Effect of agronomic practices on sudden death syndrome of soybean in Iowa

by

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CHAPTER 1. GENERAL INTRODUCTION

Sudden death syndrome, SDS, of soybean [*Glycine max* (L.) Merr.] is caused by the soilborne pathogen *Fusarium virguliforme* (formerly called, *F. solani* f. sp. *glycines*; SDS; Aoki et al., 2003) and results in chlorosis, necrosis and eventual defoliation of plants if symptoms are severe enough. Sudden death syndrome was first observed in the United States in 1971 in Arkansas. Research concerning agronomic practices and management of SDS has been conducted in the south but to our knowledge this research does not exist for Iowa. Soybean cyst nematode (*Heterodera glycines* Ichinohe; SCN) and SDS are consistently the most yield damaging soybean pathogens in Iowa, therefore research regarding SDS and agronomic practices is needed in order to provide growers in the state with appropriate management recommendations. This thesis work was conducted to evaluate the effect of agronomic practices of maturity group, planting date and row spacing on SDS foliar symptom development and soybean yield. Chapter two is a literature review. The research comprising this thesis is presented as manuscripts in chapters three, four and five.

Chapter three evaluates the effect of planting date on SDS foliar symptom development and soybean yield at two locations in Iowa. Research has demonstrated that SDS disease expression is influenced by moisture and temperature. Greatest SDS disease expression results when temperatures are cool at planting and warm during reproductive growth while moisture is adequate throughout the season. Early planting of soybean is a critical practice for producers in Iowa to maximize yield potential. Because the environmental conditions favoring early onset of SDS foliar disease expression are similar to environmental conditions in Iowa during the time of early planting, we hypothesize that

early-planted soybean in Iowa will experience higher levels of SDS disease expression than late-planted soybean.

Chapter four addresses the response of SDS foliar disease symptoms and soybean yield to row spacing and seeding rate at two locations in Iowa. Row spacing and seeding rate are agronomic practices employed by growers to achieve maximum yield. Both narrow rows and soybean seeded at higher populations exhibit greater leaf area than soybean planted in wide rows and lower populations. We hypothesize that this characteristic of narrow rows and high seeding rates will allow soybean to lessen yield loss to SDS as SDS can function to defoliate plants. No information exists on the relation of SDS foliar disease expression to row spacing and seeding rate.

Chapter five examines the effect of maturity classes and cultivar selection on SDS foliar symptom development and soybean yield. Research from Arkansas and Kentucky suggests that disease onset is a function of environment rather than maturity group. To escape yield loss to SDS in the southern US, SDS management recommendations include using early maturing cultivars so that disease onset will take place at a later growth stage compared to a later maturing cultivar. The objective of this study was to evaluate differences in SDS foliar symptom and severity among soybean cultivars with and without SDS-resistance in three classes of maturity in central Iowa. To our knowledge, this is the first study investigating the effect of classes of maturity on SDS foliar disease expression and soybean yield in Iowa.

The research presented in this thesis is the first attempt to understand the effect of common agronomic practices employed by growers in Iowa to SDS disease progression and

soybean yield. This thesis provides a foundation for further investigation regarding the relation of SDS and soybean yield to planting date, row spacing and cultivar selection. From this information, recommendations to manage SDS and maximize soybean yield for soybean producers in Iowa can be made.

CHAPTER 2. LITERATURE REVIEW

Soybean history and production

Origin and history of soybean

Soybean originated from China and the history of the crop has been reviewed in various texts (Smith et al., 1987). The earliest documentation existing that mentions soybean as one of the five main plant foods of China comes from the year 2700 B.C. (Hymowitz and Shurtleff, 2005). During the 15th and 16th Centuries, as sea and land trade routes such as the silk road were established, soybean was brought to several countries including Japan, Indonesia, the Philippines, Vietnam, Malaysia, Burma, Nepal and north India (Hymowitz, 1990). Soybean adapted to and developed in these countries. Throughout history, soybean has been a major component in East Asian diets. Europeans took note of the use of soybean in East Asian foods, and in the 17th Century soy sauce was commonly traded from the East to the West (Hymowitz, 1990).

The earliest evidence of soybean being planted in the United States comes from Georgia in 1765. Henry Yonge planted soybean on his farm after it had been introduced to the United States by Samuel Bowen. Bowen brought soybean to the United States from China (Smith et al., 1987) where it was used to produce soy sauce and vermicelli (soybean noodles) (Hymowitz, 1990). Soybean was grown and researched in the United States throughout the 1800s, documented in experiment station publications and scientific literature. In the United States, soybean was originally used as a forage crop and produced for hay and silage with cowpea (*Vigna sinensis* L.), millet (*Panicum* spp.), or sorghum [*Sorghum bicolor* (L.) Moench]. Soybean was recognized for its high yield, adaptability to various climates and

soils, and value as a silage and forage crop. Research on the crop intensified after 1890 (Smith et al., 1987).

Soybean production in the United States and Iowa

The United States plants more than 28.3 million hectares of soybean each year (National Agricultural Statistics Service, 2010). The largest number of acres planted to soybean in the United States is predicted to be 31.6 million hectares in 2010 (National Agricultural Statistics Service, 2010). In terms of future expansion of land into hectares of soybean, the amount of land in soybean hectares in the United States is projected to remain fairly constant. Land expansion to soybean has occurred outside of the United States, as observed with expansion of soybean acreage in South America, but with high costs of production this expansion has leveled-off in recent years (P. Pedersen, personal communication, 2009). In recent years, soybean yields in the United States average around 2690 kg ha^{-1} (Figure 1). The highest national average yield in the United States was 2959 kg ha^{-1} in 2009 (National Agricultural Statistics Service, 2010). As seen in Figure 1, soybean yields have gradually increased by 23.2 kg ha^{-1} yr^{-1} since 1924 due to advances in plant breeding and improved agronomic practices and management.

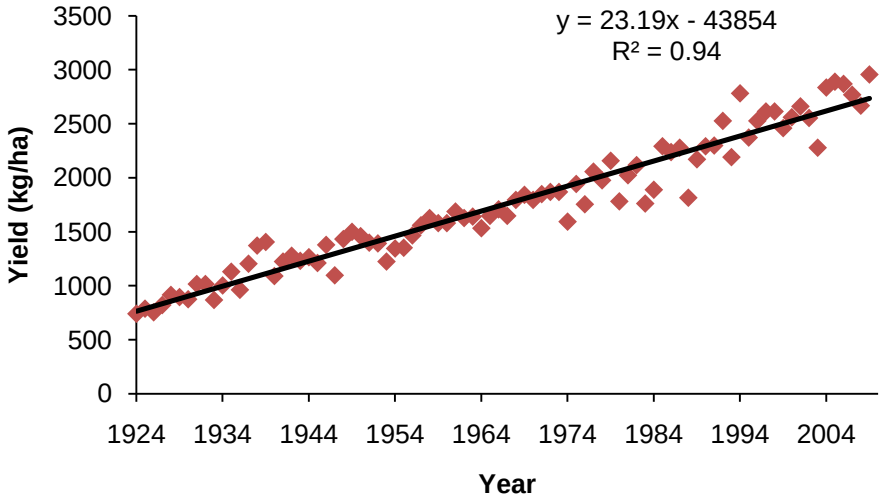

Figure 1. Average soybean yield in the United States from 1924-2009 (National Agricultural Statistic Service, 2010).

Iowa is the largest soybean producing state in the United States with a 4.6 billion dollar value of production in 2009. From 1997 to 2009 Iowa planted anywhere from 3.5 to 4.4 million hectares to soybean annually, with approximately 3.88 million hectares planted to soybean in the 2009 growing season (National Agricultural Statistic Service, 2010). From 2004 to 2009, Iowa soybean yields have averaged around 3377 kg ha^{-1} (Figure 2). These yields are higher than that of the national soybean yield average. Iowa soybean yields increase at a rate of 29.4 kg ha^{-1} yr^{-1}, which is 6.2 kg ha^{-1} yr^{-1}greater than the rate at which the national soybean yield average increases (National Agricultural Statistic Service, 2010).

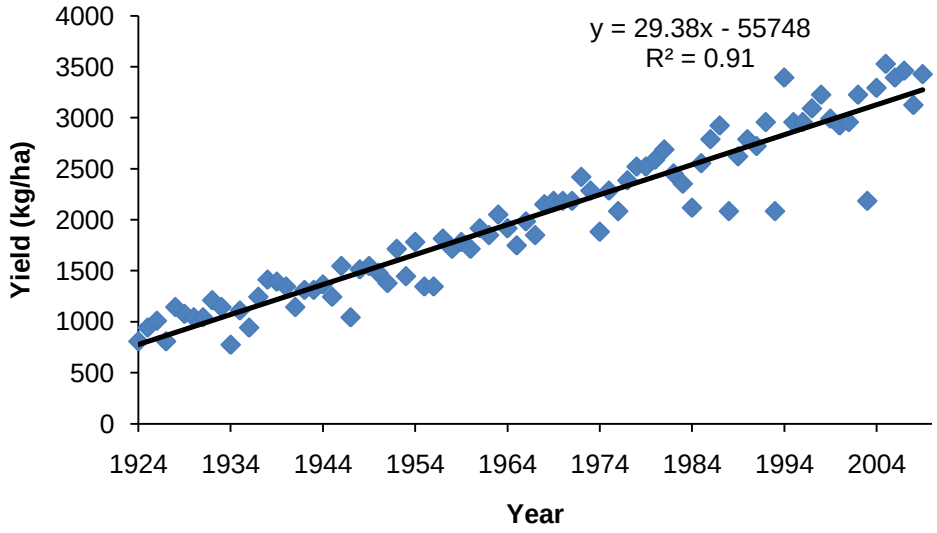

Figure 2. Average soybean yield in Iowa from 1924 to 2009 (National Agricultural Statistic Service, 2010).

Yield loss from soybean pathogens in the United States

Diseases contribute to soybean yield loss every year in the United States and Iowa (Wrather et al., 2003), which results in financial losses to the producer, rural economies and ultimately the general economy (Wrather et al., 2006). From 2003 to 2005 the United States experienced a total soybean yield reduction of approximately 29 million tons due to diseases. Soybean cyst nematode reduced soybean yields in the United States during this period more than any other disease. Phytophthora root and stem rot (*Phytophthora sojae*), SDS, and seedling diseases caused by various pathogens rank from second to fourth as diseases that suppressed soybean yield from 2003 to 2005 (Wrather et al., 2006). The severity and occurrence of these diseases vary among regions in the United States. For example, regional weather patterns (including moisture levels and temperature) in the central United States are more conducive for SDS than any other place in the United States (Scherm and Yang, 1999).

Therefore, it is no surprise that SDS reduced soybean yield more in Arkansas, Iowa, Illinois, Indiana and Tennessee during 2003 to 2005 than in any other state (Wrather et al., 2006).

Soybean growth and development

Germination

Seed germination takes place in three stages, as reported by Hadas and Russo (1974), beginning with imbibition in which water sorption is dictated by the seed's cotyledons or endosperm and is the same in non-viable and viable seeds. The next stage is development in which there is a pause while the enzymatic transformation and initiation of meristematic activities take place. The third stage is the growth stage that commences with elongation of the radical and emergence through the seed coat.

Soybean seedling emergence is influenced by the interaction of both temperature and the initial seed zone soil water content (Helms et al., 1996). In order for emergence to take place, each species has a critical value of seed water content for germination (Hadas and Russo, 1974) and for soybean the minimum seed water content for germination is 50% (Hunter and Erickson, 1952). It is important to note that fungi do not have the critical moisture requirements that seeds do. If seeds do not surpass the critical value of seed water content and germinate, they will be subject to fungi activity in this moist condition and be destroyed and decay (Hunter and Erickson, 1952).

A smaller seed-soil water contact area will slow germination by lowering the rate of water uptake (Helms et al., 1996). Thus, seedbeds should maintain an aerobic environment that maximizes seed to soil contact as well as optimizing soil water conductivity into the seed. Hobbs and Obendorf (1972) evaluated seeds that were equilibrated to specific moisture

contents and then transferred in a greenhouse. It was shown that seeds equilibrated to 13% moisture content before imbibition had higher survival rates than those equilibrated to 5% moisture content. Concerning temperature, Muendel (1986) demonstrated that percent emergence in the field was not influenced by soil temperature between 19.1 and 20.7°C. A conflicting view was presented by Fehr et al. (1973) in a study which examined three planting dates to evaluate the influence of soil temperature on emergence. Soil temperatures did indeed differ among all three planting dates, but the emergence was the same at each planting date.

Soybean seedling phase

Soybean seedling emergence will take place after seed germination and pre-emergent seedling growth in the soil (Hamman et al., 2002). Six distinct stages of soybean seedling growth are described by Muthiah et al. (1994). The germination of a soybean seed commences with seed imbibition of water and continues with the stages of testa split, radical growing 2 mm, hypocotyl-root axis reaching 10 mm, root hair development and then lateral root primordial development (Muthiah et al., 1994). Following the lateral root primordial development stage is the emergence stage as described by Fehr et al. (1971). In the emergence stage the hypocotyl arch is partly straightened and pulls the cotyledons up past the growth column (Fehr et al., 1971). When emergence takes place, seedlings have dropped their testa (Muthiah et al., 1994).

Vegetative growth stages

The first two stages of vegetative growth are VE (emergence) and VC (cotyledon) (Fehr and Caviness, 1977). Following VE and VC vegetative stages of soybean growth are

designated by counting the node number on the main stem beginning with the unifoliate node (V1), and then continues with nodes that have or have had a completely unrolled leaf, increasing as V2, V3, V4 through V(n), where (n) is the last completely developed trifoliolate leaf (Fehr et al., 1971).

Reproductive growth stages

According to Fehr et al. (1971) reproductive stages are based on development at the upper area of the main stem and can be applied to genotypes in all environments. There are eight reproductive stages: R1 and R2 describe flowering, R3 and R4 represent pod development, R5 and R6, seed development, and R7 and R8, harvest maturity (Fehr et al., 1971).

Soybean root development

Sudden death syndrome is a root disease of high-yield potential soybean (Rupe et al., 1989). The soilborne fungus can colonize and infect roots of seedlings that are 2 to 3 weeks old (Njiti et al., 1997) and maybe even earlier (Gongora-Canul and Leandro, 2007). Therefore, an understanding of soybean root development and growth is of importance to understand SDS development and pathogenesis.

Soybean root development is divided into three phases (Mitchell et al., 1971). The first phase includes from emergence to 31 days after planting. This phase is marked by downward taproot (radicle) growth associated with early rapid vegetative top growth. The taproot grows to depths of 46 to 60 cm and horizontal lateral root growth occurs in the top 10 cm of the profile. Roots enter the second phase of growth 67 to 80 days after planting. Top growth continues to take place at high rates while flowering and pod formation occurs. Roots

develop quickly to depths of 46 to 76 cm, and deeper vertical penetration of the lateral roots takes place. The final phase takes place 80 to 102 days after planting. The growth of the taproot slows while the larger lateral roots elongate quickly to depths of 122 to 183 cm. Flowering, seed set and maturity occurs during this phase of root development (Mitchell et al., 1971).

Reports describing soybean root distribution over depth and time often disagree about root growth during reproductive phases of development. Mayaki et al. (1976) report that root depth increases faster than plant height and that during seed development root dry weight decreases. Mitchell et al. (1971) report that root dry matter does not stop accumulating during flowering, pod formation and seed fill. Despite this difference, reports agree that rooting depth can increase during reproductive development (Kaspar et al., 1978). Kaspar et al. (1978) found that substantial increases in the depth of soybean root systems take place during reproduction although cultivars differ in the rates at which their roots grow downward as well as the sequence of depth increases by the roots.

Seed growth

Soybean yields are directly affected by seed number and potential seed mass (Egli, 1975). The ability of the soybean plant to fix carbon throughout the filling period or the translocation of storage carbohydrates from other plant parts is related to the accumulation of weight in the seed. Seed number is influenced by pod set and number of seeds per pod. Final seed number can be lower than seed number at the termination of flowering and is influenced by flower and pod abortion in addition to reduction in seeds per pod (Egli, 1975).

Rate and duration of a seed's growth are dynamic components of seed yield (Spaeth and Sinclair, 1984a) and together, these two components determine final seed mass (Spaeth and Sinclair, 1984b). Dry weight by a soybean seed accumulates in a pattern that follows a short exponential growth phase, relatively constant growth phase, and decreasing growth rate which lasts until the seed reaches physiological maturity and achieves a maximum dry weight (Egli, 1975).

With indeterminate soybean cultivars, seed initiation is usually sequential as opposed to one event at a certain time (Spaeth and Sinclair, 1984a). Therefore, R5 does not sufficiently describe the onset of seed-filling. The reason for disparity in date of onset of initiation of seed development is the initiation at nodes up the plant stem and the order of the raceme (Spaeth and Sinclair, 1984a). For example, in the individual 'Chippewa 64' used in a study by Spaeth and Sinclair (1984b), the seeds borne on secondary racemes initiated rapid growth initiated 6 to 9 days after those seeds from primary racemes on the same node. Seeds began rapid growth in order up the ranks of pods on a peduncle. Seed position influences seed growth rate as seeds on the main stems form earlier and have a reduced growth rate compared to seeds that form at top nodes later and have a higher seed growth rate and a shorter seed fill duration, therefore producing a lower seed mass (Spaeth and Sinclair, 1984b). Therefore, larger plants with more nodes began seed growth over a greater range of time. Seed mass decreases as node number increases, and on common nodes is smaller from secondary racemes than primary racemes (Spaeth and Sinclair, 1984b).

The effective filling period (EFP) plays a part in determining seed mass (Egli et al., 1978) A shorter EFP for late pods in indeterminate cultivars has been observed to result in

smaller seeds. There is a negative correlation between seed mass and seed number per plant that can be explained through the direct relationship between seed growth rates and seed mass (Egli et al., 1978).

Termination of soybean seed growth takes place in a pattern similar to that of initiation (Spaeth and Sinclair, 1984a). In terms of seed growth rate, it has been demonstrated that cotyledons control the genetic differences in seed growth rate and have a strong correlation with final seed size (Egli et al., 1981). Furthermore, it is believed that differences in growth rate are affected by the number of cells in the cotyledons as the cotyledon cell number represents the potential sink size of a soybean seed (Egli et al., 1981). The greater the cell number means the greater the amount of assimilate that will be allotted to fill that soybean seed.

Biomass accumulation

The nature of the environment, leaf surface area and crop physiology affect the dry matter accumulation (DMA) rate and the effectiveness of radiant energy absorption and conversion (Eastin and Gritton, 1969). In other words, it is the amount of radiant energy converted which drives the amount of dry matter accumulation (Eastin and Gritton, 1969; Shibles and Weber, 1965). It was also found that high plant population or narrow row spacing can increase the length of vegetative growth period, as they force the seed and vegetative parts of the plant to compete for carbohydrates (Shibles and Weber, 1965).

Leaf area index (LAI), DMA and crop growth rate have been related in various studies. Sivakumar et al. (1977) found that leaf dry matter reached its highest point 79 days after planting and LAI reached a maximum of 7.7 at 86 days after planting. The same report

also found that the crop growth rate was higher during rapid pod-filling than any other stage of growth. In soybean, the rate of leaf dry matter production and percent solar radiation interception increases with increasing leaf area development (Shibles and Weber, 1965).

In terms of soybean yield, increased length of yield formation period, increased efficiency of intercepted solar energy and a cultivar which demonstrates a greater diversion of photosynthate to seed production, will contribute to higher yields (Shibles and Weber, 1966).

Defoliation

Dry matter accumulation is affected by defoliation by reducing leaf area available for light interception and carbon fixation (Klubertanz et al., 1996). Higley's (1992) defoliation-light interception hypothesis states that the general mechanism of yield reduction by insect defoliation is to reduce light interception of defoliated plant canopies. An alternative view of this hypothesis was stated by Malone et al. (2002), that yield is dependent on photosynthesis during the early reproductive growth stages of soybean, and photosynthesis is dependent on canopy light interception, which can be described by LAI.

It has been shown that changes in soybean canopy light interception caused by insect defoliation at growth stages R2, R3 and R4 do have a major effect on soybean yields and linear relationships between light interception after defoliation and soybean yield losses exist (Higley, 1992). Furthermore, soybean yields can be greatly reduced if defoliation takes place during reproductive growth (Ingram et al., 1981). Extent of yield loss in soybean caused by leaf removal is dependent on the amount of foliage removed and the growth stage at which defoliation takes place as well as growth habit (Goli and Weaver, 1986). The decrease in

yield from defoliation is caused by a decline in yield components. Although conflicting reports exist, studies have demonstrated that defoliation reduces the seed number or amount of pods per plant (Caviness and Thomas, 1980) and reduces pod and seed growth rates by affecting photosynthate availability on individual seed growth rate, or canopy photosynthetic capacity (Ingram et al., 1982). Because neither seed nor pod growth duration were affected by defoliation, it has been concluded that lower levels of leaf nitrogen caused by seed growth demand lowered CO_2 assimilation but did not induce rapid canopy senescence (Ingram et al., 1982). Fehr et al. (1981) concluded that the most susceptible stage to yield loss by defoliation is R5 for both indeterminate and determinate cultivars. In that study, an 80% yield loss was reported when 100% defoliation was applied at the R5 to R5.5 period. Board (2004) reported that yield sensitivity to defoliation lessens as the seed filling period continues from R5 to R7. Compensation to defoliation might occur by improving light interception (Higley, 1992). It has been shown that soybeans compensate for defoliation with delayed leaf senescence, which includes delayed leaf abscission and altered leaf photosynthetic rates (Higley, 1992).

Sudden death syndrome

Sudden death syndrome caused by the soilborne pathogen *Fusarium virguliforme* sp. nov. (formerly called, *F. solani* f. sp. *glycines*; SDS; Aoki et al., 2003), was first observed in Arkansas in 1971 (Roy et al., 1997). Sudden death syndrome can create significant yield loss depending on cultural practices, cultivar susceptibility, soil moisture and temperature (Hartman et al., 1995; Roy et al., 1997; Rupe et al., 1991).

Symptoms

The soilborne fungus infects plants through the roots causing root necrosis and reduction in root mass (Roy et al., 1997; Rupe and Hartman, 1999). Symptoms are known to develop at or right after flowering (Roy et al., 1997). The first symptoms seen on the leaf are shaped in a circular or irregular form, scattered and interveinal and a few millimeters or more in size (Roy et al., 1997). These spots are seen as pale green or chlorotic (Roy et al., 1997) and create mottling or mosaic on the upper leaves (Navi and Yang, 2008). Young leaves with these symptoms are seen to cup slightly. As symptoms progress, the chlorotic and necrotic spots coalesce and create interveinal necrotic streaks (Scherm et al., 1998). Following this point in disease progression, leaflets demonstrating high symptomology can abscise leaving bare petioles (Roy et al., 1997). Upper leaves are always first to defoliate as the interveinal necrosis progresses more rapidly on upper rather than lower leaves. If SDS reaches severe enough levels, total plant defoliation can take place. Flower and pod abortion can take place if severe infection occurs during flowering and pod formation (Roy et al., 1997).

Root symptoms are observed when leaf symptoms become prominent or severe (Roy et al., 1997). If leaf symptoms are not present, or mild, there may be no external root symptoms. Within the root, the pith remains white, and a gray to red-brown color begins near the pith and continues out through the vascular tissue and can go throughout the taproot up the stem. When leaf symptoms are severe, the taproot and lateral roots can become necrotic and a reduction in root volume may occur. This can result in premature plant death. In the soil, a blue sporulation (macroconidia) can be produced by the pathogen on the taproot of very diseased plants. When symptoms are viewed on a large scale in the field, the distribution and disease pattern in the field is spotty (Roy et al., 1997).

Organism

Koch's postulates were completed for SDS by Roy et al. (1989) and Rupe (1989). Roy et al. (1989) found that a blue-pigmented morphological form of *Fusarium solani* is the causal agent of SDS, and designated it as form A (FSA). Now characterized as *F. virguliforme* (Aoki et al., 2003), grows slowly on potato dextrose agar (PDA) (Roy et al., 1997). The blue hue that is seen in the SDS causal agent is produced by cultures of *F. virguliforme* and the large variation in surface hue and pigmentation is caused by the large amount of sporulation. On PDA, sporulation caused by *F. virguliforme* is rapid and abundant, and sporodochia and aerial conidia normally form (Aoki et al., 2003). Aoki et al. (2003) also mention that *F. virguliforme* can be differentiated from other species within the *F. solani* complex by the comma-shaped sporodochial conidia rapidly produced on PDA.

Roy et al. (1997) found that wound-inoculated FSA into hypocotyls of soybean, with no root necrosis, leaf symptoms common to SDS were observed. This suggests that a phytotoxin is involved in leaf symptom expression. Jin et al. (1996) identified a polypeptide using culture filtrates of SDS-inciting isolates of *F. solani* from soybean that was causing certain leaf symptoms associated with SDS. Furthermore, the isolates of *F. solani* that did not incite leaf symptoms of SDS on soybean also did not produce phytotoxic polypeptide (Jin et al., 1996).

Pathogenesis

Melgar et al. (1994) report that when incubated in a soil extract solution, macroconidia of the fungus convert to chlamydospores and it is the chlamydospores that are present in sloughed cortical tissue of soybean roots in the field. Therefore, the fungus is

thought to occur in the root debris and soil primarily as chlamydospores (Roy et al., 1997). *Fusarium solani* f. sp. *glycines* is able to colonize and infect roots of seedlings that are 2 to 3 weeks old (Njiti et al., 1997). The colonization of *F. solani* f. sp. *glycines* occurs in the cortical tissue of the lower stem and root (Melgar et al., 1994). Hyphae grow intracellularly and are found in the stele after substantial root degradation, occurring as plants are expressing foliar symptoms in the late stages of growth (Melgar et al., 1994). Sporulation on the outer root is associated with root rot and is found more commonly during or right after periods of high soil moisture (Roy et al., 1997).

Sudden death syndrome in the United States

Following the first observation of SDS Arkansas in 1971 it spread to Mississippi, Missouri, Kentucky, and Tennessee by 1984 and to Illinois and Indiana by 1986 (Roy et al., 1997). Currently, SDS can be found throughout most of the soybean producing region. Outside of the United States, SDS has been observed in Argentina and Brazil as well (Roy et al., 1997).

In 1993, SDS was first observed at low intensities in four Iowa counties (Sanogo and Yang, 1999). During 1994 and 1995 the disease remained mainly in eastern Iowa, at a low prevalence. An epidemic of SDS took place in 1998 with great increases in severity and prevalence. The 1998 outbreak supported the 1996 risk assessment that SDS would become a major production concern in Iowa (Sanogo and Yang, 1999).

In 2002, the United States lost almost 10 million metric tons to disease and 728,838 or 8%, of those metric tons were lost to SDS (Wrather et al., 2003). The yield loss to all pathogens, SDS and SCN in 2003, 2004 and 2005 can be seen in Figure 3.

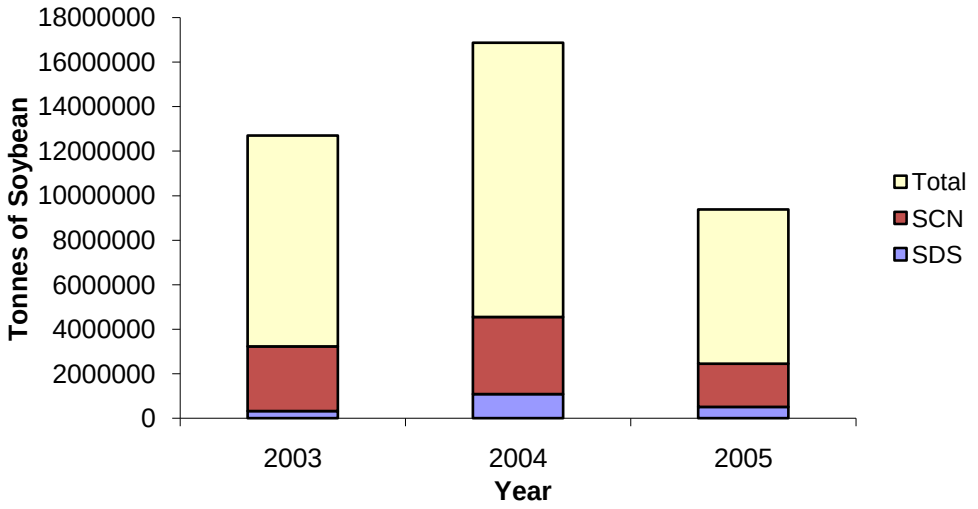

Figure 3. Estimated reduction of United States soybean yields from 2003 to 2005 due to soybean cyst nematode (SCN) and sudden death syndrome (SDS) (Wrather et al., 2006).

Sudden death syndrome is often found in moist, low and compacted areas of a field where SCN has been a problem (Roy et al., 1997). Symptoms of SDS are more severe with SCN infestation (McLean and Lawrence, 1993), although the correlation between density of cysts in the soil and SDS pathogen severity is not consistent (Rupe et al., 1993; Hershman et al., 1990). Based on reports by Wrather et al. (2006), yield suppression caused by diseases in Iowa is primarily attributed to SCN and SDS. In Iowa, SCN causes a 1.09 million kg loss every year. Sudden death syndrome and SCN cost Iowa more than 50% of the yield loss caused by soybean pathogens (Wrather et al., 2003).

Agronomic practices

Agronomic practices implemented and decisions in management need to be made in order to create the greatest yield potential for growers. Pathogens and disease can be managed using agronomic decisions and principles of crop physiology. Management

recommendations for SDS have been slow to develop due to lack of data, variability of data on the effects of cultural practices and cultivars on SDS development and progression (Hershman et al., 1990), and variability of disease levels in field experiments. Sudden death syndrome has been observed to be greatly influenced by the environment, growth stage of the plant, and cultivar susceptibility (Rupe and Gbur, 1995). Yet, difficulty still exists in trying to understand consistent relationships among SDS, cultivar and agronomic practices and this difficulty might be attributable to a variety of other factors with the ability to influence SDS development (Hershman et al., 1990).

Planting date

Njiti et al. (1997) stated that the SDS pathogen infects soybean roots early in the growing season. Studies have shown that symptoms of SDS are expressed more strongly in early rather than late planted soybeans (Hershman et al., 1990) and Navi and Yang (2008) stated that an SDS epidemic is strongly correlated with planting date and the disease will tend to be more severe in earlier planted soybeans. This observation was confirmed by Wrather et al. (1995) but only in no-tillage fields. McLean and Lawrence (1993) speculated that when a growing season begins with cool temperatures followed by high temperatures and sufficient moisture throughout the season it provides perfect conditions for SDS symptoms to develop. It has also been reported that there is a positive relationship between soil moisture and SDS incidence (Scherm and Yang, 1996). This same study presents results that demonstrate that low temperatures (15^{o}C) and high soil moisture during the early part of the growing season, followed by higher temperatures (22 to 24^{o}C) during soybean reproduction, provide prime conditions for SDS symptom expression. Rupe and Gbur (1995)

reported that early SDS symptom development correlates to accumulated degree-days from planting and that it was only in years with regular rainfall that SDS disease development continued after flowering.

Soil moisture at the V3 growth stage was greater in early (late May) rather than late planted (late June) soybean, supporting the idea of increased SDS with higher soil moisture (Hershman et al., 1990). This difference in infection, influenced by moisture and growth stage, could result in SDS symptoms being more severe in early planted rather than late planted soybean. Overall, this research suggests that SDS may be controlled by planting at later dates (Hershman et al., 1990).

In terms of moisture and cultural practices, incidence and severity of SDS were greater in irrigated than non-irrigated plants (Melgar et al., 1994). In a study by Vick et al. (2003) subsoiling, which reduces soil moisture, reduced incidence and severity of SDS foliar symptoms compared to foliar symptoms seen in no-till. Wrather et al. (1995), found SDS greater symptoms in no-tilled soybean rather than tilled soybean. Sudden death syndrome symptoms may be less in no-tilled soybean planted late rather than early planted soybean (Melgar et al., 1994). Foliar symptoms of SDS may be less in no-tilled soybean planted late rather than early, although the decline in yield as planting is delayed needs to be taken into account when considering planting date as a management practice (Wrather et al., 1995).

Row spacing

To our knowledge, no previous research exists on the relation of SDS symptom expression with row spacing and seeding rate. However, the following research concerning pathogen infection of soybean roots and soybean root growth and development provide

evidence that the cultural practices of row spacing and seeding rate may have an influence on SDS infection, development and effect on yield.

The SDS pathogen infects soybean roots early in the growing season (Njiti et al., 1997). If the level of soil moisture is high during early reproductive stages, the pathogen produces a toxin that is translocated into the parts of the plant aboveground where they induce foliar symptoms (Jin et al., 1996).

According to Mitchell and Russell (1971), soybean root growth consists of three stages: 1) downward tap and shallow horizontal lateral root growth; 2) downward root development to 76-cm depth; and 3) deep penetration of lateral roots. Studies in Iowa showed that soybean root length is greater at 0.25 m row spacing than at 1m row spacing (Mason et al., 1980). Rupe (1989) found that the highest frequency of isolation of SDS (23%) came from the epidermis of the taproot; the frequency of infection of the lateral roots was 19%. Mitchell and Russell (1971) state that the majority of the soybean root system is made up of lateral roots that emerge from the upper 10 to 15 cm of the taproot and spread out horizontally in the top 10 cm of soil. Therefore, it can be said that SDS most probably results from fungal infection taking place on areas of the root system that randomly contact the pathogen in the top 15 cm of soil (Rupe et al., 1999).

In terms of root development and row-spacing, narrow-row soybean has a greater root density than wide-row soybean at the 5-10 cm depth at the row, but wide-row soybean has more lateral growth (Scheiner et al., 2000). Across the row (within inter-row space), root densities are similar between narrow- and wide-row soybean. Planting soybean in narrow-rows results in more uniform root distribution down the soil profile (Scheiner et al., 2000).

Gray and Achenbach (1996) demonstrated that the soil inoculum level of *F. solani* influenced root rot severity. The study showed that with high amounts of inoculum a larger percentage of lateral and taproots become necrotic. In terms of SDS development, the presence of the fungus on the lateral roots seems to be important to disease development (Ortiz-Ribbing and Eastburn, 2004).

Ortiz-Ribbing and Eastburn (2004) stated that no studies have evaluated the effects of *F. solani* infection on root characteristics like surface area, length, volume, or average diameter. Rupe (1989) demonstrated that a negative relationship exists between root mass and SDS foliar symptoms while Scherm and Yang (1996) report that "no close correlation" exists between foliar disease severity and disease severity of root systems. Further research is necessary in order to understand the relation and correlation of SDS foliar symptoms to root characteristics (Ortiz-Ribbing and Eastburn, 2004).

Maturity group and cultivar selection

The onset of foliar symptoms of SDS seems dependent on the chronological age of the plant and independent of the reproductive age of the plant (Rupe et al., 1991; Rupe and Gbur, 1995). In field studies in Arkansas, for cultivars in maturity groups IV – VIII, the onset of foliar symptoms of SDS was seen at or even before flowering (Rupe et al., 1991). Further research described SDS expression during a growing season as a two-phase epidemic in which there was a rapid increase in foliar symptoms followed by a slow increase in disease expression (Rupe and Gbur, 1995). Cultivars belonging to maturity groups IV to VIII were used in this study and disease expression did not appear to be related to plant development as foliar symptom onset occurred at the same point across cultivars, R2 to R5 depending on the

cultivar. Despite this research, field trials from Kentucky show SDS symptom expression to be related to plant development and not calendar age (Hershman et al., 1990). In these studies, cultivars in maturity groups III to V were planted in late May, mid and late June and SDS symptom onset was observed R3 and R5 regardless of maturity group or planting date (Hershman et al., 1990).

Overall, this research suggests that SDS may be controlled by planting early-maturing cultivars which could result in disease onset and development at later reproductive stages which could lessen potential yield losses (Rupe et al., 1991; Rupe and Gbur, 1995). However, it is important to note that this research was conducted in the Southern United States in soybean with determinate growth belonging to maturity groups V to VIII (Rupe et al., 1991; Rupe and Gbur, 1995; Hershman et al., 1990). In Iowa and the upper Midwest soybean belonging to maturity groups I, II and III with indeterminate growth is adapted for full-season growth. The potential exists for differences between onset of SDS foliar symptoms for soybean grown in the Southern United States and Midwest, which may be due to growth type of the plant (Rupe and Gbur, 1995). With indeterminate cultivars, it is not certain if foliar symptoms of SDS are connected to growth type or another cultivar trait.

A possible method to control SDS is to select cultivars with low susceptibility or tolerance to the pathogen (Rupe et al., 1991). In terms of cultivar selection, few commercial cultivars are sold that claim to have high levels of resistance to SDS (Mueller et al., 2003). Most modern cultivars are still classified as susceptible to SDS (University of Illinois, 2008). A possible way to improve resistance to SDS in modern cultivars could be to identify new sources of resistance in the USDA Soybean Germplasm Collection (Mueller et al., 2002). As

of 2002, the USDA Soybean Germplasm Collection has 16,593 introduced *G. max* accessions (Mueller et al., 2002). Hartman et al. (1997) identified PI 567.374, PI 567.315, PI 567.441C, PI567.650B, and PI 567.664 as all having moderate resistance to sudden death syndrome. Mueller et al. (2002) identified 57 PIs as being moderately resistant to SDS and appropriate to use as sources of resistance to increase the level of resistance of cultivars in the United States.

Cultivar reactions to SDS in the field have shown to be quite variable because SDS is so dependent on location and year (Hershman et al., 1990). This could imply that the use of environmentally controlled evaluations would be more appropriate to differentiate susceptible or resistant cultivars (Hershman et al., 1990). Results from a study by Melgar and Roy (1994) suggest that the growth chamber method to evaluate is an appropriate alternative to field evaluation.

References

Aoki, T., K. O'Donnell, H. Yoshihisa, and A.R. Lattanzi. 2003. Sudden-death syndrome of soybean is caused by two morphologically and phylogenetically distinct species within the *Fusarium solani* species complex- *F. virguliforme* in North America and *F. tucumaniae* in South America. Mycologia. 95:660-684.

Board, J.E. 2004. Soybean cultivar differences on light interception and leaf area index. Agron. J. 96:305-310.

Caviness, C.E., and J.D. Thomas. 1980. Yield reduction from defoliation of irrigated and non-irrigated soybeans. Argon. J. 72:977-980.

Eastin, J.A., and E.T. Gritton. 1969. Leaf area development, light interception, and the

growth of canning (*Pisum sativum* L.) in relation to plant population and spacing. Agron. J. 61:612-615.

Egli, D.B. 1975. Rate of accumulation of dry weight in seed of soybeans and its relationship to yield. Can.J. Plant Sci. 55:215-219.

Egli, D.B., J.E. Leggett, and J.M. Wood. 1978. Influence of soybean seed size and position on the rate and duration of filling. Agron. J. 70:127-130.

Egli, D.B., J. Fraser, J.E. Leggett, and C.G. Poneleit. 1981. Control of seed growth in soya beans [*Glycine max* (L.) Merrill]. Ann. Bot. 48:171-176.

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

Fehr, W.R., J.S. Burris, and D.F. Gilman. 1973. Soybean emergence under field conditions. Agron. J. 65:740-742.

Fehr, W.R., and C.E. Caviness. 1977. Stages of soybean development. Spec. Rep. 80. Iowa Agric. Home Econ. Exp. Stn., Iowa State Univ., Ames, IA.

Fehr, W.R., B.K. Lawrence, and T.A. Thompson. 1981. Critical stages of development for defoliation of soybean. Crop Sci. 21:259-262.

Goli, A., and D.B. Weaver. 1986. Defoliation responses of determinate and indeterminate late-planted soybeans. Crop Sci. 26:156-159.

Gongora-Canul, C., and L. Leandro. 2007. Effect of inoculum level of *Fusarisum virguliforme* on timing of foliar and root symptom expression of soybean sudden death syndrome. (Abstract) Phytopathology 97:S41.

Gray, L.E., and L.A. Achenbach. 1996. Severity of foliar symptoms and root and crown rot

of soybean inoculated with various isolates and inoculums rates of *Fusarium Solani.* Plant Dis. 80:1197-1199.

Hadas, A., and D. Russo. 1974. Water uptake by seeds as affected by water stress, capillary conductivity, and seed-soil water contact. I. Experimental study. Agron J. 66:643-47.

Hamman, B., D.B. Egli, and G. Koning. 2002. Seed vigor, soilborne pathogens, preemergent growth, and soybean seedling emergence. Crop Sci. 42:451-457.

Hartman, G.L., G.R. Noel, and L.E. Gray. 1995. Occurrence of soybean sudden death syndrome in east-central Illinois and associated yield losses. Plant Dis. 79:314-318.

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

Helms, T.C., E. Deckard, R.J. Goos, J.W. Enz. 1996. Soybean seedling emergence influenced by days of soil water stress and soil temperature. Agron. J. 88:657-661.

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

Higley, L.G. 1992. New understandings of soybean defoliation and their implication for pest management. *In* L.G. Copping et al. (ed.) Pest management in soybean. Elsevier Sci. Publ., London.

Hobbs, P.R., and R.L. Obendorf. 1972. Interaction of initial seed moisture and imbibitional temperature on germination and productivity of soybean. Crop Sci. 12:664-667.

Hunter, J.R., and A.E. Erickson. 1952. Relations of seed germination to soil moisture tension. Agron J. 44:107-109.

Hymowitz, T. 1990. Soybeans: The Success Story. P. 159-163. *In* J. Janick and J. Simon. (ed.) Advances in New Crops. Timber Press, Portland, Oregon.

Hymowitz, T., and W.R. Shurtleff. 2005. Debunking soybean myths and legends in the historical and popular literature. Crop Sci. 45:473-476.

Ingram, K.T., D.C. Herzog, K.J. Boote, J.W. Jones, and C.S. Barfield. 1982. Effects of defoliating pests on soybean canopy CO_2 exchange and reproductive growth. Crop Sci. 21:961-968.

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

Kaspar, T.C., C.D. Stanley, and H.M. Taylor, H. M. 1978. Soybean root growth during the reproductive stages of development. Agron. J. 70:1105-1107.

Kluberantz, T.H., L.P. Pedigo, and R.E. Carlson. 1996. Soybean physiology, regrowth, and senescence in response to defoliation. Agron. J. 88:577-582.

Malone, S.D., A. Herbert Jr., and D.L. Holshouser. 2002. Evaluation of the LAI-2000 plant canopy analyzer to estimate leaf area in manually defoliated soybean. Agron. J. 94:1012-1019.

Mason, W.K., H.M. Taylor, A.T.P. Bennie, H.R. Rowse, D.C. Reicosky, Y. Jung, A.A. Righes, R.L. Yang, T.C. Kaspar, and J.A. Stone. 1980. Soybean row spacing and soil water supply: Their effect on growth, development, water relations, and mineral uptake. Agricultural Research, North Central Region Science and Education Administration. USDA, Peoria, IL.

Mayaki, W.C., I.D. Teare, and L.R. Stone. Top and root growth of irrigated and nonirrigated soybeans. 1976. Crop Sci. 16:92-94.

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

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

Melgar, J., K.W. Roy, and T.S. Abney. 1994. Sudden death syndrome of soybean: etiology, symptomology, and effects of irrigation and *Heterodera glycines* on incidence and severity under field conditions. Can. J. Bot. 72:1647-1653.

Mitchell, R.L., and W.J. Russell. 1971. Root development and rooting patterns of soybean (*Glycine max* (L.) Merrill) evaluated under field conditions. Agron J. 63:313-316.

Mueller, D.S., G.L. Hartman, R.L. Nelson, and W.L. Pedersen. 2002. Evaluation of *Glycine max* germ plasm for resistance to *Fusarium solani* f. sp. *glycines.* Plant Dis. 86:741-746.

Mueller, D.S., R.L. Nelson, G.L. Harman, and W.L. Pedersen. 2003. Response of commercially developed soybean cultivars and the ancestral soybean lines to *Fusarium solani* f. sp. *glycines.* Plant Dis. 87:827-831.

Muendel, H.H. 1986. Emergence and vigor of soybean in relation to initial seed moisture and soil temperature. Agron. J. 78:765-769.

Muthiah, S., D.E. Longer, and W.M. Harris. 1994. Staging soybean seedling growth from germination to emergence. Crop Sci. 34:289-291.

Navi, S.S., and X.B. Yang. 2008. Foliar symptom expression in association with early infection and xylem colonization by *Fusarium virguliforme* (formerly *F. solani* f. sp. *glycines*), the causal agent of soybean sudden death syndrome. Online. Plant Health Progress doi:10.1094/PHP-2008-0222-01-RS.

Njiti, V.N., R.J. Suttner, L.E. Gray, P.T. Gibson, and D.A. Lightfoot. 1997. Rate- reducing resistance to *Fusarium solani* f. sp. *phaseoli* underlies field resistance to soybean sudden death syndrome. Crop Sci. 37:132-138.

Ortiz-Ribbing, L.M. and D.M. Eastburn. 2004. Soybean root systems and sudden death syndrome severity: Taproot and lateral root infection. Plant Dis. 88:1011-1016.

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

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

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

Rupe, J.C., E.E. Gbur, and D.M. Marx. 1991. Cultivar responses to sudden death syndrome of soybean. Plan Dis. 75:47-50.

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

Rupe, J.C., and Gbur, E.E. Jr., 1995. Effect of plant age, maturity group, and the environment on disease progress of sudden death syndrome of soybean. Plant Dis. 79:139-143.

Rupe, J.C., and G.L. Hartman. 1999. Sudden death syndrome. In: Hartman GL, Sinclair JB, Rupe JC, eds. *Compendium of Soybean Diseases.* St Paul, MN, USA: APS Press, 37-39.

Rupe, J.C., R.T. Robbins, C.M. Becton, W.A. Sabbe, and E.E. Gbur Jr. 1999. Vertical and temporal distribution of *Fusarium solani* and *Heterodera glycines* in fields with sudden death syndrome of soybean. Soil Biol. Biochem. 31:245-251.

Sanogo, S., and X.B. Yang. 1999. Recent outbreak of soybean sudden death syndrome in Iowa. Plant Dis. 83:590.

Scheiner, J.D., F.H. Gutierrez-Boem, and R.S. Lavado. 2000. Root growth and phosphorus uptake in wide- and narrow-row soybeans. Journal of Plant Nutrition. 23(9), 1241-1249.

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

Scherm, H., and X.B. Yang. 1999. Risk assessment of sudden death syndrome of soybean in the north-central United States. Agricultural Systems 59:301-310.

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

Shibles, R.M., and C.R. Weber. 1965. Leaf area, solar radiation interception and dry matter production by soybeans. Crop Sci. 5:575-578.

Shibles, R.M., and C.R. Weber. 1966. Interception of solar radiation and dry matter production by various soybean planting patterns. Crop Sci. 6:55-59.

Sivakumar, M.V.K., H.M Taylor, and R.H. Shaw. 1977. Top and root relations of field-grown soybeans. Agron. J. 69:470-473.

Smith, K.J., and W. Huyser. 1987. World Distribution and Significance of Soybean. P. 1. *In* J.R. Wilcox (ed.) SOYBEANS: Improvement, Production, and Uses. Second Edition. ASA, CSSA, and SSSA, Madison, WI.

Spaeth, S.C., and T.R. Sinclair. 1984a. Soybean seed growth. I. Timing of individual seeds. Agron J. 76:123-127.

Spaeth, S.C., and T.R. Sinclair. 1984b. Soybean seed growth II. Individual seed mass and component compensation. Agron J. 76:128-133.

University of Illinois. 2008. Varietal Information Program for Soybean [VIPS]. Compare Varieties [Online]. Available at http://www.vipsoybeans.org/v4/vpCompare/cvResultsCS1.cfm?b=y&cvOpt=optCS1 (accessed March, 2010). UIUC, Illinois.

USDA-National Agricultural Statistics Service. 2010. Quik Stats US & All States Data – Soybeans [Online]. Available at http://www.nass.usda.gov/QuickStats/PullData_US.jsp (accessed 11 March 2010). USDA-NASS, Washington, DC.

Vick, C.M., S.K. Chong, J.P. Bond, and J.S. Russin. 2003. Response of soybean sudden death syndrome to subsoil tillage. Plant Dis. 87:629-632.

Wrather, J.A., S.R. Kendig, S.C. Anand, T.L. Niblack, and G.S. Smith. 1995. Effects of tillage, cultivar, and planting date on percentage of soybean leaves with symptoms of sudden death syndrome. Plan Dis. 79:560-562.

Wrather, J.A., S.R. Koenning, and T.R. Anderson. 2003. Effect of diseases on soybean yields in the United States and Ontario (1999-2002). Online. Plant Health Progress doi:10.1094/PHP-2003-0325-01-RV.

Wrather, J.A., and S.R. Koenning. 2006. Estimates of Disease Effects on Soybean Yields in the United States 2003 to 2005. Journal of Nematology 38:173-180.

CHAPTER 3. EFFECT OF PLANTING DATE ON SUDDEN DEATH SYNDROME IN IOWA

An article to be submitted to *Agronomy Journal*

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Abstract

Sudden death syndrome (SDS) caused by the pathogen *Fusarium virguliforme* is a disease in Iowa causing significant yield reductions in soybean [*Glycine max* (L.) Merr.]. Research in the southern United States has demonstrated that early-planted soybean exhibits more severe SDS symptoms than late-planted soybean. The objective of this study was to evaluate the effect of planting date on SDS foliar symptom incidence, severity and yield in Iowa. In 2008 and 2009, a SDS-susceptible and SDS-resistant cultivar were planted at two Iowa locations at three different planting dates in plots inoculated with and without the pathogen. Beginning at first demonstration of foliar symptoms, plots were visually assessed every ten days for disease incidence and severity. Weather conditions varied across years, resulting in inconsistent and low disease levels. Planting date had an effect on SDS disease expression with early-planted soybean demonstrating higher occurrence of SDS than late-planted soybean. Despite higher levels of disease with early-planted soybean, yield was not less than late-planted soybean. The SDS-susceptible cultivar yielded higher than the SDS-resistant cultivar in both years, despite higher levels of disease, particularly in 2009. Although disease expression was greater in early-planted versus late-planted soybean it is

still recommended that growers should continue to optimize yield by planting early. Despite higher yield observed with the SDS-susceptible cultivar, it is still recommended to use a high-yielding SDS-resistant cultivar in a SDS environment.

Introduction

Early planting in the upper Midwest of the United States is an important and inexpensive agronomic practice that increases soybean yield potential (Bastidas et al., 2008; De Bruin and Pedersen 2008a; b; Robinson et al., 2009). Experience from the upper Midwest demonstrates that planting from the last week of April through the first week of May results in the highest soybean yield (De Bruin and Pedersen, 2008a; b; Wilcox and Frankenberger, 1987). Early-planted soybean have more vegetative nodes (Bastidas et al., 2008; Wilcox and Frankenburger, 1987), increased leaf area index (Pedersen and Lauer, 2004) and greater plant biomass (Anderson and Vasilas, 1985), leading to the production of more pods (Robinson et al., 2009; Pedersen and Lauer, 2004) and a higher seed set per area (Anderson and Vasilas, 1985; De Bruin and Pedersen, 2008b).

More than 50% of the soybean yield lost to soybean pathogens in Iowa is caused by soybean cyst nematode (*Heterodera glycines* Ichinohe; SCN) and sudden death syndrome (*Fusarium virguliforme* formerly called, *F. solani* f. sp. *glycines*; SDS; Aoki et al., 2003) (Wrather et al., 2003). Sudden death syndrome is often observed in low, moist or compacted areas of fields and often where SCN is also a problem (Roy et al., 1997). *Fusarium virguliforme*, a soilborne pathogen, infects the plant through the roots (Navi and Yang, 2008) early in the growing season (Njiti et al., 1997). Foliar symptoms normally develop at flowering and include chlorosis, necrosis, defoliation, flower and pod abortion as well as

total plant defoliation (Roy et al., 1997). Root necrosis and reduction in the root mass are also observed with increasing foliar symptoms (Roy et al., 1997; Rupe and Hartman, 1999).

Environmental conditions, plant growth stage, and cultivar susceptibility greatly influence SDS development (Rupe and Gbur, 1995). Increased SDS severity has been associated with early planting (Hershman et al., 1990; Wrather et al., 1995). In Kentucky, Hershman et al. (1990) found that symptoms of SDS seemed more severe in early as opposed to late-planted soybean and stated that soil moisture at the V3 growth stage (Fehr and Caviness, 1977) was greater in early-planted soybean than late plantings, which may have enhanced infection of plants. These observations regarding soil moisture, growth stages and SDS disease expression were corroborated by the work of Scherm and Yang (1996) who reported a positive relationship between soil moisture and SDS incidence. Their results indicate that low temperatures (15°C) and high soil moisture during vegetative growth stages (V3) followed by higher temperatures (22 to 24°C) and adequate moisture during reproductive stages (Roy et al., 1989; Rupe and Gbur, 1995) are optimal conditions for SDS symptom expression. These cool, wet conditions that favor SDS disease development are the soil conditions in late April and early May when growers in Iowa are planting soybean to maximize yield potential (De Bruin and Pedersen, 2008b).

Soilborne pathogens, such as SCN and brown stem rot (*Phialophora gregata*; BSR), can negate the yield benefits of early planting if not managed (De Bruin and Pedersen, 2008a; Grau et al., 1994). Previously, it was documented that SDS symptoms must be severe before the R5 growth stage in order to significantly decrease seed yield (Stephens et al., 1993). Cultivar selection with tolerance and even resistance to SDS is essential to manage

SDS (Roy et al., 1997; Rupe et al., 1991). Hershman et al. (1990) recommend, based on research from the southern United States, that yield loss due to SDS could be reduced through delayed planting or planting early-maturing cultivars, two methods that would result in disease development at a later reproductive stage, resulting in less yield loss (Rupe and Gbur, 1995). However, early planting in the southern United States at the time the research was conducted was considered to be mid-May (Hershman et al., 1990; Rupe and Gbur, 1995; Wrather et al., 1995). Soybean producers in Iowa and the upper Midwest, where over 80% of the United States' soybean production occurs (National Agricultural Statistics Service, 2010), plant in the last week of April and first week of May to maximize yield (Bastidas et al., 2008; De Bruin and Pedersen 2008a; b; Robinson et al., 2009). Therefore, SDS management recommendations based on research considering early planting to be mid-May are not appropriate for growers in Iowa and the upper Midwest. Currently, growers in Iowa do not have information on the effect that cultivar resistance will have on SDS development and yield for various planting dates.

Early planting of soybean is important to maximize yield, yet the yield advantage of early planting could be reduced if early-planted soybean are at risk for an increase of SDS that impacts yield. As SDS has become more common in Iowa, information regarding agronomic practices and interactions with SDS disease development is critical. We hypothesize that although early-planted soybean will experience optimal environmental conditions for disease infection and therefore more incidence and severity of SDS than late-planted soybean, SDS-resistant cultivars will demonstrate a positive yield response to early planting. The objective for this research was to evaluate differences in incidence of SDS

foliar symptoms and severity and soybean seed yield among three planting dates using an SDS-resistant and SDS-susceptible cultivar in Iowa.

Materials and Methods

Studies were conducted in Iowa during 2008 and 2009 near Jefferson and Nevada in fields with a prior history of SDS (Table 1). The experimental design was a randomized complete block in a split-plot arrangement with four replications. Main plots were three planting dates of early May, mid-May, and late May/June (Table 1) and sub-plots were a factorial combination of two inoculation treatments (with and without SDS inoculum) and two cultivars. One cultivar was classified as resistant to SDS (K-285; Kruger Seed, Dike, IA), and one cultivar was classified as susceptible to SDS (K-275; Kruger Seed, Dike, IA), as indicated by seed company assessments. Both cultivars had similar genetic background and contained PI88788 SCN resistance. Sorghum (*Sorghum bicolor* (L.) Moench) seed was inoculated with *F. virguliforme* as outlined by Farias Neto et al. (2006) and planted with the soybean seed in the furrow with 125 ml of inoculum per plot (approximately 3.3 g of infested sorghum seed per meter of row) (Farias Neto et al., 2006). Three pathogenic *F. virguliforme* isolates (Clinton 1.b, Scott F21 11a, Scott B2) were used to produce the inoculum. Isolates were collected and isolated by H. Scherm and X.B. Yang (Sanogo et al., 2000, Scherm et al., 1998) and grown on one-third strength Difco Potato Dextrose Agar (PDA). Media on which the isolate grew was cut into three equal-sized pieces in each plate. One piece of isolate bearing media, from each of the three isolates, was placed in a sealed, plastic bag containing 2.27 kg of sorghum seed. Bags were incubated at room temperature for 15 days. After 15 days, sorghum seed was removed from bags and allowed to dry at room temperature.

Field locations were chisel-plowed in the fall and field cultivated twice in the spring. Pre-emergent herbicide consisting of s-metolachlor [2-chloro-*N*-(2-ethyl-6-methylphenyl)-*N*-(-2-methoxy-1-methylethyl) acetamide]) at a rate of 0.92 kg a.i. ha^{-1} and fomesafen 5-[2-chloro-4-9trifluoromethyl) phenoxy]-N-(methysulfonyl)-2-nitrobenzamide)) at a rate of 0.20 kg a.i. ha^{-1} was applied to manage weeds, followed by two post emergence applications of glyphosate [N-(phosphonomethyl)glycine] at a rate of 1.12 kg a.i. ha^{-1}. Plot size was 3 by 7.6 m and plots were planted with an Almaco grain drill (Almaco, Nevada, IA) in 76-cm rows at a seeding rate of 371,000 seeds ha^{-1}.

Disease assessments were conducted in field plots every 10 days, beginning when plants first demonstrated foliar symptoms until R7 (Table 2). Two measures of disease were obtained: disease incidence and disease severity. Disease incidence was defined as a percentage of plants in a plot demonstrating leaf symptoms (Njiti et al., 1996; 1998), while disease severity was scored on a scale of 1 to 9 (mild to severe symptoms), based on the percentage of the leaf area displaying symptoms on plants. 1 = 0 to 10% chlorosis or 1 to 5% necrosis, 2 = 10 to 20% chlorosis or 6 to 10% necrosis, 3 = 20 to 40% chlorosis or 10 to 20% necrosis, 4 = 40 to 60% chlorosis or 20 to 40% necrosis, 5 = > 60% chlorosis or > 40% necrosis, 6 = up to 33% premature defoliation, 7 = up to 66% premature defoliation, and 9 = premature death of the plant (Njiti et al., 1996).

Seed yield was determined by harvesting the center two rows of each plot with an Almaco plot combine, and harvest weights were adjusted to 130 g kg^{-1} moisture for final yield determination. Additional data acquired at harvest were plant height, lodging, plant density and seed mass (based on a sample weight of 300 seeds). Lodging values were scored

on a scale of 1 to 5 with 1 representing completely erect plants and 5 representing completely prostrate plants.

Yield data were analyzed using PROC MIXED in SAS 9.2 (SAS Institute, 2008), treating planting date, cultivar, and inoculum as fixed effects and environment and replication as random effects. Disease data were analyzed using a repeated measures mixed model with a compound symmetry covariance structure selected based on Akaike's Information Criteria (AIC). Environment, planting date, cultivar, inoculum and date of disease assessment were treated as fixed effects with replication treated as a random effect. Because of variation in disease levels and number of disease assessments, all data were analyzed by year and using a Kenward-Rogers approximation to calculate degrees of freedom.

Results and Discussion

Air temperature during both years was slightly below average at both locations. Rainfall varied considerably between years. During May, June and July monthly rainfall totals were above normal in 2008 (ranging from 1 to 160 mm above normal) and below normal in 2009 (ranging from -13 to -59 mm below normal), with the exception of July 2009 at Jefferson which was 24 mm above normal.

Yield

An interaction between planting date and cultivar on yield was observed in 2008 (Table 4). At the early planting date, the SDS-susceptible cultivar K-275 yielded 8% greater than the SDS-resistant cultivar K-285. No yield difference between the two cultivars at the later two planting dates was observed (data not shown).

In 2008, mid-planted soybean yielded 6% greater than the late-planted soybean, respectively (Table 4). This yield response is in accordance with previous observations and results seen for yield response to planting date (De Bruin and Pedersen, 2008b; Wilcox and Frankenburger, 1987). In 2009 there was evidence ($P = 0.06$) that later planting increased yield (Table 4). Late-planted soybeans typically yield less than early-planted soybeans in Iowa (De Bruin and Pedersen, 2008b) but the combination of extreme cool growing conditions (Table 3) and SDS may have reduced yield of early-planted soybean..

The SDS-susceptible cultivar, K-275, yielded greater than the SDS-resistant cultivar, K-285 by 4% in 2008 and by 5 % in 2009 (Table 4). The higher yield response of the susceptible cultivar was not expected, yet is not unusual as yield-penalties have been previously shown for planting SCN-resistant and Phytophthora-resistant cultivars in environments with low disease pressure (Donald et al., 2006; Tooley and Grau, 1986). We speculate that low yield potential of K-285, low levels of overall disease pressure, and presence of other diseases may have contributed to this yield response.

In 2008 there was no yield difference between inoculated and non-inoculated plots (Table 4). However, SDS expression in 2009 was greater than 2008 and inoculated plots yielded 7% yield less compared to non-inoculated plots in 2009 (Table 4).

Final Plant Population

In 2008 there was an interaction between planting date, cultivar and inoculation for final plant population (Table 4). In both years late-planted soybean had higher final plant population than early- and mid-planted soybean (Table 4), which agrees with previous studies showing that plant establishment increases in late planted soybean because of

increased soil temperature (Oplinger and Philbrook, 1992). In both years, the SDS-susceptible cultivar K-275 had a higher final plant population than the SDS-resistant cultivar K-285 (Table 4). Inoculation did not have an effect on final plant population in either year (Table 4). These results indicate final plant population was not affected by SDS.

Plant Height

An interaction was observed between planting date and cultivar in 2009 for plant height (Table 4). Late planting of the SDS-resistant cultivar K-285 resulted in 6% greater plant height than the SDS-susceptible cultivar K-285 planted at the earlier dates (data not shown). This reduction in height at the early and mid-planting dates contradicts the response of plant height to planting date found by Bastidas et al. (2008) and Wilcox and Frankenburger (1987). Early and mid-planted soybean experienced greater disease symptoms than late-planted soybean (Table 5), therefore the height reduction at the early and mid-planting dates may be attributed to *F. virguliforme* infection. This is in agreement with previous work on the response of plant height to *F. solani* isolates (Rupe, 1989). A planting date by inoculation interaction for plant height was also observed in 2009 (Table 4). Late-planted soybean in non-inoculated plots were 7% taller than soybean in non-inoculated plots at the earlier date (data not shown).

Overall, late-planted soybean was taller than mid- and early-planted soybean in 2008, and early-planted soybean in 2009 (Table 4) which agrees with work conducted by Pedersen and Lauer (2003) but conflicts with that of Bastidas et al. (2008). In both years, K-275 demonstrated greater plant height than K-285 (by 11% in 2008 and by 16% in 2009) (Table 4). Inoculation did not have an effect on plant height in either year (Table 4).

Seed Mass, Oil and Protein

An interaction between planting date and inoculation was observed for seed mass in 2008 (Table 4). For late-planted soybean, inoculated plots had 3% greater seed mass than non-inoculated plots whereas no difference in seed mass between inoculated and non-inoculated plots existed at the earlier dates (data not shown). This increase in seed mass for late planting dates contradicts results found by Anderson and Vasilas (1985) and Raymer and Bernard (1988) that seed mass decreases with delayed planting. Interactions between planting date and cultivar and also between cultivar with inoculation were found in 2009 for protein content (Table 4). The interaction between planting date and cultivar was attributed to low protein content of K-285 planted at the mid-planting date (data not shown). The interaction between cultivar with inoculation is attributed to low protein content of K-285 in inoculated plots compared to control plots (data not shown). These differences in protein content were small and because of the variable response of the cultivars to disease there is no disease-related explanation for these interactions.

Seed mass increased with delayed planting in both years (Table 4). Although a tendency for late planting to increase seed mass has been documented (Pedersen and Lauer, 2004) our finding disagrees with previous findings indicating that seed mass decreased with delayed planting (Anderson and Vasilas, 1985; De Bruin and Pedersen, 2008a; Raymer and Bernard, 1988). This suggests that SDS affected seed fill of early and mid-planted soybean as these planting dates experienced higher levels of disease expression than late-planted soybean (Table 5).

In general, protein and oil content decreased with delayed planting in both years (Table 4). Several studies have documented oil concentration to decrease with delaying planting (Bastidas et al., 2008; Kane et al., 1997; Robinson et al., 2009). However, our data contradict previous observations on the response of planting date on protein content. Other studies have documented that protein content increases with later planting dates (Bastidas et al., 2008; Kane et al., 1997; Robinson et al., 2009) and that protein accumulation is sensitive to reduced source strength (Proulx and Naeve, 2009). Inoculation did not affect protein or oil content in either year (Table 4). Therefore, we attribute the decrease in protein concentration with delayed planting to below average temperatures throughout the growing seasons (Table 3).

In 2008, the SDS-susceptible cultivar K-275 had a higher seed mass than the SDS-resistant cultivar K-285 (Table 4). Over both years K-275 demonstrated higher oil content than K-285. In 2009, K-275 had a higher protein content than K-285 (Table 4). Except for seed mass in 2009, no differences were observed between inoculated and non-inoculated plot for seed mass, protein and oil content. In 2009, soybean of inoculated plots had a lower seed mass compared to non-inoculated plots (Table 4) supporting previous findings that SDS reduces seed size (Roy et al., 1997).

Disease Incidence and Severity

Soil moisture is an important factor associated with development of SDS (Rupe et al., 1989; Scherm and Yang, 1996); therefore the variation in rainfall could have contributed to the difference in disease levels observed between years (Table 3). Although SDS is favored by high soil moisture, previous work has shown too much rainfall during vegetative growth

may reduce or delay SDS (Rupe and Gbur, 1995). Therefore, heavy rainfall in May and June of 2008 could have played a role in the low levels of disease in this study. Previous observation has shown SDS to be limited with hot and dry conditions but increased when moisture is not a limiting factor (Hershman et al., 1990). Therefore, SDS progression later in the 2008 growing season could have been limited by below normal rainfall totals in August (Table 3) while plants were in growth stages R3 – R6 (Table 2).

Several interactions were observed each year for disease incidence and severity. An interaction for planting date with date of disease assessment was found for disease incidence and severity in both years. Early-planted soybean had a greater foliar symptom incidence and severity at the final four assessment dates in 2008 and on 22 June, 1, 9 July, 10, 21 August, and 3 September in 2009 (Fig. 1). Mid-planted soybean had greater disease incidence and severity than late-planted soybean on 10 September 2008 (Fig. 1). Our results and disease progression in each of the planting dates throughout the season indicate that disease development is a function of timing between growth stages and environment rather than a function of specific growth stage (Fig. 1; Table 2). In 2008, early-planted soybean had 0% disease incidence at growth stage R3 (21 July) compared with 8.8% at growth stage R5 (11 August). In comparison, late-planted soybean had 1.4% disease incidence at R3 (30 July) and decreased to 0.6% by R5 (3 September). The time period between 21 July and 3 September appears to be an important time for disease progression if soybean have reached R3 but not that critical for soybean only in the late vegetative stages (V7). Our data support previous work that environmental conditions and plant growth stage are critical for SDS development (Rupe and Gbur, 1995).

In 2008, there was an interaction between cultivar with date of disease assessment on disease severity (Fig. 2). The interaction is attributed to differences between the two cultivars in level of foliar symptom severity on 30 July and 3 September, with K-275 having less disease severity than K-285 on 30 July and greater disease severity than K-285 on 3 September.

In 2009 an interaction of cultivar and date of disease assessment was found for disease incidence and severity (Fig. 2). No difference in cultivar performance was detected before 21 August, at which date K-275 demonstrated greater levels of disease incidence and severity than K-285 (Fig. 2). These data illustrate the variability in cultivar response to SDS and indicate that difference in cultivar performance is not detected until plants reach early reproductive periods (R2 in 2008 and R4 in 2009) (Table 2). The tendency of K-285 to have an earlier onset of disease incidence than K-275 and K-275 to have greater disease expression later in the growing seasons (Fig 2) may explain why K-275 yielded greater than K-285 by 4% in 2008 and by 5 % in 2009 (Table 4).

In 2009, there were interactions observed among planting date, cultivar and inoculation for disease incidence and severity; planting date, inoculation and timing of disease assessment for disease incidence and severity; and cultivar, inoculation and timing of disease assessment for disease incidence (Table 5). Considering environmental and disease variability as well as the variable response of the cultivars to SDS, explanations for these interactions were non-conclusive (data not shown).

In 2008, early-planted soybean had greater levels of disease incidence (5.4%) than the two later planting dates (3.7 and 0.6%), respectively (Table 5). However, this did not

translate into a yield reduction (Table 4). One explanation may be that the overall levels of disease in 2008 were low (Table 3; 5). In 2009, early-planted soybean had a higher disease incidence and severity than mid and late-planted soybean (Table 5). Combined with the observation that later planting increased yield ($P = 0.06$) (Table 4), this data suggests that SDS reduced yield in early-planted soybean. The higher levels of disease in the early compared to late-planted soybean in both years is consistent with previous findings indicating that SDS symptoms are more severe with earlier planting (Hershman et al., 1990).

In 2008, no differences were found between cultivars for disease incidence or severity (Table 5). This suggests that in environments with low-levels of disease there is no differentiable response of the cultivars in our study. In 2009, K-275 had a higher SDS disease incidence and severity than K-285 (Table 5) but no yield reduction was found (Table 4). In 2008 there was no difference between inoculated and non-inoculated plots for disease incidence or disease severity (Table 5). The low disease pressure in 2008 may be attributed to the cool and dry conditions that year in August (Table 3) or ineffective inoculum. However, in 2009, inoculated plots had a greater SDS disease incidence and severity than non-inoculated plots (Table 5), which translated into a 7% yield reduction in inoculated plots versus non-inoculated plots (Table 4).

Conclusion

Little research has previously been conducted from Iowa and the upper Midwest on planting date and SDS disease incidence and severity. This study documented that early-planted soybean have greater SDS symptom severity and incidence than late-planted soybean. However, the expression of SDS symptoms in the early-planted soybean did not

translate to a significant reduction in yield compared to the late-planted soybean. Although data from 2009 provided evidence that with greater levels of disease and optimum moisture for SDS development the yield advantage of earlier planting could be negated, disease levels were inconsistent across years and further research is necessary to confirm this observation. The results of this study support previous work demonstrating greater SDS occurrence in early-planted soybean yet these data do not provide conclusive evidence to recommend growers to sacrifice the yield maximizing practice of early planting to combat SDS. It is still recommended to plant early in Iowa to maximize yield though use of a high-yielding SDS-resistant cultivar in a field with a history of SDS.

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References

Aoki, T., K. O'Donnell, H. Yoshihisa, and A.R. Lattanzi. 2003. Sudden-death syndrome of soybean is caused by two morphologically and phylogenetically distinct species within the *Fusarium solani* species complex-*F. virguliforme* in North America and *F. tucumaniae* in South America. Mycologia 95:660-684.

Anderson, L.R., and B.L. Vasilas. 1985. Effects of planting date on two soybean cultivars: Seasonal dry matter accumulation and seed yield. Crop Sci. 25:999-1004.

Bastidas, A.M., T.D. Setiyono, A. Dobermann, K.G. Cassman, R.W. Elmore, G.L Graef, and

J.E. Specht. 2008. Soybean sowing date: The vegetative, reproductive and agronomic impacts. Crop Sci. 48:727-740.

De Bruin, J.L., and P. Pedersen. 2008a. Soybean cultivar and planting date response to soil fumigation. Agron. J. 100:965-970.

De Bruin, J.L., and P. Pedersen. 2008b. Soybean seed yield response to planting date and seeding rate in the upper midwest. Agron. J. 100:696-703.

Donald, P.A., P.E. Pierson, S.K. St. Martin, P.R. Sellers, G.R. Noel, A.E. MacGuidwin, J. Faghihi, V.R. Ferris, C.R. Grau, D.J. Jardine, H. Malakeberhan, T.L. Niblack, W.C. Stienstra, G.L. Tylka, T.A. Wheeler, and D.S. Wysong. 2006. Assessing *Heterodera Glycines*-resistant and susceptible cultivar yield response. J Nematol. 38(1):76-82.

Farias Neto, A.F., G.L. Hartman, W.L. Pedersen, S. Li, and B.W. Diers. 2006. Irrigation and inoculation methods that increase the severity of soybean sudden death syndrome in the field. Crop Sci. 46:2547-2554.

Fehr, W.R., and C.E. Caviness. 1977. Stages of soybean development. Spec. Rep. 80. Iowa Agric. Home Econ. Exp. Stn., Iowa State Univ., Ames.

Grau, C.R., E.S. Oplinger, E.A. Adee, E.A. Hinkens, and M.J. Martinka. 1994. Planting date and row width effect on severity of brown stem rot and soybean productivity. J. Prod. Agric. 7:347-351.

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

Kane, M.V., C.C. Steele, L.J. Grabau, C.T. MacKown, and D.F. Hildebrand. 1997. Early-

maturing soybean cropping systems: III. Protein and oil contents and oil composition. Agron. J. 89;464-469.

Navi, S.S., and X.B. Yang. 2008. Foliar symptom expression in association with early infection and xylem colonization by *Fusarium virguliforme* (formerly *F. solani* f. sp. *glycines*), the causal agent of soybean sudden death syndrome. Online. Plant Health Progress doi:10.1094/PHP-2008-0222-01-RS.

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

Njiti, V.N., R.J. Suttner, L.E. Gray, P.T. Gibson, and D.A. Lightfoot. 1997. Rate- reducing resistance to *Fusarium solani* f. sp. *phaseoli* underlies field resistance to soybean sudden death syndrome. Crop Sci. 37:132-138.

Njiti, V.N., M.A. Shenaut, R.J. Suttner, M.E. Schmidt, and P.T. Gibson. 1998. Relationship between soybean sudden death syndrome disease measures and yield components in F_6-derived lines. Crop Sci. 38:673-678.

Oplinger, E.S. and B.D. Philbrook. 1992. Soybean planting date, row width, and seeding rate response in three tillage systems. J. Prod. Agric. 5:94-99.

Pedersen, P., and J.G. Lauer. 2003. Soybean response to management systems. Agron J. 95:1146-1151.

Pedersen, P., and J.G. Lauer. 2004. Response of soybean yield components to management system and planting date. Agron. J. 96:1372-1381.

Proulx, R.A. and S.L. Naeve. 2009. Pod removal, shade, and defoliation effects on soybean yield, protein, and oil. Agron J. 101:971-978.

Raymer, P.L., and R.L. Bernard, 1988. Response of current midwestern soybean cultivars to late planting. Crop Sci. 28:761-764.

Robinson, A.P., S.P. Conley, J.J. Volenec, and J.B. Santini. 2009. Analysis of high yielding, early-planted soybean in Indiana. Agron. J. 101:131-139.

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

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

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

Rupe, J.C., M.C. Hirrel, and D.E. Hershman. 1989. Sudden death syndrome. In: Sinclair JB, Backman PA, eds. *Compendium of Soybean Diseases*. St. Paul, MN, USA: APS Press, 84-85.

Rupe, J.C., E.E. Gbur, and D.M. Marx. 1991. Cultivar responses to sudden death syndrome of soybean. Plant Dis. 75:47-50.

Rupe, J.C., and Gbur, E.E. Jr., 1995. Effect of plant age, maturity group, and the environment on disease progress of sudden death syndrome of soybean. Plant Dis. 79:139-143.

Rupe, J.C., and G.L. Hartman. 1999. Sudden death syndrome. In: Hartman GL, Sinclair JB,

Rupe JC, eds. *Compendium of Soybean Diseases.* St Paul, MN, USA: APS Press, 37-39.

Sanogo, S., X.B. Yang, and H. Scherm. 2000. Effects of herbicides on *Fusarium solani* f. sp. *glycines* and development of sudden death syndrome in glyphosate-tolerant soybean. Phytopathology 90:57-66.

SAS Institute. 2008. The SAS system for Windows. v. 9.2. SAS Inst. Cary, NC.

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

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

Stephens, P.A., C.D. Nickell, C.K. Moots, and S.M. Lim. 1993. Relationship between field and greenhouse reactions of soybean to *Fusarium solani*. Plant Dis. 77:163-166.

Tooley, P.W. and C.R. Grau. 1986. Microplot comparison of rate-reducing and race-specific resistance to *Phytophthora megasperma* f. sp. *glycinea* in soybean. Phytopathology. 76:554-557.

USDA-National Agricultural Statistics Service. 2010. Quik Stats US & All States Data – Soybeans [Online]. Available at http://www.nass.usda.gov/QuickStats/PullData_US.jsp (accessed 11 March 2010). USDA-NASS, Washington, DC.

Wilcox, J.R., and E.M. Frankenberger. 1987. Indeterminate and determinate soybean responses to planting date. Agron. J. 79:1074-1078.

Wrather, J.A., S.R. Kendig, S.C. Anand, T.L. Niblack, and G.S. Smith. 1995. Effects of tillage, cultivar, and planting date on percentage of soybean leaves with symptoms of sudden death syndrome. Plant Dis. 79:560-562.

Wrather, J.A., S.R. Koenning, and T.R. Anderson. 2003. Effect of diseases on soybean yields in the United States and Ontario (1999-2002). Plant Health Progress. Doi:10.1094/PHP-2003-0325-01-RV.

Table 1. Field characteristics at the Jefferson and Nevada field sites in 2008 and 2009.

Location	Jefferson		Nevada	
Year	2008	2009	2008	2009
Soil Series	Canisteo clay loam	Canisteo clay loam	Webster clay loam	Webster clay loam
Soil Family	Fine-loamy, Mixed, Superactive, Mesic Typic Hapludolls	Fine-loamy, Mixed, Superactive, Mesic Typic Hapludolls	Fine-loamy, Mixed, Superactive, Mesic Typic Hapludolls	Fine-loamy, Mixed, Superactive, Mesic Typic Hapludolls
pH	6.7	6.8	7.5	6.8
P (mg kg^{-1})	23	8	38	68
K (mg kg^{-1})	176	188	197	280
OM (g kg^{-1})†	47	42	55	46
Planting Dates	1 May	5 May	5 May	5 May
	17 May	19May	17 May	19 May
	16 June	31 May	16 June	31 May
Harvest Dates				
	1 Oct	28 Sept	2 Oct	29 Sept
	10 Oct	11 Oct	11 Oct	11 Oct

† OM, organic matter

Table 2. Soybean growth stages from three different planting dates at the disease assessment dates across the two locations in Iowa during 2008 and 2009.

2008				2009			
Date of disease assessment	Growth Stage†			Date of disease assessment	Growth Stage		
	E‡	M	L		E	M	L
30 June	R1	V5	V1	10 June	V3	V1	VE
10 July	R2	R1	V3	22 June	V5	V3	V1
21 July	R3	R2	V7	1 July	R1	V5	V3
30 July	R4	R3	R2	9 July	R2	R1	V5
11 August	R5	R4	R3	22 July	R3	R2	R1
21 August	R6	R5	R4	30 July	R4	R3	R2
3 September	R6	R6	R5	10 August	R5	R4	R3
10 September	R7	R7	R6	21 August	R6	R5	R4
-	-	-	-	3 September	R7	R6	R5

† Growth stages as described by Fehr and Caviness, 1977.
‡ E, M and L represent early May, mid May and late May/early June planting dates, respectively.

Table 3. Monthly mean air temperature and precipitation totals recorded at two experimental locations in 2008 and 2009. Deviations from the 20-yr average are reported in parentheses.

		May		June		July		August		September	
Year	Location	Air temp.	Rainfall	Air temp.	Rainfall	Air temp.	Rainfall	Air temp.	Rainfall	Air temp.	Rainfall
		°C	mm	°C	mm	°C	mm	°C	mm	°C	mm
2008	Jefferson	14 (-2.1)†	232 (117)	21 (-0.6)	184 (62)	23 (-1.1)	115 (1)	21 (-2.1)	46 (-62)	17 (-1.0)	47 (-29)
	Nevada	16 (-1.0)	216 (94)	21 (-0.4)	271 (160)	23 (0.05)	234 (123)	21 (-1.0)	53 (-73)	18 (-0.3)	78 (0)
2009	Jefferson	16 (-0.7)	77 (-42)	20 (-1.5)	115 (-13)	20 (-3.7)	135 (24)	21 (-1.5)	110 (12)	18 (0.3)	28 (-48)
	Nevada	16 (-0.8)	102 (-26)	71 (-0.3)	104 (-16)	21 (-2.7)	70 (-59)	21 (-1.6)	89 (-25)	18 (-0.3)	31 (-48)

† Twenty-year averages based on Iowa Environmental Mesonet locations near Jefferson and Nevada, IA.
Available at: http://mesonet.agron.iastate.edu/climodat/index.phtml

Table 4. Main effect means of planting date, cultivar and inoculation with *Fusarium virguliforme* or seed yield, final plant population, plant height, seed mass, protein and oil at Jefferson and Nevada in 2008 and 2009.

Treatment	Seed yield		Final plant population		Plant height		Seed mass		Protein		Oil	
	2008	2009	2008	2009	2008	2009	2008	2009	2008	2009	2008	2009
	kg ha^{-1}		Plants ha^{-1}		cm		g 100 seed^{-1}		g kg^{-1}		g kg^{-1}	
Planting Date (D)†												
Early	4202	4413	215900	226800	80	92	15.5	15.7	32.1	32.5	19.2	18.3
Mid	4273	4634	233200	236000	87	95	15.9	16.4	32.1	32.0	19.2	18.3
Late	3998	4822	287200	295300	95	98	16.6	17.1	31.7	32.3	18.7	17.8
LSD (0.05)	214	NS‡	21800	33200	6	4	0.4	0.5	0.3	0.3	0.3	0.3
Cultivar (C)§												
K-275	4245	4749	254800	276500	92	103	15.8	16.3	32.0	32.5	19.2	18.3
K-285	4071	4497	236000	229000	82	87	16.2	16.5	31.9	32.0	18.9	18.0
LSD (0.05)	123	190	17800	23700	3	3	0.2	NS	NS	0.3	0.1	0.3
Inoculation (I)												
Control	4198	4781	251700	259800	88	95	16.0	16.6	32.0	32.3	19.0	18.1
Inoculated	4118	4464	239200	245600	86	96	16.1	16.2	31.9	32.2	19.1	18.2
LSD (0.05)	NS	190	NS	NS	NS	NS	NS	0.3	NS	NS	NS	NS
ANOVA												
D X C	*	NS	NS	NS	NS	*	NS	NS	NS	*	NS	NS
D X I	NS	NS	NS	NS	NS	*	*	NS	NS	NS	NS	NS
C X I	NS	NS	NS	NS	NS	NS	NS	NS	NS	**	NS	NS
D X C X I	NS	NS	*	NS	NS	NS	NS	NS	NS	NS	NS	NS

† Early planting date was early May, mid planting date was mid-May, and late planting date was mid-June in 2008 and late May in 2009.

‡ NS, not significantly different at $P \leq 0.05$; *,**,*** significantly different at $P \leq 0.05$, 0.01, and 0.001 levels, respectively.

§ K-275 was susceptible to SDS, K-285 was resistant to SDS.

Table 5. Response of sudden death syndrome incidence and severity to planting date, cultivar, inoculation, and sampling time at Jefferson and Nevada in 2008 and 2009.

Treatment	Disease Incidence		Disease Severity	
	2008	2009	2008	2009
	%		1 – 9†	
Planting Date (D)‡				
Early	5.4	7.3	0.3	1.2
Mid	3.7	2.7	0.4	0.7
Late	0.6	0.3	0.1	0.1
LSD (0.05)	2.0	1.5	0.1	0.2
Cultivar (C)§				
K-275	3.3	4.3	0.27	0.79
K-285	3.1	2.6	0.23	0.51
LSD (0.05)	NS¶	1.3	NS	0.14
Inoculation (I)				
Control	3.1	1.0	0.3	0.2
Inoculated	3.4	5.9	0.3	1.1
LSD (0.05)	NS	1.3	NS	0.1
ANOVA				
D X C	NS	NS	NS	NS
D X I	NS	***	NS	***
C X I	NS	NS	NS	NS
D X T#	***	***	***	***
C X T	NS	***	*	***
I X T	NS	***	NS	***
D X C X I	NS	NS	NS	NS
D X C X T	NS	***	NS	**
D X I X T	NS	***	NS	*
C X I X T	NS	***	NS	NS
D X C X I X T	NS	NS	NS	NS

† If disease was present, foliar symptom severity assessed according to increasing severity on a scale of 1 – 9 as outlined by Njiti et al. (1996).

‡ Early planting date was early May, mid planting date was mid-May, and late planting date was mid-June in 2008 and late May in 2009.

§ K-275 was susceptible to SDS, K-285 resistant to SDS.

¶ NS, not significantly different at $P \leq 0.05$; *,**,*** significantly different at $P \leq 0.05$, $P \leq 0.01$, and $P \leq 0.001$ levels, respectively.

# Denotes timing of disease assessment treatment.

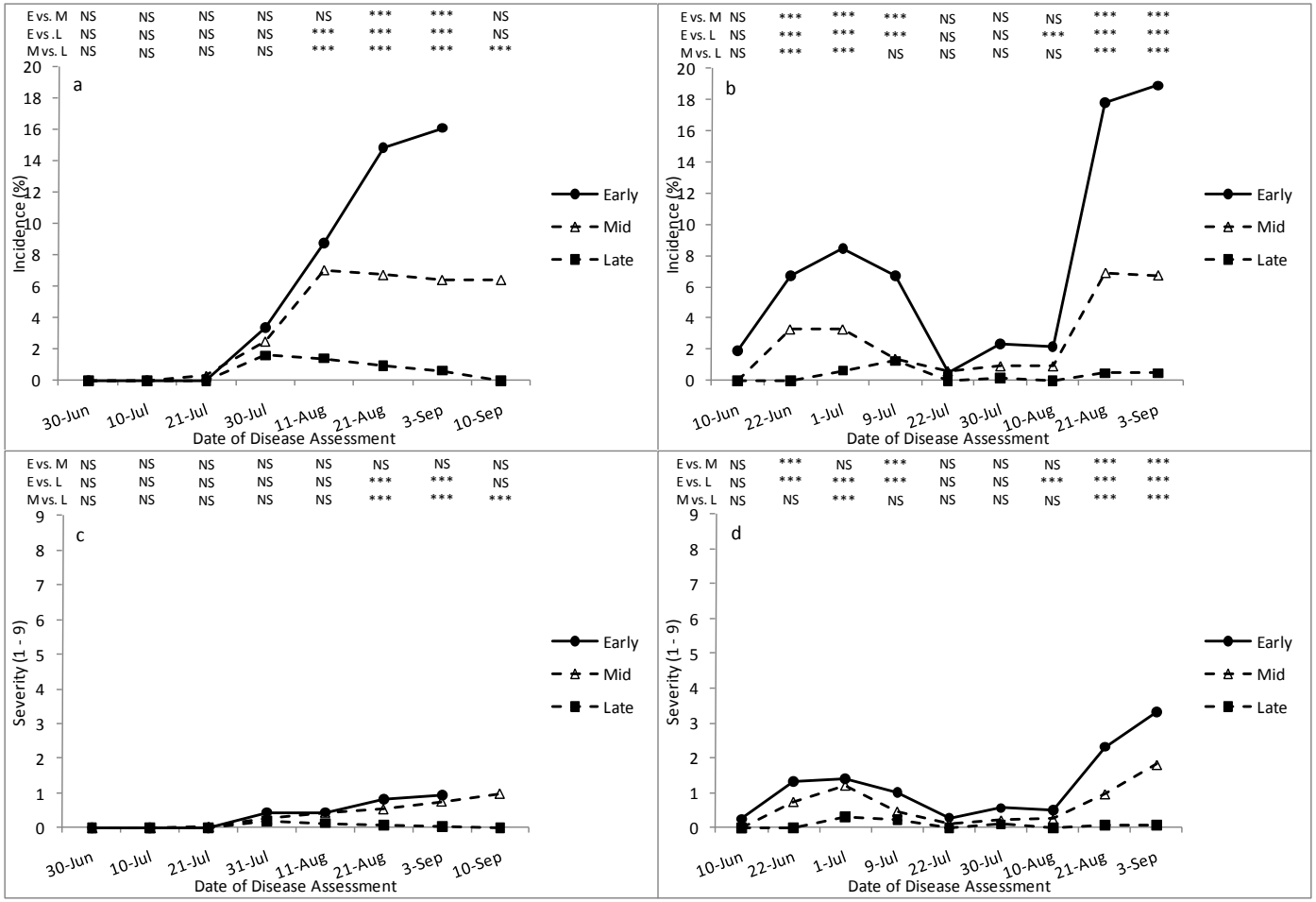

Figure 1. Disease incidence and severity in (a), (c) 2008, and (b), (d) 2009 in Iowa for three planting dates (early, mid and late) at each assessment time. Each point is the mean incidence or severity value of one SDS-susceptible cultivar (K-275) and one SDS-resistant cultivar (K-285) over inoculated and non-inoculated plots at one of three planting dates. *** represent significant differences ($P \leq 0.001$) and NS represents no significant differences ($P \leq 0.001$) in disease incidence or severity between respective planting dates at specific dates of disease assessment. E, M and L indicate early, mid, and late planting dates, respectively. Growth stages of soybean plants at each date of disease assessment and planting date are indicated in Table 2.

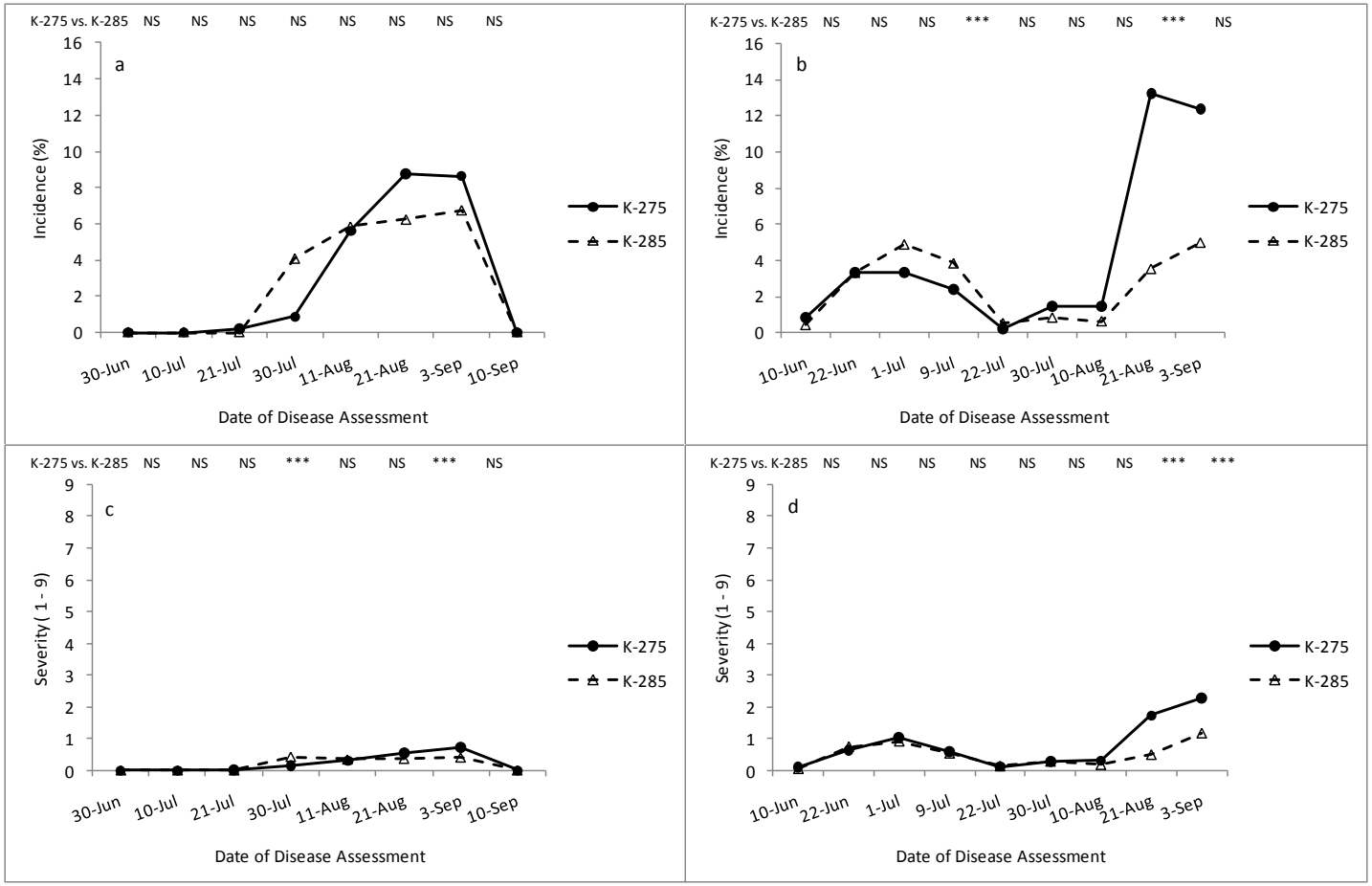

Figure 2. Disease incidence and severity (respectively) in (a), (c) 2008 and (b), (d) 2009 for the cultivars K-275 (SDS-susceptible) and K-285 (SDS-resistant) at each disease assessment date. Each point is the mean incidence or severity value of either the SDS-susceptible cultivar (K-275) or the SDS-resistant cultivar (K-285) over inoculated and non-inoculated plots at the corresponding date of disease assessment. *** represent significant differences ($P \leq 0.001$) and NS represents no significant differences ($P \leq 0.001$) in disease incidence or severity between the two cultivars at that specific date of disease assessment.

CHAPTER 4. SOYBEAN YIELD RESPONSE TO ROW SPACING AND SEEDING RATE IN SUDDEN DEATH SYNDROME ENVIRONMENTS

An article to be submitted to *Agronomy Journal*

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Abstract

Sudden death syndrome (SDS), caused by the pathogen *Fusarium virguliforme*, causes significant yield reductions in soybean [*Glycine max* (L.) Merr.] in the United States. Appropriate recommendations to manage SDS for growers in Iowa and the upper Midwest are limited. The research objective was to determine the response of SDS foliar disease incidence, severity and yield to row spacing and seeding rate. In 2008 and 2009, at two Iowa locations, in fields with histories of SDS, a SDS-susceptible and SDS-resistant cultivar were planted in 38- and 76-cm rows at seeding rates of 185,000; 309,000; and 432,000 seeds ha^{-1} in plots inoculated with and without the pathogen. Very low SDS incidence and severity was observed however differences were observed between inoculated and non-inoculated plots. Inoculated plots had greater SDS disease incidence and severity than non-inoculated plots. A row spacing by inoculation interaction indicated 7% greater yield in narrow rows (38-cm) than wide rows (76-cm) in non-inoculated plots, with no yield advantage to narrow rows in inoculated plots. Inoculation reduced soybean seed mass (7%) in narrow rows, explaining the yield reduction for narrow rows with greater SDS. The two highest seeding rates had increased SDS incidence, but yielded 9% greater than the lowest seeding rate. The susceptible cultivar had greater SDS incidence, severity and yielded 7% less than the

resistant cultivar. Despite no clear evidence, this study indicates that in inoculated plots with greater SDS symptom expression the yield advantage of narrow rows may be negated, therefore cultivar selection is crucial when planting in narrow rows to maximize yield.

Introduction

Row spacing and seeding rates are cultural practices that growers can utilize to achieve maximum soybean [*Glycine max* (L.) Merr.] yield (De Bruin and Pedersen, 2008a). Decreasing row spacing at equal plant populations creates a more equidistant plant distribution which results in greater canopy leaf area development and light interception earlier in the growing season (Shibles and Weber, 1966; Weber et al., 1966). Increased canopy leaf area development and light interception increases the crop growth rate, dry matter accumulation, and seed yield (Andrade et al., 2002; Bullock et al., 1998). Many growers still use seeding rates depending on row spacing even though research has demonstrated that in row spacings of 38-cm and 76-cm >95% of maximum yield can be reached with initial seeding rates of 309,000 seeds ha^{-1} regardless of row spacing (De Bruin and Pedersen, 2008a). For soybean, harvest plant populations needed to attain maximum yield are from 200,000 to 230,000 plants ha^{-1}. There is no yield increase with final plant populations higher than this range (De Bruin and Pedersen, 2009).

Sudden death syndrome, caused by the soilborne pathogen *Fusarium virguliforme* (formerly called, *F. solani* f. sp. *glycines*; SDS; Aoki et al., 2003), is a soybean disease favored by high-yield environments (Rupe et al., 1989). The disease is considered one of the most important limiting factors to soybean production in the United States (Aoki et al., 2003) and along with soybean cyst nematode (*Heterodera glycines* Ichinohe; SCN), accounts for more than 50% of soybean yield suppression caused by pathogens in Iowa (Wrather et al.,

2003). Infection takes place early in the growing season (Njiti et al., 1997), with the pathogen entering the plant through the root (Navi and Yang, 2008). Symptoms of the root infection include necrosis and reduction in root mass (Roy et al., 1997; Rupe and Hartman, 1999). Foliar symptoms are observed beginning at flowering and consist of necrosis and chlorosis that can lead to defoliation, flower and pod abortion (Roy et al., 1997) developing later in the season during the reproductive growth stages (Navi and Yang, 2008). If symptoms are severe enough, total plant defoliation occurs (Roy et al., 1997).

Soybean yield response to narrow row spacing is positive in Iowa (De Bruin and Pedersen, 2008a). Research has shown narrow rows have higher leaf area and light interception compared to wide rows over the course of a growing season (Shibles and Weber, 1966; Weber et al., 1966) and soybean planted at high populations produce greater leaf area than soybean planted at lower populations (Shibles and Weber, 1966). We hypothesize that the increased leaf area and light interception of narrow row and high population soybean may be beneficial by protecting against yield loss in a SDS environment because SDS can defoliate plants (Roy et al., 1997). However, increased seeding rate and plant to plant competition may increase stress on the canopy and can limit the benefit to narrow row spacing when environmental conditions limit plant growth (Devlin et al., 1995; Elmore, 1998).

Soybean diseases pose a threat to high-yield practices if they are not managed carefully (Grau and Radke, 1984; Grau et al., 1994). Grau et al. (1994) documented that brown stem rot (BSR) (caused by *Phialophora gregata*) was able to negate any yield benefits from early planting date and narrow row spacing when a susceptible cultivar (Corsoy 79) was planted. However, the positive yield response to planting date and row spacing was observed

if a resistant cultivar was planted (Grau et al., 1994). Furthermore, it has been speculated that occurrence of SCN lessens the advantage of soybean planted in narrow rows (Pedersen and Lauer, 2003). This is an observation of importance to soybean growers as SDS is commonly observed together with SCN in low, moist, and compacted areas of fields (Roy et al., 1997).

Currently, information is lacking on the effect of row spacing, plant population, cultivar susceptibility on SDS and the impact on yield. Sudden death syndrome is considered a disease problematic in high-yielding environments (Rupe et al., 1989) and it is uncertain if the yield benefits observed with narrow row spacing and plant populations will still be achieved in environments with high incidence of SDS. Based on the greater effective leaf area that narrow rows produce, we hypothesize that narrow row spaced soybean will tolerate SDS and maintain greater yield than when planted in wide rows. The objective of this study was to determine differences in SDS foliar symptom severity between soybean planted in narrow and wide row spacing at three seeding rates using a SDS-resistant and SDS-susceptible cultivar.

Materials and Methods

Studies were established in 2008 and 2009 in Iowa near Jefferson and Nevada in fields selected based on previous history of SDS (Table 1). The location at Nevada was abandoned in 2008 due to flooding. The experimental design was a randomized complete block in a split-plot arrangement with four replications. Main plots were row spacings of 38- and 76-cm. Sub plots were a factorial combination of three seeding rates, two cultivars and two inoculation treatments (with and without *F. virguliforme* inoculum). Seeding rates were 185,000; 309,000; and 432,000 seeds ha^{-1}. Cultivars were K-285 and K-283 (Kruger Seed, Dike, IA), classified as resistant and susceptible to SDS, respectively, according to seed

company assessments. Plots were inoculated by planting sorghum (*Sorghum bicolor* (L.) Moench) seed, infested with *F. virguliforme* according to methods as described by Farias Neto et al. (2006), with soybean seed in the furrow at an amount of 125 ml of inoculum per plot (approximately 3.3 g of infested sorghum seed per meter of row) (Farias Neto et al., 2006). Inoculum was produced using three pathogenic *F. virguliforme* isolates (Clinton 1.b, Scott F21 11a, Scott B2), collected and isolated by H. Scherm and X.B. Yang (Sanogo et al., 2000; Scherm et al., 1998), grown on one-third strength Difco Potato Dextrose Agar (PDA). For an individual plate, the isolate-bearing media was cut into three equal-sized pieces and one piece of media from each of the three isolates was placed into a bag containing 2.27 kg of sorghum seed. Bags were incubated at room temperature for 15 days. After incubation, sorghum seed was removed from the bags and allowed to air dry at room temperature.

Fields were chisel-plowed in the fall and cultivated twice in the spring. To manage weeds, pre-emergent herbicide consisting of s-metolachlor [2-chloro-*N*-(2-ethyl-6-methylphenyl)-*N*-(-2-methoxy-1-methylethyl) acetamide]) at a rate of 0.92 kg a.i. ha^{-1} and fomensafen 5-[2-chloro-4-9trifluoromethyl) phenoxy]-N-(methysulfonyl)-2-nitrobenzamide)) at a rate of 0.20 kg a.i. ha^{-1} was applied, followed by two post emergence applications of glyphosate [N-(phosphonomethyl)glycine] at a rate of 1.12 kg a.i. ha^{-1}. An Almaco grain drill (Almaco, Nevada, IA) was used to plant seeds in 38- and 76-cm rows into plots measuring 3 by 7.6 m.

One visual disease assessment for SDS was conducted on each plot when plants were at growth stage R6 (Fehr and Caviness, 1977). Two disease measures were obtained, incidence and severity. Disease incidence was defined as a percentage of plants within a plot exhibiting foliar symptoms of SDS (Njiti et al., 1996; 1998), while disease severity was rated

using a scale of 1 to 9 (mild to severe symptoms) based on percentage of the leaf demonstrating symptoms based on Njiti et al. (1996). Ratings were as follows: 1 = 0 to 10% chlorosis or 1 to 5% necrosis, 2 = 10 to 20% chlorosis or 6 to 10% necrosis, 3 = 20 to 40% chlorosis or 10 to 20% necrosis, 6 = up to 33% premature defoliation, 7 = up to 66% premature defoliation, 8 = > 66% premature defoliation, and 9 = premature death of the plant.

To determine seed yield, the center two and four rows (of 76- and 38-cm row spacing, respectively) were harvested with an Almaco plot combine and harvest weights were adjusted to 130 g kg^{-1} moisture for final yield determination. Additional measures at harvest included plant height, lodging, plant population, and seed mass (based on a sample weight of 300 seeds). Plots were scored for lodging according to a scale of 1 to 5 with 1 corresponding to completely erect plants and 5 corresponding to completely prostrate plants. Statistical analyses were conducted using PROC MIXED in SAS 9.2 (SAS Institute, 2008) for disease and yield data. Both years and all three locations of data were analyzed together with location considered an environment (Miliken and Johnson, 1994). Row spacing, plant population, cultivar and inoculum were set as fixed effects and environment and replication as random effects. Because of variation in disease levels, degrees of freedom were calculated using a Kenward-Rogers approximation.

Results

Air temperature at all environments was below the 20-yr average. Rainfall varied between years (Table 2). In 2008, rainfall totals were well above the 20-yr average in May and June (ranging from 62 to 117 mm above normal) and below the 20-yr average in May and June in 2009 (ranging from -13 to -42 mm below normal) (Table 2).

Yield was reduced by 7% in inoculated plots compared to non-inoculated plots (Table 3). An interaction between row spacing and inoculation for yield showed that narrow rows (38-cm) yielded 7% more than wide rows (76-cm) in non-inoculated plots while no yield difference existed between narrow and wide rows within the inoculated plots (Table 4). Soybean seeded at 185,000 seeds ha^{-1} yielded 9% and 12% less than the higher two seeding rates (Table 3). The SDS-resistant cultivar K-285 yielded 7% greater than the SDS-susceptible cultivar, K-283 (Table 3).

Final plant population was not affected by inoculation (Table 3). No interactions were observed among the main effects for final plant population. Overall, final plant population increased as seeding rate increased (Table 3). The SDS-susceptible cultivar had a higher final plant population than the SDS-resistant cultivar, although the difference was negligible (Table 3).

Overall, there was no difference in plant height between inoculated and non-inoculated plots (Table 3). However, a row spacing by inoculation interaction was observed (Table 5). In non-inoculated plots, soybean planted in 38-cm rows was 4 cm taller than soybean planted in 76-cm rows while no difference in height was found for soybean planted in 38- and 76-cm rows in inoculated plots. Row spacing had no effect on plant height (Table 3). Soybean planted at the lowest seeding rate was 3 and 5 cm shorter than soybean planted at the middle and highest seed rates (Table 3). Seed mass was reduced 6% in inoculated plots compared to non-inoculated plots and oil content of non-inoculated plots was less than inoculated plots (Table 3) suggesting that SDS influenced oil content. An interaction between row spacing and inoculation for seed mass showed that seed mass was reduced in inoculated plots by 6% for 38-cm soybean and 3% for 76-cm soybean compared to non-inoculated plots

(Table 6). For seed composition, the highest seeding rate had the greatest protein content (32.4%), greater than that of the middle seeding rate (32.2%), which in turn was greater than the protein content of the lowest seeding rate (32.0%) (Table 3). Conversely, soybean planted at the lowest seeding rate had the greatest oil content (18.7%), greater than soybean at the middle seeding rate (18.6%), which in turn was greater than the oil content of soybean planted at the highest seeding rate (18.4%) (Table 3).

Inoculated plots had greater levels of disease incidence (7% vs. 2%) and severity (0.90 vs. 0.27) than non-inoculated plots, respectively (Table 7). A cultivar by inoculation interaction indicated that the susceptible cultivar K-283 had greater SDS disease incidence and severity compared to the SDS-resistant cultivar K-285 in both inoculated and non-inoculated plots but this difference was greater in inoculated plots than in the non-inoculated plots (Table 8). Overall, K-285 demonstrated less disease incidence (2% vs. 7%) and severity (0.3 vs. 0.9) than K-283, respectively, translating into a 7% greater yield for K-285 compared to K-283 (Tables 3 and 7). Despite no interaction between row spacing and inoculation for disease incidence, there is evidence ($P = 0.07$) of an interaction between the two treatments for disease severity. This evidence showed no difference in disease severity between 38- and 76-cm row spacing for non-inoculated plots whereas in inoculated plots narrow rows demonstrated greater disease severity than wide rows (data not shown). Overall, there was no difference in disease incidence or severity when comparing narrow and wide rows (Table 7). Seeding rate affected disease incidence but not severity with the two highest seeding rates having increased disease incidence compared to the lowest seeding rate (Table 7).

Discussion

The variation in rainfall between years could have played a role in variation in the disease levels observed (Tables 2 and 7). Excessive rainfall during vegetative growth has been shown to reduce or delay SDS (Rupe and Gbur, 1995), therefore rainfall in May and June 2008 may have contributed to lower disease levels that year. Except for Jefferson in 2009, rainfall totals in August and September were below the 20-yr average. Sudden death syndrome is limited with hot and dry conditions during reproductive growth periods and is increased when moisture is sufficient (Hershman et al., 1990). Therefore, moisture could have been a limiting factor for disease occurrence in 2008, contributing to lower levels of disease expression.

The inoculation treatment was successful in increasing SDS disease pressure as demonstrated by greater levels of disease incidence (7% vs. 2%) and severity (0.9 vs. 0.3) in inoculated compared to non-inoculated plots, respectively (Table 7). The greater disease pressure in the inoculated plots translated into a 7% yield reduction compared to non-inoculated plots (Tables 3 and 7).

The yield response of soybean to narrow rows in non-inoculated plots (Table 3) agrees with results of De Bruin and Pedersen (2008a) who found a yield advantage when planting soybean in 38-cm row spacings compared to 76-cm. When combined with the data that there was increased disease severity in narrow row spacings (Tables 3 and 7), this suggests that *F. virguliforme* acted to negate the yield advantage of narrow rows that was seen in low disease and non-inoculated plots and agrees with work regarding brown stem rot and soybean cyst nematode (Grau et al., 1994; Pedersen and Lauer, 2003). This result is counter to our original hypothesis that increased leaf area produced by narrow row soybean can reduce the effects of SDS (Table 3). There was no overall difference in SDS for narrow

and wide rows whereas inoculated plots had greater SDS than non-inoculated plots (Table 7). Therefore, this study provided evidence that row spacing affected yield in environments where low-levels of *F. virguliforme* existed and not in environments where greater levels of *F. virguliforme* existed.

The lack of significant difference in yield between the highest two seeding rates is not consistent with previous studies that showed that yield increased with increasing seeding rate (Oplinger and Philbrook, 1992). In addition, the difference in yield between the lowest seeding rate and the two highest seeding rates conflicts with previous research from Iowa that demonstrated that yield increases are negligible for seeding rates >185,300 seeds ha^{-1} (De Bruin and Pedersen, 2008b). However, in this study, it was found that the lowest and middle seeding rates had final plant populations below the 200,000 to 230,000 plants ha^{-1} harvest populations needed to achieve maximum yield in Iowa (De Bruin and Pedersen, 2009). Increased levels of SDS for the two highest seeding rates did not appear to reduce the yield advantage of the highest two seeding rates (Tables 3 and 7), most likely due to the greater leaf area found for soybean plants at these plant populations (Shibles and Weber, 1966) which could have allowed for some leaf area loss due to the effects of SDS without impacting yield.

The importance of cultivar selection to reduce the impact of SDS was found based on the 7% yield advantage for K-285 (resistant to SDS) compared to K-283 (susceptible to SDS). This result is consistent with previous research that indicated cultivar selection is critical for managing SDS (Roy et al., 1997; Rupe et al., 1991).

It was found that the final plant population and yield responses were not related to SDS, in part because inoculation had no effect on final plant population (Table 3). The

increase in final plant population as seeding rate increased (Table 3) supports previous results of De Bruin and Pedersen (2008a). The lowest and middle seed rates had final plant populations (Table 3) below the range of 200,000 to 230,000 plants ha^{-1} needed to achieve maximum yield (De Bruin and Pedersen, 2009). We speculate that the low final plant stands of the lowest and middle seeding rates were caused by unseen interactions involving *F. virguliforme* with other soilborne pathogens.

No difference was found for plant height in inoculated and non-inoculated plots (Table 3), a result that is different to previous results that have shown that infection by *F. solani* led to reduced plant height (Rupe, 1989). However, the row spacing by inoculation interaction that demonstrated a height reduction of narrow row soybean in inoculated plots compared to non-inoculated plots (Table 5) contradicts research showing plant height to be unaffected by row spacing (De Bruin and Pedersen, 2008a). This response of plant height for narrow rows in inoculated plots (Table 5) and evidence ($P = 0.07$) for narrow rows to have greater disease severity than wide rows in inoculated plots suggest that *F. virguliforme* stunted soybean height in narrow rows. Reduced plant height in response to *F. solani* isolates has also been reported by Rupe (1989). Our results showing the overall effect of row spacing to have no influence on plant height agree with the findings of Oplinger and Philbrook (1992). Taller plant height demonstrated in the soybean at the two highest seeding rates compared to soybean at the lowest seeding rate (Table 3) agrees with results by De Bruin and Pedersen (2008a) and Elmore (1998).

Seed mass was reduced by 6% in inoculated plots compared to non-inoculated plots and this suggests that *F. virguliforme* had a key role in reducing seed mass (Roy et al., 1997). Furthermore, there was a 7% reduction in seed mass for K-283 compared to K-285 indicating

that cultivar selection is critical for managing SDS when conditions are favorable for the pathogen (Roy et al., 1997; Rupe et al., 1991). The increased reduction in seed mass for soybean planted in narrow rows was a function of inoculation because there was no difference between seed mass of narrow and wide rows in non-inoculated plots (Table 6). This indicated that there is an increased effect of SDS in narrow row spacings when conditions for the disease are favorable as previous studies have shown that seed mass is reduced in narrow rows (De Bruin and Pedersen, 2008a; Egli, 1994; Elmore 1998; Ethredge et al., 1989).

Results from this study that showed an increased protein content and decreased oil content as seeding rates increased agree with previous research (Butler et al., 2010; Weber et al., 1966). Based on these results, there was no impact of SDS on protein or oil content at the different soybean seeding rates.

Conclusion

This paper is the first to address the effect of row spacing, seeding rate and cultivar susceptibility on SDS and soybean yield. Although overall SDS disease expression was low across the environments, there were several important discoveries. In particular, the yield advantage of narrow row spacing could be negated when the risk of SDS is high as shown under inoculated conditions. Also, while there was a higher disease incidence in the highest seeding rates, yield was still found to be greater than at the lowest seed rating indicating that the additional leaf area and light interception for the highest seeding rate may be able to tolerate a low level of disease without impacting yield. Furthermore, cultivar selection is important for reducing the risk of yield loss due to SDS. Overall, the supporting evidence is not strong enough to suggest that growers in Iowa should sacrifice yield-maximizing

agronomic practices such as narrow row spacing and reduced seeding rate to escape yield-loss to SDS. It was concluded that in environments favorable for increased SDS disease incidence and severity, growers may not observe the yield advantage of narrow rows but should still use SDS-resistant cultivars regardless of row spacing.

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References

Aoki, T., K. O'Donnell, H. Yoshihisa, and A.R. Lattanzi. 2003. Sudden-death syndrome of soybean is caused by two morphologically and phylogenetically distinct species within the *Fusarium solani* species complex- *F. virguliforme* in North America and *F. tucumaniae* in South America. Mycologia 95:660-684.

Andrade, F.H., P. Calvino, A. Cirilo, and P. Barbieri. 2002. Yield responses to narrow rows depend on increased radiation interception. Agron. J. 94:975-980.

Bullock, D., S. Khan, and A. Rayburn. 1998. Soybean yield response to narrow rows is largely due to enhanced early growth. Crop Sci. 38:1011-1016.

Butler, J.A., J.L. De Bruin, and P. Pedersen. 2010. Plant density effect on reduced linolenic acid soybean cultivars. Agron. J. 102:348-354.

De Bruin, J.L., and P. Pedersen. 2008a. Effect of row spacing and seeding rate on soybean yield. Agron. J. 100:704-710.

De Bruin, J.L., and P. Pedersen. 2008b. Soybean seed yield response to planting date and seeding rate in the Upper Midwest. Agron. J. 100:696-703.

De Bruin, J.L., and P. Pedersen. 2009. New and old soybean cultivar responses to plant density and intercepted light. Crop Sci. 49:2225-2232.

Devlin, D.L., D.L. Fjell, J.P. Shroyer, W.B. Gordon, B.H. Marsh, L.D. Maddux, V.L. Martin, and S.R. Duncan. 1995. Row spacing and seeding rates for soybean in low and high yielding environments. J. Prod. Agric. 8:215-222.

Egli, D.B. 1994. Mechanisms responsible for soybean yield response to equidistant planting patterns. Agron. J. 86:1046-1049.

Elmore, R.W. 1998. Soybean cultivar responses to row spacing and seeding rates in rainfed and irrigated environments. J. Prod. Agric. 11:326-331.

Ethredge, W.J., Jr., D.A. Ashley, and J.M. Woodruff. 1989. Row spacing and plant population effects on yield components of soybean. Agron. J. 81:947-951.

Farias Neto, A.F., G.L. Hartman, W.L. Pedersen, S. Li, and B.W. Diers. 2006. Irrigation and inoculation methods that increase the severity of soybean sudden death syndrome in the field. Crop Sci. 46:2547-2554.

Fehr, W.R., and C.E. Caviness. 1977. Stages of soybean development. Spec. Rep. 80. Iowa Agric. Home Econ. Exp. Stn., Iowa State Univ., Ames.

Grau, C.R., and V.L. Radke. 1984. Effects of cultivars and cultural practices on Sclerotinia stem rot of soybean. Plant Dis. 68:56-58.

Grau, C.R., E.S. Oplinger, E.A. Adee, E.A. Hinkens, and M.J. Martinka. 1994. Planting date and row width effect on severity of brown stem rot and soybean productivity. J. Prod. Agric. 7:347-351.

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

Miliken, G.A., and D.E. Johnson.1994. Analysis of messy data, Vol. 1: Designed experiments. Chapman & Hall, London.

Navi, S.S., and X.B. Yang. 2008. Foliar symptom expression in association with early infection and xylem colonization by *Fusarium virguliforme* (formerly *F. solani* f. sp. *glycines*), the causal agent of soybean sudden death syndrome. Online. Plant Health Progress doi:10.1094/PHP-2008-0222-01-RS.

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

Njiti, V.N., R.J. Suttner, L.E. Gray, P.T. Gibson, and D.A. Lightfoot. 1997. Rate-reducing resistance to *Fusarium solani* f. sp. *phaseoli* underlies field resistance to soybean sudden death syndrome. Crop Sci. 37:132-138.

Njiti, V.N., M.A. Shenaut, R.J. Suttner, M.E. Schmidt, and P.T. Gibson. 1998. Relationship between soybean sudden death syndrome disease measures and yield components in F_6-derived lines. Crop Sci. 38:673-678.

Oplinger, E.S., and B.D. Philbrook. 1992. Soybean planting date, row width, and seeding rate response in three tillage systems. J. Prod. Agric. 5:94-99.

Pedersen, P., and J.G. Lauer. 2003. Corn and soybean response to rotation sequence, row spacing, and tillage systems. Agron. J. 95:965-971.

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

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

Rupe, J.C., M.C. Hirrel, and D.E. Hershman. 1989. Sudden death syndrome. p. 84-85. *In* J.B. Sinclair and P.A. Backman (ed.) Compendium of soybean diseases. 3rd ed. APS Press, St. Paul, Minnesota.

Rupe, J.C., E.E. Gbur, and D.M. Marx. 1991. Cultivar responses to sudden death syndrome of soybean. Plant Dis. 75:47-50.

Rupe, J.C. and E.E. Gbur. 1995. Effect of plant age, maturity group, and the environment on disease progress of sudden death syndrome of soybean. Plant Dis. 79:139-143.

Rupe, J.C., and G.L. Hartman. 1999. Sudden death syndrome. p. 37-39. *In* G.L. Hartman et al. (ed.) Compendium of soybean diseases. 4th ed. APS Press, St. Paul, Minnesota.

Sanogo, S., X.B. Yang, and H. Scherm. 2000. Effects of herbicides on *Fusarium solani* f. sp. *glycines* and development of sudden death syndrome in glyphosate-tolerant soybean. Phytopathology 90:57-66.

SAS Institute. 2008. The SAS system for Windows. v. 9.2. SAS Inst. Cary, NC.

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

Shibles, R.M., and C.R. Weber. 1966. Interception of solar radiation and dry matter production by various soybean planting patterns. Crop Sci. 6:55-59.

Weber, C.R., R.M. Shibles, and D.E. Byth. 1966. Effect of plant population and row spacing on soybean development and production. Agron. J. 58:99-102.

Wrather, J.A., S.R. Koenning, and T.R. Anderson. 2003. Effect of diseases on soybean yields in the United States and Ontario (1999-2002). Online. Plant Health Progress doi:10.1094/PHP-2003-0325-01-RV.

Table 1. Field characteristics at the Jefferson and Nevada field sites in 2008 and 2009.

Location	Jefferson		Nevada
Year	2008	2009	2009
Soil Series	Canisteo clay loam	Canisteo clay loam	Webster clay loam
Soil Family	Fine-loamy, Mixed, Superactive, Mesic Typic Hapludolls	Fine-loamy, Mixed, Superactive, Mesic Typic Hapludolls	Fine-loamy, Mixed, Superactive, Mesic Typic Hapludolls
pH	6.7	6.8	6.8
P (mg kg^{-1})	23	8	68
K (mg kg^{-1})	176	188	280
OM (g kg^{-1})†	47	42	46
Planting date	1 May	5 May	5 May
Harvest date	1 Oct	28 Sept	29 Sept

† OM, organic matter

Table 2. Monthly mean air temperature and precipitation totals recorded at two experimental locations in 2008 and 2009. Deviations from the 20-yr average are reported in parentheses.

		May		June		July		August		September	
Year	Location	Air temp.	Rainfall	Air temp.	Rainfall	Air temp.	Rainfall	Air temp.	Rainfall	Air temp.	Rainfall
		°C	mm	°C	mm	°C	mm	°C	mm	°C	mm
2008	Jefferson	14 (-2.1)†	232 (117)	21 (-0.6)	184 (62)	23 (-1.1)	115 (1)	21 (-2.1)	46 (-62)	17 (-1.0)	47 (-29)
	Nevada	16 (-1.0)	216 (94)	21 (-0.4)	271 (160)	23 (0.05)	234 (123)	21 (-1.0)	53 (-73)	18 (-0.3)	78 (0)
2009	Jefferson	16 (-0.7)	77 (-42)	20 (-1.5)	115 (-13)	20 (-3.7)	135 (24)	21 (-1.5)	110 (12)	18 (0.3)	28 (-48)
	Nevada	16 (-0.8)	102 (-26)	71 (-0.3)	104 (-16)	21 (-2.7)	70 (-59)	21 (-1.6)	89 (-25)	18 (-0.3)	31 (-48)

† Twenty-year averages based on Iowa Environmental Mesonet locations near Jefferson and Nevada, IA.
Available at: http://mesonet.agron.iastate.edu/climodat/index.phtml

Table 3. Main effect means of row spacing, seeding rate, cultivar and inoculation with *Fusarium virguliforme* for soybean seed yield, final plant population, plant height, seed mass, protein and oil at Jefferson and Nevada in 2008 and 2009.

Treatment	Yield	Final plant population	Plant height	Seed mass	Protein	Oil
	kg ha^{-1}	Plants ha^{-1}	cm	g 100 seed^{-1}	%	
Row Spacing (R)						
38 cm	4255	199 600	82	14.9	32.2	18.5
76 cm	4142	184 900	81	15.2	32.2	18.6
LSD (0.05)	NS†	10 300	NS	0.3	NS	NS
Seeding Rate (S)						
185 000	3889	134 300	79	15.0	32.0	18.7
309 000	4269	195 400	82	15.0	32.2	18.6
432 000	4436	247 000	84	15.2	32.4	18.4
LSD (0.05)	208	12 600	3	NS	0.2	0.1
Cultivar (C)‡						
K-283	4052	197 500	81	14.5	32.4	18.5
K-285	4344	187 000	82	15.5	32.0	18.6
LSD (0.05)	169	10 300	NS	0.2	0.2	NS
Inoculation (I)						
Control	4363	194 100	82	15.8	32.3	18.5
Inoculated	4033	190 400	81	14.8	32.1	18.6
LSD (0.05)	169	NS	NS	0.2	NS	0.1
ANOVA						
R X S	NS	NS	NS	NS	NS	NS
R X C	NS	NS	NS	NS	NS	NS
S X C	NS	NS	NS	NS	*	NS
R X I	*	NS	**	**	NS	NS
S X I	NS	NS	NS	NS	NS	NS
C X I	NS	NS	NS	NS	NS	NS
R X S X C	NS	NS	NS	NS	NS	NS
R X C X I	NS	NS	NS	NS	NS	NS
R X S X I	NS	NS	NS	NS	NS	NS
S X C X I	NS	NS	NS	NS	NS	NS
R X S X C X I	NS	NS	NS	NS	NS	NS

† NS, not significantly different at $P \leq 0.05$.

‡ K-283 is rated as susceptible to SDS and K-285 is rated as resistant to SDS (Kruger Seed, Dike, IA).

*,**,*** significantly different at $P \leq 0.05$, 0.01, and 0.001 levels, respectively.

Table 4. Row spacing by inoculation interaction for soybean yield across three environments in Iowa during 2008 and 2009.

	Yield	
Treatment	Control	Inoculated
Row Spacing	kg ha^{-1}	
38 cm	4530	3979
76 cm	4196	4088
LSD (0.05)	310	

Table 5. Soybean plant height by inoculation interaction across three environments in Iowa during 2008 and 2009.

	Plant Height	
Treatment	Control	Inoculated
Row Spacing	cm	
38 cm	84.5	80.0
76 cm	80.3	82.1
LSD (0.05)	3.7	

Table 6. Row spacing by inoculation interaction for soybean seed mass across three environments in Iowa during 2008 and 2009.

	Seed Mass	
Treatment	Control	Inoculated
Row Spacing	g 100 seed^{-1}	
38 cm	15.3	14.5
76 cm	15.4	15.0
LSD (0.05)	0.3	

Table 7. Response of sudden death syndrome (SDS) incidence and severity in soybean at growth stage R6 to row spacing, seeding rate, cultivar and inoculation across three environments in Iowa during 2008 and 2009.

Treatment	Disease Incidence	Disease Severity
	%	1 – 9†
Row Spacing (R)		
38 cm	4.8a‡	0.6a
76 cm	4.1a	0.6a
Seeding Rate (S)		
185 000	3.1b	0.5a
309 000	6.0a	0.7a
432 000	4.3ab	0.6a
Cultivar (C)§		
K-283	7.3a	0.90a
K-285	1.6b	0.28b
Inoculation (I)		
Control	2.1b	0.3b
Inoculated	6.9a	0.9a
ANOVA		
R X S	NS¶	NS
R X C	NS	NS
S X C	NS	NS
R X I	NS	NS
S X I	NS	NS
C X I	**	**
R X S X C	NS	NS
R X C X I	NS	NS
R X S X I	NS	NS
R X C X I	NS	NS
S X C X I	NS	NS
R X S X C X I	NS	NS

† If disease was present, foliar symptom severity assessed according to increasing severity on a scale of 1 – 9 as outlined by Njiti et al. (1996).
‡ Values followed by the same letter not significantly different at $P \leq 0.05$.
§ K-283 is rated as susceptible to SDS and K-285 is rated resistant to SDS (Kruger Seed, Dike, IA).
¶ NS, not significantly different at $P \leq 0.05$.
*,**,*** significantly different at $P \leq 0.05$, 0.01, and 0.001 levels, respectively.

Table 8. Cultivar by inoculation interaction across three environments in Iowa during 2008 and 2009.

	Disease Incidence		Disease Severity	
Treatment	Control	Inoculated	Control	Inoculated
Cultivar†	%		1 - 9‡	
K-283	3.66	10.96	0.44	1.36
K-285	0.44	2.82	0.10	0.45
LSD (0.05)	2.7		0.3	

† K-283 is rated as susceptible to SDS and K-285 is rated as resistant to SDS (Kruger Seed, Dike, IA).

‡ If disease was present, foliar symptom severity assessed according to increasing severity on a scale of 1 – 9 as outlined by Njiti et al. (1996).

CHAPTER 5. EFFECT OF MATURITY GROUP AND CULTIVAR ON SUDDEN DEATH SYNDROME OF SOYBEAN

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Abstract

Soybean [*Glycine max* (L.) Merr.] yield is reduced every year by the disease sudden death syndrome (SDS), caused by the pathogen *Fusarium virguliforme* sp. nov. Agronomic recommendations for SDS management in the upper Midwest are lacking. Our objective was to evaluate differences in SDS severity, incidence and soybean yield among cultivars belonging to three classes of maturity (MG). A SDS-susceptible and SDS-resistant cultivar within three classes of maturity (early MG II, late MG II, and MG III) for central Iowa were planted in plots inoculated with and without *F. virguliforme* at two locations in SDS-prone Iowa fields in 2008 and 2009. Low disease levels were observed during both years of this study. Early and late MG II cultivars had greater SDS than MG III cultivars both years. Maturity group III cultivars yielded 388 kg ha^{-1} more than early MG II cultivars in 2008 but no differences were observed among any of the MG in 2009. Sudden death-resistant cultivars had less disease than SDS-susceptible cultivars both years. The early MG II SDS-resistant cultivar had less SDS and greater yield than the early MG II SDS-susceptible cultivar by 387 and 297 kg ha^{-1} in 2008 and 2009 ($P = 0.09$), respectively. The SDS-resistant MG III cultivar had less SDS both years and yielded 470 kg ha^{-1} more in 2009 than the SDS-susceptible MG III cultivar. Despite low and inconsisten levels of SDS, it is still recommended that to

maximize yield potential in Iowa, growers should plant full season adapted cultivars with SDS-resistance when soybean is planted in high-risk SDS environments.

Introduction

Cultivar selection is the most important decision a producer makes to maximize soybean (*Glycine max* (L.) Merrill) yield (De Bruin and Pedersen, 2008a). An ideal soybean cultivar has yield stability and can achieve maximum yield in many environments regardless of environmental conditions (De Bruin and Pedersen, 2008a). Both abiotic and biotic stresses in an environment can influence plant growth and reduce yield (Cook, 2000). In Iowa and the upper Midwest, management decisions regarding cultivar selection and early planting (De Bruin and Pedersen, 2008b; Robinson et al., 2009) are implemented to increase yield. In Iowa, De Bruin and Pedersen (2008a) found that cultivar selection should be based on yield, yield stability, and disease resistance to soybean cyst nematode (*Heterodera glycines* Ichinohe; SCN). Sudden death syndrome (*Fusarium virguliforme* sp. nov. formerly called, *F. solani* f. sp. *glycines*; SDS; Aoki et al., 2003) is often associated with SCN (Roy et al., 1989), and together, these two diseases account for more than 50% of the yield suppression caused by soybean pathogens in Iowa (Wrather et al., 2003).

It is important that growers in Iowa take maturity group (MG) into consideration when selecting a cultivar since the growing season in Iowa is short and yield is made much faster than further south in the Corn Belt. A full-season MG increases light interception and maximize daily photosynthesis (Shibles and Weber, 1966) and short-season cultivars may not produce sufficient leaf area to take complete advantage of available light during flowering and seed fill (Board et al., 1992).

Sudden death syndrome is a significant constraint to US soybean production (Aoki et al., 2003) with foliar symptoms developing during reproductive stages, following pathogen infection of the roots (Navi and Yang, 2008). Cultivar selection is a vital component of an effective management program to control SDS (Roy et al., 1997). Cultivar susceptibility has a strong effect on SDS disease development and currently the only reliable method of control available to producers is to use cultivars with tolerance or low susceptibility to the pathogen (Rupe et al., 1991). Few commercial cultivars are marketed as having high levels of resistance to SDS (Mueller et al., 2003), and most modern cultivars are considered susceptible to SDS (University of Illinois, 2008) which requires growers to turn to other decisions such tillage, planting date and maturity group (MG) to potentially control SDS.

Sudden death syndrome is influenced by environmental conditions and cultivar susceptibility (Rupe and Gbur, 1995). Resistance to SDS is expressed in most cultivars as a delay in disease onset as well as a reduction in rate of disease progression (Rupe and Gbur, 1995). Onset of foliar symptoms appears dependent on the chronological age of the plant and independent of the reproductive age of the plant (Rupe et al., 1991). Rupe and Gbur (1995) found the earliest increases in SDS around the same number of days after planting for all maturity groups (MG V – VIII). Their study showed rapid disease progression takes place during reproductive development (R2 to R5) at the same time across maturity groups but at no specific growth stage.

Yield differences among maturity groups may exist if more or less than favorable environmental conditions coincide with critical growth stages (Bunting, 1971). Soybean yield is highly correlated to seed m^{-2} (Shibles et al., 1975) and is also a function of length of seed fill (Gay et al., 1980; Smith and Nelson, 1986a). Seed m^{-2} is determined during the early

stages of reproductive growth and closely associated with dry matter accumulation (Egli, 1993; Egli and Zhen-wen, 1991). Sudden death syndrome foliar symptoms consist of chlorosis and necrosis, which develop at flowering, while defoliation, flower and pod abortion (Roy et al., 1997) can occur later during reproductive growth if symptoms are severe enough (Navi and Yang, 2008). Research examining the effect of defoliation on soybean yield shows that greatest yield losses exist when total defoliation occurs in early seed fill (R5 to R5.6) (Board et al., 2010). The magnitude of soybean yield loss caused by defoliation lessens the later during seed fill that defoliation stress occurs (Board et al., 2010). Therefore, plant growth stage at symptom development onset and whether or not symptoms progress rapidly and become severe contribute to the effect of SDS on yield components (Roy et al., 1997).

Studies from Arkansas and Kentucky using cultivars belonging to MG III-VIII show that using early-maturing cultivars within a region has often resulted in SDS development at a later plant reproductive stage (R5 or later), in turn, reducing the risk of serious yield losses (Roy et al., 1997; Rupe and Gbur, 1995; Hershman et al., 1990). This agrees with reports from Egli (1993) that an advantage of a short season cultivar is a possible reduction in disease problems and implies that for growers in central Iowa, a region which qualifies as an optimum zone of adaptation for cultivars belonging to late MG II, yield losses caused by SDS could be reduced by planting cultivars that mature earlier than MG II. Research on the relation of MG and SDS does not exist for Iowa.

As farms are getting larger, producers want to start harvesting earlier, in turn planting cultivars belonging to shorter MG than the full season adapted cultivars for their area. Earlier maturing cultivars have shorter reproductive stages compared to later maturing cultivars,

therefore, early maturing cultivars have less opportunity for disease onset to coincide with critical yield formation growth stages of the plant. We hypothesize that class of maturity, or the time from flowering to harvest maturity, will affect the relationship between SDS over time and yield. Our objective was to evaluate differences in SDS severity and incidence and soybean yield among three classes of maturity (early, mid, and late maturity) in central Iowa.

Materials and Methods

The experiment was conducted in 2008 and 2009 near Jefferson and Nevada, Iowa, in fields with a history of SDS (Table 1). In 2008, the experimental design was a randomized complete block design in a split-plot arrangement with four replications. Main plot consisted of three classes of maturity and the sub-plots were a factorial combination of six cultivars and two inoculation treatments (with and without SDS inoculum). Three cultivars were classified as resistant to SDS (AG2002, K-285, K-348) and three were classified as susceptible (K-204, K-283, K-321) according to seed company assessments (Table 2) (Kruger Seed, Dike, IA; Monsanto, St. Louis, MO). Cultivars had similar genetic backgrounds and all contained PI88788 resistance to SCN. In 2009, the experimental design was a randomized complete block design with no split and the same six cultivars as in 2008 in factorial combination with two inoculation treatments (with and without SDS inoculum).

Plots were inoculated with infested sorghum (*Sorghum bicolor* (L.) Moench) seed prepared according to methods described by Farias Neto et al. (2006). Sorghum seed was planted in the furrow with the soybean seed, each plot receiving 125 ml of inoculum (approximately 3.3 g of infested sorghum seed per meter of row) (Farias Neto et al., 2006). Inoculum was produced from three pathogenic *Fusarium virguliforme* isolates (Clinton 1.b, Scott F21 11a, Scott B2) collected and isolated by H. Scherm and X. B. Yang (Sanogo et al.,

2000; Scherm et al., 1998). Isolates were grown on one-third strength PDA (Difco Potato Dextrose Agar). To inoculate, the media on which the isolate was growing was cut into three equal-sized pieces in the plate. Each bag, containing 2.27 kg of sorghum seed, received one piece of the isolate bearing media from a plate of each of the three isolates. Bags were incubated at room temperature for 15 days, after which sorghum seed was removed from the bags and allowed to dry until planting.

Prior to planting, fields were chisel-plowed in the fall and field cultivated twice in the spring. Weeds were managed with a pre-emergent herbicide consisting of s-metolachlor [2-chloro-*N*-(2-ethyl-6-methylphenyl)-*N*-(-2-methoxy-1-methylethyl) acetamide]) at a rate of 0.92 kg a.i. ha^{-1} and fomensafen [5-[2-chloro-4-(trifluoromethyl) phenoxy]-N-(methysulfonyl)-2-nitrobenzamide] at a rate of 0.20 kg a.i. ha^{-1}. This was followed by two post emergence applications of glyphosate [N-(phosphonomethyl)glycine] each at a rate of 0.865 kg a.e. ha^{-1}.

Studies were planted on 1 and 5 May in 2008 and 2009 at Jefferson and on 5 May in both years at Nevada (Table 1). Plots were planted in 76-cm row spacing at a seeding rate of 371,000 seeds ha^{-1} in plots sized at 3 by 7.6 m using an Almaco grain drill (Almaco, Nevada, IA). Field plots were given visual disease assessments every 10 days starting at the onset of foliar disease symptoms until plants reached growth stage R7 (Fehr and Caviness, 1977) (Table 2). In 2008, SDS could not be differentiated from early plant senescence and late season diseases after 3 September (Table 2). Therefore, 2008 had two fewer dates of disease assessment than 2009. At each date of disease assessment a general growth stage was determined for each plot. At plot level, foliar disease rating scored disease incidence as a percentage of plants within a plot displaying visible leaf symptoms (Njiti et al., 1996; 1998).

Disease severity was rated on a scale of 1 to 9 (mild to severe symptoms). This scale was based on percentage of the leaf area demonstrating symptoms with 1 = 0 to 10% chlorosis or 1 to 5% necrosis, 2 = 10 to 20% chlorosis or 6 to 10% necrosis, 3 = 20 to 40% chlorosis or 10 to 20% necrosis, 4 = 40 to 60% chlorosis or 20 to 40% necrosis, 5 = > 60% chlorosis or > 40% necrosis, 6 = up to 33% premature defoliation, 7 = up to 66% premature defoliation, 8 = > 66% premature defoliation, and 9 = premature death of the plant (Njiti et al., 1996).

At maturity the center two rows of each plot were harvested with an Almaco plot combine to determine yield. Harvest weights were adjusted to 130 g kg^{-1} moisture for final yield determination. Other measurements at harvest were plant height, lodging, plant density, and seed mass (based on a sample weight of 300 seeds). Lodging scores were based on a scale of 1 to 5 with 1 representing completely erect plants and 5 representing completely prostrate plants. Yield data were analyzed using PROC MIXED SAS 9.2 (SAS Institute, 2008) treating cultivar and inoculum as fixed effects and location and replication as random effects. Disease data were analyzed using a repeated measures mixed model with a compound symmetry covariance structure based on Akaike's Information Criteria (AIC). All data were analyzed by year, using a Kenward-Rogers approximation to calculate degrees of freedom because of the variation in disease levels and number of disease assessments across years. For both yield and disease data, cultivars were grouped according to class of maturity and SDS-resistance or susceptibility, and compared using estimates. In 2008, the maturity group split was not considered in the final analysis since the error associated with that split did not contribute more than the overall residual error term in the model.

Results and Discussion

Variation in rainfall totals between 2008 and 2009 could have contributed to differences in disease levels between years as SDS disease progress is influenced by soil moisture (Rupe et al., 1989; Scherm and Yang, 1996). In May and June monthly rainfall totals were above the 20-year average in 2008 (ranging from 62 to 160 mm above average) and slightly below average in 2009 (ranging from 13 to 42 mm below average) (Table 3). At both locations in both years air temperature was below average (Table 3).

Yield

In 2008, there was no yield difference between inoculated and non-inoculated plots, an indication of overall low disease pressure that year (data not shown). In 2009, inoculated plots yielded 9% less than non-inoculated plots (data not shown).

Yield differences existed among cultivars ranging from 3216 to 3874 kg ha^{-1} in 2008 and from 3799 to 4338 kg ha^{-1} in 2009 (data not shown). In 2008, MG III cultivars yielded 388 kg ha^{-1} greater than early MG II cultivars with no differences found among classes of maturity in 2009 (Table 4). There was evidence ($P = 0.15$) in 2008 and ($P = 0.07$) in 2009 that SDS-resistant cultivars yielded 186 and 183 kg ha^{-1} greater than SDS-susceptible cultivars, respectively (Table 5). There were indications in both 2008 ($P = 0.09$) and 2009 ($P = 0.10$) that the early MG II SDS-resistant cultivar (AG2002) yielded 387 and 297 kg ha^{-1} more than the early MG II SDS-susceptible cultivar (K-204), respectively (Table 6). In 2009, the MG III SDS-resistant cultivar K-348 yielded 470 kg ha^{-1} greater than the MG III SDS-susceptible cultivar K-321 (Table 6).

The yield advantage of the MG III cultivars over the early MG II cultivars agrees with previous work and observation showing the highest soybean yields to be achieved when cultivars have a total growth cycle that uses the majority of the available growing season

(Edwards and Purcell, 2005a; b). The difference in yield between early MG II SDS-resistant and SDS-susceptible cultivars in both years contradicts the hypothesis (Table 6) and illustrates the importance of planting a SDS-resistant cultivar as early-maturing cultivars with short reproductive growth periods may experience increased susceptibility to stress and have less time to potentially recover (Egli, 1993).

Final Plant Population

Inoculation did not have an effect on final plant population in either year (data not shown). In 2008, AG2002, K-283 and K-321 had the highest final plant population with 242,100 plants ha^{-1} and K-348 had the lowest with 197,000 plants ha^{-1} (data not shown). In 2009, AG2002 had the highest final plant population with 270,100 plants ha^{-1} and K-321 had the lowest at 159,200 plants ha^{-1} (data not shown). All cultivars, except for K-348 in 2008 and K-285 and K-321 in 2009, had final plant populations close to or greater than 230,000 plants ha^{-1} , populations at which maximum yield potential is reached and with further increases in population no yield increase results (De Bruin and Pedersen, 2009). Therefore in this study potential yield differences cannot fully be explained by different plant population.

Plant Height

Plant height was not affected by inoculation in either growing season (data not shown) which is in disagreement with work showing *F. solani* isolates to reduce plant height (Rupe, 1989). Plant height differed among the cultivars (data not shown) with the general trend during both years showing plant height to increase with increasing MG (Table 4). This observation agrees with previous observations (Zhang et al., 2001) and research documenting that when reproductive growth periods increase larger plants result (greater node number and increased vegetative mass per plant; Egli, 1993). No differences in plant height were found

between the SDS-susceptible and SDS-resistant cultivars (Table 5). Within each class of maturity, the early MG II SDS-resistant cultivar AG2002 was 4.7 cm taller than the SDS-susceptible cultivar K-204 in 2009 with no other differences observed in either year (Table 6).

Seed Mass

Seed mass was not influenced by inoculation in either year (data not shown) contradicting previous research showing SDS to influence seed fill (Roy et al., 1997). Differences existed among cultivars (data not shown) with late MG II cultivars having greater seed mass than early MG II cultivars by 1.2 g 100 seeds^{-1} in both years (Table 4). In 2008, late MG II cultivars had 0.5 g 100 seeds^{-1} greater seed mass than MG III cultivars (Table 4). Maturity group III cultivars had a greater seed mass than early MG II cultivars by 0.7 g 100 seeds^{-1} in 2008 and by 1.5 g 100 seeds^{-1} in 2009 (Table 4). Overall, SDS-susceptible cultivars had a greater seed mass than SDS-resistant cultivars in 2008 but no differences were observed in 2009 (Table 5). Within the early MG II cultivars, the SDS-susceptible cultivar had greater seed mass than the SDS-resistant cultivar in both years (Table 6). Within the late MG II cultivars, the SDS-resistant cultivar had a greater seed mass than the SDS-susceptible cultivar which is contrary to the overall trend of SDS-susceptible cultivars having greater seed mass than SDS-resistant cultivars in 2008, (Table 6).The increased seed mass of late MG II and MG III cultivars compared to early MG II cultivars (Table 4) along with results showing MG III cultivars to have a yield advantage over early MG II cultivars, supports work demonstrating that the duration of seed fill is a critical yield-determining factor (Gay et al., 1980; Smith and Nelson, 1986a).

Protein and Oil

Protein and oil were not affected by inoculation either year (data not shown). Maturity class did not influence protein content in 2008 or oil content in 2009 (Table 4). In 2009, early MG II cultivars had higher protein content than cultivars in the later maturity groups, and late MG II cultivars had higher protein content than MG III cultivars (Table 4). In 2008 early MG II cultivars had greater oil content than late MG II and MG III cultivars (Table 4). This conflicts with findings by Naeve and Huerd (2008) observing an increase in oil concentration with late season increases in temperature and that later maturing cultivars have greater oil concentration than earlier maturing cultivars. Sudden death syndrome-susceptible cultivars tended to have higher protein content than the SDS-resistant cultivars in both years ($P = 0.07$) (Table 5). Sudden death syndrome-susceptible cultivars had greater oil content than SDS-resistant cultivars in 2008 whereas SDS-resistant cultivars had higher oil content than SDS-susceptible cultivars in 2009 ($P = 0.06$) (Table 5). In both years, AG2002 had higher protein content than K-204 (Table 6). In both years K-321 had higher protein content than K-348 and lower oil content than K-348 in 2009 (Table 6). In 2009 K-283 had higher protein content than K-285 (Table 6). In 2008, SDS-susceptible cultivars had higher oil contents than SDS-resistant cultivars within each maturity group ($P = 0.08$ for cultivars in MG III) (Table 6). Overall, response of protein and oil were variable between years and no conclusions can be made as to direct relationships between SDS, susceptibility to SDS, and protein and oil contents.

Disease Incidence and Severity

Heavy rainfall in May and June of 2008 (Table 3) could have influenced the low levels of disease that year because excessive rainfall during vegetative growth might lessen or slow SDS (Rupe and Gbur, 1995). During August 2008, while plants were in growth

stages R3 – R6 (Table 2), rainfall totals were below normal (Table 3), which could have lessened SDS disease development because SDS is restricted in hot and dry environments and increased when moisture is adequate (Hershman et al., 1990).

Interactions between inoculation and date of disease assessment were observed in both years for disease incidence and severity (Table 7). In 2008, there were no differences in disease incidence or severity between inoculated and non-inoculated plots at the first four assessment dates on 30 June, 10 July, 21 July, and 30 July (data not shown) during which growth stages of maturity classes ranged from R1 to R4 (Table 2). At 11 August, 21 August, and 3 September inoculated plots demonstrated greater SDS than non-inoculated plots. Based on growth stages of plants at these dates seed m^{-2} had been primarily determined in early and late MG II (R4 – R7) cultivars while seed m^{-2} was still being determined in MG III cultivars (R3 – R5) (Table 2) (Egli, 1993; Fehr and Caviness, 1977). In 2009, SDS was greater in inoculated plots than non-inoculated plots at every disease assessment date with the exception of disease incidence at 30 July and 10 August (data not shown).

A cultivar by inoculation interaction was found in 2008 for disease incidence and in 2009 for disease incidence and severity (Table 7). In 2008, this interaction showed no difference in disease incidence for K-204 (highest disease incidence among cultivars) and K-348 (lowest disease incidence among cultivars) in inoculated and non-inoculated plots where as every other cultivar had greater levels of SDS incidence in inoculated plots compared to non-inoculated plots (data not shown). In 2009, each cultivar had greater disease incidence and severity in inoculated plots compared to non-inoculated plots, and the interaction is attributed to K-204 and K-283 having the same amount of SDS in inoculated plots but K-283 having less SDS in non-inoculated plots compared to inoculated plots. An explanation for the

variable responses of K-283 and K-348 to inoculation between years is unclear. However, such variable responses are not surprising as past research has shown differences in disease development across years to be consistent with some cultivars and not with others (Rupe et al., 1991). The response of the SDS-resistant cultivar K-348 in 2008 is similar to findings of Farias Neto et al. (2006) showing no difference in the amount of disease expressed by the SDS-resistant cultivar AG3302 between inoculation and control treatments. The other cultivars' demonstration of greater disease in inoculated versus non-inoculated plots agrees with the response of the SDS-susceptible cultivar AG3003 that was found in the aforementioned study (Farias Neto et al., 2006).

Interactions for cultivar with date of disease assessment were seen in both years for disease incidence and severity (Table 7). In both years disease onset occurred at the same time across cultivars (data not shown). This agrees with previous work showing SDS onset to be independent of the reproductive age of the plant (Rupe et al., 1991; Rupe and Gbur, 1995). In 2008 there was no difference in disease expression among the cultivars until 11 August for disease incidence and 30 July for disease severity (data not shown). In 2009, there were no differences in disease incidence among the cultivars except for at 1 July, 21 August, and 3 September (data not shown). Cultivars had the same levels of disease severity until 22 July, after which there were differences at each date of disease assessment (data not shown).

Although SDS was greater in inoculated versus non-inoculated plots in both years (Table 7), an influence of SDS on yield was only observed in 2009 when inoculated plots experienced a 9% yield reduction compared to non-inoculated plots (data not shown). These data support the observation that timing of SDS development plays a critical role in seed yield losses (Farias Neto et al., 2006). Disease was greater in inoculated plots than non-

inoculated plots in 2008, yet there was no effect on yield because differences in disease levels between inoculated and non-inoculated plots were not observed until 11 August as explained by the inoculation by date of disease assessment interaction for disease incidence and severity (data not shown). At this date of disease assessment early and late MG II and MG III cultivars had reached growth stages R5, R4, and R3, respectively (Table 2). In 2009, however, overall disease pressure was greater than in 2008 (Table 7) and inoculated plots had in general greater disease severity than non-inoculated plots at all disease assessment dates and greater disease incidence (data not shown). Therefore, SDS was greater in inoculated plots than non-inoculated plots while yield was being formed in 2009 but not necessarily in 2008, which supports the observation by Stephens et al. (1993) that SDS must be severe before R5 in order to influence seed yield.

Early MG II cultivars had greater disease incidence and severity than MG III cultivars in both years which translated into a yield reduction of early MG II cultivars in 2008 (Table 4 and 8). Greater SDS in earlier maturing cultivars compared to later maturing cultivars contradicts findings of Mueller et al. (2003) showing earlier maturing cultivars demonstrated less SDS foliar symptoms than later maturing cultivars in greenhouse studies. The yield difference in 2008 contradicts the findings of the inoculation by date of disease assessment found for disease incidence and severity (data not shown). In 2008, inoculated plots did not demonstrate more disease than non-inoculated plots until 11 August (data not shown), at which point early MG II cultivars were at R5 (Table 2) whereas during 2009 disease was severe while yield of the early MG II cultivars might have still been forming. Since disease must be severe before R5 in order to affect yield (Stephens et al., 1993) the yield difference between early MG II and MG III cultivars should have been observed in 2009. However, a

lack of association between SDS symptoms and seed yield could be related to the differential resistance of the cultivars to the disease (Farias Neto et al., 2006). The yield advantage of the MG III cultivars over the early MG II cultivars in 2008 is attributed to the yield benefit of a full-season cultivar for the respective area of adaptation as the reactions of the cultivars to SDS at each disease assessment date throughout the growing seasons were inconsistent and not conclusive (data not shown).

In 2008 and 2009 late MG II cultivars had greater disease incidence and severity than MG III cultivars but no difference in yield between the two maturity groups was observed (Tables 4 and 8). Early MG II cultivars had greater disease incidence and severity than MG III cultivars in both years (Table 8). Again, earlier maturing cultivars exhibiting greater SDS contradicts work by Mueller et al. (2003) showing earlier maturing cultivars to show less SDS than later maturing cultivars. Resistant cultivars demonstrated less SDS than susceptible cultivars in both years yet the only indication ($P = 0.07$) that SDS reduced yield of the SDS-susceptible cultivars was in 2009 (Table 5; 9). The interaction for cultivar with date of disease assessment for disease incidence and severity demonstrated inconsistent reactions of the cultivars to SDS at each date of disease assessment throughout the growing seasons, therefore clear relations among disease occurrence, levels and yield cannot be understood based on this study and there are not conclusive explanations for these results. Despite the lack of strong evidence for a decrease in yield seen when SDS-susceptible cultivars demonstrate more disease than SDS-resistant cultivars in our study due to low levels of disease, previous work has demonstrated a linear relationship between yield decrease and SDS for cultivar susceptibility (Farias Neto et al., 2006). The variable and unclear responses

of cultivars to SDS in this study further demonstrate the significant differences in cultivar response to SDS (Hershman et al., 1990; Rupe and Gbur, 1995).

Our results provide evidence that when planting an early maturing cultivar, SDS-resistance is important as K-204 had greater SDS and lower yield than AG2002 in both years (Tables 6 and 10). Susceptibility to SDS and greater disease expression of K-283 did not translate into a yield reduction compared to K-285 in either year (Tables 6 and 10). For MG III cultivars K-321 demonstrated greater SDS and lower yield than K-348 in 2009 (Tables 6 and 10). Research shows that earlier maturing cultivars form more yield per day than later maturing cultivars (Egli, 1993; Salado-Navarro et al., 1986). We speculate that the yield reduction of K-204 is the result of early maturing cultivars forming a greater amount of yield during a shorter time period than later maturing cultivars. In 2008, SDS progression was not strong in August, during which MG III cultivars could form yield whereas early MG II cultivars had formed the majority of their yield. In 2009, SDS progression was more consistent throughout the season and K-321 experienced a yield reduction, illustrating the importance of using a SDS resistant-cultivar to protect yield against severe disease progression during yield formation. Other than yield, no other harvest component was influenced by inoculation and therefore conclusions regarding effect of SDS on maturity class for those factors cannot be addressed from this study (data not shown).

Summary

Results of this study support past research from the southern United States showing disease onset to be independent of maturity group, and also that cultivar response to SDS was variable throughout both growing seasons. Although SDS onset occurred at a later growth stage in early MG II cultivars compared to later maturing cultivars, early MG II cultivars did

not demonstrate a yield advantage over late MG II and MG III cultivars in SDS environments. In agreement with past studies, our results indicate that yield loss to SDS appears to be a function of disease onset and progression throughout the growing season. This study indicates that in order to achieve full yield potential in SDS environments, growers in Iowa and the upper Midwest should plant a full season SDS-resistant cultivar adapted to their location.

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References

Aoki, T., K. O'Donnell, H. Yoshihisa, and A.R. Lattanzi. 2003. Sudden-death syndrome of soybean is caused by two morphologically and phylogenetically distinct species within the *Fusarium solani* species complex- *F. virguliforme* in North America and *F. tucumaniae* in South America. Mycologia. 95:660-684.

Board, J.E., M. Kamal, and B.G. Harville. 1992. Temporal importance of greater light interception to increased yield in narrow-row soybean. Agron. J. 84:575-579.

Board, J.E., S. Kumudini, J. Omielan, E. Prior, and C.S. Kahlon. 2010. Yield response of soybean to partial and total defoliation during the seed-filling period. Crop Sci. 50:703-712.

Bunting, A.H. 1971. Productivity and profit or is your vegetative phase really necessary? Ann. Appl. Biol., 67:265-285.

Cook, R.J. 2000. Advances in plant health management in the twentieth century. Annu. Rev. Phytopathol. 38:95-116.

De Bruin, J.L., and P. Pedersen. 2008a. Yield improvement and stability for soybean cultivars with resistance to *Heterodera Glycines* Ichinohe. Agron J. 100:1354-1359.

De Bruin, J.L., and P. Pedersen. 2008b. Soybean cultivar and planting date response to soil fumigation. Agron. J. 100:965-970.

De Bruin, J.L., and P. Pedersen. 2009. New and old soybean cultivar responses to plant density and intercepted light. Crop Science. 49:2225-2232.

Edwards, J.T., and L.C. Purcell. 2005a. Soybean yield and biomass responses to increasing Plant population among maturity groups: I. Agronomic characteristics. Crop Sci. 45:1770-1777.

Edwards, J.T., L.C. Purcell, and D.E. Karcher. 2005b. Soybean yield biomass responses to increasing plant population among diverse maturity groups: II. Light interception and utilization. Crop Sci. 45:1778-1785.

Egli, D.B., and Y. Zhen-wen. 1991. Crop growth rate and seeds per unit area in soybean. Crop Sci. 31:439-442.

Egli, D.B. 1993. Cultivar maturity and potential yield of soybean. Field Crops Res. 32;147-158.

Farias Neto, A.F., G.L. Hartman, W.L. Pedersen, S. Li, and B.W. Diers. 2006. Irrigation and inoculation methods that increase the severity of soybean sudden death syndrome in the field. Crop Sci. 46:2547-2554.

Fehr, W.R., and C.E. Caviness. 1977. Stages of soybean development. Spec. Rep. 80. Iowa Agric. Home Econ. Exp. Stn., Iowa State Univ., Ames.

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

Gay, S., Egli, D.B., and Reicosky, D.A. 1980. Physiological basis of yield improvement in soybeans. Agron. J. 72:387-391.

Naeve, S.L., and S.C. Huerd. 2008. Year, region, and temperature effects on the quality of Minnesota's soybean crop. Agron. J. 100:690-695.

Navi, S.S., and X.B. Yang. 2008. Foliar symptom expression in association with early infection and xylem colonization by *Fusarium virguliforme* (formerly *F. solani* f. sp. *glycines*), the causal agent of soybean sudden death syndrome. Online. Plant Health Progress doi:10.1094/PHP-2008-0222-01-RS.

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

Njiti, V.N., R.J. Suttner, L.E. Gray, P.T. Gibson, and D.A. Lightfoot. 1997. Rate- reducing resistance to *Fusarium solani* f. sp. *phaseoli* underlies field resistance to soybean sudden death syndrome. Crop Sci. 37:132-138.

Njiti, V.N., M.A. Shenaut, R.J. Suttner, M.E. Schmidt, and P.T. Gibson. 1998. Relationship between soybean sudden death syndrome disease measures and yield components in F_6-derived lines. Crop Sci. 38:673-678.

Mueller, D.S., R.L. Nelson, G.L. Hartman, and W.L. Pedersen. 2003. Response of commercially developed soybean cultivars and the ancestral soybean lines to *Fusarium solani* f. sp. *glycines.* Plant Dis. 87:827-831.

Robinson, A.P., S.P. Conley, J.J. Volenec, and J.B. Santini. 2009. Analysis of high yielding,

early-planted soybean in Indiana. Agron. J. 101:131-139.

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

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

Rupe, J.C., E.E. Gbur, and D.M. Marx. 1991. Cultivar responses to sudden death syndrome of soybean. Plant Dis. 75:47-50.

Rupe, J.C., and E.E. Gbur Jr. 1995. Effect of plant age, maturity group, and the environment on disease progress of sudden death syndrome of soybean. Plant Dis. 79:139-143.

Salado-Navarro, L.R., T.R. Sinclair, and K. Hinson. 1986. Yield and reproductive growth of simulated and field-grown soybean. II. Dry matter allocation and seed growth rates. Crop Sci. 26:971-975.

Sanogo, S., X.B. Yang, and H. Scherm. 2000. Effects of herbicides on *Fusarium solani* f. sp. *glycines* and development of sudden death syndrome in glyphosate-tolerant soybean. Phytopathology 90:57-66.

SAS Institute. 2008. The SAS system for Windows. v. 9.2. SAS Inst. Cary, NC.

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

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

Shibles, R.M., and C.R. Weber. 1966. Interception of solar radiation and dry matter

production by various soybean planting patterns. Crop Sci. 6:55-59.

Shibles, R.M., I.C. Anderson, and A.H. Gibson. 1975. Soybean. p. 151-190. *In*: L.T. Evans (ed) Crop physiology. Cambridge Univ. Press, Cambridge.

Smith, J.R., and R.L. Nelson. 1986a. Relationship between seed-filling period and yield among soybean breeding lines. Crop Sci. 26:496-472.

Stephens, P.A., C.D. Nickell, and S.M. Lim. 1993. Sudden death syndrome development in soybean cultivars differing in resistance to *Fusarium solani*. Crop Sci. 33:63-66.

Wrather, J.A., S.R. Koenning, and T.R. Anderson. 2003. Effect of diseases on soybean yields in the United States and Ontario (1999-2002). Plant Health Progress. Doi:10.1094/PHP-2003-0325-01-RV.

Zhang, L.X., Wang, R. and J.D. Hesketh. 2001. Effects of Photoperiod on Growth and development of soybean floral bud in different maturity. Agron J. 93:944-948.

Table 1. Field characteristics at the Jefferson and Nevada field sites in 2008 and 2009.

Location	Jefferson		Nevada	
Year	2008	2009	2008	2009
Soil Series	Canisteo clay loam	Canisteo clay loam	Webster clay loam	Webster clay loam
Soil Family	Fine-loamy, Mixed, Superactive, Mesic Typic Hapludolls	Fine-loamy, Mixed, Superactive, Mesic Typic Hapludolls	Fine-loamy, Mixed, Superactive, Mesic Typic Hapludolls	Fine-loamy, Mixed, Superactive, Mesic Typic Hapludolls
pH	6.7	6.8	7.5	6.8
P (mg kg^{-1})	23	8	38	68
K (mg kg^{-1})	176	188	197	280
OM (g kg^{-1})	47	42	55	46
Planting Dates	1 May	5 May	5 May	5 May
Harvest Dates	1 Oct	28 Sept	2 Oct	29 Sept

†OM, organic matter

Table 2. Soybean growth stages of cultivars according to classes of maturity at the disease assessment dates across the two locations in Iowa during 2008 and 2009. Growth stages determined at each assessment time based on evaluation of each plot growth stage.

2008				2009			
Date of disease assessment	Maturity class†			Date of disease assessment	Maturity class		
	E	M	L		E	M	L
30 June	R1‡	R1	R1	10 June	V3	V3	V3
10 July	R2	R2	R1	22 June	V5	V5	V5
21 July	R3	R2	R2	1 July	R1	R1	R1
30 July	R4	R3	R2	9 July	R2	R2	R1
11 August	R5	R4	R3	22 July	R3	R2	R2
21 August	R6	R5	R4	30 July	R4	R3	R3
3 September	R7	R6	R5	10 August	R5	R4	R3
-	-	-	-	21 August	R6	R5	R4
-	-	-	-	3 September	R7	R6	R5

†E, M and L represent early MG II, late MG II and MG III maturing cultivars for central Iowa, respectively.

‡Growth stages determined as described by Fehr and Caviness (1977).

Table 3. Monthly mean air temperature and precipitation totals recorded at two experimental locations in 2008 and 2009. Deviations from the 20-yr average are reported in parentheses.

		May		June		July		August		September	
Year	Location	Air temp.	Rainfall	Air temp.	Rainfall	Air temp.	Rainfall	Air temp.	Rainfall	Air temp.	Rainfall
		°C	mm	°C	mm	°C	mm	°C	mm	°C	mm
2008	Jefferson	14 (-2.1)†	232 (117)	21 (-0.6)	184 (62)	23 (-1.1)	115 (1)	21 (-2.1)	46 (-62)	17 (-1.0)	47 (-29)
	Nevada	16 (-1.0)	216 (94)	21 (-0.4)	271 (160)	23 (0.05)	234 (123)	21 (-1.0)	53 (-73)	18 (-0.3)	78 (0)
2009	Jefferson	16 (-0.7)	77 (-42)	20 (-1.5)	115 (-13)	20 (-3.7)	135 (24)	21 (-1.5)	110 (12)	18 (0.3)	28 (-48)
	Nevada	16 (-0.8)	102 (-26)	71 (-0.3)	104 (-16)	21 (-2.7)	70 (-59)	21 (-1.6)	89 (-25)	18 (-0.3)	31 (-48)

† Twenty-year averages based on Iowa Environmental Mesonet locations near Jefferson and Nevada, IA.
Available at: http://mesonet.agron.iastate.edu/climodat/index.phtml

Table 4. Mean differences between classes of maturity for soybean seed yield, final plant population, plant height, seed mass, protein and oil at Jefferson and Nevada in 2008 and 2009.

	2008						2009					
Maturity Group	Seed yield	Final plant population	Plant height	Seed mass	Protein	Oil	Seed yield	Final plant population	Plant height	Seed mass	Protein	Oil
	kg ha^{-1}	Plants ha^{-1}	cm	g 100 seed^{-1}	g kg^{-1}	g kg^{-1}	kg ha^{-1}	Plants ha^{-1}	cm	g 100 seed^{-1}	g kg^{-1}	g kg^{-1}
Early vs. Mid†												
Estimate	-218	12500	-5.8	-1.2	0.13	0.80	-1	45300	-5.6	-1.2	0.88	0.12
P-value	0.17	0.29	0.01	<0.01	0.28	<0.01	0.99	<0.01	<0.01	<0.01	<0.01	0.18
Mid vs. Late												
Estimate	-170	8700	-2.7	0.5	0.06	-0.03	-155	24100	-4.7	-0.4	0.39	-0.01
P-value	0.28	0.47	0.20	<0.01	0.64	0.71	0.22	0.04	<0.01	0.16	0.01	0.94
Early vs. Late												
Estimate	-388	21200	-8.4	-0.7	0.19	0.77	-15	69400	-10.4	-1.6	1.26	0.11
P-value	0.02	0.08	<0.01	<0.01	0.13	<0.01	0.22	<0.01	<0.01	<0.01	<0.01	0.21

† Early, Mid and Late represent early MG II, late MG II and MG III cultivars for central Iowa, respectively.

Table 5. Differences between soybean cultivars without and with sudden death syndrome (SDS) resistance at two locations in Iowa (Jefferson and Nevada) in 2008 and 2009.

	2008		2009	
SDS-Susceptible vs. SDS-Resistant	Estimate	*P*-value	Estimate	*P*-value
Yield, kg ha^{-1}	-186	0.15	-183	0.08
Final Plant Population, plants ha^{-1}	11900	0.22	-17400	0.07
Plant Height, cm	-1.8	0.29	-0.8	0.53
Seed Mass, g 100 $seed^{-1}$	0.4	<0.01	-0.3	0.12
Protein, g kg^{-1}	0.18	0.07	0.60	<0.01
Oil, g kg^{-1}	0.31	<0.01	-0.14	0.06

Table 6. Mean differences between soybean cultivars without and with sudden death syndrome (SDS) resistance within three classes of maturity at two locations in Iowa (Jefferson and Nevada) in 2008 and 2009.

	2008						2009					
Maturity Group	Seed yield	Final plant population	Plant height	Seed mass	Protein	Oil	Seed yield	Final plant population	Plant height	Seed mass	Protein	Oil
	kg ha^{-1}	Plants ha^{-1}	cm	g 100 seed^{-1}	g kg^{-1}	g kg^{-1}	kg ha^{-1}	Plants ha^{-1}	cm	g 100 seed^{-1}	g kg^{-1}	g kg^{-1}
Early†												
Estimate	-387	-21000	-2.9	1.6	-0.97	0.46	-298	-40100	-4.7	0.9	-0.61	0.13
P-value	0.09	0.21	0.33	<0.01	<0.01	<0.01	0.10	0.02	0.03	<0.01	<0.01	0.29
Mid												
Estimate	-323	12300	-3.3	-0.7	0.02	0.28	217	30800	1.3	-1.5	1.00	-0.36
P-value	0.15	0.46	0.25	<0.01	0.90	0.02	0.22	0.07	0.56	<0.01	<0.01	<0.01
Late												
Estimate	154	44400	0.8	0.2	1.48	0.20	-470	-43000	1.1	-0.4	1.40	-0.20
P-value	0.49	0.01	0.79	0.29	<0.01	0.08	0.01	0.01	0.59	0.25	<0.01	0.13

† Early, Mid and Late represent early MG II, late MG II and MG III cultivars for central Iowa, respectively.

Table 7. Response of sudden death syndrome (SDS) incidence and severity to planting date, soybean cultivar, inoculation, and sampling time at Jefferson and Nevada in 2008 and 2009.

Treatment	Disease Incidence		Disease Severity	
	2008	2009	2008	2009
	%		1 – 9†	
Cultivar (C)‡				
AG 2002	4.4	5.1	0.4	0.9
K-204	13.0	13.5	0.8	2.1
K-283	9.3	12.8	0.8	1.9
K-285	6.6	7.6	0.8	1.3
K-321	6.8	9.7	0.6	1.5
K-348	1.2	3.7	0.1	0.8
LSD (0.05)	3.3	2.5	0.3	0.3
Inoculation (I)				
Non-inoculated	4.2	2.7	0.4	0.5
Inoculated	9.5	14.7	0.8	2.4
LSD (0.05)	1.9	1.5	0.1	0.2
ANOVA				
C X I	NS§	**	**	**
C X T¶	***	***	***	***
I X T	***	***	***	***
C X I X T	NS	NS	NS	NS

† If disease was present, foliar symptom severity assessed according to increasing severity on a scale of 1 – 9 as outlined by Njiti et al. (1996).

‡ AG 2002, K-285, K-348 were resistant to SDS; K-204, K-283, K-321 were susceptible to SDS.

§ NS, not significantly different at $P \leq 0.05$.

¶ Denotes timing of disease assessment treatment.

,* significantly different at $P \leq 0.01$, and $P \leq 0.001$ levels, respectively.

Table 8. Differences in sudden death syndrome (SDS) disease incidence and severity between soybean classes of maturity at two locations in Jefferson and Nevada, Iowa in 2008 and 2009.

	2008		2009	
	Disease Incidence	Disease Severity	Disease Incidence	Disease Severity
	%	1 – 9†	%	1 – 9
Early vs. Mid‡				
Estimate	0.7	-0.2	-0.9	-0.1
P-value	0.55	0.03	0.33	0.59
Mid vs. Late				
Estimate	3.9	0.4	3.5	0.4
P-value	<0.01	<0.01	<0.01	<0.01
Early vs. Late				
Estimate	4.6	0.2	2.6	0.4
P-value	<0.01	<0.01	<0.01	<0.01

† If disease was present, foliar symptom severity assessed according to increasing severity on a scale of 1 – 9 as outlined by Njiti et al. (1996).
‡ Early, Mid and Late represent early MG II, late MG II and MG III cultivars for central Iowa, respectively.

Table 9. Mean differences between soybean cultivars without and with sudden death syndrome (SDS) resistance at two locations at Jefferson and Nevada, Iowa in 2008 and 2009.

	2008		2009	
	Estimate	*P*-value	Estimate	*P*-value
Disease Incidence, %	5.6	<0.01	6.5	<0.01
Disease Severity, 1 - 9†	0.4	<0.01	0.8	<0.01

† If disease was present, foliar symptom severity assessed according to increasing severity on a scale of 1 – 9 as outlined by Njiti et al. (1996).

Table 10. Mean differences between cultivars without and with sudden death syndrome (SDS) resistance within classes of maturity at two locations in Jefferson and Nevada, Iowa in 2008 and 2009.

	2008		2009	
	Disease Incidence	Disease Severity	Disease Incidence	Disease Severity
	%	1 – 9†	%	1 - 9
Early‡				
Estimate	8.5	0.5	8.3	1.2
P-value	<0.01	<0.01	<0.01	<0.01
Mid				
Estimate	2.8	0.1	5.2	0.5
P-value	0.10	0.52	<0.01	<0.01
Late				
Estimate	5.6	0.5	6.0	0.7
P-value	<0.01	<0.01	<0.01	<0.01

† If disease was present, foliar symptom severity assessed according to increasing severity on a scale of 1 – 9 as outlined by Njiti et al. (1996).

‡ Early, Mid and Late represent early MG II, late MG II and MG III cultivars for central Iowa, respectively.