Epidemiology and management of *Pantoea stewartii* in the Stewart's disease of corn/corn flea beetle pathosystem

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This thesis is dedicated to my mother Celirose Noel and my father Richard Menelas who had made a great deal of sacrifices for their children to receive a better education. I would not have completed this endeavor without the strong determination, love and support of my sisters Elizabeth and Anella and my brothers Riche, Gary, Frandre, and Icher.

J'ai appris une chose et je sais en mourant
Qu'elle vaut pour chacun:
Vos bons sentiments, que signifient-ils
Si rien n'en parait en dehors?
Et votre savoir, qu'en est-il
S'il reste sans consequences?
[...]
Je vous le dis:
Souciez-vous, en quittant ce monde,
Non d'avoir ete bon, cela ne suffit pas,
Mais de quitter un monde bon!

Bertolt Brecht,
Sainte Jeanne des abbatoirs

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INTRODUCTION

Stewart's disease of corn (*Zea mays* L.) caused by *Pantoea* (*Erwinia*) *stewartii* (Smith) Dye (Mergaert et al., 1993) is an economically important disease of both sweet and seed corn (Pataky et al., 1995a). The disease was first discovered in New York in 1897 by F. C. Stewart (Stewart 1897), however, it was not until 1932, when considerable losses were recorded in the U.S. corn belt and the Northeastern U.S., that its importance was determined (Poos and Elliot, 1936; Poos, 1939 and 1955). In the 1990's, Stewart's disease has become an extremely important problem in Iowa seed corn production as evidenced by the fact that disease prevalence increased from 13% of the fields in 1995 to 58% in 2000 (Esker and Nutter, 2000). This increase was due, in part, to the occurrence of six successive mild winters that favored the survival of the corn flea beetle (*Chaetocnema pulicaria* Melsh.), the primary vector for this pathogen. Stewart's disease is important to the seed corn industry because phytosanitary regulations restrict the sale of seed lots originating from seed corn fields where *P. stewartii* was found to occur (Block et al., 1999).

Pathogen

Pantoea stewartii is an insect-borne pathogen. However, *P. stewartii* is not known to be vertically transferred from corn flea beetle generation to the next corn flea beetle generation (vertical transmission) (Dill, 1979). According to Pepper (1967), *P. stewartii* is a non-motile, non-flagellate, non-spore-forming, capsule-forming gram-negative rod about 0.4–0.8 μm by 0.9–2.2 μm (Pepper, 1967). Pepper also reported that the bacterium was aerobic to facultatively anaerobic, based on the environmental conditions. However, Smith reported the bacterium as being strictly aerobic with a single polar flagellum (Smith, 1914), but this was most likely a contaminant. Since Smith's first account of the disease, the bacterium has undergone several name changes (*Pseudomonas stewartii*, *Bacterium stewarti, Aplanobacter stewarti*, *Bacillus stewarti, Phytomonas stewarti*, *Xanthomonas stewarti, Xanthomonas stewartii* and *Erwinia stewartii*). Based on the examination of electropherograms of soluble proteins, the pathogen was renamed *Pantoea stewartii* in 1993 (Mergaert et al., 1993).

Epidemiology of *Pantoea stewartii*

The primary source of inoculum for Stewart's disease is the corn flea beetle (*Chaetocnema pulicaria,* Melsh) (Stevens, 1934). *Pantoea stewartii* overwinters in the gut of adult corn flea beetles and the adult beetles are believed to overwinter in grassy areas that border corn fields (Poos, 1955; Dill, 1979). Corn flea beetles become active in early spring and typically feed on alternative grass species such as *Dactylis glomerata* L. (orchard grass), *Panicum capillare* L. (witchgrass), *Digitaria sanguinalis* (L.) Scrop. (crabgrass), *Setaria lutescens* (Weigel) Hubb (yellow foxtail), and *Poa pratenis* L. (Kentucky bluegrass), especially if corn has not yet emerged (Poos, 1955). Following corn emergence, the beetles migrate from grassy areas to corn fields and begin to feed on corn seedlings. The pathogen is transmitted to susceptible corn seedlings by infectious beetles. The bacteria propagate within the vascular tissues of corn plants and subsequent generations of corn flea beetles can acquire and then transmit the pathogen, after feeding on infected corn plants. In Iowa, there is an overwintering adult corn flea beetle generation that emerges in the spring, followed by two summer generations of corn flea beetles in the field (Esker et al., 2002).

Pantoea stewartii has been shown to survive in infected corn seeds; however, studies have shown that seed-to-seedling transmission is epidemiologically unimportant (Elliot and Poos, 1940; Block et al., 1998; Michener et al., 2002a). Although these studies have demonstrated that there is a low risk of seed transmission from infected seeds, zero tolerance phytosanitary regulations remain in place and are a major issue for seed corn companies. Early reports by Smith (1898) and Thomas (1924) concluded that infested soil was the primary source of inoculum for Stewart's disease of corn, but numerous studies since then have found no evidence to support the hypothesis that the bacterium can be transmitted from soil (Reddy, 1921; Rand and Cash, 1933; Frutchey, 1936). Ivanoff (1933) was able to isolate *P. stewartii* from infested soil and infected plant residue using a selective medium, however, transmission studies conducted by Rand and Cash (1933) involving infested plant residue found no conclusive evidence for the role of plant residue in this pathosystem. The risk of Stewart's disease epidemics is dependent upon several risk factors. Disease risk is higher (i) when mild winters favor the survival of the vector (*C. pulicaria*), (ii) in areas where the disease was prevalent during the previous growing season, (iii) in areas where there is a high overwintering population of corn flea beetles, and (iv) when fall populations of the corn flea beetles are infested with the bacterium (Esker et al., 2002; Esker and Nutter, 2002; Esker and Nutter, 2003; Nutter et al., 2002).

Pathogen vectors

The corn flea beetle is the primary vector of *P. stewartii* (Stevens, 1934), but other insects have also been documented as occasional vectors of *P. stewartii*. Rand and Cash (1933) were among the first to investigate other potential vectors of *P. stewartii*. In 1933, they conducted experiments with the twelve-spotted cucumber beetle (*Diabrotica duodecimpunctata* Fab.), and the toothed flea beetle (*Chaetocnema denticula* Ill.). Their results showed that these beetles could also acquire and transmit the bacterium, but not nearly as efficiently as *C. pulicaria*. Furthermore, Ivanoff (1933) was able to isolate *P.*

stewartii from corn rootworm larvae (*Diabrotica barberi* Smith) feeding on infected plants and demonstrated their role in transmission. These larvae were placed individually at the base of corn seedlings growing in the greenhouse and a high percentage of the plants (82%) exhibited Stewart's disease symptoms. Isolation from leaf tissue of these plants yielded typical *P. stewartii* colonies on agar plates. The seed corn maggot (*Delia platura* Meigen) was also found to be capable of transmitting the bacterium, but *C. pulicaria* (corn flea beetle) remains the most epidemiologically important vector for Stewart's disease of corn (Frutchey, 1936; Elliot and Poos, 1940).

Investigating seasonal development of *C. pulicaria* Elliot and Poos (1940) reported an estimated 20 to 30% of approximately 4,800 corn flea beetles were infested with the pathogen. In 1955, Roberts tested the percentage of overwintering corn flea beetles infested with *P. stewartii* and found that 10 to 20% of the beetles carried the bacterium and that the incidence of *P. stewartii*-infested corn flea beetles reached 75% during the midsummer months. Esker and Nutter, 2003 attempted to compare the proportion of *P. stewartii*-infested corn flea beetles at the end of one growing season to the proportion of *P. stewartii*-infested corn flea beetles present at the beginning of the next growing season for several location-years in Iowa. Results from some locations revealed no significant differences in the proportion of *P. stewartii*-infested corn flea beetles present from fall to spring, whereas, significant differences between fall and spring levels were observed for Ames, Chariton, and Nashua. For these locations, the proportion of *P. stewartii*-infested corn flea beetles was lower in spring 2000 compared to fall 1999 infestations.

Disease symptoms

Stewart's disease of corn occurs in two phases, an early wilt phase and a late leaf blight phase (Pepper, 1967). During the early wilt phase, typical symptoms on seedlings include leaves with linear, water-soaked, pale green to yellow lesions that elongate along the leaf veins (Smith, 1914; Rand and Cash, 1933; Pepper, 1967). This is followed by the wilting of seedlings, and when epidemics are severe, seedling death can occur (Dillard and Kline, 1989; Pataky and Eastburn, 1993). Typical symptoms of the late leaf blight phase include yellowish, water-soaked lesions or streaks that soon become necrotic (APS Corn Compendium, 1999). These symptoms are most often observed along the leaf veins of the corn leaves. Sweet corn is typically the more susceptible to *P. stewartii* than other types of corn, and significant yield losses can occur in susceptible or moderately susceptible sweet corn hybrids (Suparyono and Pataky, 1989; Pataky et al., 1990 and 1995b). Stewart's disease can also cause significant yield reductions in corn hybrids, if resistant hybrids are not used (Pataky et al., 1990).

Disease management

Stevens (1934) states the amount of primary inoculum present at the beginning of the season is related to the number of corn flea beetles that survive the winter. Various disease management strategies have been used to control the primary vector of *P. stewartii, C. pulicaria.* Disease forecasting based on the mean monthly temperatures for December, January and February has been used to predict the likelihood that corn flea beetle populations will survive the winter (Boewe, 1949; Esker and Nutter, 2000; Nutter et al., 1998; Stevens, 1934). These analyses help to indicate whether or not systemic insecticide seed treatments would be useful, as well as whether or not the date of planting should be altered to avoid the emergence of high overwintering populations of corn flea beetles. Disease risk has also been mapped using geographic information systems software to allow seed corn growers the option of choosing planting sites with lower disease risk (Nutter et al., 2002). Chemical seed treatments, such as the insecticides imidacloprid (Gaucho®) and thiamethoxan (Cruiser®), have been shown to effectively manage corn flea beetle populations (Munkvold et al., 1996; Pataky et al., 2000; Kuhar et al., 2001). However, there is no evidence to suggest that chemical seed treatments provide protection beyond the fifth leaf stage (V5 growth stage) of corn (Dill, 1979; Munkvold et al., 1996). The application of foliar insecticides after the V5 growth stage would be aimed at reducing corn flea beetle feeding (and therefore pathogen transmission) that might occur after the first and second field generations of corn flea beetles have emerged (Esker and Nutter, 2002; Nutter et al., 2002). Therefore, biologically-based information as to when to apply foliar insecticides after the V5 growth stage are needed in order to develop an effective, cost-efficient disease management program for Stewart's disease.

Altering the time of planting to avoid peak emergence periods of the overwintering corn flea beetle populations may also potentially reduce the risk of early season infection by *P. stewartii.* For instance, sweet corn is often planted over an extended period of time from early spring to mid-summer. This practice poses a serious threat to sweet corn growers because early season infection by *P. stewartii* can result in severe damage to corn seedlings when seedling emergence coincides with peak periods of corn flea beetle emergence. From 1934 to 1937, Elliot and Poos (1940) conducted several studies in Virginia to determine the planting dates of sweet corn that were most exposed to disease risk and damage caused by different generations of *C. pulicaria*. They reported that disease incidence was more severe in late plantings of a susceptible Golden Bantam variety, compared to earlier plantings (Elliot and Poos, 1940). After observing 14 successive plantings of Jubilee (a susceptible sweet corn variety), Heichel (1977) suggested that Stewart's disease can be controlled by adjusting the time of planting so that corn seedlings emerge when corn flea beetle populations are low. One potential drawback of this tactic, however, is that yield reductions may occur in association with certain late planting dates (Pataky et al., 1995). For sweet corn growers in Iowa, knowledge concerning when to plant susceptible sweet corn hybrids in order to avoid yield losses caused by Stewart's disease is of paramount importance.

Finally, the most effective management tactic to control Stewart's disease is the use of resistant varieties. In a recent study, Kuhar et al. (2002) indicated genetic resistance provided better control than insecticide seed treatment. They reported that disease incidence in two resistant sweet corn varieties (Dynamo and Bonus) was low ($\leq 5\%$) in plots that were treated or non-treated with insecticide seed treatments. However, growers should not rely solely on resistant varieties, because high corn flea beetle populations can cause severe damage even to resistant varieties and early infection of seedlings can result in substantial damage (Suparyono and Pataky, 1989).

Justification

The prevalence of Stewart's disease in Iowa increased dramatically between 1995 and 2000 and the need for better management strategies is critical, because phytosanitary regulations prevent the exportation of seed contaminated with *P. stewartii.* Corn flea beetles are the primary source of inoculum for Stewart's disease epidemics, yet, there is a lack of quantitative information concerning how long it takes for corn flea beetles to acquire and transmit *P. stewartii.* Knowledge about how corn flea beetles acquire and transmit the pathogen is important for a better understanding of this pathosystem and may provide valuable information as to how to better manage disease risk. For instance, knowing the length of time required for *P. stewartii*-infested corn flea beetles to transmit the pathogen to noninfected corn plants will help to determine the optimum time frame to apply foliar insecticides. Therefore, acquisition and transmission studies to monitor the length of time needed for a corn flea beetle to acquire and transmit the bacterium are needed.

It has been reported that insecticide seed treatments have provided partial control of the insect vector up to corn growth stage V5, resulting in a lower incidence of Stewart's disease early in the season. However, disease incidence may be further minimized if foliar insecticide application programs can be developed that optimize the timing of applications to coincide with specific corn growth stages. The development of a degree-day model to time foliar applications is a second option. Another possible method to time foliar insecticide applications would be the development of an action threshold based upon insect scouting. Corn growth stage, degree day models, and corn flea beetle action threshold timing methods may prove valuable as a means to determine when the application of foliar insecticides is most effective and cost-efficient, but these tactics need to be developed and tested experimentally.

The purpose of this study was to evaluate the effects of using an integrated approach that includes the time of planting, and the benefits of combining seed and foliar insecticides to improve disease management. Therefore, the objectives of this study were:

1. To quantify and model the acquisition access and transmission feeding periods required by corn flea beetles to acquire and to transmit *P. stewartii* in the Stewart's disease of corn pathosystem.

2. To determine the efficacy of using both seed and foliar insecticides to reduce corn flea beetle populations and Stewart's disease of corn.
3. To determine the method that best times foliar insecticide applications to reduce the corn flea beetle populations and the incidence of Stewart's disease of corn.
4. To determine the effects of time of planting on Stewart's disease of corn in Iowa.

MATERIALS AND METHODS

Acquisition – transmission studies

Studies were conducted under greenhouse conditions at Iowa State University to quantify the acquisition access and transmission feeding periods required by corn flea beetles (*Chaetocnema pulicaria*) to acquire and transmit *P. stewartii* in the Stewart's disease of corn pathosystem. The susceptible sweet corn variety Jubilee (Syngenta Seeds Inc., Boise, ID) was planted in 0.15-m-diameter plastic pots containing a steam pasteurized 1:2:1 mixture of peat, perlite, and soil. Two seeds were planted per pot, and later thinned to one plant per pot after emergence. The plants were watered every other day and fertilized weekly with a solution of 21-5-20 (N-P-K) fertilizer (200 ppm N, Miracle-Gro Excel, Marysville, OH).

Inoculum and leaf inoculum

Pantoea stewartii isolate ES Rif-9A was obtained from Dr. Charles C. Block of the USDA Plant Introduction Station at Iowa State University, Ames, IA. This isolate is a rifampicin and nalixidic acid-resistant isolate derived from a wild-type strain of *P. stewartii* (SS104) using the gradient plate method (Lamka et al., 1991). Cultures were incubated for 48 hours at 25 °C on nutrient broth yeast agar (NBY) amended with cycloheximide (100 µg / ml of ethanol), rifampicin (50 µg / ml of methanol), and nalidixic acid (40 µg / ml of 0.1M NaOH). Through serial dilution, the bacterial suspensions were adjusted to approximately 1 X 10^8 CFU/ml in normal saline solution (0.85 % NaCl). At corn growth stage V5 (fifth-leaf stage), the second newest leaf of each seedling was inoculated with *P. stewartii* (ES Rif-9A) and kept at a temperature between 18 to 21° C. A tong mounted with a 2.5 cm rubber stopper and a piece of sponge with protruding pins on one end, and another rubber stopper and a piece of sponge on the opposite end, was used to inoculate each corn leaf. For each inoculated plant, the tong-inoculator was dipped into the bacterial suspension and the second newest leaf was punctured with the tong-inoculator to simulate wounding and feeding scars by corn flea beetles. To ensure successful infection, the plants were reinoculated five days later with the same bacterial isolate and concentration.

Acquisition study

After symptoms were visible on the inoculated leaves (7–9 days later), one field-collected corn flea beetle was placed in a cage consisting of a 2.5 cm diameter acrylic hollow rod that was approximately 2.5 cm long. The top end was sealed with a polyester mesh (32 X 32 per 2.5 cm) and a piece of armaflex insulation sheet was placed on the opposite end affixed with two-1.25 cm nails (Fig. 1). Samples of field-collected corn flea beetles were separated from plant debris and other insects collected during field sampling by emptying the contents of a sample bag into a 5.6 liter plastic pan that was half-filled with tap water. Individual corn flea beetles were collected using a wet, small-bristle paintbrush and one corn flea beetle was placed inside each cage. Each cage (containing one beetle) was placed on the edge of *P.* ***stewartii*** lesions located on a diseased corn leaf. A diseased leaf was one where lesions typical of *P. stewartii* were observed along the leaf veins. Corn flea beetles were then allowed to feed for the appropriate acquisition access period. For the acquisition experiments, there were six treatments (6, 12, 24, 36, 48, and 72 hours) and 45 beetles (replications) per treatment. Upon removal, beetles were individually ground in 300 µl of 1X PBST buffer (8.0 g of NaCl, 1.15 g of Na_2HPO_4, 0.2 g KH_2PO_4, 0.2 g of KCl, 0.5 g of Tween 20, and 1,000 ml of dH_2O, pH 7.4) in an autoclaved microcentrifuge tube using a 20 cm drill press (Delta Machinery, Jackson, TN) operated at a speed of 1100 rpm. One hundred-microliters of each suspension was placed and streaked onto nutrient broth yeast agar amended with cycloheximide, rifampicin and nalidixic acid to isolate *P. stewartii*. Each plate was duplicated and the bacterial cultures were placed in an incubator at 25° C under a light/dark cycle of 12L:12D. After 48 hours, the plates were examined for colonies typical of *P. stewartii*. Pathogenicity tests were conducted to confirm that the colonies were *P. stewartii*. This was done by inoculating corn seedlings with individual isolates at corn growth stage V2 (two-leaf stage) using 1×10^8 CFU/ml in normal saline solution (0.85 % NaCl). Plants were observed for disease symptoms after 8 days. Acquisition experiment was performed three times.

Data analysis

Data from the acquisition experiments were analyzed using ANOVA and mean separations were performed using the Waller-Duncan K-ratio test ($P \leq 0.05$) (Statistical Analysis System, SAS Institute, Gary, NC). After plotting percentage acquisition with respect to acquisition access period using Sigma Plot® (SPSS INC., Chicago, IL), the mean values for pathogen acquisition were transformed to determine which model (logistic, linear, Gompertz, monomolecular, and logarithmic) best fit the data. The criteria used to select the best model were: (i) *F*-test for the overall model, (ii) coefficient of determination (R^2), (iii) the standard error of the estimate for y (SEEy), (iv) and the T-statistic for the slope. After selecting the most appropriate population growth model and transforming pathogen acquisition data, linear regression was used to estimate the slope (rate of pathogen acquisition with respect to time) and the intercept, and to calculate regression statistics.

Transmission study

Based on the results obtained from the acquisition study, the transmission studies were conducted using a single acquisition period of 48 hours. For each transmission experiment, corn flea beetles (270) were placed in cages (one per cage) and allowed to feed for 48 hours on diseased plants. After the 48 hr acquisition access period, each cage was removed from the diseased plant and then transferred to a healthy plant at corn growth stage V3 (three-leaf stage). There were seven treatments and 30 replications per treatment. The duration of the transmission periods were: 3, 6, 12, 24, 36, 48, or 72 hours. After the appropriate transmission period, corn flea beetles were removed and the plant leaves were carefully examined for feeding scars. The beetles were individually placed in autoclaved microcentrifuge tubes containing 300 µl of 1X PBST buffer and ground using a 20 cm drill press operated at a speed of 1100 rpm. From these microcentrifuge tubes, 200 µl were placed onto 100 X 15 mm bi-partitional plates (100 µl/partition) containing rifampicin-nalidixic acid selective media. The leaves were sampled seven days later by taking 0.3 g of leaf tissue in the area of the feeding scars, and grinding in 800 µl of 1X PBST buffer using the same drill press. The leaf tissue weight was based on the volume of 1X PBST buffer required for grinding. Two hundred microliters of leaf sap obtained from the ground leaf samples was plated onto the same selective medium to isolate the pathogen using the same incubation conditions as described previously. Pathogenicity tests were also conducted on these cultures as described previously.

Data analysis

Data from the transmission experiments were analyzed using ANOVA and mean separations were performed using the Waller-Duncan K-ratio test ($P \leq 0.05$) (Statistical Analysis System, SAS Institute, Gary, NC). After plotting percent transmission with respect to transmission feeding period using Sigma Plot®, the mean values for pathogen transmission were transformed to determine which model (logistic, linear, Gompertz, monomolecular, or logarithmic) best fit the data. The criteria used to select the best model were: (i) *F*-test for the overall model, (ii) coefficient of determination (R^2), (iii) the standard error of the estimate for y (SEEy), (iv) and the T-statistic for the slope. After selecting the most appropriate population growth model and transforming pathogen transmission data, linear regression was used to estimate the slope (rate of pathogen transmission) and the intercept, and to calculate regression statistics.

Insecticide study

To determine the effectiveness of seed and/or foliar insecticides in reducing the incidence of Stewart's disease of corn, Gaucho® (Gustafson, Inc., Dallas, TX), or Cruiser® (formerly Adage) (Syngenta, Inc.,Greensboro, NC) insecticide seed treatments were used alone or in combination with the foliar insecticide Warrior® (ZENECA Ag Products, Wilmington, DE). The insecticide treatments were evaluated in field experiments conducted in 2001 and 2002 at both the ISU Southeast Research Farm, Crawfordsville, IA and at the Pioneer Research Farm, Johnston, IA. The experimental sites were previously cropped with soybean at Crawfordsville and with corn at Johnston. At Crawfordsville, field preparations consisted of disking in the fall, followed by field cultivation in the spring. Ammonium nitrate was applied at 57 kg/ha and post-emergent herbicides (1.057 L Laddok S-12 + 1.057 L Crop oil concentrate) were applied after seedlings emergence (fifth-leaf stage). At Johnston, the field was cultivated prior to planting, followed by the application of nitrogen (87 kg/ha) and herbicide applications (0.79 L Dual II Magnum + 1.0 lb atrazine).

Experimental design

Two Stewart's disease-susceptible corn inbred lines were used for the field experiments. The inbred line A634 × CM105 (MBS INC., Story City, IA) was used in 2001 at both locations, and A632 Ht Block (Holdens Foundation Seeds Inc., Williamsburg, IA) was used in 2002 at both locations. The seeding rate was 70,000 plants per hectare. Seeds were sown at Johnston and Crawfordsville respectively on 24 April and 2 May in 2001 and on 16 and 26 April in 2002. The inbreds were planted in a randomized complete block design with four replications and there was a 12 m alley between replications at both locations. Each experimental unit (plot) at Crawfordsville consisted of eight rows, 15.24 m long that were spaced 0.91 m apart. Experimental units at Johnston were four rows wide and 12.19 m long, spaced 0.91 m apart.

Insecticide treatments

There were 12 treatments for both experimental sites (Table 1). The first two treatments consisted of corn seed that was treated with either Gaucho® (2.5 g a.i./kg seed) or Cruiser® (2.0 g a.i./kg seed). There were three methods used to determine when foliar sprays of Warrior® were applied. These applications were made according to (i) corn growth stage (ii) a degree-day model or (iii) according to an action threshold based on the number of corn flea beetles trapped on yellow sticky cards. Using the corn growth stage method to time insecticide sprays, foliar insecticides were first applied at corn growth stage V5 (fifth-leaf stage). The V5 stage was chosen because this is the growth stage beyond which chemical seed treatments have been reported to be no longer effective (Dill, 1979; Munkvold et al., 1996). The timing of foliar insecticide sprays was based upon the accumulation of degree-day or heat units beginning on 1 January to predict corn flea beetle development. Degree-days were calculated by subtracting the corn flea beetle developmental threshold reported by Dill (16) from the average daily temperature (DD = average daily temperature - 16 °C). Spray applications were made after 350 degree days to coincide with the emergence of the corn flea beetles for the first summer generation and after 650 degree days for the predicted emergence of the second generation.

For the action threshold method to time foliar insecticide applications of Warrior®, yellow sticky card traps (16 × 16 cm) were attached horizontally at a height of 30 cm above the soil line. Yellow sticky cards were attached with two medium binder clips to a 16 by 16 by 1.3 cm piece of plywood that was nailed to a 0.6 m wooden stake. One trap was placed in the center of each replicate plot of treatments 5, 8, and 11 (action threshold treatments) to determine when foliar applications of Warrior® would be applied (Table 1). Traps used were similar to the design described by Esker (2002). The action threshold was triggered at each site when there was an average of one corn flea beetle per trap from the twelve traps. The yellow sticky cards were changed and monitored weekly for *C. pulicaria.* A foliar insecticide application of Warrior® was then applied the same day the action threshold was exceeded.

The spray equipment used to apply Warrior® insecticide consisted of a four-row spray boom with four nozzles (R & D Sprayer, Opelousas, LA) and a pressurized backpack sprayer (Model TBAC CO_2, The Cornelius Co., Anoka, MN). Because the spray boom covered only four rows, two passes were required to cover each plot at Crawfordsville, whereas at Johnston, only one pass was required to treat the four row plots. One person carried the sprayer and walked at approximately 6.44 km/h to deliver 3 liters / plot at Johnston and 9 liters / plot at Crawfordsville at a pressure of 276 kPa (40 psi). The nozzles (8002VS, *TeeJet*) attached to the boom dispensed the chemical downward onto the foliage. Seed and foliar insecticide rates, and foliar insecticide application dates are shown in Tables 2 and 3, respectively.

Stand counts

Stand counts were obtained at the second and fifth leaf stage (V2, V5). Two-15 m transects per plot at Crawfordsville and two-12 m transects per plot at Johnston were used to determine the number of corn plants per meter of row. The average number of corn plants per meter of row was determined and treatment means were compared using the Waller-Duncan K-ratio test ($P \leq 0.05$).

Plant height

The plant height (cm) of corn plants in each plot was measured at corn growth stage V5 (fifth-leaf stage). Ten plants from the two center rows of each plot (5 per row) were measured from the base of the plant to the tip of the newest leaf. The average plant height for each plot was determined and treatment means were compared using the Waller-Duncan K-ratio test ($P \leq 0.05$).

Disease assessment methods

Fifty plants per plot from the two center rows (25 plants per row) were selected for disease incidence assessments. All plants located along a 9.14 m transect in the middle section of each row were assessed. The top two leaves on each plant were visually assessed for symptoms typical of Stewart's disease of corn (leaf streaking, stunting and/or wilting). Disease incidence (%) was defined as the number of plants exhibiting *P. stewartii* symptoms, divided by the total number of plants assessed, multiplied by 100. Disease incidence assessments were performed seven times during the 2001 growing season and six times during the 2002 growing season. Disease progress curves for each replicate plot were obtained by plotting disease incidence with respect to time using Sigma Plot® (SPSS INC., Chicago, IL). Area under the disease progress curves were calculated for each plot using the trapezoidal integration method (Campbell and Madden, 1990). Standardized AUDPC values for each replicate plot were obtained by dividing each AUDPC value by the duration of the epidemic (Campbell and Madden, 1990). Data were analyzed using Proc GLM in SAS and treatment means were compared using the Waller-Duncan K-ratio test ($P \leq 0.05$).

Corn flea beetle sampling methods

Corn flea beetle populations (*C. pulicaria*) were quantified throughout each growing season in 2001 and 2002 using sweep nets and yellow sticky cards, as described by Esker et al. 2002. Corn flea beetle samples were collected weekly using a 38.1-cm-diameter sweep net (Gempler's, Belleville, WI). Three, 6-m transect samples were taken from the center two rows of each plot. A 6-m transect sample consisted of ten sweeps over the plant canopy. Once collected, the contents of each sample was placed in a freezer bag, taken to the laboratory, and placed in a freezer for 24 hours at -17.8 °C to immobilize the insects. The contents of each sample were then emptied onto a white sheet of paper. To determine the number of corn flea beetles per plot, the number of corn flea beetles per 10-sweep sample was counted and the three samples were then averaged. Area under the cumulative corn flea beetle progress curves were calculated to determine the effect of insecticide treatment programs on corn flea beetle populations over the course of the growing season. Progress curves were obtained using Sigma Plot® and data were analyzed using Proc GLM in SAS and treatment means were compared using the Waller-Duncan K-ratio test ($P \leq 0.05$).

Analysis of disease progress curves

Data from field experiments were analyzed using ANOVA and mean separations were performed using the Waller-Duncan K-ratio test ($P \leq 0.05$) (Statistical Analysis System, SAS Institute, Gary, NC). After plotting disease incidence versus time, the mean values for pathogen incidence were transformed to determine which model (logistic, linear, Gompertz, monomolecular, or logarithmic) best fit the data. The criteria used to select the best model were: (i) *F*-test for the overall model, (ii) coefficient of determination (R^2), (iii) the standard error of the estimate for y (SEEy), (iv) and the T-statistic for the slope. After selecting the most appropriate population growth model and transforming disease incidence data, linear regression was used to estimate the slope (rate of disease progress with respect to time) and the intercept and to calculate regression statistics.

Harvest and yield data

Prior to harvest, both the number of corn plants and the number of ears per row were counted in the center three rows of each plot at Crawfordsville and for the center two rows of each plot in Johnston. The middle three rows of each plot at Crawfordsville, were machine-harvested and the seed moisture was obtained using Moisture Trac Model 5010 (Shiwers, Corydon, IA). At Johnston, the center two rows of each plot were hand-harvested. Seed quality tests were performed for all four location-years using an INFRATEC 1229 grain analyzer (Tecator, Hoganas, Sweden). The yield of harvested grain (kg/ha) for each plot was calculated using the formula: Yield (kg/ha) = sample weight (lbs) [(1 - measured moisture) / (1 - adjusted moisture) / 56 / Area] * 62.71; where 56 is the corn conversion factor for bushels per acre and 62.71 is the corn conversion factor for kilograms per hectare. Harvest dates were 1 October 2001 and 30 September 2002 for Crawfordsville, and 3 October 2001 and 28 August 2002 for Johnston. The effects of seed and foliar insecticide treatment programs on yield and seed quality were analyzed using Proc GLM in SAS and treatment means were compared using the Waller-Duncan K-ratio test ($P \leq 0.05$).

Planting date study

To determine the effect of planting date on the incidence of Stewart's disease of corn, the susceptible sweet corn variety Jubilee (Syngenta Seeds INC., Boise, ID) was planted on five sequential weekly planting intervals at Boone, IA (2001) and Crawfordsville, IA (2002). Planting dates in 2001 were: 18 April, 25 April, 2 May, 9 May, and 16 May. In 2002, planting dates were 23 April, 30 April, 7 May, 14 May, and 21 May. Plots were arranged in a randomized complete block design with three replications and five treatments (planting dates). Each experimental unit consisted of eight rows, 9.14 m long and spaced 0.91 m apart. There was a 9.14 m alley between replications. Sixty seeds per row were planted and after emergence, the stands were thinned to 45 plants per row. Plots were not artificially inoculated with *P. stewartii* and neither foliar insecticides nor chemical seed treatments were used to control *C. pulicaria* in this study.

Disease assessment methods

Incidence (%) of Stewart's disease was assessed at five different growth stage (V5, V10, V15, VT and R5) during the growing season as previously described. Area under the disease progress curves were calculated for each plot using the trapezoidal integration method (Campbell and Madden, 1990). Standardized AUDPC values for each replicate plot were obtained by dividing each AUDPC value by the duration of the epidemic (Campbell and Madden, 1990). Progress curves were obtained using Sigma Plot and data were analyzed using Proc GLM in SAS and treatment means were compared using the Waller-Duncan K-ratio test ($P \leq 0.05$).

Assessing corn flea beetle populations

Chaetocnema pulicaria populations were sampled weekly in all plots using sweep nets and yellow sticky cards as previously described (Esker et al., 2002). Traps (3 per plot) were located in the center of each plot between row numbers 2-3, 4-5, and 6-7. Area under the cumulative corn flea beetle progress curves were calculated to determine the effect of date of planting on corn flea beetle populations over the course of the growing season. Progress curves were obtained using Sigma Plot® (SPSS INC., Chicago, IL) and data were analyzed using Proc GLM in SAS and treatment means were compared using the Waller-Duncan K-ratio test ($P \leq 0.05$).

Analysis of disease progress curves

Data from field experiments were analyzed using ANOVA and mean separations were performed using the Waller-Duncan K-ratio test ($P \leq 0.05$) (Statistical Analysis System, SAS Institute, Gary, NC). After plotting disease incidence versus time, the mean values for pathogen incidence were transformed to determine which model (logistic, linear, Gompertz, monomolecular, or logarithmic) best fit the data. The criteria used to select the best model were: (i) *F*-test for the overall model, (ii) coefficient of determination (R^2), (iii) the standard error of the estimate for y (SEEy), (iv) and the T-statistic for the slope. After selecting the most appropriate population growth model and transforming disease incidence data, linear regression was used to estimate the slope (rate of disease progress with respect to time) and the intercept, and to calculate regression statistics.

Marketable and unmarketable sweet corn yield

Prior to harvesting plots by hand, the number of plants and corn ears were counted in the four center rows of each plot. The mass of unhusked and husked ears, as well as the length and diameter of ears, were determined post-harvest. For each plot, husked ears were separated into marketable ears and unmarketable ears and the mass of each was measured to determine the yield. A filled cob with no insect damage was considered marketable, whereas an unfilled cob or a filled cob with insect damage was considered unmarketable. Harvest dates were from 27 July to 9 August in 2001 for Boone, and from 3 August to 6 August in 2002 for Crawfordsville. The effects of date of planting on yield were analyzed using Proc GLM in SAS and treatment means were compared using the Waller-Duncan K-ratio test ($P \leq 0.05$). Regression of final disease incidence versus marketable and unmarketable yields was calculated to determine if there was any linear relationship.

Table 1. Treatments and number of foliar applications of Warrior® to control corn flea beetles and Stewart's disease of corn at Crawfordsville and Johnston, Iowa in 2001 and 2002.

Treatment	Number of foliar applications
1- Nontreated control (no insecticide)	Zero
2- Gaucho® seed treatment	Zero
3- Cruiser® seed treatment	Zero
4- Warrior® at V5[a]	One
5- Warrior® using a beetle threshold[b]	Two
6- Warrior® using DD-model[c]	Two
7- Gaucho® + Warrior® at V5, VT, R3[d]	Three
8- Gaucho® + Warrior® using threshold	Two
9- Gaucho® + Warrior® using DD-model	Two
10- Cruiser® + Warrior® at V5, VT, R3	Three
11- Cruiser® + Warrior® using threshold	Two
12- Cruiser® + Warrior® using DD-model	Two

[a] V5 is the stage of growth when corn plants had five leaves with a visible collar.
[b] The action threshold was reached when there was an average of one beetle per trap per week.
[c] Both sprays were applied according to the accumulation of degree days using a developmental threshold of 16 °C. The first spray was applied after 350 degree days had accumulated and the second spray was applied after 650 degree days had accumulated.
[d] VT is the tassel stage; the R3 corn growth stage is when the inner fluid of the cob is milky.

Table 2. Insecticides and the rates of application used to control corn flea beetles and to reduce the incidence of Stewart's disease of corn at Crawfordsville and Johnston, Iowa in 2001 and 2002.

Trade name	Common name	Rate
Gaucho®	Imidacloprid	250 g a.i./100 kg seed
Cruiser®	Thiamethoxam	200 g a.i./100 kg seed
Warrior®	Lambda-cyhalothrin	0.10 kg a.i./ha

Table 3. Dates and growth stages that foliar applications of Warrior® insecticide were applied at Crawfordsville and Johnston, Iowa in 2001

	Crawfordsville				Johnston			
	Treatment dates (DOY)							
Treatment	**6/20 (171)**	**6/29 (180)**	**7/19 (200)**	**8/3 (215)**	**6/21 (172)**	**7/3 (184)**	**7/13 (194)**	**8/16 (228)**
2- Gaucho® seed treatment								
3- Cruiser® seed treatment								
4- Warrior® at V5 [a]	V5				V5			
5- Warrior® using threshold [b]								
6- Warrior® using DD-model [c]		V8		R3		V11		R3
7- Gaucho® + Warrior® at V5, VT, R3 [d]	V5		VT	R3	V5		VT	R3
8- Gaucho® + Warrior® using threshold								
9- Gaucho® + Warrior® using DD-model		V8		R3		V11		R3
10- Cruiser® + Warrior® at V5, VT, R3	V5		VT	R3	V5		VT	R3
11- Cruiser® + Warrior® using threshold								
12- Cruiser® + Warrior® using DD-model		V8		R3		V11		R3

[a] V5 is the stage of growth when corn plants had five leaves with a visible collar.
[b] The action threshold was reached when an average of one beetle per trap per week was reached.
[c] Both sprays were applied according to the accumulation of degree days using a developmental threshold of 16 °C. The first spray was applied after 350 degree days had accumulated and the second spray was applied after 650 degree days had accumulated.
[d] VT is the tassel stage; the R3 corn growth stage is when the inner fluid of the cob is milky.

Table 4. Dates and growth stages of foliar applications of Warrior® insecticide were applied at Crawfordsville and Johnston, Iowa in 2002.

	Crawfordsville						Johnston					
	Treatment dates (DOY)											
Treatment	6/6 (157)	6/29 (180)	7/4 (185)	7/15 (196)	8/2 (214)	8/6 (218)	6/7 (158)	7/1 (182)	7/10 (191)	7/23 (204)	8/4 (216)	8/8 (220)
2- Gaucho® seed treatment												
3- Cruiser® seed treatment												
4- Warrior® at V5 [a]	V5						V5					
5- Warrior® using threshold [b]			V11			R3				R1		R3
6- Warrior® using DD-model [c]		V10			R3			V10			R3	
7- Gaucho® + Warrior® at V5, VT, R3 [d]	V5			VT-R1	R3		V5		VT-R1		R3	
8- Gaucho® + Warrior® using threshold			V11			R3				R1		R3
9- Gaucho® + Warrior® using DD-model		V10			R3			V10			R3	
10- Cruiser® + Warrior® V5, VT, R3	V5			VT-R1	R3		V5		VT-R1		R3	
11- Cruiser® + Warrior® using threshold			V11			R3				R1		R3
12- Cruiser® + Warrior® using DD-model		V10			R3			V10			R3	

[a] V5 is the stage of growth when corn plants had five leaves with a visible collar.
[b] The action threshold was reached when an average of one beetle per trap per week was reached.
[c] Both sprays were applied according to the accumulation of degree days using a developmental threshold of 16 °C. The first spray was applied after 350 degree days had accumulated and the second spray was applied after 650 degree days had accumulated.
[d] VT is the tassel stage; the R3 corn growth stage is when the inner fluid of the cob is milky.

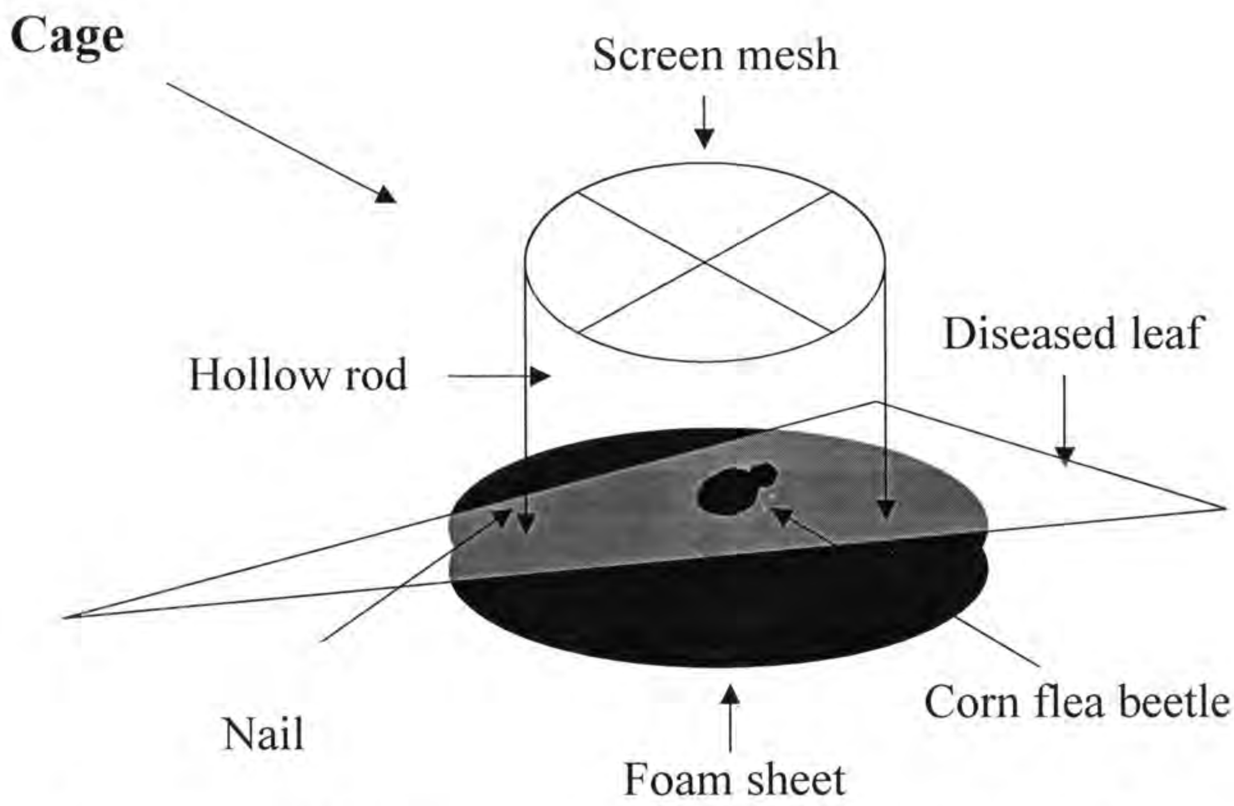


Figure 1. Diagram of the cage used to quantify the time periods required for corn flea beetles to acquire and transmit *Pantoea stewartii* on the sweet corn variety Jubilee.

RESULTS

Acquisition experiment

The earliest measured acquisition of *P. stewartii* by corn flea beetles occurred within three to six hours of feeding on diseased corn plants. The percentage of beetles that acquired the bacterium after a six hour access period ranged from 0–26.7 % (Fig. 2A). Acquisition increased at the greatest rate from 12 to 36 hours, and then increased more slowly between 36 and 72 hours (Fig. 2A). After 72 hours, the percentage of corn flea beetles, that had acquired the bacterium, ranged from 68.2 to 93.8 % (Fig. 2A). Overall, the percentage acquisition of *P. stewartii* with respect to duration of the acquisition access period was best described by the Gompertz model (Fig. 2B). The coefficient of determination (R^2) values for the three experiments were 0.91, 0.99, and 0.91, respectively (Fig. 2B), indicating greater than 90 % of the variation in percentage acquisition (gompits) of *P. stewartii* by corn flea beetles was explained by the duration of the acquisition access period. The standard errors of estimate for y (SEEy) for the three transformed acquisition curves were 0.38, 0.15, and 0.19, respectively. The slopes relating percentage acquisition of *P. stewartii* by corn flea beetles to acquisition access period for the three experiments were 0.043, 0.058, and 0.022 gompits per hour, respectively (Fig. 2B). Using linear regression equations (Fig. 2B), the duration of the access period required for 50% of the corn flea beetles to acquire *P. stewartii* in the three experiment were 53.4, 28.3, and 33.3 hours, respectively. The average acquisition time for 50 % of the corn flea beetles to acquire *P. stewartii* was 38.3 ± 10.1 hours. The experiments could not be combined in a single model because there were significant differences ($P \leq 0.05$) among the slopes for the individual experiments.

Transmission experiment

Percent transmission of *P. stewartii* by *P. stewartii*-infested corn flea beetles to healthy corn seedlings (Fig. 3A) increased the fastest early in the transmission feeding period and then slowed with increasing transmission feeding time. The increase in the percent transmission of *P. stewartii* from ***P. stewartii***-infested corn flea beetles to corn seedlings with respect to increasing transmission feeding period for the three transmission experiments were best described by the monomolecular model (Fig. 3B). This model showed that transmission feeding period explained 77 to 97 % of the variation in the change of transformed percentage transmission of *P. stewartii* by corn flea beetles. The R^2 values using this model transformation were 0.77, 0.97, and 0.93, respectively for the three experiments and standard errors of the estimate for y (SEEy) were 0.12, 0.04, and 0.13, respectively (Fig. 3B). The rates of *P. stewartii* transmission using the monomolecular model for each experiment were 0.008, 0.007, and 0.017, respectively, indicating that, for each hour of transmission feeding period, ln (1/1-y) percent transmission increased by 0.008, 0.007, and 0.017 per hour (Fig. 3B). The minimum time required for *C. pulicaria* to transmit *P. stewartii* after a 48 hour of acquisition access period was between zero to three hours in experiment three (Fig. 3A). The percentage transmission from 0 to 6 hours for all three experiments ranged from 0 to 33.3 % (Fig. 3A). Using monomolecular model, the predicted times required for 50 % pathogen transmission in the three experiments were 29.3, 28.3 and 27.2 hours, respectively (Fig. 3B). The mean time to reach 50 % pathogen transmission was 28.3 ± 5.4 hours. After a 72 hour feeding period, percentage transmission of *P. stewartii* from corn flea beetles to corn seedlings for the three transmission experiments was 58.3, 60.0, and 71.4 %, respectively

(Fig 3A). Significant differences ($P \leq 0.05$) among the slopes for the three experiments indicated that the experiments could not be combined into one single model.

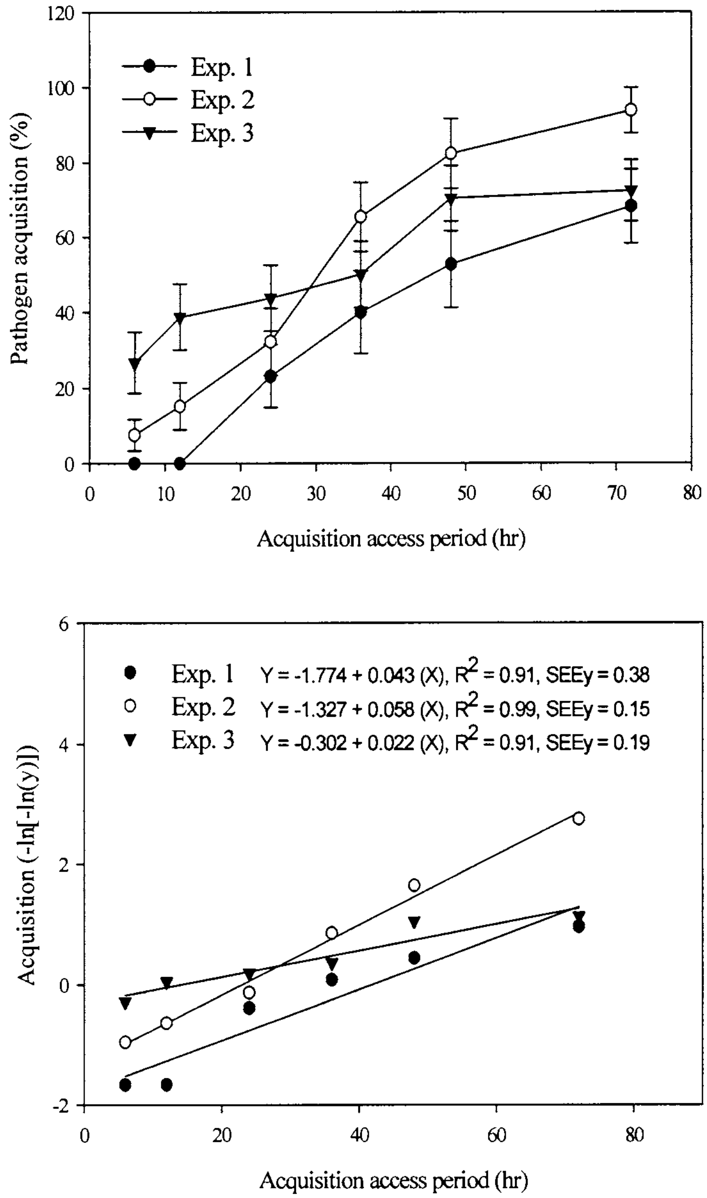


Figure 2. (**A**) Effect of acquisition access period on pathogen acquisition (%) by corn flea beetles after feeding on corn plants inoculated with *P. stewartii* (isolate ES-Rif 9A), and (**B**) linear regression lines using the Gompertz model (-ln [-ln(y)]) versus time to transform *P. stewartii* acquisition percentage data by corn flea beetles.

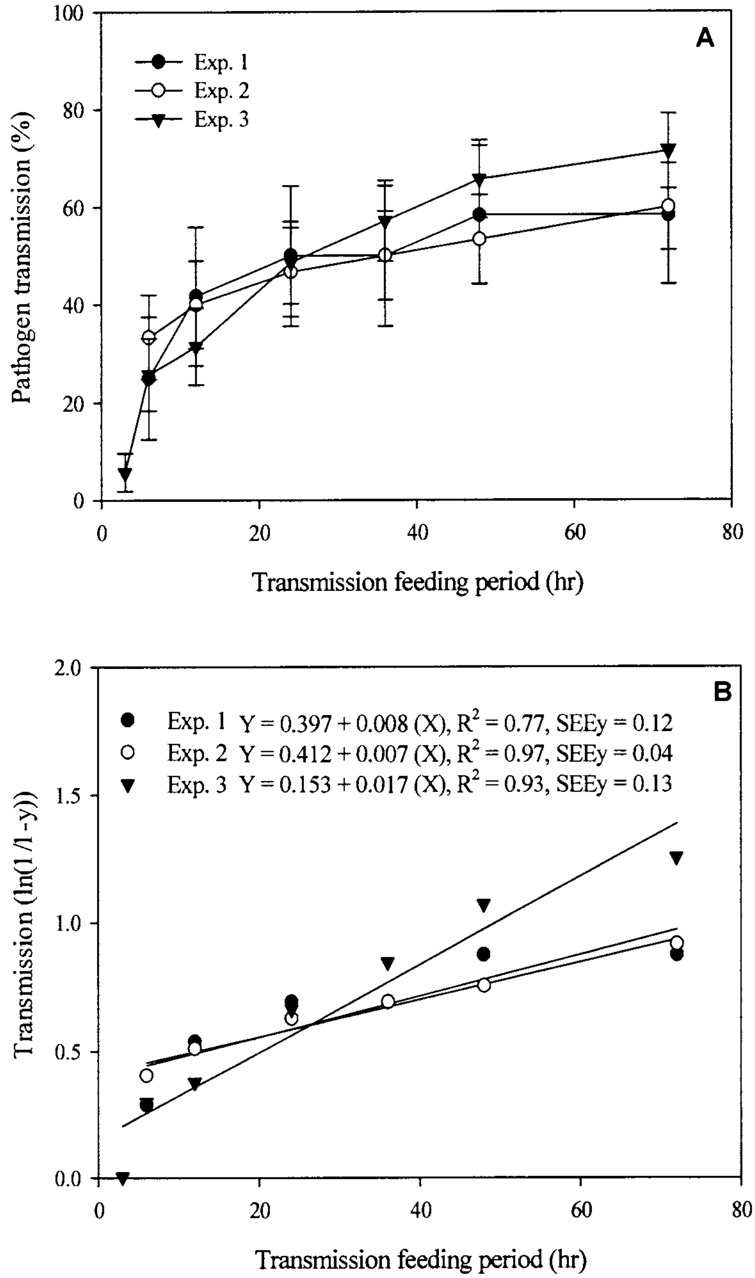


Figure 3. (**A**) Effect of transmission feeding period on pathogen transmission (%) by *P. stewartii*-infested corn flea beetles (isolate ES-Rif 9A) after feeding on healthy corn plants for different periods of time, and (**B**) the relationship between transmission feeding period and ln (1/1-y) percentage transmission of *P. stewartii* from infested corn flea beetles to corn seedlings.

Effect of seed insecticides on plant height and plant stand

None of the insecticide seed treatment applications resulted in plant height and stand counts that were significantly higher than the nontreated control. In fact, there were cases in which some insecticide seed treatments resulted in reduced plant heights and lower stand counts than the nontreated control (Tables 5 and 6), indicating that there was probably no effect on plant height or stand.

Effect of seed and foliar insecticides on Stewart's disease

When disease incidence was assessed at corn growth stage V5 (fifth-leaf stage) in 2001 prior to the time that foliar insecticides of Warrior® had been applied, incidence of Stewart's disease was 3.8 % (treatment 11) at Crawfordsville on DOY 163 and 1.3 % (treatment 9) at Johnston on DOY 168 (Tables 7 and 8). By comparison, disease incidence at the V5 growth stage in the nontreated control plots were significantly higher ($P \leq 0.05$) at Crawfordsville (16.3 %) and Johnston (12.5 %) (Tables 7 and 8). Final disease incidence in the nontreated control plots on DOY 247 was 34.5 % at Crawfordsville and 28.8 % on DOY 245 at Johnston. Although the final disease incidence in plots treated with seed and/or foliar insecticides were lower than the nontreated controls at both locations, there were no significant differences in final disease incidence among treatments at both Crawfordsville and Johnston in 2001. The final incidence of Stewart's disease across all treatments was higher at Crawfordsville compared to Johnston in 2001 (Tables 7 and 8).

When standardized area under the disease progress curves (STD AUDPC) were analyzed, STD AUDPC values for treatments at Crawfordsville in 2001 showed that Cruiser® combined with three foliar sprays of Warrior® applied using the corn growth stage method (12.2) and Cruiser® with Warrior® applied using the degree day model (12.4) were significantly lower ($P \leq 0.05$) compared to the nontreated control treatment (29.6) (Table 7). Treatments in which Warrior® was applied in addition to Cruiser® or Gaucho® seed treatments were not significantly different from treatments using Cruiser® or Gaucho® alone based on STD AUDPC values. No significant differences in STD AUDPC values among treatments were detected at Johnston in 2001.

Disease assessments at growth stage V5 in 2002 indicated that treatments using Gaucho® seed treatment alone or in combination with foliar application of Warrior® resulted in the lowest incidence of Stewart's disease on DOY 155 at Crawfordsville (2.0 %), and on DOY 154 at Johnston (4.0 %) (Tables 9 and 10). These results were significantly ($P \leq 0.05$) different when compared to the nontreated control treatment at Crawfordsville (13.0 % on DOY 155), however, they were not significantly different from the nontreated control treatment at Johnston (8.0 % on DOY 154) (Tables 9 and 10). In 2002, the final incidence of Stewart's disease was significantly higher in the nontreated control plots at Crawfordsville (36.0% on DOY 232) compared to all other treatments (Table 9). At Johnston, final disease incidence in the nontreated control (32.0% on DOY 231) was significantly higher than for all other treatments, except Warrior® at V5 (treatment 4), Warrior® using the DD-model (treatment 6), and Cruiser® plus Warrior® using the threshold method (treatment 11) (Tables 9 and 10).

Final disease incidence on DOY 232 in 2002 at Crawfordsville, IA was lowest in the Gaucho® treatment plus two applications of Warrior® applied according to the degree day model (12.0 %), and in the Cruiser® treatment plus three foliar sprays of Warrior® applied at growth stages V5, VT, and R3 (12.0 %), however, these two treatments were not statistically different from using Gaucho® or Cruiser® alone. Cruiser® plus foliar sprays of Warrior®

provided disease control that was statistically equal to Gaucho® plus foliar sprays of Warrior® insecticide. Final disease incidence at Johnston, IA on DOY 231 was lowest in the Gaucho® plus Warrior® sprays applied at the V5, VT, and R3 growth states (14.0 %), the Gaucho® plus Warrior® treatment applied twice using a beetle threshold model (14.0 %), and the Gaucho® plus Warrior® treatment applied twice using a degree-day model (14.0 %), however, these treatments were not statistically different from using Gaucho® or Cruiser® alone. Cruiser® plus Warrior® applied using the three different timing methods resulted in final disease incidence levels that were somewhat higher (16 - 23 % final incidence), but statistically equivalent to, Gaucho® plus Warrior® using any of the three timing methods.

Analysis of standardized area under the disease progress curves (STD AUDPC) at Crawfordsville showed that all treatments provided disease control that was significantly better than the nontreated control (24.4), except for Warrior® alone applied once at the V5 growth stage (17.4, Treatment 4), and the single application of Warrior® using the degree day model (17.3, Treatment 6). At Johnston, STD AUDPC values were highest for the nontreated control treatment (20.9), while Gaucho® and Cruiser® applied alone or in combination with Warrior® foliar applications resulted in significantly lower STD AUDPC values. The application of Warrior® foliar sprays without Gaucho® or Cruiser® had STD AUDPC values that were not significantly different from the nontreated control (TRT 4, 17.8; TRT 5, 17.9; TRT 6, 16.5). The Cruiser® plus Warrior® treatment using the threshold model (TRT 11, 14.0) was also not significantly different from the nontreated control.

Based on the analysis of STD AUDPC values, there were no significant differences among the three methods used to time foliar applications of Warrior® for all location/ years. Combinations of Gaucho® or Cruiser® plus Warrior® were not statistically better than

Gaucho® or Cruiser® used alone, except in 2002 at Johnston where STD AUDPC values indicated the Gaucho® plus foliar sprays of Warrior® applied at V5, VT, and R3 (Treatment 7) was significantly ($P \leq 0.05$) better than either seed treatment used alone.

Disease incidence progress curves for 2001 and 2002 at Crawfordsville and Johnston, IA in 2001(Fig. 4) and 2002 (Fig. 5) showed that Stewart's disease incidence over time was highest in the nontreated control plots, where no seed or foliar insecticides had been applied. To obtain a linear relationship between transformed disease incidence with respect to time, five disease progress models were evaluated. Based upon model evaluation criteria, the logistic model best explained the change in transformed disease incidence with respect to time for all four locations-years (Tables 11, 12, 13 and 14). At Johnston in 2001, the linear model represented the data equally as well as the logistic model, but the logistic model was chosen to maintain consistency, thereby facilitating comparisons among treatments, location and years using the same model. The *F*-statistics for models ranged from 24.40 to 351.28 and all were highly significant at $P \leq 0.0001$, indicating that there was a strong linear relationship between the change in logit Stewart's disease incidence with respect to time (Tables 11, 12, 13 and 14). The coefficients of determination (R^2) values ranged from 0.83 to 0.99, indicating that time explained 83 to 99 % of the variation in the change in logit Stewart's disease incidence with respect to time. Furthermore, the standard errors of the estimate for y were very low, ranging from 0.0552 to 0.2373, indicating that time could be used to accurately predict the increase in logit Stewart's disease incidence. The rate of disease progress among treatments was slow at both locations during both years, ranging from 0.011 to 0.0220 logits per day. The transformed regression lines showed that there was little difference in the rate of logit disease incidence with respect to time among treatments, compared to the nontreated control for both locations in 2001 (Fig. 6, Tables 11 and 12). In 2002, the rate of disease progress was fastest in the nontreated control (treatment 1) (Fig. 7, Tables 13 and 14). Treatments 5 and 10 (Warrior® alone using threshold and Cruiser® plus Warrior® at V5, VT and R3) had the slowest rate (Tables 13 and 14).

Corn flea beetle assessments

Corn flea beetle populations did not differ among treatments at both locations in 2001. The action threshold for corn flea beetles was never reached in 2001, and therefore, no foliar insecticide applications of Warrior® were applied using this timing method in 2001 (Treatments 5, 8, and 11). Corn flea beetle populations were sparse throughout the growing season in 2001, and therefore, it was not possible to perform any data analysis on corn flea beetle population densities. Late in the 2001 season after the corn plants had begun to senesce, corn flea beetle populations began to increase in grassy areas that bordered corn fields at Crawfordsville.

In 2002 at Crawfordsville, the number of corn flea beetles sampled with respect to time for treatment 1 is shown in Fig. 9A and depicts the seasonal population dynamics of *C. pulicaria* in the nontreated control. The average number of corn flea beetles sampled in the nontreated control were plotted against date of sampling (day of year) along with the average number of corn flea beetles captured for treatments grouped according to one of the three timing methods used to trigger foliar insecticide applications (Fig. 9 A-D). In 2002, the first corn flea beetle captured by sweep netting in the grassy areas bordering corn fields (prior to corn emergence) was recorded on 11 April at Crawfordsville and on 15 April at Johnston. At Crawfordsville, on DOY 218 (6 August), when the mean numbers of beetles in the nontreated control was highest (6.5), the treatment means for growth stage, threshold, and degree day timing methods ranged from 0.5 to 6.75, 0.25 to 1.25, and 0 to 1.25 corn flea beetles per 10 sweeps, respectively (Fig. 9 A-D). At Johnston, corn flea beetle populations were low throughout much of the growing season and populations only began to recover after corn had senesced (Fig. 8 A-D). The insecticide treatments did not delay corn flea beetle population peaks (Fig. 8 A-D and Fig. 9 A-D).

At Crawfordsville in 2002, cumulative corn flea beetle progress curves showed that corn flea beetle populations were low early in the season in all treatments until approximately DOY 205 (Fig. 10A-D), and final cumulative corn flea beetle populations were highest in the Cruiser® seed treatment (Treatment 3), compared to all other treatments (Fig. 10A). Cruiser® plus Warrior® using the degree day model had the lowest cumulative corn flea beetle populations (Fig. 10D) compared to all other treatments. Cumulative corn flea beetle populations at Johnston did not reveal any clear effects of the insecticide treatments on reducing cumulative corn flea beetle population densities (Fig. 11 A-D). However, at Crawfordsville, the cumulative number of corn flea beetles in plots that were sprayed once with Warrior® (treatment 4) or never sprayed with Warrior® (treatments 1, 2, and 3) ranged from 54 to 79 beetles (Fig. 10 A and B). Plots receiving more than one application of Warrior® (treatments 5 to 12) had lower cumulative numbers of corn flea beetles, ranging from 13 to 32 beetles (Fig. 10 B-D).

The standardized area under the cumulative beetle progress curves (STD AUCBPC) at Crawfordsville indicated that STD AUCPBC values for the treatments using the three timing methods to schedule sprays of Warrior® were significantly different ($P \leq 0.05$) from the nontreated control, ranging from 40.54 to 93.07. Cruiser® or Warrior® used alone (treatments 3 and 4), had significantly higher STD AUCPBC values than any of the treatments using Cruiser® or Gaucho® plus Warrior® (Table 15). Cruiser® used alone had the highest STD AUCPBC value (233.28) compared to all other treatments. At Johnston, there were no significant differences among treatments based upon STD AUCBPC values (Table 15).

Yield and seed quality

In 2001, there were no significant differences in corn yields among treatments at Crawfordsville (Table 16). However, there were significant treatment effects on corn yields at Johnston (Table 17). At Johnston, the highest yield was achieved by treatment 10 (5611.9 kg/ha) and the lowest yields were obtained from the plots that received only the Cruiser® seed treatment (4801.7 kg/ha). There were only small differences in treatment effects on corn protein and oil at Crawfordsville (Table 16). There were no differences in percent protein or oil content for the harvested grain at Johnston (Table 17). Regression of final Stewart's disease incidence on yield at both locations indicated no significant linear relationship between incidence and yield in 2001 (Fig. 9).

In 2002, there were no significant differences in yield, protein, or oil among treatment at Crawfordsville and Johnston (Tables 18 and 19); except for percent oil content at Crawfordsville that revealed some significant differences with treatment 8 resulting in the highest oil content and treatment 7 resulting in the lowest oil content (Table 18). Regression of final Stewart's disease incidence on yield at Johnston in 2002 showed no linear relationship (Fig. 12 A-B). At Crawfordsville, however, there was a significant linear relationship in 2002 between final Stewart's disease incidence and yield ($P \leq 0.02$, $R^2 = 0.42$) (Fig. 13B).

Table 5. The effects of seed insecticides on corn plant height and stand as affected by Stewart's disease caused by *P. stewartii* on inbred A632 Ht block plots at Crawfordsville and Johnston, Iowa 2001.

Treatment	**Crawfordsville**		**Johnston**	
	Plant height (cm)	**Plant stand** [z]	**Plant height (cm)**	**Plant stand** [z]
1- Nontreated control (no insecticide)	20.6 ab[x]	42.5 ab	53.7 ab	52.0 abc
2- Gaucho® seed treatment	22.9 a	42.9 ab	54.8 a	52.1 abc
3- Cruiser® seed treatment	22.0 ab	45.1 a	54.6 ab	53.3 ab
4- Warrior® at V5 (1)[y]	20.1 ab	39.9 b	54.3 ab	51.6 abc
5- Warrior® using threshold (2)	19.1 b	41.4 ab	53.5 ab	54.0 a
6- Warrior® using DD-model (2)	19.3 b	39.1 b	53.8 ab	52.9 ab
7- Gaucho® + Warrior® at V5, VT, R3	21.1 ab	40.8 b	52.7 ab	48.1 c
8- Gaucho® + Warrior® using threshold (2)	21.3 ab	40.0 b	51.8 b	49.4 bc
9- Gaucho® + Warrior® using DD-model (2)	20.6 ab	40.3 b	55.1 a	50.6 abc
10- Cruiser® + Warrior® at V5, VT, R3	19.5 b	40.6 b	53.4 ab	52.0 abc
11- Cruiser® + Warrior® using threshold (2)	21.5 ab	41.5 ab	53.7 ab	52.3 abc
12- Cruiser® + Warrior® using DD-model (2)	21.5 ab	41.4 ab	53.2 ab	50.8 abc

[x] Means with the same letters are not significantly different ($P \leq 0.05$) based on the Waller Duncan K-ratio test.
[y] Number in ( ) indicates the number of foliar application of Warrior® insecticides.
[z] Average number of plants in two-15 m transects.

Table 6. The effects of seed insecticides on corn plant height and stand as affected by Stewart's disease caused by *P. stewartii* on inbred A632 Ht block plots at Crawfordsville and Johnston, Iowa 2002.

	Crawfordsville		Johnston	
Treatment	**Plant height (cm)**	**Plant stand** [z]	**Plant height (cm)**	**Plant stand** [z]
1- Nontreated control (no insecticide)	21.9 a[x]	38.5 ab	19.0 ab	34.9 e
2- Gaucho® seed treatment	20.7 a	28.1 d	19.0 ab	36.4 cde
3- Cruiser® seed treatment	20.8 a	36.4abc	19.1 ab	41.6 bcd
4- Warrior® at V5 (1)[y]	21.7 a	39.5a	18.6 ab	36.5 cde
5- Warrior® using threshold (2)	22.2 a	39.8a	17.8 b	35.3 de
6- Warrior® using DD-model (2)	21.6 a	38.6 ab	17.7 b	36.1 cde
7- Gaucho® + Warrior® at V5, VT, R3 (3)	22.1 a	37.4 ab	18.2 ab	42.6 bc
8- Gaucho® + Warrior® using threshold (2)	20.8 a	34.1 bc	18.9 ab	41.9 bcd
9- Gaucho® + Warrior® using DD-model (2)	20.8 a	32.3 cd	19.2 ab	42.3 bc
10- Cruiser® + Warrior® at V5, VT, R3 (3)	20.4 a	37.9 ab	19.5 ab	51.6a
11- Cruiser® + Warrior® using threshold (2)	20.9 a	36.9 ab	18.7 ab	43.3 b
12- Cruiser® + Warrior® using DD-model (2)	21.0 a	32.3 cd	20.2 a	41.8 bcd

[x] Means with the same letters are not significantly different ($P \leq 0.05$) based on the Waller Duncan K-ratio test.
[y] Number in ( ) indicates the number of foliar application of Warrior® insecticides.
[z] Average number of plants in two-15 m transects.

Table 7. The effects of seed and foliar insecticides on the incidence of Stewart's disease of corn on the inbred line A634XCM105 for seven assessment dates and the standardized area under the disease progress curve (STD AUDPC) at Crawfordsville, Iowa in 2001.

Treatment (number of foliar sprays)	Incidence (%)							
	DOY 163 (12-June)	**DOY 198 (17-July)**	**DOY 205 (24-July)**	**DOY 211 (30-July)**	**DOY 219 (07-Aug)**	**DOY 233 (21-Aug)**	**DOY 247 (04-Sep)**	**STD[y] AUDPC**
1- Nontreated control (no insecticide)	16.3 a[x]	27.5 a	29.8 a	33.3 a	33.5 a	34.3 a	34.5 a	29.6 a
2- Gaucho® seed treatment	6.3 bc	22.5 a	26.3 a	28.8 a	30.0 a	31.3 a	31.3 a	21.3 a b
3- Cruiser® seed treatment	11.3 ab	25.0 a	27.5 a	28.8 a	31.0 a	31.0 a	31.3 a	22.2 a b
4- Warrior® at V5 (1)[z]	10.0 b	17.5 a	21.3 a	23.8 a	25.0 a	26.3 a	26.3 a	17.8 a b
5- Warrior® using threshold	7.5 bc	21.3 a	26.3 a	30.0 a	31.3 a	31.3 a	31.3 a	21.1 a b
6- Warrior® using DD-model (2)	10.0 b	15.0 a	17.5 a	21.3 a	22.5 a	25.0 a	26.3 a	15.8 a b
7- Gaucho® + Warrior® at V5, VT, R3 (3)	8.8 bc	20.0 a	23.8 a	25.0 a	27.5 a	27.5 a	27.5 a	20.3 a b
8- Gaucho® + Warrior® using threshold	7.5 bc	20.0 a	25.0 a	27.5 a	28.8 a	30.0 a	30.0 a	19.5 a b
9- Gaucho® + Warrior® using DD-model (2)	8.8 bc	11.3 a	15.0 a	22.5 a	25.0 a	26.3 a	26.3 a	15.2 a b
10- Cruiser® + Warrior® at V5, VT, R3 (3)	6.3 bc	12.5 a	15.0 a	16.3 a	18.8 a	18.8 a	20.0 a	12.2 b
11- Cruiser® + Warrior® using threshold	3.8 c	17.5 a	21.3 a	25.0 a	27.5 a	27.5 a	27.5 a	16.9 a b
12- Cruiser® + Warrior® using DD-model (2)	6.3 bc	10.0 a	12.5 a	18.8 a	22.5 a	22.5 a	23.8 a	12.4 b

[x] Means with the same letters within columns are not significantly different ($P \leq 0.05$) based on the Waller Duncan K-ratio test.
[y] STD AUDPC is calculated by dividing AUDPC values ($AUDPC = \sum (y_i + y_{i+1})/2)(t_n - t_1)$) by the length of the epidemics.
[z] Number in ( ) indicates the number of foliar application of Warrior® insecticide which began on DOY 171 in some treatments.

Table 8. The effects of seed and foliar insecticides on the incidence of Stewart's disease of corn on the inbred line A634XCM105 for seven assessment dates and the standardized area under disease progress curve (STD AUDPC) at Johnston, Iowa in 2001.

	Incidence (%)							
Treatment (number of foliar sprays)	**DOY 168 (17-June)**	**DOY 207 (26-July)**	**DOY 214 (02-Aug)**	**DOY 221 (09-Aug)**	**DOY 233 (21-Aug)**	**DOY 239 (27-Aug)**	**DOY 245 (02-Sep)**	**STD[y] AUDPC**
1- Nontreated control (no insecticide)	12.5 a[x]	18.8 a	19.5 a	24.5 a	26.8 a	28.5 a	28.8 a	21.6 a
2- Gaucho® seed treatment	3.8 bcd	8.8 a	11.3 a	15.0 a	16.3 a	16.3 a	16.3 a	11.4 a
3- Cruiser® seed treatment	8.8 ab	13.8 a	17.5 a	20.0 a	21.3 a	21.3 a	21.3 a	16.6 a
4- Warrior® at V5 (1)[z]	7.5 abc	12.5 a	15.0 a	17.5 a	18.8 a	20.0 a	20.0 a	14.9 a
5- Warrior® using threshold	7.5 abc	13.8 a	16.3 a	21.3 a	23.8 a	25.0 a	26.3 a	17.2 a
6- Warrior® using DD-model (2)	6.3 bcd	11.3 a	14.5 a	15.3 a	16.8 a	17.0 a	17.8 a	12.8 a
7- Gaucho® + Warrior® at V5, VT, R3 (3)	2.5 cd	13.8 a	17.5 a	20.0 a	20.0 a	20.0 a	20.0 a	15.1 a
8- Gaucho® + Warrior® using threshold	6.3 bcd	11.5 a	11.8 a	13.5 a	15.3 a	18.3 a	18.8 a	13.3 a
9- Gaucho® + Warrior® using DD-model (2)	1.3 d	11.3 a	15.0 a	17.5 a	18.8 a	18.8 a	18.8 a	12.9 a
10- Cruiser® + Warrior® at V5, VT, R3 (3)	8.8 ab	13.8 a	16.3 a	17.5 a	18.8 a	18.8 a	18.8 a	15.0 a
11- Cruiser® + Warrior® using threshold	6.3 bcd	10.0 a	12.5 a	15.3 a	16.5 a	16.5 a	17.3 a	12.3 a
12- Cruiser® + Warrior® using DD-model (2)	2.5 cd	9.0 a	13.8 a	16.3 a	17.5 a	18.3 a	18.8 a	12.7 a

[x] Means with the same letters within columns are not significantly different ($P \leq 0.05$) based on the Waller Duncan K-ratio test.
[y] STD AUDPC is calculated by dividing AUDPC values (AUDPC = $\sum (y_i + y_{i+1})/2)(t_n - t_1))$ by the length of the epidemics.
[z] Number in ( ) indicates the number of foliar application of Warrior® insecticides which began on DOY 172 in some treatments.

Table 9. The effects of seed and foliar insecticides on the incidence of Stewart's disease of corn on the inbred line A632 Ht Block for six assessment dates and the standardized area under disease progress curve (STD AUDPC) at Crawfordsville, Iowa in 2002.

	Incidence (%)						
Treatment (number of foliar sprays)	**DOY 155 (04-June)**	**DOY 176 (25-June)**	**DOY 199 (18-July)**	**DOY 214 (02-Aug)**	**DOY 225 (13-Aug)**	**DOY 232 (20-Aug)**	**STD[y] AUDPC**
1- Nontreated control (no insecticide)	13.0 a[x]	18.0 a	27.0 a	30.0 a	35.0 a	36.0 a	24.4 a
2- Gaucho® seed treatment	4.0 ab	8.0 ab	13.0 bc	14.0 b	17.0 bcd	18.0 bcd	11.2 b
3- Cruiser® seed treatment	4.0 ab	8.0 ab	10.0 bc	13.0 b	15.0 bcd	15.0 bcd	9.9 b
4- Warrior® at V5 (1)[z]	10.0 ab	15.0 ab	19.0 abc	20.0 ab	23.0 b	23.0 b	17.4 ab
5- Warrior® using threshold (2)	10.0 ab	12.0 ab	14.0 bc	15.0 b	16.0 bcd	16.0 bcd	13.5 b
6- Warrior® using DD-model (2)	9.0 ab	15.0 ab	20.0 ab	20.0 ab	21.0 bc	22.0 bc	17.3 ab
7- Gaucho® + Warrior® at V5, VT, R3 (3)	2.0 b	8.0 ab	14.0 bc	17.0 b	18.0 bcd	19.0 bcd	11.9 b
8- Gaucho® + Warrior® using threshold (2)	2.0 b	5.0 b	7.0 c	11.0 b	13.0 cd	13.0 cd	7.4 b
9- Gaucho® + Warrior® using DD-model (2)	2.0 b	5.0 b	11.0 bc	11.0 b	12.0 cd	12.0 d	8.2 b
10- Cruiser® + Warrior® at V5, VT, R3 (3)	5.0 ab	9.0 ab	9.0 bc	11.0 b	11.0 d	12.0 d	9.2 b
11- Cruiser® + Warrior® using threshold (2)	5.0 ab	7.0 ab	10.0 bc	11.0 b	12.0 cd	13.0 cd	8.8 b
12- Cruiser® + Warrior® using DD-model (2)	4.0 ab	8.0 ab	12.0 bc	14.0 b	15.0 bcd	15.0 bcd	10.6 b

[x] Means with the same letters within columns are not significantly different ($P \leq 0.05$) based on the Waller Duncan K-ratio test.
[y] STD AUDPC is calculated by dividing AUDPC values (AUDPC = $\sum (y_i + y_{i+1})/2)(t_n - t_1)$) by the length of the epidemics.
[z] Number in ( ) indicates the number of foliar application of Warrior® insecticides which began on DOY 157 in some treatments.

Table 10. The effects of seed and foliar insecticides on the incidence of Stewart's disease of corn on the inbred line A632 Ht Block for six Assessment dates and the standardized area under disease progress curve (STD AUDPC) at Johnston, Iowa in 2002.

	Incidence (%)						
Treatment (number of foliar sprays)	**DOY 154 (03-June)**	**DOY 175 (24-June)**	**DOY 188 (08-July)**	**DOY 203 (22-July)**	**DOY 219 (07-Aug)**	**DOY 231 (19-Aug)**	**STD[y] AUDPC**
1- Nontreated control (no insecticide)	8.0 a[x]	15.0 a	23.0 a	28.0 a	31.0 a	32.0 a	20.9 a
2- Gaucho® seed treatment	4.0 a	9.0 ab	12.0 cd	18.0 bcd	20.0 bc	21.0 bc	12.4 cd
3- Cruiser® seed treatment	3.0 a	8.0 ab	14.0 bcd	19.0 abcd	21.0 bc	21.0 bc	12.8 bcd
4- Warrior® at V5 (1)[z]	7.0 a	15.0 a	20.0 ab	23.0 ab	23.0 ab	24.0 ab	17.8 ab
5- Warrior® using threshold (2)	7.0 a	15.0 a	20.0 ab	20.0 abcd	21.0 bc	22.0 bc	17.0 abc
6- Warrior® using DD-model (2)	9.0 a	13.0 ab	17.0 abc	21.0 abc	23.0 ab	24.0 ab	16.5 abcd
7- Gaucho® + Warrior® at V5, VT, R3 (3)	2.0 a	6.0 b	8.0 d	11.0 d	13.0 c	14.0 c	8.0 e
8- Gaucho® + Warrior® using threshold (2)	2.0 a	8.0 ab	13.0 bcd	13.0 cd	13.0 c	14.0 c	10.1 cde
9- Gaucho® + Warrior® using DD-model (2)	2.0 a	7.0 b	11.0 cd	14.0 bcd	14.0 bc	14.0 c	9.6 de
10- Cruiser® + Warrior® at V5, VT, R3 (3)	5.0 a	9.0 ab	13.0 bcd	16.0 bcd	16.0 bc	17.0 bc	11.8 bcde
11- Cruiser® + Warrior® using threshold (2)	5.0 a	10.0 ab	14.0 bcd	20.0 abcd	22.0 abc	23.0 abc	14.0 abcde
12- Cruiser® + Warrior® using DD-model (2)	4.0 a	8.0 ab	12.0 cd	14.0 bcd	15.0 bc	16.0 bc	10.6 cde

[x] Means with the same letters within columns are not significantly different ($P \leq 0.05$) based on the Waller Duncan K-ratio test.
[y] STD AUDPC is calculated by dividing AUDPC values (AUDPC = $\sum (y_i + y_{i+1})/2)(t_n - t_1)$) by the length of the epidemics.
[z] Number in ( ) indicates the number of foliar application of Warrior® insecticides which began on DOY 158 in some treatments.

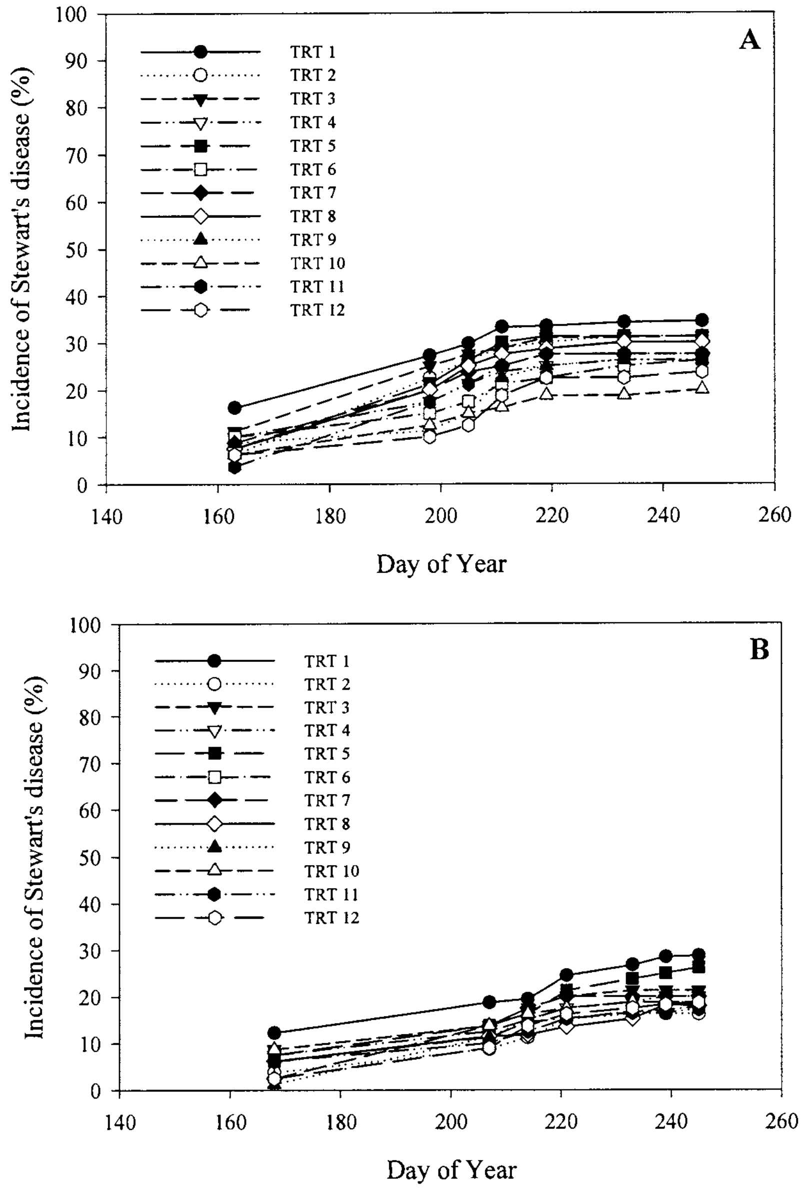
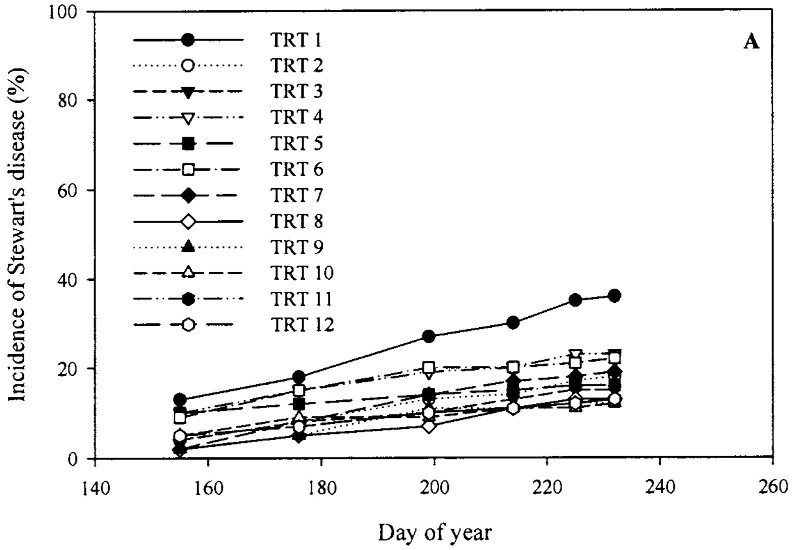


Figure 4. Disease incidence progress curves for Stewart's disease of corn as affected by insecticide treatments applied to inbred line A634XCM105 at (**A**) Crawfordsville and (**B**) Johnston, IA during 2001.



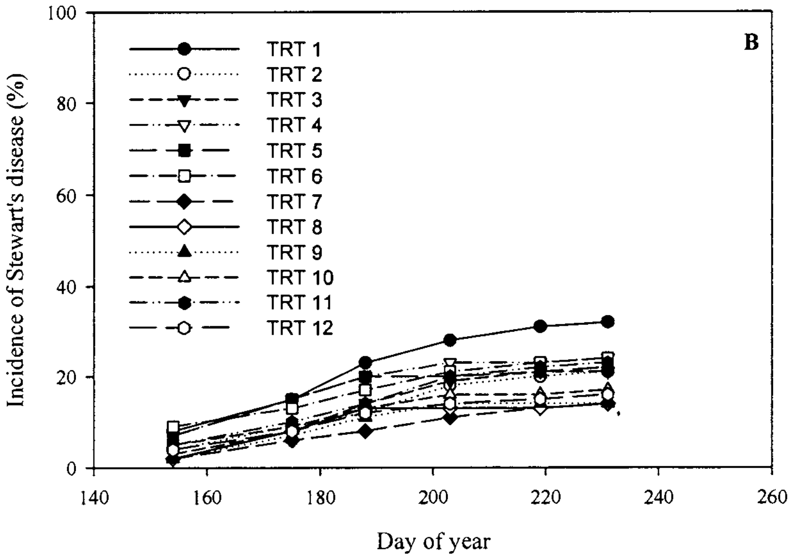


Figure 5. Disease incidence progress curves for Stewart's disease of corn as affected by insecticide treatments applied to inbred line A632 Ht Block at (**A**) Crawfordsville and (**B**) Johnston, IA during 2002.

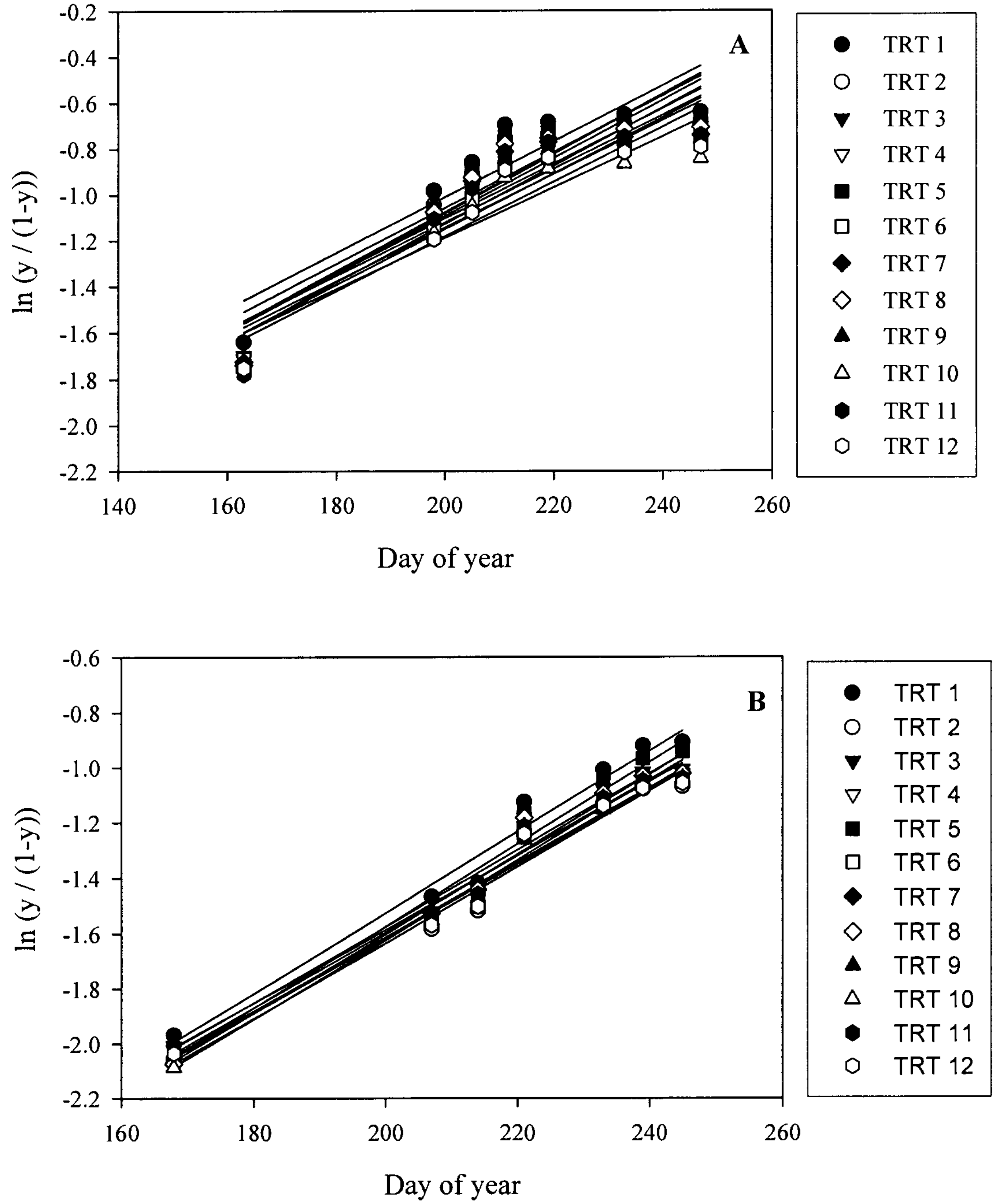


Figure 6. Linear regression lines using the logistic model ln (y / (1-y)) versus time to transform Stewart's disease incidence in corn inbred A632 Ht Block at (**A**) Crawfordsville and (**B**) Johnston, IA in 2001.

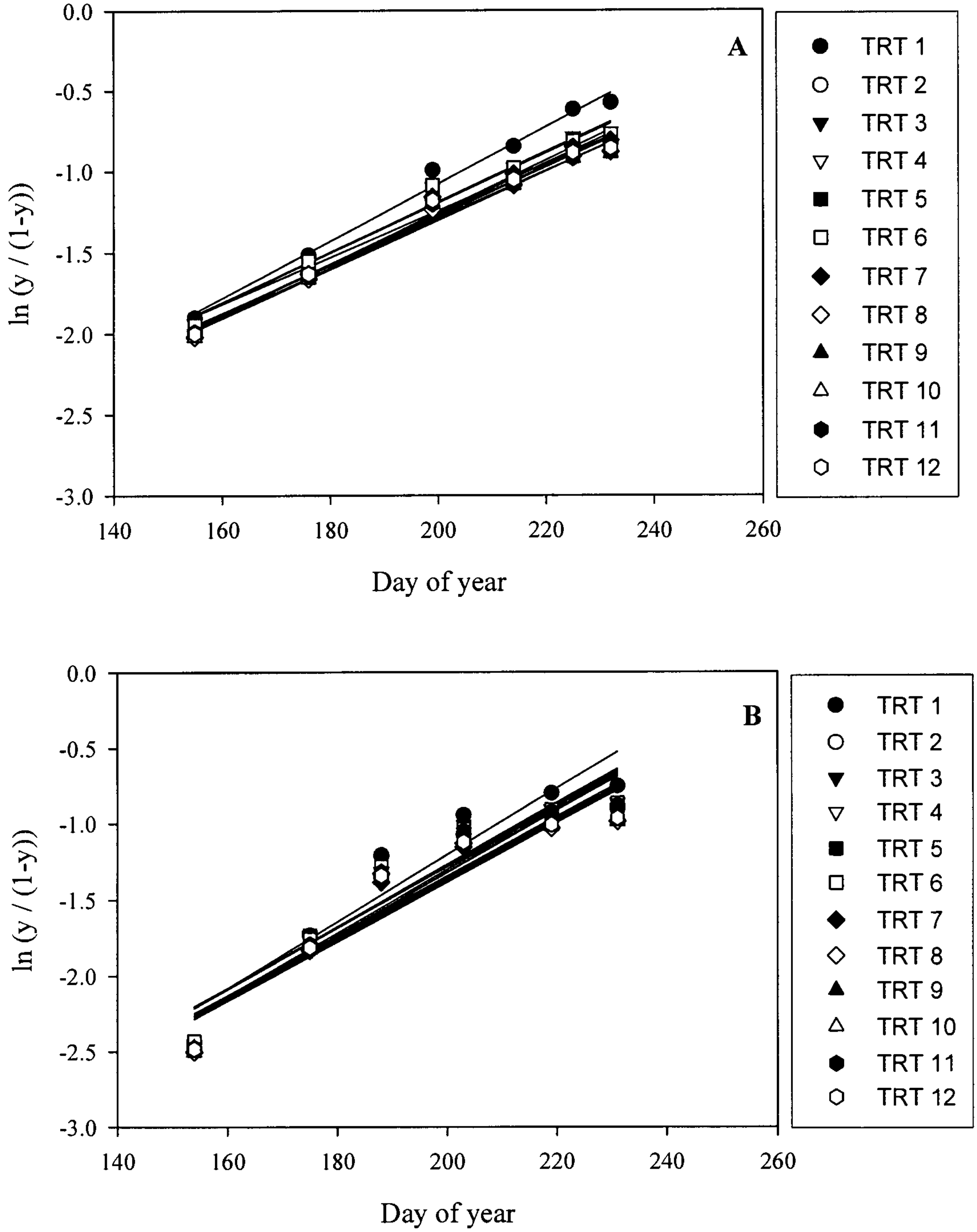


Figure 7. Linear regression lines using the logistic model ln (y / (1-y)) versus time to transform Stewart's disease incidence in corn inbred A632 Ht Block at (**A**) Crawfordsville and (**B**) Johnston, IA in 2002

Table 11. Logistic model parameters and statistics describing the change in logit disease incidence of Stewart's disease of corn with respect to time as affected by insecticide treatments on corn inbred A634XCM105 at Crawfordsville, IA during 2001.

	Crawfordsville 2001				
Treatment	*F-statistic*	Intercept	Slope	R^2	SEEy
1- Nontreated control (no insecticide)	25.26	-3.44	0.012 ± 0.0024	0.835	0.159
2- Gaucho® seed treatment	25.47	-3.66	0.013 ± 0.0026	0.836	0.169
3- Cruiser® seed treatment	24.40	-3.50	0.012 ± 0.0025	0.830	0.163
4- Warrior® at V5	27.97	-3.45	0.012 ± 0.0022	0.848	0.145
5- Warrior® using threshold	24.59	-3.64	0.013 ± 0.0026	0.831	0.171
6- Warrior® using DD-model	36.18	-3.47	0.012 ± 0.0024	0.879	0.128
7- Gaucho® + Warrior® at V5, VT, R3	25.25	-3.50	0.012 ± 0.0024	0.835	0.157
8- Gaucho® + Warrior® using threshold	26.63	-3.61	0.013 ± 0.0024	0.842	0.161
9- Gaucho® + Warrior® using DD-model	34.71	-3.58	0.012 ± 0.0021	0.874	0.136
10- Cruiser® + Warrior® at V5, VT, R3	28.57	-3.40	0.011 ± 0.0021	0.851	0.137
11- Cruiser® + Warrior® using threshold	26.29	-3.67	0.013 ± 0.0025	0.840	0.163
12- Cruiser® + Warrior® using DD-model	35.36	-3.56	0.012 ± 0.0020	0.876	0.132

Table 12. Logistic model parameters and statistics describing the change in logit disease incidence of Stewart's disease of corn with respect to time as affected by insecticide treatments on corn inbred A634XCM105 at Johnston, IA during 2001.

	Johnston 2001				
Treatment	*F-statistic*	Intercept	Slope	R^2	SEEy
1- Nontreated control (no insecticide)	175.98	-4.46	0.015 ± 0.0011	0.972	0.070
2- Gaucho® seed treatment	168.38	-4.38	0.014 ± 0.0011	0.971	0.067
3- Cruiser® seed treatment	182.55	-4.35	0.014 ± 0.0010	0.973	0.065
4- Warrior® at V5	200.54	-4.35	0.014 ± 0.0010	0.976	0.062
5- Warrior® using threshold	181.57	-4.55	0.015 ± 0.0011	0.973	0.070
6- Warrior® using DD-model	236.65	-4.31	0.014 ± 0.0009	0.979	0.056
7- Gaucho® + Warrior® at V5, VT, R3	192.40	-4.49	0.014 ± 0.0010	0.975	0.066
8- Gaucho® + Warrior® using threshold	230.47	-4.33	0.014 ± 0.0009	0.979	0.057
9- Gaucho® + Warrior® using DD-model	204.51	-4.51	0.014 ± 0.0010	0.976	0.064
10- Cruiser® + Warrior® at V5, VT, R3	208.57	-4.26	0.013 ± 0.0009	0.977	0.059
11- Cruiser® + Warrior® using threshold	185.18	-4.31	0.014 ± 0.0010	0.974	0.063
12- Cruiser® + Warrior® using DD-model	204.51	-4.48	0.014 ± 0.0010	0.976	0.063

Table 13. Logistic model parameters and statistics describing the change in logit disease incidence of Stewart's disease of corn with respect to time as affected by insecticide treatments on corn inbred A632 Ht Block at Crawfordsville, IA during 2002.

	Crawfordsville 2002				
Treatment	*F-statistic*	Intercept	Slope	R^2	SEEy
1- Nontreated control (no insecticide)	351.28	-4.60	0.018 ± 0.0009	0.989	0.063
2- Gaucho® seed treatment	257.76	-4.36	0.016 ± 0.0010	0.985	0.064
3- Cruiser® seed treatment	286.90	-4.29	0.015 ± 0.0009	0.986	0.059
4- Warrior® at V5	231.66	-4.28	0.016 ± 0.0010	0.983	0.068
5- Warrior® using threshold	230.11	-4.12	0.014 ± 0.0009	0.983	0.063
6- Warrior® using DD-model	174.83	-4.27	0.015 ± 0.0012	0.978	0.077
7- Gaucho® + Warrior® at V5, VT, R3	233.12	-4.44	0.016 ± 0.0011	0.983	0.070
8- Gaucho® + Warrior® using threshold	332.77	-4.32	0.015 ± 0.0008	0.988	0.055
9- Gaucho® + Warrior® using DD-model	177.13	-4.29	0.015 ± 0.0011	0.978	0.075
10- Cruiser® + Warrior® at V5, VT, R3	244.48	-4.16	0.014 ± 0.0009	0.984	0.061
11- Cruiser® + Warrior® using threshold	245.21	-4.21	0.015 ± 0.0009	0.984	0.062
12- Cruiser® + Warrior® using DD-model	219.25	-4.29	0.015 ± 0.0010	0.982	0.068

Table 14. Logistic model parameters and statistics describing the change in logit disease incidence of Stewart's disease of corn with respect to time as affected by insecticide treatments on corn inbred A632 Ht Block at Johnston, IA during 2002.

	Johnston 2002				
Treatment	*F-statistic*	Intercept	Slope	R^2	SEEy
1- Nontreated control (no insecticide)	37.36	-5.60	0.022 ± 0.0036	0.903	0.229
2- Gaucho® seed treatment	37.54	-5.43	0.021 ± 0.0034	0.904	0.214
3- Cruiser® seed treatment	34.68	-5.47	0.021 ± 0.0035	0.897	0.225
4- Warrior® at V5	30.03	-5.35	0.020 ± 0.0037	0.882	0.237
5- Warrior® using threshold	28.82	-5.27	0.020 ± 0.0037	0.878	0.237
6- Warrior® using DD-model	36.20	-5.34	0.020 ± 0.0034	0.901	0.215
7- Gaucho® + Warrior® at V5, VT, R3	34.95	-5.30	0.020 ± 0.0033	0.897	0.211
8- Gaucho® + Warrior® using threshold	28.34	-5.25	0.019 ± 0.0036	0.876	0.232
9- Gaucho® + Warrior® using DD-model	30.15	-5.29	0.020 ± 0.0036	0.883	0.228
10- Cruiser® + Warrior® at V5, VT, R3	31.69	-5.27	0.020 ± 0.0035	0.889	0.221
11- Cruiser® + Warrior® using threshold	37.52	-5.45	0.021 ± 0.0034	0.904	0.216
12- Cruiser® + Warrior® using DD-model	32.30	-5.27	0.020 ± 0.0035	0.890	0.220

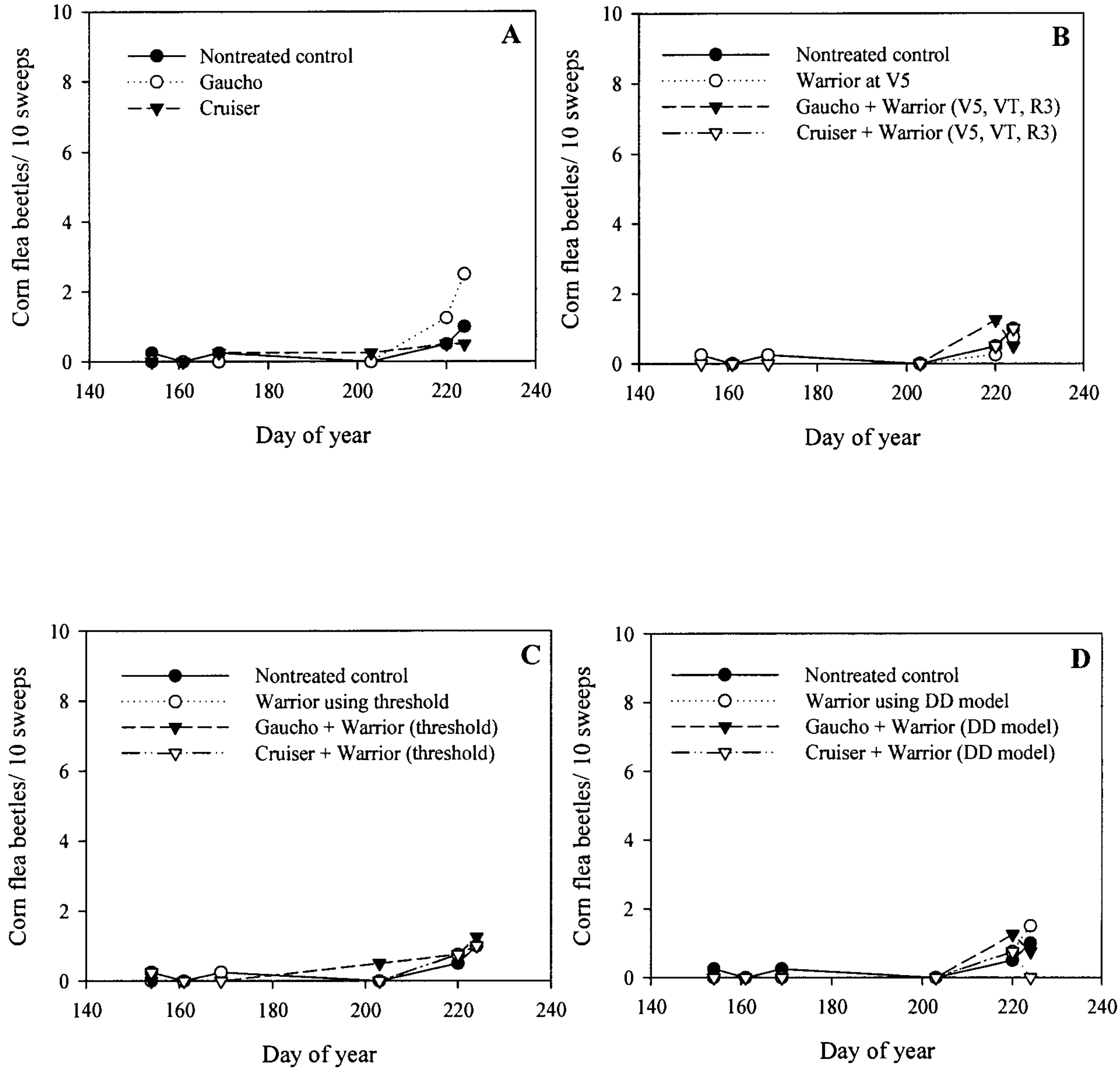


Figure 8. Mean number of corn flea beetles captured per 10 sweeps (6 m transect) at Johnston, IA during 2002, using different insecticide programs: (**A**) Nontreated control versus seed treatment alone, (**B**) Nontreated control versus Gaucho® or Cruiser® using corn growth stage to time Warrior® applications, (**C**) Nontreated control versus Gaucho® or Cruiser® plus Warrior® applied according to a corn flea beetle action threshold, and (**D**) Nontreated control versus Gaucho® or Cruiser® plus Warrior® applied according to a degree day model.

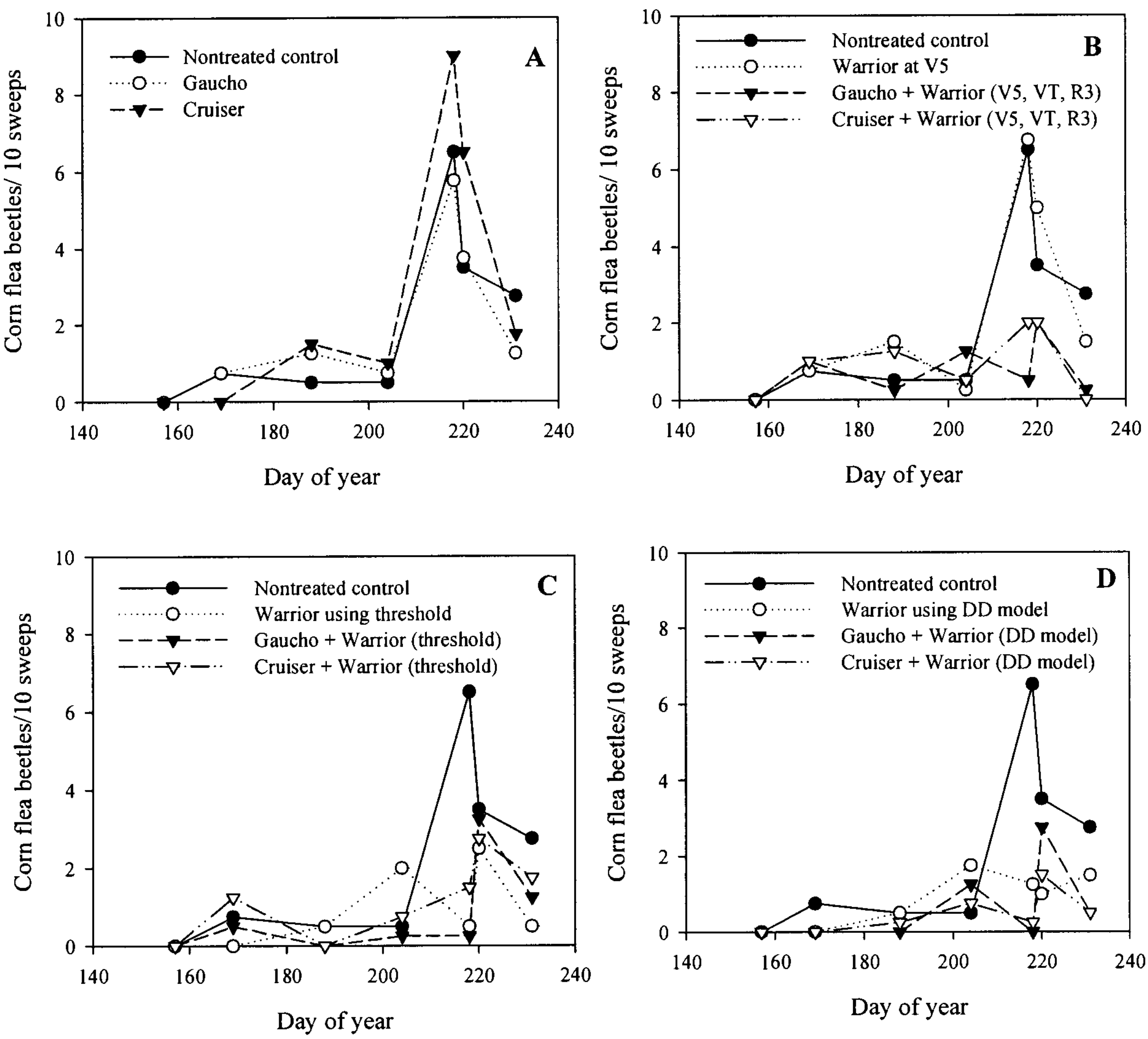


Figure 9. Mean number of corn flea beetles captured per 10 sweeps (6 m transect) at Crawfordsville, IA during 2002, using different insecticide programs: (**A**) Nontreated control versus seed treatment alone, (**B**) Nontreated control versus Gaucho® or Cruiser® using corn growth stage to time Warrior® applications, (**C**) Nontreated control versus Gaucho® or Cruiser® plus Warrior® applied according to a corn flea beetle action threshold, and (**D**) Nontreated control versus Gaucho® or Cruiser® plus Warrior® applied according to a degree day model.

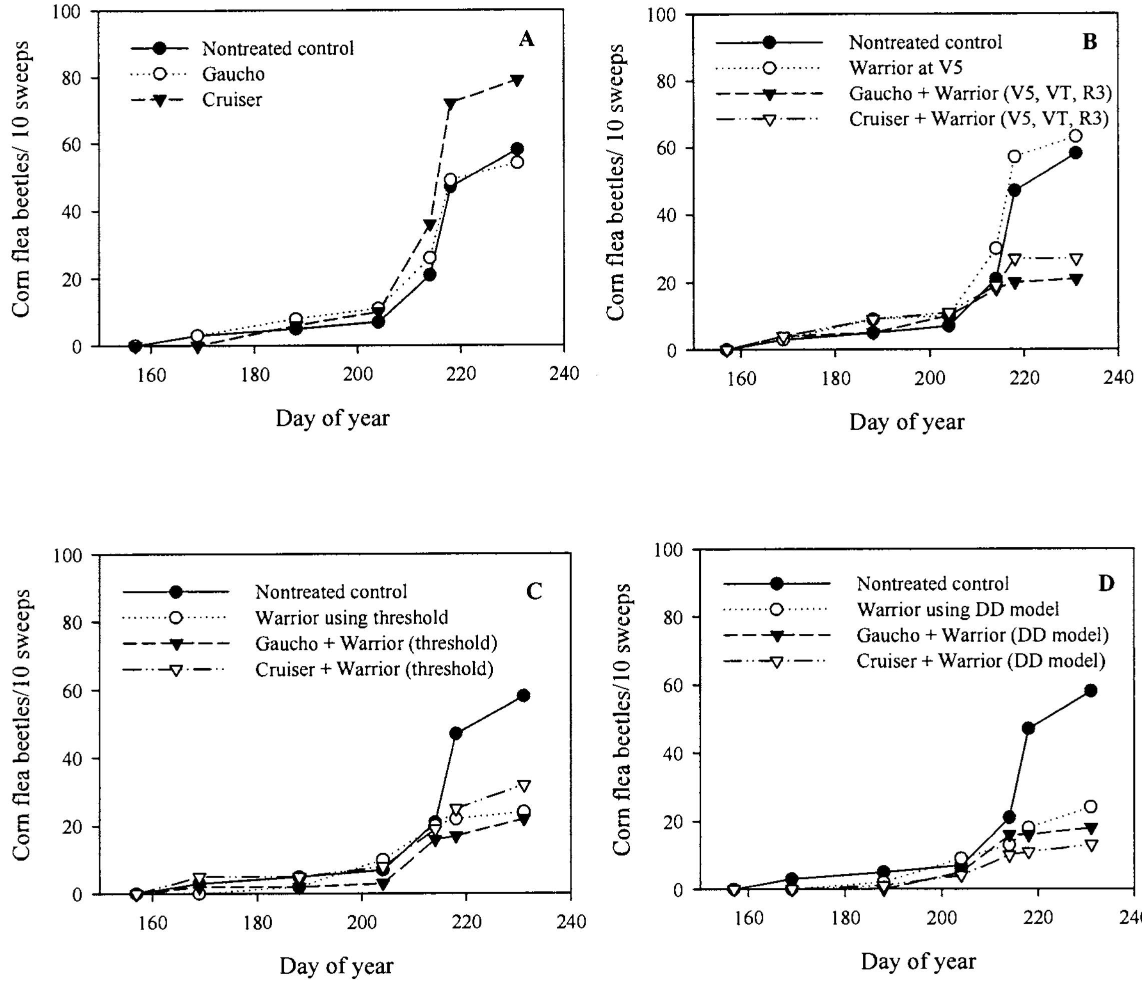


Figure 10. Cumulative number of corn flea beetles captured per 10 sweeps (6 m transect) at Crawfordsville, IA during 2002, using different insecticide programs: (**A**) Nontreated control versus seed treatments alone, (**B**) Nontreated control versus Gaucho® or Cruiser® using corn growth stage to time three applications of Warrior®, (**C**) Nontreated control versus Gaucho® or Cruiser® plus two applications of Warrior® applied according to a corn flea beetle action threshold, and (**D**) Nontreated control versus Gaucho® or Cruiser® plus two applications of Warrior® applied according to a degree day model.

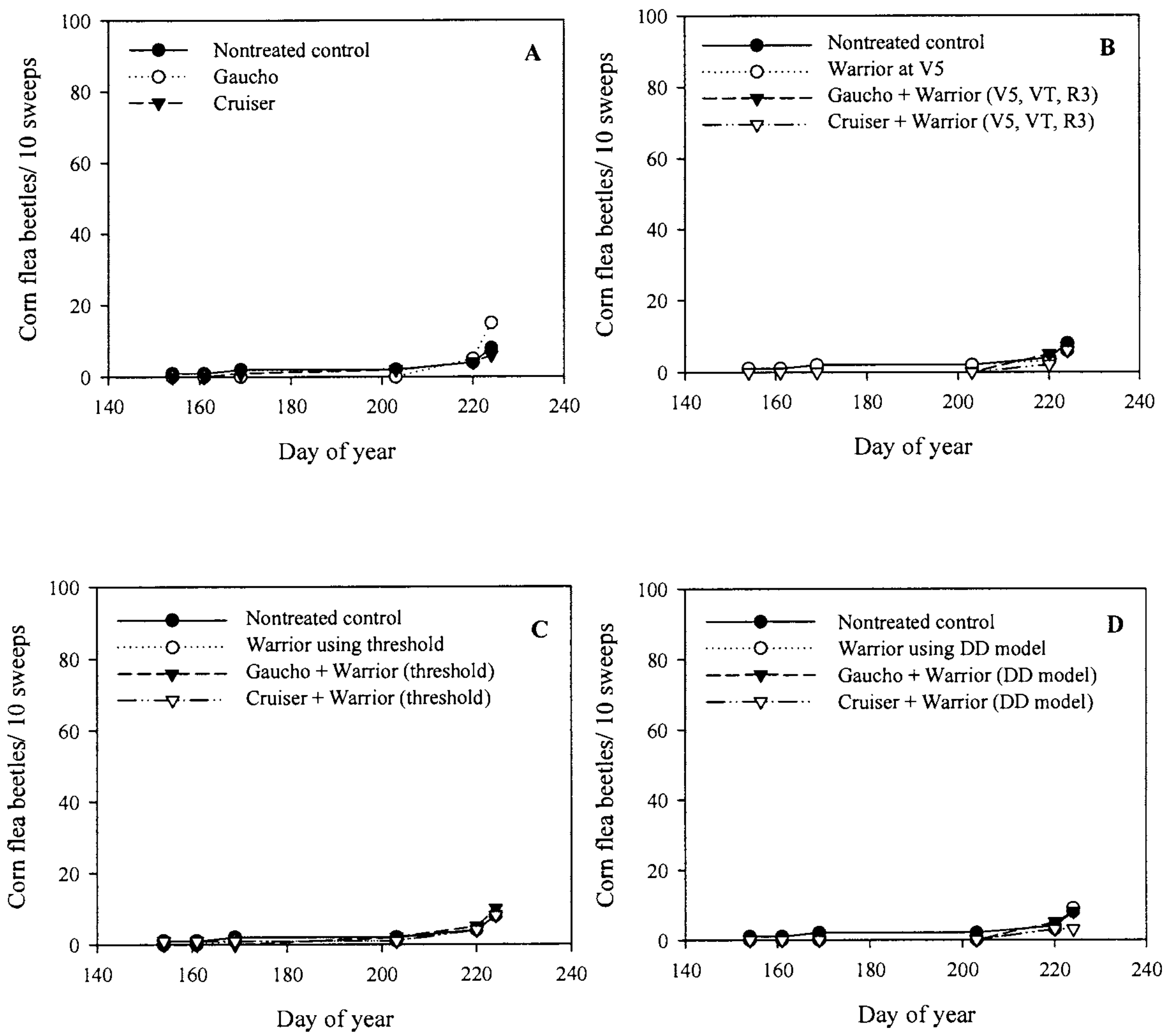


Figure 11. Cumulative number of corn flea beetles captured per 10 sweeps (6 m transect) at Johnston, IA during 2002, using different insecticide programs: (**A**) Nontreated control versus seed treatments alone, (**B**) Nontreated control versus Gaucho® or Cruiser® using corn growth stage to time three applications of Warrior®, (**C**) Nontreated control versus Gaucho® or Cruiser® plus two applications of Warrior® applied according to a corn flea beetle action threshold, and (**D**) Nontreated control versus Gaucho® or Cruiser® plus two applications of Warrior® applied according to a degree day model

Table 15. The effects of seed and foliar insecticide treatment programs on the standardized area under the cumulative beetle progress curves (STD AUCBPC) at Crawfordsville and Johnston, Iowa in 2002.

	STD AUCBPC	
Treatment	**Crawfordsville**	**Johnston**
1- Nontreated control (no insecticide)	162.16 b[x]	17.86 a
2- Gaucho® seed treatment	164.86 b	25.89 a
3- Cruiser® seed treatment	233.28 a	25.54 a
4- Warrior® at V5 (1)[y]	187.50 ab	13.39 a
5- Warrior® using threshold (2)	80.74 c	21.61 a
6- Warrior® using DD-model (2)	79.56 c	15.54 a
7- Gaucho® + Warrior® at V5, VT, R3 (3)	67.57 c	20.18 a
8- Gaucho® + Warrior® using threshold (2)	55.41 c	33.04 a
9- Gaucho® + Warrior® using DD-model (2)	52.36 c	20.89 a
10- Cruiser® + Warrior® at V5, VT, R3 (3)	93.07 c	10.36 a
11- Cruiser® + Warrior® using threshold (2)	87.84 c	14.11 a
12- Cruiser® + Warrior® using DD-model (2)	40.54 c	11.25 a

[x] Means with the same letters within columns are not significantly different ($P \leq 0.05$) based on the Waller Duncan K-ratio test.

[y] Number in ( ) indicates the number of foliar application of Warrior® insecticide.

Table 16. The effects of seed and foliar insecticide treatments on corn yield, protein and oil content as affected by Stewart's disease caused by *P. stewartii* on inbred A634XCM105 plots at Crawfordsville, Iowa, in 2001.

	Yield/Quality		
Treatment	**Yield (Kg/ha)**	**Protein (%)**	**Oil (%)**
1- Nontreated control (no insecticide)	3538.0 a[x]	9.55 a	3.17c
2- Gaucho® seed treatment	4140.0 a	9.50 ab	3.23 abc
3- Cruiser® seed treatment	4467.7 a	9.43 ab	3.25 bc
4- Warrior® at V5 (1)[y]	3793.9 a	9.45 ab	3.23 bc
5- Warrior® using threshold	3731.8 a	9.33 b	3.30 abc
6- Warrior® using DD-model (2)	3961.7 a	9.08 c	3.30 abc
7- Gaucho® + Warrior® at V5, VT, R3 (3)	4185.0 a	9.33 b	3.35 abc
8- Gaucho® + Warrior® using threshold	3976.4 a	9.53 ab	3.25 bc
9- Gaucho® + Warrior® using DD-model (2)	3564.0 a	9.53 ab	3.30 abc
10- Cruiser® + Warrior® at V5, VT, R3 (3)	4363.0 a	9.43 ab	3.40 ab
11- Cruiser® + Warrior® using threshold	4306.8 a	9.48 ab	3.30 abc
12- Cruiser® + Warrior® using DD-model (2)	4916.7 a	9.48 ab	3.45 a

[x] Means with the same letters within columns are not significantly different ($P \leq 0.05$) based on the Waller Duncan K-ratio test.

[y] Number in ( ) indicates the number of foliar application of Warrior® insecticides.

Table 17. The effects of seed and foliar insecticide treatments on corn yield, protein and oil content as affected by Stewart's disease caused by *P. stewartii* on inbred A634XCM105 plots at Johnston, Iowa in 2001.

Treatment	**Yield/Quality**		
	Yield (Kg/ha)	**Protein (%)**	**Oil (%)**
1- Nontreated control (no insecticide)	4969.6 bc[x]	11.10 a	3.13 a
2- Gaucho® seed treatment	4984.8 bc	11.25 a	3.20 a
3- Cruiser® seed treatment	4786.7 c	11.30 a	3.18 a
4- Warrior® at V5 (1)[y]	5365.9 ab	11.20 a	3.23 a
5- Warrior® using threshold	4847.6 bc	11.43 a	3.03 a
6- Warrior® using DD-model (2)	4969.6 bc	11.23 a	3.10 a
7- Gaucho® + Warrior® at V5, VT, R3 (3)	5183.0abc	11.05 a	3.20 a
8- Gaucho® + Warrior® using threshold	4801.9 c	11.55 a	3.13 a
9- Gaucho® + Warrior® using DD-model (2)	5061.0abc	8.80 a	4.43 a
10- Cruiser® + Warrior® at V5, VT, R3 (3)	5594.6a	11.08 a	3.33 a
11- Cruiser® + Warrior® using threshold	4984.8 bc	11.38 a	3.25 a
12- Cruiser® + Warrior® using DD-model (2)	5106.8 abc	11.13 a	3.33 a

[x] Means with the same letters within columns are not significantly different ($P \leq 0.05$) based on the Waller Duncan K-ratio test.

[y] Number in ( ) indicates the number of foliar application of Warrior® insecticides.

Table 18. The effects of seed and foliar insecticide treatments on corn yield, protein and oil content as affected by Stewart's disease caused by *P. stewartii* on inbred A632 Ht block plots at Crawfordsville, Iowa in 2002.

	Yield/Quality		
Treatment	**Yield (Kg/ha)**	**Protein (%)**	**Oil (%)**
1- Nontreated control (no insecticide)	2121.2 a[x]	12.90 a	3.08 abc
2- Gaucho® seed treatment	1828.0 a	13.03 a	3.23 ab
3- Cruiser® seed treatment	2029.6 a	12.93 a	3.10 abc
4- Warrior® at V5 (1)[y]	2190.8 a	12.93 a	3.08 abc
5- Warrior® using threshold (2)	1739.2 a	12.73 a	3.10 abc
6- Warrior® using DD-model (2)	1818.6 a	12.50 a	3.10 abc
7- Gaucho® + Warrior® at V5, VT, R3 (3)	1732.8 a	12.85 a	2.93 c
8- Gaucho® + Warrior® using threshold (2)	1875.0 a	12.73 a	3.32 a
9- Gaucho® + Warrior® using DD-model (2)	1651.9 a	12.68 a	3.03 bc
10- Cruiser® + Warrior® at V5, VT, R3 (3)	1797.3 a	12.80 a	3.08 abc
11- Cruiser® + Warrior® using threshold (2)	1606.2 a	12.75 a	3.10 abc
12- Cruiser® + Warrior® using DD-model (2)	1845.9 a	12.88 a	3.20 ab

[x]Means with the same letters within columns are not significantly different ($P \leq 0.05$) based on the Waller Duncan K-ratio test.

[y] Number in ( ) indicates the number of foliar application of Warrior® insecticides.

Table 19. The effects of seed and foliar insecticide treatments on corn yield, protein and oil content as affected by Stewart's disease caused by *P. stewartii* on inbred A632 Ht block plots at Johnston, Iowa in 2002.

	Yield/Quality		
Treatment	**Yield (Kg/ha)**	**Protein (%)**	**Oil (%)**
1- Nontreated control (no insecticide)	1432.9 a[x]	12.80 a	3.03 a
2- Gaucho® seed treatment	1265.3 a	12.75a	3.35 a
3- Cruiser® seed treatment	914.6 a	13.25 a	3.28 a
4- Warrior® at V5 (1)[y]	1067.1 a	12.68 a	3.20 a
5- Warrior® using threshold (2)	1051.8 a	13.00 a	3.13 a
6- Warrior® using DD-model (2)	868.9 a	13.43 a	3.10 a
7- Gaucho® + Warrior® at V5, VT, R3 (3)	2210.4 a	12.65 a	2.90 a
8- Gaucho® + Warrior® using threshold (2)	1493.9 a	12.68 a	3.00 a
9- Gaucho® + Warrior® using DD-model (2)	990.9 a	13.38 a	3.20 a
10- Cruiser® + Warrior® at V5, VT, R3 (3)	2225.6 a	12.33 a	2.98 a
11- Cruiser® + Warrior® using threshold (2)	1021.4 a	12.88 a	3.30 a
12- Cruiser® + Warrior® using DD-model (2)	1920.8 a	12.55 a	3.17 a

[x] Means with the same letters within columns are not significantly different ($P \leq 0.05$) based on the Waller Duncan K-ratio test.

[y] Number in ( ) indicates the number of foliar application of Warrior® insecticides.

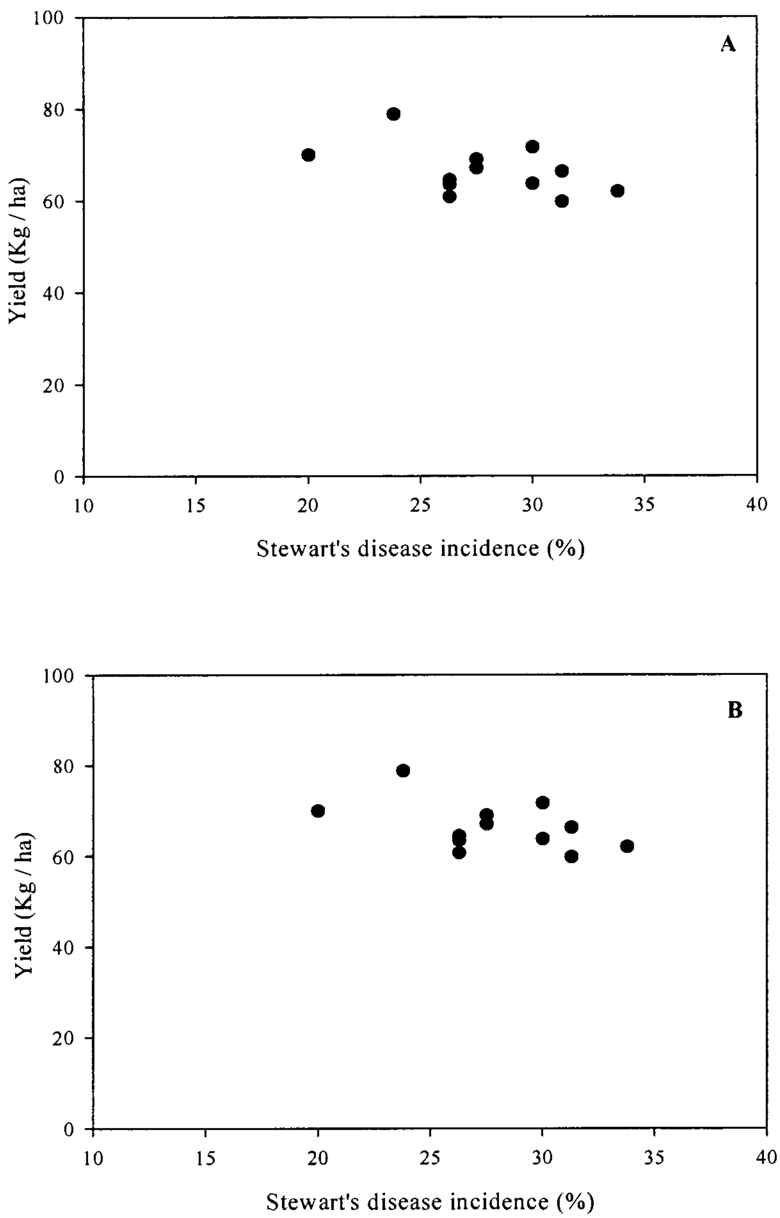


Figure 12. Relationship between final incidence of Stewart's disease of corn and yield at (**A**) Johnston and (**B**) Crawfordsville, in 2001.

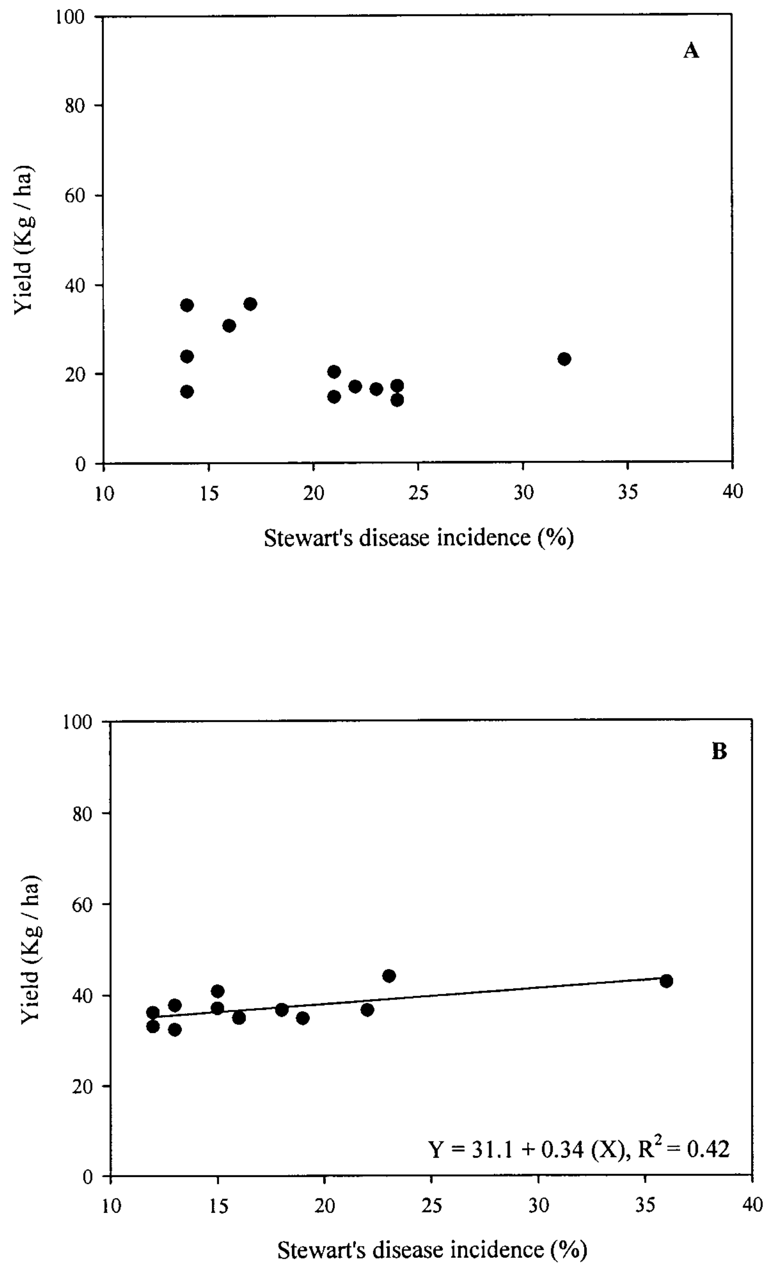


Figure 13. Relationship between final incidence of Stewart's disease of corn and yield at (**A**) Johnston and (**B**) Crawfordsville, in 2002.

Effect of date of planting on Stewart's disease of corn

The incidence of Stewart's disease varied between seasons. In 2001, final disease incidence ranged from just 0 to 3.67 % (Table 20), whereas at Crawfordsville in 2002, final disease incidence ranged from 26.7 to 48.7 % (Table 21). Stewart's disease incidence on DOY 150 (30 May) in 2001 was significantly higher in the first two plantings of Jubilee, compared to the latter three plantings at Boone, IA (Table 20). Disease incidence on day of year 150 ranged from 0 to 3.20 % (Fig. 14A, Table 20). In comparison, disease incidence at Crawfordsville on DOY 157 (6 June) ranged from 5.3 to 12.7 % in 2002 (Fig. 14B, Table 21).

In 2001 at Boone, final disease incidence on DOY 214 (2 August) was low and ranged from 0.17 to 3.67 % in the first three planting dates and no disease incidence was recorded in the latter two planting dates (Table 20). Final disease incidence at Boone, IA in 2001 on DOY 227 was significantly lower in the third (0.17 %), fourth (0 %), and fifth (0 %) planting dates compared to the first two planting dates (Table 20). In 2002, at Crawfordsville, the first, second, and third planting dates had the highest incidence up until DOY 190 (9 July) (Fig. 14B, Table 21). After DOY 190, only the first and third planting dates had significantly higher disease incidence, compared to the other treatments. Final disease incidence on DOY 214 in 2002 was significantly higher in the first planting date (48.7 %) in comparison to treatments 2, 4, and 5 (second, fourth and fifth planting dates) (Table 21).

Disease incidence progress curves for 2001 at Boone (Fig. 14A) and 2002 at Crawfordsville, IA (Fig. 14B), showed that disease incidence over time was higher in the first planting date compared to all other planting dates. The logistic population growth years (Tables 22 and 23). In 2001, the coefficients of determination (R^2) for the first two planting dates were 0.611 and 0.613, indicating approximately 61 % of the variation in Stewart's disease incidence was explained by the change in time. The standard errors for the estimate for y (SEEy) were low and ranged from 0.039 to 0.040 logits (Table 22). In 2001, the rate of disease progress (change in logit Stewart's disease incidence with respect to time) was very low among all treatments (0.0015) logits per day (Table 22). In 2002, the coefficients of determination, (R^2), ranged from 0.97 to 0.98, indicating that 97 to 98 % of the variation in Stewart's disease incidence was explained by the change in time (Table 23). Standard errors of the estimate for y were lowest with the logistic model, ranging from 0.10 to 0.16. The rate of Stewart's disease progress during the 2002 season ranged from 0.0296 to 0.0349 logits per day, with the fastest rate occurring during the first planting date 23 April 2002 and the rate was slowest for the last planting date 21 May 2002, with other treatment rates being intermediate (Table 23).

Using standardized area under the disease progress curve (STD AUDPC) values to evaluate and compare planting date treatments for 2001, STD AUDPC values were significantly higher ($P \leq 0.05$) for the first planting date (69.55) compared to all other planting dates (Table 20). The second planting date (54.17) had a STD AUDPC value that was significantly lower than the first planting date, but significantly higher compared to the third, fourth, and fifth planting dates. The latter three planting dates resulted in significantly lower STD AUDPC values compared to the first and second planting dates, ranging from 0 to 1.21 (Table 18).

In 2002, the STD AUDPC values were significantly higher ($P \leq 0.05$) for the first (60.9) and third (52.3) planting dates compared to all treatments, followed by the second

In 2002, the STD AUDPC values were significantly higher ($P \leq 0.05$) for the first (60.9) and third (52.3) planting dates compared to all treatments, followed by the second (42.6) and fourth (40.8) planting dates which were also significantly different from the other three planting dates (Table 21). In 2002, the STD AUDPC value for the fifth planting date (31.9) was significantly lower ($P \leq 0.05$) compared to all other planting dates (Table 21).

Corn flea beetle assessments

In 2001, corn flea beetle populations were extremely low and beetles were only occasionally observed in the field trial at Boone (data not shown). In 2002 at Crawfordsville, corn flea beetle population densities were very low early in the season (Fig. 16A-E), but populations were significantly higher compared to Boone in 2001. In 2002 on DOY 148 (28 May) at Crawfordsville, (date when beetle samples were first collected), the average number of beetles in the third and fifth planting dates were zero, compared to the other three planting dates which ranged from 0.44 to 0.67 beetles per day. Corn flea beetle numbers for the rest of the 2001 season were too low to analyze. In 2002 at Crawfordsville, corn flea beetle population densities remained low during the growing season until approximately DOY 180 (29 June). However, corn flea beetle populations were significantly higher in the first and second planting dates on DOY 200. The highest populations of corn flea beetles were observed in early August (DOY 214), ranging from 3.67 to 10.56 beetles per day (Fig. 16E). Among planting dates in 2002, the lowest populations on DOY 214 were observed in the first planting date, and the highest populations were observed in the fifth planting date.

In 2001 at Boone, cumulative corn flea beetle progress curves were too low to analyze by comparing the area under the cumulative beetle progress curves (AUCBPC). In 2002 at Crawfordsville, however, the cumulative beetle progress curves showed that corn flea beetles populations were low in all planting dates early in the season until approximately DOY 184, when cumulative corn flea beetle populations began to increase exponentially (Fig. 17). Although cumulative corn flea beetle populations were highest in the fifth planting date, there were no significant differences among treatments for cumulative corn flea beetle curves based on STD AUCBPC values (1337.9 to 1575.5) (Table 24).

Yield assessments

The effect of date of planting on marketable and unmarketable ears in 2001 is shown in Table 25. Although there were significant differences in marketable and unmarketable ears in 2001 at Boone, IA, these yield differences can not be attributed to Stewart's disease, because disease incidence among planting dates were extremely low in 2001. The number of unmarketable corn ears for each of the first four planting dates in 2001 (39.08, 31.50, 26.58 and 22.17) were greater than the number of marketable ears (20.33, 15.42, 19.50 and 20.92), for corresponding planting dates, respectively (Table 25). In 2002, the variation in Stewart's disease incidence among planting dates was not reflected in the yield data, as there were no significant differences in marketable or unmarketable ears among planting dates in 2002 using ANOVA and the Waller-Duncan K-ratio test ($P \leq 0.05$) (Table 25). Regression analysis of final disease incidence (X) on yield (Y) also indicated that there were no linear relationships between disease incidence and yield, except in 2001 at Boone where there was a significant linear (positive) relationship ($P \leq 0.015$, $R^2 = 0.90$) indicating that for every 1% increase in final Stewart's disease incidence, unmarketable yield increased by 0.015 %.

Table 20. The effect of planting date on the incidence of Stewart's disease of sweet corn variety Jubilee in Boone, Iowa in 2001.

	Incidence (%)						
Planting dates	**DOY 150 (30-May)**	**DOY 164 (13-June)**	**DOY 178 (27-June)**	**DOY 197 (16-July)**	**DOY 211 (30-July)**	**DOY 227 (15-Aug)**	**STD AUDPC**
DOY (108) (18-April)	3.20 a[x]	3.5 a	3.67 a	3.67 a	3.67 a	3.67 a	69.55 a
DOY (115) (25-April)	2.53 a	2.66 a	2.83 a	2.83 a	2.83 a	2.83 a	54.17 b
DOY (122) (02-May)	0 b	0 b	0.17 b	0.17 b	0.17 b	0.17 b	1.21 c
DOY (129) (09-May)	0 b	0 b	0 b	0 b	0 b	0 b	0 c
DOY (136) (16-May)	0 b	0 b	0 b	0 b	0 b	0 b	0 c

[x] Means with the same letters within columns are not significantly different ($P \leq 0.05$) based on the Waller Duncan K-ratio test.

Table 21. The effect of planting date on the incidence of Stewart's disease of sweet corn variety Jubilee in Crawfordsville, Iowa in 2002.

	Incidence (%)					
Planting dates	**DOY 157 (06-June)**	**DOY 176 (25-June)**	**DOY 190 (09-July)**	**DOY 203 (22-July)**	**DOY 214 (02-Aug)**	**STD AUDPC**
DOY 113 (23-April)	12.7 a[x]	18.0 a	26.0 a	42.7 a	48.7 a	60.9 a
DOY 120 (30-April)	10.7 ab	13.3 ab	19.3 ab	28.0 bc	32.0 bc	42.6 b
DOY 127 (07-May)	12.0 a	15.3 ab	22.0 ab	36.7 ab	40.7 ab	52.3 a
DOY 134 (14-May)	6.7 bc	12.0 ab	20.0 ab	27.3 bc	31.3 bc	40.8 b
DOY 141 (21-May)	5.3 c	9.3 b	14.7 b	21.3 c	26.7 c	31.9 c

[x] Means with the same letters within columns are not significantly different ($P \leq 0.05$) based on the Waller Duncan K-ratio test.

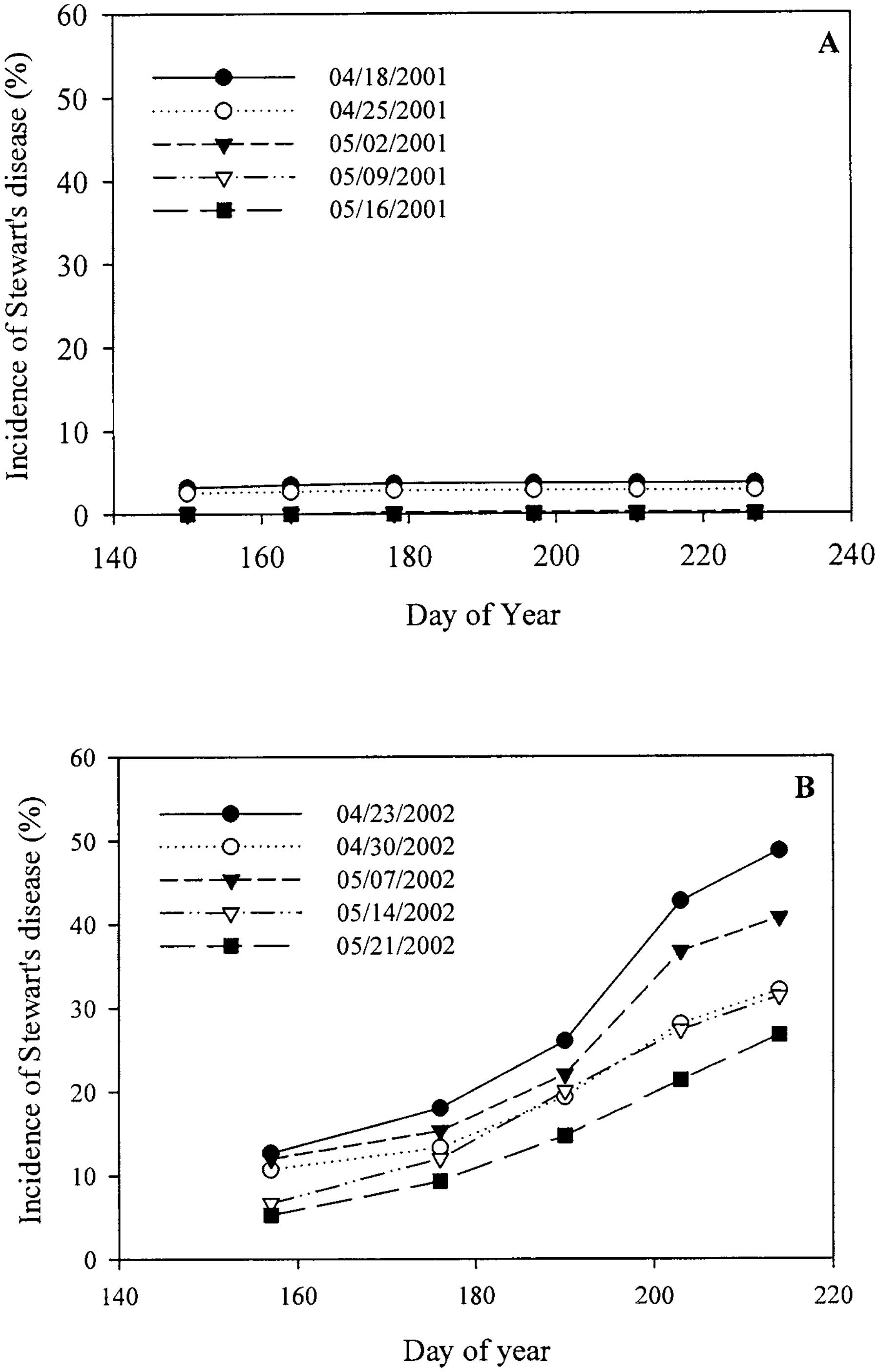


Figure 14. Disease incidence progress curves for Stewart's disease of corn on sweet corn variety Jubilee plots in (**A**) Boone, IA in 2001 and in (**B**) Crawfordsville, IA in 2002.

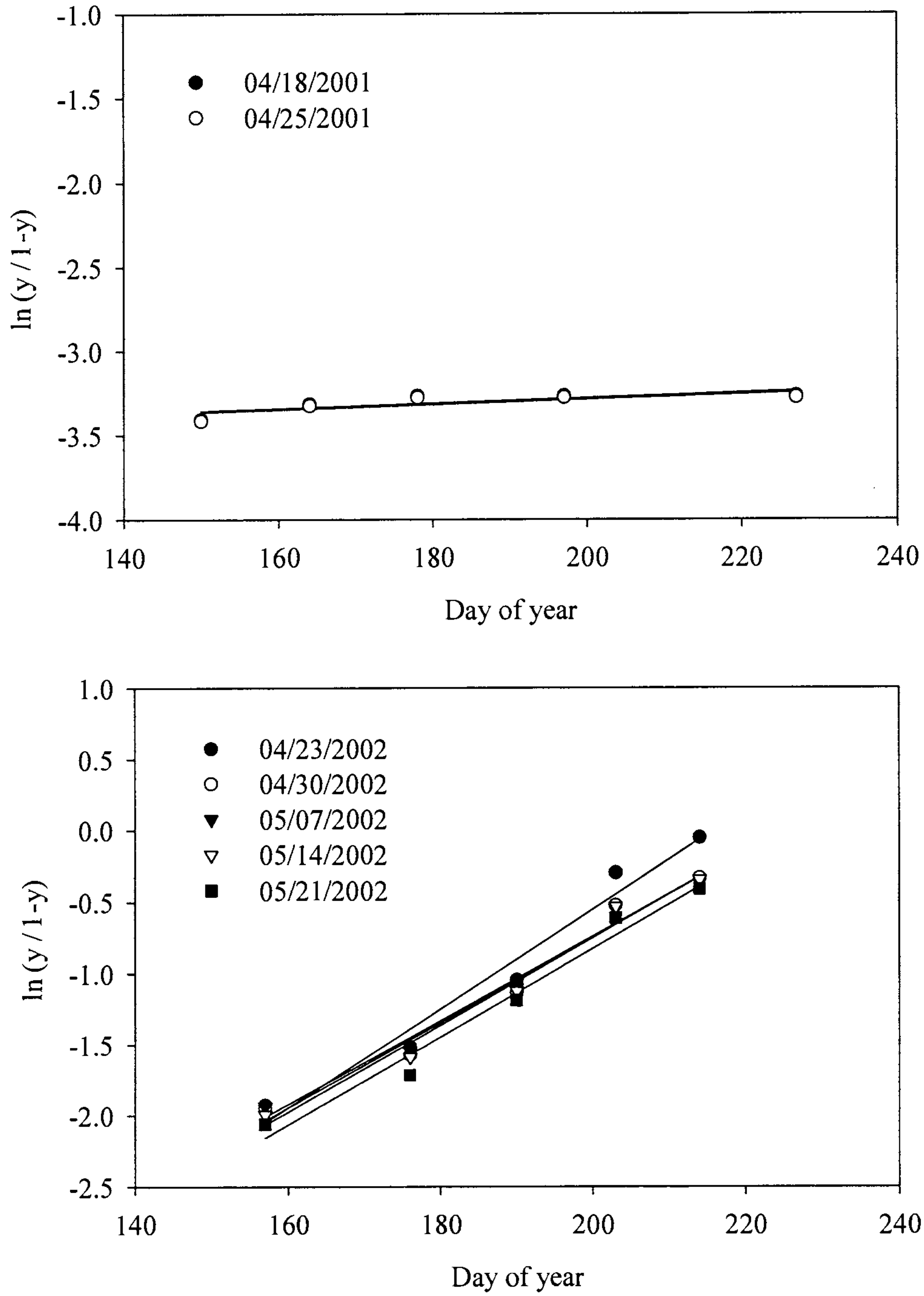


Figure 15. Linear regression lines using the logistic model ln (y / (1-y)) versus time to transform Stewart's disease incidence data in sweet corn variety Jubilee at (**A**) Boone, IA in 2001, and (**B**) Crawfordsville, IA in 2002.

Table 22. Logistic model parameters and statistics describing the disease incidence progress curves of Stewart's disease of sweet corn variety Jubilee in Boone, Iowa in 2001.

	Boone 2001				
Planting dates	*F-statistic*	Intercept	Slope	R^2	SEEy
DOY (108) (18-April)	6.29	-3.59	0.002 ± 0.0006	0.611	0.040
DOY (115) (25-April)	6.34	-3.59	0.002 ± 0.0006	0.613	0.039
DOY (122) (02-May)	NA[z]	NA	NA	NA	NA
DOY (129) (09-May)	NA	NA	NA	NA	NA
DOY (136) (16-May)	NA	NA	NA	NA	NA

[z] indicates data not available.

Table 23. Logistic model parameters and statistics describing the disease incidence progress curves of Stewart's disease of sweet corn variety Jubilee in Crawfordsville, Iowa in 2002.

	Crawfordsville 2002				
Planting dates	*F-statistic*	Intercept	Slope	R^2	SEEy
DOY 113 (23-April)	96.22	-7.52	0.035 ± 0.0036	0.970	0.1594
DOY 120 (30-April)	124.68	-6.76	0.030 ± 0.0027	0.978	0.1209
DOY 127 (07-May)	95.25	-7.15	0.033 ± 0.0033	0.970	0.1494
DOY 134 (14-May)	155.05	-6.89	0.031 ± 0.0025	0.981	0.1105
DOY 141 (21-May)	156.65	-6.73	0.030 ± 0.0024	0.981	0.1061

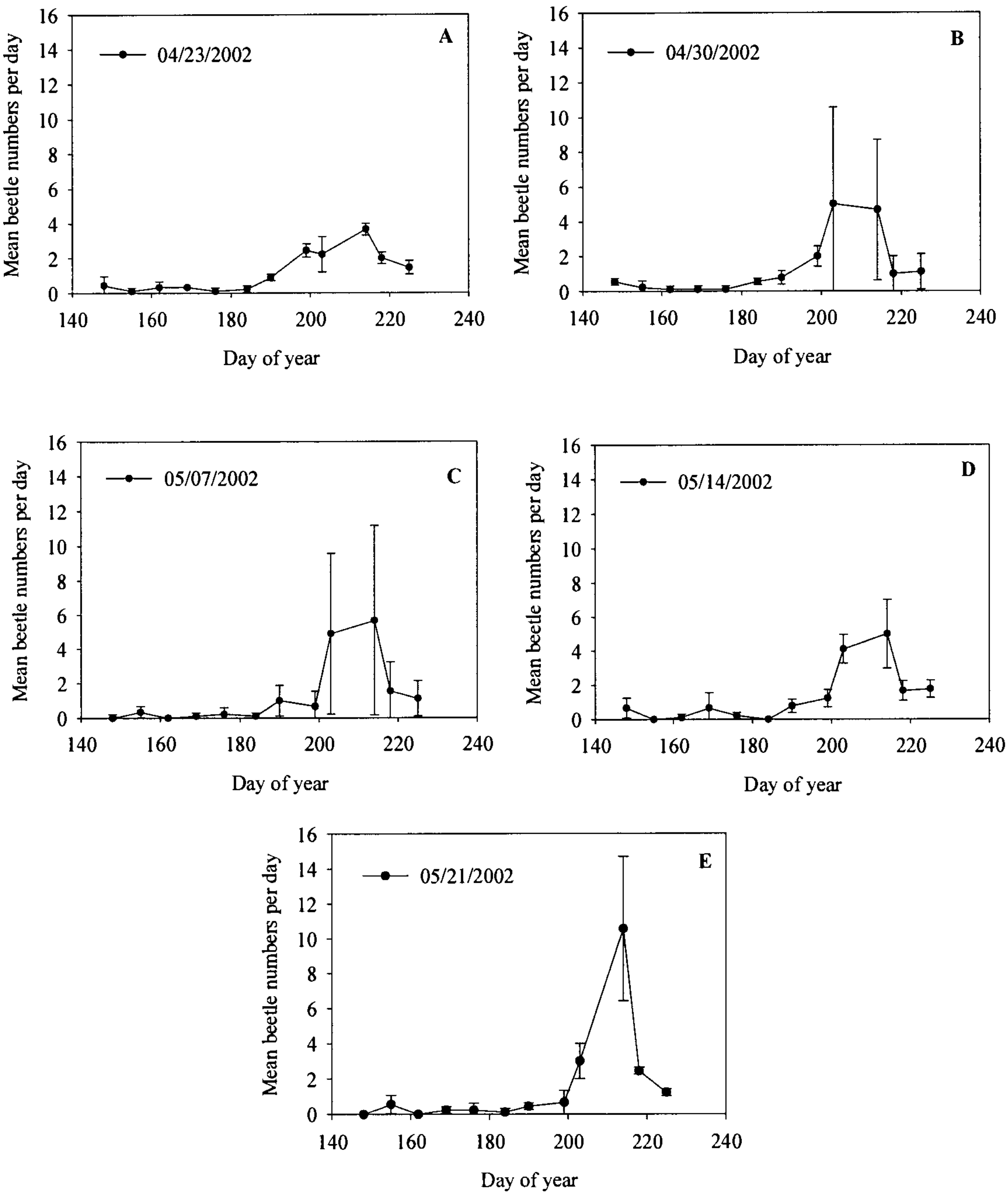


Figure 16. Effect of planting date on the mean numbers of corn flea beetles caught using yellow sticky cards at Crawfordsville, IA during the 2002 growing season.

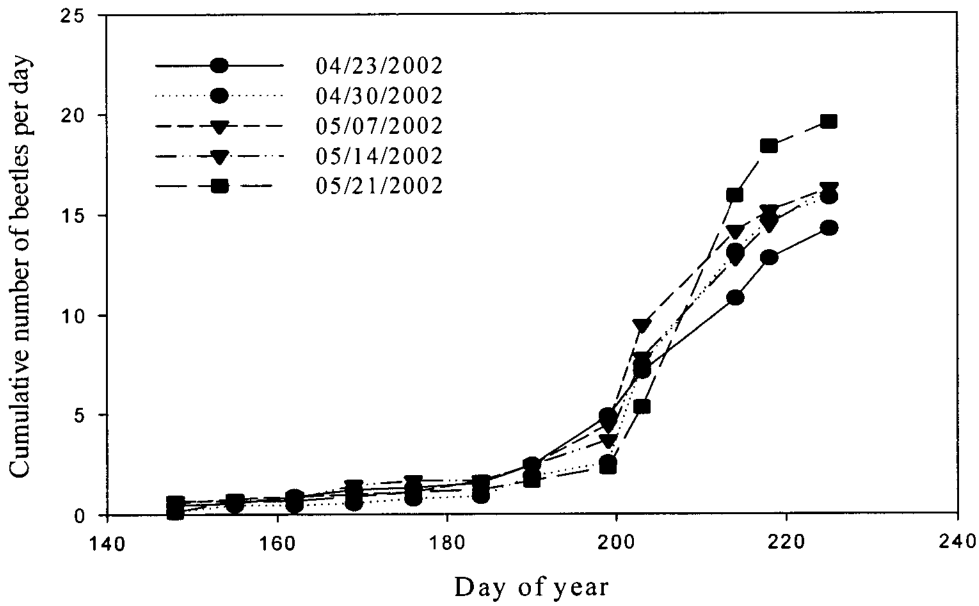


Figure 17. Effect of planting date on the cumulative number of corn flea beetles caught using yellow sticky cards at Crawfordsville, IA during the 2002 growing season.

Table 24. The effect of altering date of planting on the standardized area under the cumulative beetle progress curves at Crawfordsville, Iowa in 2002.

	Crawfordsville 2002
Planting dates	**STD AUCBPC**
DOY 113 (23-April)	1345.9 a[x]
DOY 120 (30-April)	1337.9 a
DOY 127 (07-May)	1575.5 a
DOY 134 (14-May)	1477.7 a
DOY 141 (21-May)	1515.4 a

[x] Means with the same letters within columns are not significantly different ($P \leq 0.05$) based on the Waller Duncan K-ratio test.

Table 25. The effects of date of planting on marketable and unmarketable yield of the sweet corn variety Jubilee at Boone (2001) and Crawfordsville (2002), IA.

Location/Year		**Yield** (kg / plot)	
Boone 2001	**Treatment** (Planting date)	**Marketable**	**Unmarketable**
	DOY (108) (18-April)	20.33 ab[x]	39.08 a
	DOY (115) (25-April)	15.42 c	31.50 ab
	DOY (122) (02-May)	19.50 b	26.58 bc
	DOY (129) (09-May)	20.92 ab	22.17 c
	DOY (136) (16-May)	22.83 a	21.00 c
Crawfordsville 2002			
	DOY 113 (23-April)	8.17 a	20.08 a
	DOY 120 (30-April)	14.00 a	17.08 a
	DOY 127 (07-May)	13.83 a	21.83 a
	DOY 134 (14-May)	9.25 a	14.33 a
	DOY 141 (21-May)	9.41 a	23.00 a

[x] Means with the same letters within columns are not significantly different ($P \leq 0.05$) based on the Waller Duncan K-ratio test.

DISCUSSION

Acquisition study

This study was the first to quantify the acquisition access and transmission feeding periods required by the corn flea beetle to acquire and transmit *Pantoea (Erwinia) stewartii,* the causal agent of Stewart's disease of corn. In previous studies, Munkvold et al. (1996) arbitrarily used an acquisition access period of 9 to 10 days on diseased plants to study the effects of insecticide seed treatments on corn flea beetle leaf feeding and transmission of *P. stewartii* to corn plants. Dill (1979) also allowed corn flea beetles to feed on corn plants infected with *P. stewartii,* but the duration of the acquisition access period and the efficiency of acquisition with respect to feeding time were not reported.

It has been reported that *P. stewartii* is not passed from adult corn flea beetles to eggs (Dill, 1979). Therefore, the first and second generations of *C. pulicaria* must acquire *P. stewartii* from diseased plants in the field. This study provides new quantitative information to explain how quickly corn flea beetles can acquire *P. stewartii* from diseased corn plants in the field. Esker and Nutter (2003) observed that the first summer generation of *P. stewartii*-infested corn flea beetles was as high as 20 to 40 % when corn flea beetles were sampled and tested by ELISA just after the beetle-free period. The beetle-free period occurs in early-to-mid June after the overwintering corn flea beetle generation has completed their life cycle, but before the first summer generation of corn flea beetles has not yet emerged from eggs laid by the overwintering generation (Esker et al., 2002). In another study, Esker and Nutter (2003) found that the incidence of *P. stewartii*-infested corn flea beetles reached levels as high as 85.6 % by late August. Our findings that acquisition of *P. stewartii* by corn flea beetles can occur within six hours and that the time required for *P. stewartii*-infested corn flea beetle populations to reach 50 % acquisition is approximately 38.3 ±10.1 hrs supports field observations by Esker et al. (2003) regarding the population dynamics of *P. stewartii*-infested corn flea beetles during the growing season. Epidemiologically, the ability of the corn flea beetles to acquire the pathogen within 6 hours facilitates rapid increase in secondary inoculum, which contributes directly to the increase in disease incidence with respect to time. We did not examine the length of time that *C. pulicaria* remains infectious (retention time), but previous studies by Robert (1953) and Dill (1979) have postulated that corn flea beetles remain infectious throughout a single generation once the bacterium is acquired. Therefore, it would be important in future studies to examine whether or not *C. pulicaria* can acquire *P. stewartii* and transmit it to more than one plant. This study is the first to report that the Gompertz population growth model best describes pathogen acquisition by corn flea beetles over time. *Pantoea stewartii* acquisition increases fastest early in the acquisition access period and acquisition slows after reaching an inflection point at 0.37 (37 % incidence).

Transmission study

In the transmission study, there was a positive relationship between duration of the feeding period and the increase in percent transmission to test plants. The longer the beetles remained on the test plants, the higher the percentage transmission of *P. stewartii* from corn flea beetles to corn seedlings. This study is the first to report that transmission of *P. stewartii* by corn flea beetles is best described by the monomolecular model. Transmission percentage of *P. stewartii* is greatest at the beginning of the transmission feeding period and the rate of transmission decreases with respect to time without an inflection point. Following an acquisition access period of 48 hours on diseased plants, the minimum feeding period for transmission occurred within three hours. Under normal field conditions, the actual percentage of diseased plants resulting from the transmission of a fixed population size of *P. stewartii*-infested corn flea beetles may be higher (after a three hour feeding period) than the 5.7 % transmission reported in this study, because corn flea beetles move readily from plant to plant, and a single infectious corn flea beetle may lead to many infected plants. This hypothesis, however, needs to be examined experimentally.

We did not test the possibility that transmission efficiency may increase with increasing acquisition access period, because our study was based on a single acquisition access period (48 hr). However, our results did indicate that there is a small window of opportunity for insecticide seed treatments or foliar insecticides to effectively prevent transmission of *P. stewartii* by *P. stewartii*-infested corn flea beetles.

It was observed in our study that non-transmission of *P. stewartii* by corn flea beetles that were known to be carrying *P. stewartii* (based on positive tests for *P. stewartii* by plating beetle contents on rifampicin-nalidixic acid amended media) sometimes occurred. Non-transmission by infectious corn flea beetles could be due to factors such as insufficient feeding time (in cages) to transmit the bacterium, the need for a period of latency in the corn flea beetle, possible injury to beetles when transferring them from diseased to healthy test plants, and/or a prolonged settling time for caged beetles to initiate feeding. Dill's 1979 study is the only study to date that provides information concerning the transmission efficiency of *P. stewartii* by corn flea beetles. Dill's study determined that there was a minimum amount of leaf tissue that needed to be consumed by the corn flea beetles (as measured by the length of the feeding scars), before beetles could successfully transmit *P. stewartii* to corn plants. Dill reported that *P. stewartii*-infested corn flea beetles were able to transmit the pathogen after they had consumed at least 9 mm of leaf tissue, and that *P.*

stewartii transmission ranged from 0 to 36 % for feeding scars ranging from 3 mm to 18 mm in length. In contrast, the present study was based on exposing healthy corn plants to predetermined transmission feeding periods by *P. stewartii*-infested corn flea beetles to quantify the relationship between duration of feeding period and percentage transmission by corn flea beetles. Similar trends can be inferred from both studies; the larger the amount of leaf tissue consumed or the longer the duration of the transmission feeding period, the higher the percentage transmission by corn flea beetles.

Insecticide study

This is the first study to quantify and evaluate the application of foliar insecticides to control *C. pulicaria* and Stewart's disease of corn when both foliar insecticide sprays and insecticide seed treatments are applied. This is also the first study to report that the change in Stewart's disease incidence with respect to time was best described by the logistic model. This model shows that the rate of disease incidence increases as incidence approaches 50 %, and that the rate decreases after reaching an inflection point of 0.5 (50 % incidence) due to limited nondiseased plants. In the present study, early season control of Stewart's disease of corn, using either imidacloprid (Gaucho®) or thiamethoxam (Cruiser®) seed treatments significantly reduced disease incidence. Similar results have been reported by Munkvold et al. (1996), Pataky et al. (2000), and Kuhar et al. (2002) in that they also reported a significant reduction in Stewart's disease incidence in susceptible inbred and sweet corn varieties when using insecticide seed treatments.

Esker and Nutter (2003) have described three critical periods during the corn growing season that may affect the amount of inoculum available for Stewart's disease epidemics. Seed treatments typically provide protection for the first critical period, which is the seedling wilt phase of the disease. During this first critical period, the emergence of overwintering adult corn flea beetles (that had already acquired *P. stewartii* the previous growing season), coincides with the emergence of corn seedlings.

The second critical period occurs in mid-to-late June and continues into July (Esker et al., 2002; Esker and Nutter, 2003). This critical period plays an important role in the late leaf blight phase of Stewart's disease, because *P. stewartii* is transmitted by corn flea beetles from diseased to healthy corn plants during this period. If infection occurs during this period, visual symptoms of Stewart's disease will be observed during the time that phytosanitary field inspections are conducted (early to mid August). If field inspectors find even a single corn plant with Stewart's disease, the entire field would fail inspection and seed from the diseased field would not meet the export requirements of many countries (Block et al., 1999).

It is believed that the longer that infection can be delayed in the field, the less likely that plant-to-seed transmission of *P. stewartii* will occur (Block et al., 1999). Resistant or moderately resistant plants seem to delay the movement of the bacterium within corn plants. Block et al. (1999) observed that no *P. stewartii*-infected seed was detected from five resistant and nine moderately resistant maize lines. Michener et al. (2002a), however, reported 0.1 % and 0.01% seed infection from moderately-resistant and resistant plant genotypes, respectively. Even in susceptible corn genotypes, the later that plant infection occurs in the field, the lower the probability, that seeds will be infected with *P. stewartii*. Thus, seed harvested from a field with very low incidence of Stewart's disease or from fields where corn plants were infected late in the season due to the application of foliar insecticides might still pass phytosanitary requirements based on ELISA tests of seed in the laboratory (even if the field had previously failed the field inspection).

It has been hypothesized that the use of seed and foliar insecticides would significantly reduce and delay the incidence of the late leaf blight phase of Stewart's disease. This hypothesis was rejected in our study because foliar applications of Warrior® that were used in addition to insecticide seed treatments did not significantly reduce the final incidence of Stewart's disease compared to the use of seed insecticides alone. There were some treatment differences in the incidence of Stewart's disease across all four location-years, however, these differences did not translate into yield differences among treatments at all four location-years.

The third critical period for Stewart's disease of corn occurs in August when corn flea beetles migrate from corn fields to grassy areas bordering corn fields. This migration begins just after the time that corn begins to senesce (Esker and Nutter, 2002). Control of corn flea beetles during this critical period may reduce the amount of inoculum available for the following growing season, however, this hypothesis needs to be tested experimentally.

The three different timing methods used to schedule foliar applications of Warrior® (degree day accumulation, corn flea beetle threshold, and corn growth stage) were not significantly different in reducing Stewart's disease incidence. Although there were no significant differences among the three foliar insecticide timing methods, growers may prefer using the corn growth stage insecticide method because less work is needed for its implementation. This program is based on crop physiology, whereby spray applications are made at corn growth stages V5, VT, and R3. This program does not account for year-to-year variation in corn flea beetle populations or Stewart's disease epidemics. It is suggested to spray at growth stage (V5) to protect against the early wilt phase, especially if there is a high population of overwintering adult corn flea beetles in the field. This is the growth stage, after which, seed treatments no longer provide adequate crop protection (Dill, 1979; Munkvold et al., 1996). Suparyono and Pataky (1989) reported that even resistant plants can be adversely affected when corn flea beetle populations are high. For the other two corn growth stages, there is a question as to whether or not foliar insecticide applications might miss peak periods of corn flea beetle emergence. The corn flea beetle threshold timing method minimizes the potential risk of disease spread associated with the initial emergence of corn flea beetle populations. A disadvantage of the action threshold timing method (one corn flea beetle per trap per week) is the requirement for constant monitoring of fields for corn flea beetles. The advantage of the degree day model timing method is that, unlike the other two timing methods, continual monitoring of the crop is not required. However, growers will need to obtain reliable air temperature data to calculate the accumulation of degree day units.

The amount of primary inoculum present at the beginning of the season is related to the number of corn flea beetles that survive the winter (Stevens, 1934). Therefore the seasonal intensity of Stewart's disease is related, in part, to the year-to-year variation in overwintering corn flea beetle population densities. Following six successive mild winters that favored the survival of large population of corn flea beetles, the prevalence of Stewart's disease in Iowa reached record highs (58 %) in both 1999 and 2000 (Esker and Nutter, 2002). However, following the more severe (cold) winters of 2000-2001 and 2001-2002, corn flea beetle populations at Crawfordsville and Johnston during the 2001 and 2002 seasons were low-to-moderate and the prevalence of Stewart's disease during the 2001 and 2002 growing seasons ranged from just 2 to 4 % in Iowa (unpublished data). Under these conditions, the use of foliar insecticide applications of Warrior® appeared to be impractical, as there was no significant reduction in final Stewart's disease incidence compared to the use of seed treatments alone (Gaucho® or Cruiser®). The lack of significant results could be due to unknown factors such as the migratory movement of corn flea beetles from plot to plot (interplot interference), resulting in lack of significant differences in final Stewart's disease incidence among insecticide treatments (Gourmet et al., 1994). The plot sizes were not identical for both locations (Crawfordsville: 8 rows of 15.24 m by 0.91 m, Johnston: 4 rows of 12.9 m by 0.91 m), due to limited space available at Johnston. Alleys between plots and larger plot sizes may help minimize the effects of interplot interference. Therefore, further investigations concerning corn flea beetle migration and distribution patterns are needed to better determine the size of plots needed to evaluate the use of foliar insecticide spray programs. Such experiments also need to be conducted when corn flea beetle populations and levels of incidence of Stewart's disease are both high. The economic implications concerning the use of using foliar insecticides by seed corn producers and the environmental effects of insecticides on the agroecosystem also need to be addressed before a general recommendation can be made.

Planting date study

The need to reduce chemical inputs in food production and the development of environmentally-friendly approaches to managing plant diseases has been mandated by the U.S. Congress. Cultural practices such as altering the date of planting to avoid exposure to pathogen populations is one disease management alternative that could be used as part of an integrated pest management program for Stewart's disease of corn. We examined the effect of altering the date of planting on the susceptible sweet corn variety, Jubilee. Our hypothesis was that if the emergence of sweet corn coincides with the emergence of the overwintering generation of *P. stewartii*-infested corn flea beetles, then a higher incidence of Stewart's disease would occur during the seedling wilt phase. This study showed that disease incidence was highest in the early plantings, compared to the later plantings in Iowa. Although the effectiveness of cultural practices are sometimes a function of the geographic location, similar observations regarding reductions in disease incidence as affected by date of planting have been reported by Pataky et al. (1995). In their study, nine sequential field plantings were evaluated for disease reduction in Delaware, Illinois, and Missouri. They found that early plantings were most affected by Stewart's disease at all three locations. Heichel et al. (1977) also reported a higher incidence of Stewart's disease on plantings made prior to 27 May, and lower disease incidence for plantings after 2 June. However, Elliot and Poos (1940) found that the incidence of Stewart's disease increased as the date of planting was delayed for studies conducted between 1934 and 1937. Late-planted sweet corn may escape the emergence of overwintering adult corn flea beetle populations during the susceptible seedling wilt growth stages (emergence to corn growth stage V5), however, the cumulative number of corn flea beetles caught during the growing season was far greater at the end of the season in late-planted sweet corn, compared to early-planted sweet corn. This may have been because more green leaf tissue was present in late plantings compared to early plantings that had already begun to senesce.

This study reports for the first time that the change in Stewart's disease incidence with respect to time was best described by the logistic model. The rate of disease incidence increases fastest as disease incidence approaches 50 %, and rate slows after reaching an inflection point of 0.5 (50 % incidence) due to limited healthy plants. The rates of disease progress in 2001 at Boone were not as fast compared to the rates recorded at Crawfordsville in 2002. This was due to the higher population density of the overwintering adult populations of *P. stewartii*-infested corn flea beetles and the presence of higher population densities throughout the 2002 growing season compared to 2001. However, one interesting finding in 2001 was that disease incidence remained almost unchanged throughout the entire growing season. In addition, the last two planting dates that had completely avoided infection by the overwintering beetle population remained completely disease-free for the entire growing season. Variation in epidemics between both years could be related to environmental factors, such as winter temperatures, which affect the survival of the overwintering *P. stewartii*-infested corn flea beetle populations, spring precipitation, disease prevalence and vector populations during the previous year, and crop growth stage (Esker and Nutter, unpublished; Pataky et al., 1990; Pataky et al., 1995; Lam et al., 2001; Esker and Nutter, 2002). In all likelihood, this was due to factors such as the previous harsh winter that considerably reduced the number of surviving overwintering corn flea beetles (Esker and Nutter, unpublished; Pataky et al., 1990).

The results from these experiments suggest that incidence of Stewart's disease decreased as date of planting was delayed. This practice should reduce the risk of Stewart's disease in seed corn fields, thus providing an economic advantage to seed corn producers. However, to take full advantage of the length of the growing season and other economic considerations, sweet corn growers most likely will not delay planting to avoid potential risk associated with early planting. One additional or alternative measure would be to use an insecticide seed treatment to further reduce potential disease risk during the seedling wilt phase, because the occurrence of Stewart's disease during this phase could result in significant economic losses to sweet corn producers.

LITERATURE CITED

1. Block, C. C., Hill, J. H., and McGee, D. C. 1999. Relationship between late-season severity of Stewart's bacterial wilt and seed infection in maize. Plant Dis.83:527-530.

2. Block, C. C., Hill, J. H., and McGee, D. C. 1998. Seed transmission of *Pantoea stewartii* in field and sweet corn. Plant Dis. 82:775-780.

3. Boewe, G. H. 1949. Late season incidence of Stewart's disease on sweet corn and winter temperature in Illinois, 1944-1948. Plant Dis. Reptr. 33:192-194.

4. Campbell, C. L., and Madden, L. V. 1990. Introduction to Plant Disease Epidemiology. John Wiley & Sons, New York. 532p.

5. Castor, L. L., Ayers, J. E., MacNab, A. A., and Krause, R. A. 1975. Computerized forecasting system for Stewart's bacterial disease of corn. Plant. Dis. Reptr. 59:533-536.

6. Dill, J. F. 1979. Biology and management of the corn flea beetle, C*haetocnema pulicaria* Melsheimer, relative to the incidence of Stewart's disease in corn. Ph.D. diss. Purdue University, West Lafayette, IN. 167 p.

7. Dillard, H. R., and Kline, W. L. 1989. An outbreak of Stewart's bacterial wilt of corn in New York State. Plant Dis. 73:273.

8. Elliot, C., and Poos, F. W. 1940. Seasonal development, insect vectors, and host range of bacterial wilt of sweet corn. J. Agric. Res. 60:645-686.

9. Esker, P. D. 2001. Geographical and temporal dynamics of *Chaetocnema pulicaria* populations and their role in Stewart's disease of corn in Iowa. M.Sc. diss. Iowa State University, Ames, IA. 92 p.

10. Esker, P. D., and Nutter, F. W., Jr. 2002. Assessing the risk of Stewart's disease of corn through improved knowledge of the role of the corn flea beetle vector. Phytopathology 92:688-670.

11. Esker, P. D., and Nutter, F. W., Jr. 2000. Monitoring the dynamics of corn flea beetles infested with *Pantoea stewartii* by ELISA. Phytopathology 90:S22.

12. Esker, P. D., and Nutter, F. W., Jr. 2003. Temporal dynamics of corn flea beetle populations infested with *Pantoea stewartii,* causal agent of Stewart's disease of corn. Phytopathology 93:210-218.

13. Esker, P. D., Obrycki, J., and Nutter, F. W., Jr. 2002. Temporal distribution of *Chaetocnema pulicaria* (Coleoptera: Chrysomelidae) populations in Iowa. J. Econ. Entomol. 95(4): 739-747.

14. Frutchey, C. W. 1936. A study of Stewart's disease of sweet corn caused by *Phytomonas stewartii.* Mich. Agric. Exp. Stn. Tech. Bull. 152. 25 p.

15. Gourmet, C., Hewing, A. D., Kolb, F. L., Smyth, C. A., 1994. Effect of imidacloprid on nonflight movement of *Rhopalosiphum padi* and the subsequent spread of barley yellow dwarf virus. Plant Dis. 78:1098-1101.

16. Heichel, G. H., Sands, D. C., and Kring, J. B. 1977. Seasonal patterns and reduction by carbofuran of Stewart's bacterial wilt of sweet corn. Plant Dis. Reptr. 61:149-153.

17. Ivanoff, S.S. 1933. Stewart's wilt disease of corn with emphasis on the life history of *Phytonomas stewartii* in relation to pathogenicity. J. Agri. Res. 47:749-770.

18. Kuhar, T. P., Sivers-Young, L. J., Hoffmann, M. P., and Taylor, A. G. 2002. Control of corn flea beetle and Stewart's wilt in sweet corn with imidacloprid and thiamethoxan seed treatments. Crop Protection 21:25-31.

19. Lam, W. K. F., Pedigo, L. P., and Hinz, P. N. 2001. Population dynamics of bean leaf beetles (Coleoptera: Chrysomelidae) in central Iowa. J. Econ. Entomol. 30(3):362-367.

20. Lamka, G. L., Hill, J. H., McGee, D. C., and Braun, E. J. 1991. Development of an immunosorbent assay for seedborne *Erwinia stewartii* in corn seeds. Phytopathology 81:839-846.

21. Mergaert, J., Verdonck, L., and Kersters, K. 1993. Transfer of *Erwinia ananas* (synonym, *Erwinia uredovora*) and *Erwinia stewartii* to the genus *Pantoea* emend. as *Pantoea ananas* (Serrano 1928) comb. nov. and *Pantoea stewartii* (Smith 1898) comb. nov., respectively and description of *Pantoea stewartii* subsp. nov. Int. J. Syst. Bacteriol. 43:162-173.

22. Michener, P. M., Freeman, N. D., and Pataky, J. K. 2003. Relationships between reactions of sweet corn hybrids to Stewart's wilt and incidence of systemic infection by *Erwinia stewartii.* Plant Dis. 87:223-228.

23. Michener, P. M., Pataky, J. K., and White, D. G. 2002b. Rates of transmitting *Erwinia stewartii* from seed to seedlings of a sweet corn hybrid susceptible to Stewart's wilt. Plant Dis. 86:1031-1035.

24. Michener, P. M., Pataky, J. K., and White, D. G. 2002a. Transmission of *Erwinia stewartii* from plants to kernels and reactions of corn hybrids to Stewart's wilt. Plant Dis. 86:167-172.

25. Munkvold, G. P., McGee, D. C., and Iles, A. 1996. Effects of imidacloprid seed treatment of corn on foliar feeding and *E. stewartii* transmission by the corn flea beetle. Plant Dis. 80:747-749.

26. Munkvold, G. P., and Rice, E. M. 1998. Risk of Stewart's disease high in 1998. ISU University Extension. Iowa State University, Ames, IA. IC-480 (5).

27. Nutter, F. W., Jr., and Gaunt, R. E. 1996. Recent developments in methods for assessing disease losses in forage/pasture crops. Pages 93-118 in: Pasture and Forage Crop Pathology. Chakraborty, S., Leath, D. T., Skipp, R. A., Pederson, G. A, Bray, R. A., Latch, G. C. M., and Nutter, F. W., Jr., eds. American Soc. Agronomy, Crop Sci. Soc. America and Soil Sci. Soc. America, Madison, WI.

28. Nutter, F. W., Jr., and Parker, S. K. 1996. Fitting disease progress curves using EPIMODEL. Pages 24-28 in: Exercises in Plant Disease Epidemiology. L. Francl and D. Neher, eds. The American Phytopathological Society, St. Paul, MN.

29. Nutter, F. W., Jr., Rubsam, R. R., Taylor, S. E., Harri, J. A. and Esker, P. D. 2002. Use of geospatially-referenced disease and weather data to improve site-specific forecasts for Stewart's disease of corn in the US corn belt. Comp. and Elect. in Ag. 37: 7-14.

30. Nutter, F. W., Jr., Teng, P. S., and Shokes, F. M. 1991. Disease assessment terms and concepts. Plant Dis. 75:1187-1188.

31. Nutter, F. W., Jr., Wegulo, S. N., Rubsam, R. R., Taylor, S. E., and Harri, J. A. 1998. Use of geospatially-referenced disease and weather data to improve site-specific forecasts for Stewart's disease of corn. Phytopathology 88:S68.

32. Pataky, J. K., Du Toit, L. J., and Freeman, N. D. 2000. Stewart's wilt reduction of an international collection *Zea mays* germplasm inoculated with *Erwinia stewartii.* Plant Dis. 84:901-906.

33. Pataky, J. K., Du Toit, L. J., Kunkel, T. E., and Schmitt, R. A. 1995. Severe Stewart's wilt in central Illinois on sweet corn hybrids moderately resistant to *Erwinia stewartii.* Plant Dis. 79:104.

34. Pataky, J. K., and Eastburn, D. M. 1993. Using hybrid disease nurseries and yield loss studies to evaluate level of resistance in sweet corn. Plant Dis. 77:760-765.

35. Pataky, J. K., Hawk, J. A., Weldekidan, T., and Fallah Moghaddam, P. 1995. Incidence and severity of Stewart's bacterial wilt on sequential plantings of resistant and susceptible sweet corn hybrids. Plant Dis. 79:1202-1207.

36. Pataky, J. K., Michener, P. M., Freeman, N. D., Weinzierl, R. A., and Teyker, R. H. 2000. Control of Stewart's wilt in sweet corn with seed treatment insecticides. Plant Dis. 84:1104-1108.

37. Pataky, J. K., Suparyono, Hawk, J. A., Gardiner, M. L., and Pauly, M. H. 1990. Associations between Stewart's wilt ratings and maturity of sweet corn hybrids. Plant Dis. 74:792-796.

38. Pepper, E. H. 1967. Stewart's wilt of corn. Monograph No. 4. The American Phytopathology Society, St. Paul, MN.

39. Poos, F. W. 1939. Host plants harboring *Aplanobacter stewartii* without showing external symptoms after inoculation by *Chaetocnema pulicaria.* J. Econ. Ent. 32:881-882.

40. Poos, F. W. 1955. Studies of certain species of *Chaetocnema.* J. Econ. Entomol. 48: 555-563.

41. Poos, F. W., and Elliot, C.1936. Certain insect vectors of *Aplanobacter stewartii.* J. Agric. Res. 52:585-608.

42. Rand, F. V., and Cash, L. C. 1933. Bacterial wilt of corn. USDA Tech. Bull. 362: 1-22.

43. Rand, F. V., and Cash, L. C. 1924. Further evidence of insect dissemination of bacterial wilt of corn. Science 59:67-69.

44. Reddy, C. H. 1921. Experiments with Stewart's disease on dent, flint, and sweet corn (Abstr.) Phytopathology 11:31.

45. Robert, A. L., Jenkins, M. T., Findley, W. R., Jr. 1953. *Helminthosporium turcicum*, *Helminthosporium maydis*, and *Bacterium stewarti* leaf blight ratings on corn at the Plant Industry Station, Beltsville, MD (mimeo.), Div. of Cereal Crops and Diseases, Plant Industry Sta., Beltsville, MD.

46. Smith, E. F. 1909. Corn as a means of disseminating *Bacterium stewartii.* Science 30:223-224.

47. Smith, E. F. 1898. Notes on Stewart's sweet-corn germ, *Pseudonomas stewartii* n.sp. Proc. Am. Assoc. Adv. Sci. 47:422-426.

48. Smith E. F. 1914. Stewart's disease of sweet corn (maize). Pages 89-147 in: Bacteria in Relation to Plant Diseases. Carnegie Institute of Washington, Washington, D.C.

49. Stevens, N. E.1934. Stewart's disease in relation to winter temperatures. Plant Dis. Reptr. 18:141-149.

50. Stewart, F.C. 1897. A bacterial disease of sweet corn. New York Agric. Exp. Stn. Bull. 130:422-439.

51. Suparyono and Pataky, J. K. 1989. Influence of host resistance and growth stage at the time of inoculation on Stewart's wilt and Goss's wilt development and sweet corn hybrid yield. Plant Dis. 73:339-345.

52. Suparyono and Pataky, J. K. 1989. Relationships between incidence and severity of Stewart's and Goss's bacterial wilts and yield of sweet corn hybrids. Crop Prot. 8:363-368.

53. Thomas, R. C. 1924. Stewart's disease or bacterial wilt of sugar corn. Ohio Agric. Exp. Stn. Mon. Bull. 9:81-84.

54. White, D. G. 1999. APS Corn Compendium. APS Press. St. Paul, MN. 128 p.

55. Woods, T. I., Israel, H. W., and Scherf, A. F. 1981. Isolation and partial characterization of a bacteriophage of *Erwinia stewartii* from the corn flea beetle *Chaetocnema pulicaria.* Prot. Ecol. 3:229-236.

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