan 2009	COM1	2	280	260	280	1.0
Manhattan 2009	COM1	3	220	200	0	0.0
Manhattan 2009	COM1	4	320	280	260	0.8
Manhattan 2009	COM2	1	80	80	80	1.0
Manhattan 2009	COM2	2	480	540	620	1.3
Manhattan 2009	COM2	3	100	520	680	6.8
Manhattan 2009	COM2	4	160	220	240	1.5
Manhattan 2009	COM3	1	60	60	60	1.0
Manhattan 2009	COM3	2	220	180	80	0.4
Manhattan 2009	COM3	3	260	480	560	2.2
Manhattan 2009	COM3	4	340	200	100	0.3
Manhattan 2009	KS3406RR	1	240	400	880	3.7
Manhattan 2009	KS3406RR	2	460	1200	2960	6.4
Manhattan 2009	KS3406RR	3	120	540	680	5.7
Manhattan 2009	KS3406RR	4	100	140	360	3.6

Table A.5. Continued.

Environment	Genotype	Rep	SCN Pi	SCN Pm	SCN Pf	SCN Rf
Morganville 2009	COM1	1	380	180	0	0.0
Morganville 2009	COM1	2	600	500	100	0.2
Morganville 2009	COM1	3	140	160	0	0.0
Morganville 2009	COM1	4	360	220	40	0.1
Morganville 2009	COM2	1	180	300	280	1.6
Morganville 2009	COM2	2	100	100	80	0.8
Morganville 2009	COM2	3	180	160	120	0.7
Morganville 2009	COM2	4	260	240	260	1.0
Morganville 2009	COM3	1	380	400	60	0.2
Morganville 2009	COM3	2	140	120	120	0.9
Morganville 2009	COM3	3	140	100	80	0.6
Morganville 2009	COM3	4	100	80	40	0.4
Morganville 2009	KS3406RR	1	240	540	760	3.2
Morganville 2009	KS3406RR	2	380	600	780	2.1
Morganville 2009	KS3406RR	3	120	100	40	0.3
Morganville 2009	KS3406RR	4	300	400	580	1.9
Rossville 2008	COM1	1	280	1520	1820	6.5
Rossville 2008	COM1	2	480	1020	1000	2.1
Rossville 2008	COM1	3	860	1880	860	1.0
Rossville 2008	COM1	4	440	9820	2280	5.2
Rossville 2008	COM2	1	240	680	1100	4.6
Rossville 2008	COM2	2	440	880	1060	2.4
Rossville 2008	COM2	3	900	2400	1380	1.5
Rossville 2008	COM2	4	600	540	1760	2.9
Rossville 2008	COM3	1	80	620	840	10.5
Rossville 2008	COM3	2	180	1400	1340	7.4
Rossville 2008	COM3	3	860	640	720	0.8
Rossville 2008	COM3	4	360	720	1300	3.6
Rossville 2008	KS3406RR	1	280	960	560	2.0
Rossville 2008	KS3406RR	2	360	440	880	2.4
Rossville 2008	KS3406RR	3	300	680	1340	4.5
Rossville 2008	KS3406RR	4	620	420	920	1.5

Table A.5. Continued.

Environment	Genotype	Rep	SCN Pi	SCN Pm	SCN Pf	SCN Rf
Rossville 2009	COM1	1	20	180	400	20.0
Rossville 2009	COM1	2	160	600	500	3.1
Rossville 2009	COM1	3	140	1020	1220	8.7
Rossville 2009	COM1	4	120	540	760	6.3
Rossville 2009	COM2	1	60	240	420	7.0
Rossville 2009	COM2	2	120	1800	2040	17.0
Rossville 2009	COM2	3	140	1200	1160	8.3
Rossville 2009	COM2	4	220	460	720	3.3
Rossville 2009	COM3	1	140	320	620	4.4
Rossville 2009	COM3	2	60	1220	1580	26.3
Rossville 2009	COM3	3	120	760	900	7.5
Rossville 2009	COM3	4	280	220	240	0.9
Rossville 2009	KS3406RR	1	60	420	660	11.0
Rossville 2009	KS3406RR	2	240	880	1200	5.0
Rossville 2009	KS3406RR	3	60	80	100	1.7
Rossville 2009	KS3406RR	4	200	620	600	3.0
Topeka 2008	COM1	1	140	380	760	5.4
Topeka 2008	COM1	2	180	480	680	3.8
Topeka 2008	COM1	3	220	720	440	2.0
Topeka 2008	COM1	4	180	620	900	5.0
Topeka 2008	COM2	1	240	460	520	2.2
Topeka 2008	COM2	2	220	480	720	3.3
Topeka 2008	COM2	3	240	700	740	3.1
Topeka 2008	COM2	4	240	900	1000	4.2
Topeka 2008	COM3	1	260	700	840	3.2
Topeka 2008	COM3	2	40	660	760	19.0
Topeka 2008	COM3	3	180	880	560	3.1
Topeka 2008	COM3	4	280	520	1080	3.9
Topeka 2008	KS3406RR	1	180	880	3120	17.3
Topeka 2008	KS3406RR	2	120	1460	1100	9.2
Topeka 2008	KS3406RR	3	220	980	1320	6.0
Topeka 2008	KS3406RR	4	400	600	600	1.5

Table A.5. Continued.

Environment	Genotype	Rep	SCN Pi	SCN Pm	SCN Pf	SCN Rf
Topeka 2009	COM1	1	140	140	40	0.3
Topeka 2009	COM1	2	60	680	1100	18.3
Topeka 2009	COM1	3	240	1560	1540	6.4
Topeka 2009	COM1	4	280	1820	3480	12.4
Topeka 2009	COM2	1	500	480	500	1.0
Topeka 2009	COM2	2	300	140	100	0.3
Topeka 2009	COM2	3	360	360	840	2.3
Topeka 2009	COM2	4	120	580	960	8.0
Topeka 2009	COM3	1	140	80	0	0.0
Topeka 2009	COM3	2	1000	680	500	0.5
Topeka 2009	COM3	3	680	440	120	0.2
Topeka 2009	COM3	4	180	880	1200	6.7
Topeka 2009	KS3406RR	1	140	100	60	0.4
Topeka 2009	KS3406RR	2	280	460	520	1.9
Topeka 2009	KS3406RR	3	380	400	260	0.7
Topeka 2009	KS3406RR	4	450	780	1160	2.6

Table A.6. Pearson's correlation coefficients for yield, *F. virguliforme* soil populations at planting (FVPl), midseason (FVMd), and harvest (FVHv), *F. virguliforme* root population (FVRt), SCN soil populations at planting (SCN Pi), midseason (SCN Pm), and harvest (SCN Pf), SCN reproductive factor (SCN Rf), and AUDPC for COM1 (A), COM2 (B), COM3 (C), and KS3406RR (D) by replication (n=112).

				Variables A					
Variables	FVPl	FVMd	FVHv	FVRt	SCN Pi	SCN Pm	SCN Pf	SCN Rf	AUDPC
Yield	0.11	0.33	0.14	-0.02	-0.01	0.05	0.24	0.03	-0.43*
FVPl		0.65**	0.57**	-0.09	0.12	-0.07	-0.11	-0.16	0.28
FVMd			0.71**	-0.20	0.21	0.03	-0.09	-0.46*	0.27
FVHv				0.21	0.10	0.20	0.03	-0.18	0.47*
FVRt					-0.04	0.05	0.01	0.27	-0.09
SCN Pi						0.25	-0.09	-0.35	0.31
SCN Pm							0.67**	0.25	0.39*
SCN Pf								0.47*	-0.05
SCN Rf									-0.11

				Variables B					
Variables	FVPl	FVMd	FVHv	FVRt	SCN Pi	SCN Pm	SCN Pf	SCN Rf	AUDPC
Yield	0.10	0.26	0.42*	-0.57**	-0.19	0.04	0.07	0.06	-0.59
FVPl		0.35	0.28	-0.19	0.29	0.34	0.35	-0.05	0.11
FVMd			0.75**	-0.15	0.17	0.02	0.01	-0.05	0.29
FVHv				0.02	0.02	-0.04	-0.03	-0.01	0.07
FVRt					-0.17	-0.25	-0.19	0.09	0.27
SCN Pi						0.24	0.24	-0.69**	0.56**
SCN Pm							0.90**	0.41*	-0.10
SCN Pf								0.36*	-0.07
SCN Rf									-0.37

Variables C									
Variables	FVPl	FVMd	FVHv	FVRt	SCN Pi	SCN Pm	SCN Pf	SCN Rf	AUDPC
Yield	-0.33	-0.15	-0.16	-0.41*	-0.04	0.25	0.23	0.24	-0.70**
FVPl		0.76**	0.65**	-0.07	0.25	-0.18	-0.25	-0.47*	0.48*
FVMd			0.56**	-0.10	0.01	-0.18	-0.21	-0.31	0.36*
FVHv				0.12	0.07	-0.27	-0.13	-0.65**	0.23
FVRt					0.06	-0.03	0.23	-0.08	0.24
SCN Pi						0.25	-0.01	-0.39*	0.40*
SCN Pm							0.75**	0.48**	0.13
SCN Pf								0.48**	-0.04
SCN Rf									-0.27

Variables D									
Variables	FVPl	FVMd	FVHv	FVRt	SCN Pi	SCN Pm	SCN Pf	SCN Rf	AUDPC
Yield	-0.31	-0.04	-0.27	-0.56**	-0.34	0.08	0.07	0.41*	-0.76**
FVPl		-0.11	0.05	0.16	0.36*	0.37*	0.33	-0.04	0.36
FVMd			0.78**	-0.20	0.34	-0.03	0.01	-0.19	0.45**
FVHv				-0.10	0.37*	-0.07	-0.01	-0.24	0.59**
FVRt					-0.17	-0.37*	-0.27	-0.18	0.15
SCN Pi						0.43*	0.36*	-0.45*	0.57**
SCN Pm							0.84**	0.38*	0.06
SCN Pf								0.55**	0.01
SCN Rf									-0.43**

*, ** indicates significant difference at $p \leq 0.05$ and $p \leq 0.01$, respectively.