Epidemiological study of foliar diseases with strip intercropping rotation in Iowa

Kayimbi Mendha Tubajika

Iowa State University

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Epidemiological study of foliar diseases with strip intercropping rotation in Iowa

Tubajika, Kayimbi Mendha, Ph.D.

Iowa State University, 1992
Epidemiological study of foliar diseases with strip intercropping rotation in Iowa

by

Kayimbi Mendha Tubajika

A Dissertation Submitted to the Graduate Faculty in Partial Fulfillment of the Requirements for the Degree of DOCTOR OF PHILOSOPHY

Major: Plant Pathology

Approved:

Signature was redacted for privacy.

In Charge of Major Work

Signature was redacted for privacy.

For the Major Department

Signature was redacted for privacy.

For the Graduate College

Iowa State University
Ames, Iowa

1992
DEDICATION

To my parents, Evariste Mbolela and Anastasia Tshingutu; my wife, Mathilda Lusamba; my sons, Thierry Mbolela Tubajika and Claude Francois Mukendi Tubajika; and my daughter, Carina Kalanga Tubajika
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SYMBOLS AND ABBREVIATIONS

AUDPC  Area under the disease progress curve
ANOVA  Analysis of variance
D      Doublet
E(D)   Expected doublet
E(U)   Expected runs
ln     Natural logarithm
LRA    Linear regression analysis
m      Number of infected plants in a row
n      Number of row
N      Total number of plants in the row
r      Coefficient of correlation
R²     Coefficient of determination
S      Distance from inoculum source
S-shape Skewed shaped
Su     Standard deviation of runs
t      Time
U      Number of runs
y      Disease intensity
Z      Standardized statistic
GENERAL INTRODUCTION

The four primary measures for foliar disease control in agronomic crops are crop rotation, clean tillage to bury the debris from the previous crop, resistant varieties, and fungicides. These are especially appropriate for those diseases where the pathogen overwinters in the infected crop debris (Sumner et al., 1981; Boosalis et al., 1981). Rotation from the susceptible crop to a nonsusceptible crop for one to two years will usually allow the crop debris from the prior crop to decompose before the susceptible crop is replanted (Cook et al., 1978). During decomposition of the debris the pathogens in the debris are commonly killed by antagonistic microorganisms in the soil. Clean plowing with a moldboard plow will bury the debris and place a soil barrier between the inoculum source in the debris and the foliage of the susceptible crop. The plowing will also accelerate decomposition of the crop debris in the biologically active soil environment. Disease resistance is always a desirable disease control measure, but unfortunately resistance is not available for all diseases, and may be unstable, especially under severe disease pressure. Fungicides are not a feasible alternative for the culture of field crops, because effective fungicides may not be available or registered, are cost prohibitive, or are not an environmentally attractive alternative.

Conservation tillage is a crop management practice that has become requisite for most farmers, because the crop debris left on the soil surface reduces soil erosion by wind and water, reduces water evaporation, and increases infiltration of water in the
soil (Andrews and Kassam, 1976; Boosalis et al., 1981). Therefore, the control of most foliar diseases has relied on crop rotation and the use of resistant varieties where available.

Strip intercropping can be understood here as a production of two or more crops simultaneously in strips wide enough to permit independent cultivation but narrow enough for crops to interact (Andrews and Kassam, 1976). This practice not only stops soil erosion, but also increases yield (Whigham, 1985; Francis et al., 1986) through more efficient use of resources such as light (Allen et al., 1976). Strip planting exposes more rows of maize to receive more sunlight, even though some soybean rows are shaded by the maize.

Intercropping can also be understood here as a growing of two or more crops simultaneously in the same field during the same year. Land equivalent ratio (LER) (Andrews and Kassam, 1976), relative yield total (Andrews and Kassam, 1976; Whigham, 1985; Francis et al., 1986; Willey, 1979a,b), and gross income from strip crops have been used to evaluate the efficiency of the intercropping in several instances.

Considered as a primitive practice of production in developing countries, intercropping is practiced today in some developed countries (Willey, 1979a). Yield increase and land use efficiency are the keys to making intercropping more important than single cropping (Andrews and Kassam, 1976; Willey, 1979a; Whigham, 1985; Francis et al., 1986).
The increase of maize yield on strip intercropping over solid planting has been repeatedly reported by Whigham (1985), Francis et al. (1986), and Andrews and Kassam (1976). They found that when alternating strips of maize with strips of soybeans the maize yield is enhanced and the yield is even higher when maize is bordered by soybeans than when bordered by maize itself (Lang and Hilst, 1949).

Strip intercropping, which is the crop management practice being studied, involves three-year crop rotations with one edge of every strip contiguous to the debris of the same crop from the prior year. Strip intercropping usually requires management practices of no-tillage, ridge tillage, or minimum tillage to maintain the proper spacing of the strips; therefore, the infected crop debris will be on the soil surface unless the stems and foliage were removed for animal feed or bedding. Clean removal is almost impossible. Burning the debris is possible, but often is environmentally or agronomically unwise. Strip intercropping may lead to serious disease problems if inocula of the pathogens spread from crop debris into contiguous strips and develop into economic problems (Kimber, 1967; Sanderson, 1974; Brown et al, 1978). Soil-borne root infecting pathogens, e.g. those fungi causing root rots, wilt and vascular diseases, and stem and crown rots, have the potential to be more serious in the row contiguous to the strip planted to the same crop the prior year. The soil shared by the outside rows will have roots of the same crop for two of the three years.
Stalk rot of maize is a stress regulated disease. The causes of stalk rots are fungi and include several *Fusarium* spp., *Stenocarpella maydis* (Berk.) Sutton, and *Colletotrichum graminicola* (Ces.) G. W. Wils (Christensen and Wilcoxson, 1966; Hooker, 1976; Lipps, 1983a,b, 1985; Naylor and Leonard, 1977; White et al., 1979). These are soil-borne and debris-borne pathogens. The inoculum can be air borne. Infection of the plant is normally not a limiting factor, as the pathogens may be isolated easily from the stalks, crowns and roots of maize. Stalk rot usually does not develop until some stress is imposed upon the plant and the stress results in lowered food reserves in the stalk and a concomitant loss of resistance to the resident pathogen (Agrios, 1988; Dodd, 1980a,b; Halbert et al., 1935). The factors of the plant and environment that result in a decreased photosynthate supply for the stalk, root, and crown tissues will increase stalk rot if they are imposed after pollination (Dodd, 1977; Halbert et al., 1935; De Turk et al., 1937). These may be defoliating insects, virus diseases, drought, cloudy weather, and mineral deficiencies (Agrios, 1988; Shurtleff, 1980; Halbert et al., 1935; Vanderplank, 1984). The kernels developing after pollination are a photosynthate sink of highest priority, and photosynthates will be partitioned to the developing ear at the expense of the root, crown, and stalk tissues (Halbert et al., 1935; Mortimore and Ward, 1964). The kernel sink is directly related to the number of kernels pollinated. Plant population is another form of stress (Mortimore and Ward, 1964; Dodd, 1980a,b). The strip intercropping system could influence stalk rot by providing a source of debris-borne inoculum, and rows within
the strip with different levels of leaf disease, insect exposure, plant crowding, and light exposure.

The objectives of this research were to:

1) Determine the crop diseases that could be potential problems in strip intercropping rotation of maize, soybean and oat.

2) To determine whether agronomic practices (tillage, weed control, N fertilizer, crop rotation) may affect disease development

3) To describe the spatial and temporal spread of pathogens from the residue source (debris) across the strip.

Explanation of Dissertation Format

This dissertation follows the alternate format described in the Iowa State University Graduate College Thesis Manual. The dissertation has been divided into three sections; each is in the form of a complete manuscript that will be submitted to a professional journal.

The three sections describe experiments conducted to determine the potential diseases in strip intercropping rotations of maize, soybean, and oat and to model disease spread across the strips. Section I reports on an analysis of the temporal and spatial pattern of Septoria blight of oat as influenced by strip intercropping of maize, soybean, and oat. Section II examines the effect of tillage and strip intercropping on incidence and severity of brown spot and bacterial blight disease of soybeans. Section
III presents the disease incidence and severity in maize grown with strip intercropping.

The three sections of the dissertation are preceded by a general introduction and a literature review and are followed by a general discussion. References cited in the General Introduction, Literature Review, and General Discussion are listed in the Additional Literature Cited following the General Discussion. The Appendix contains supplemental tables and figures not included in any section.
LITERATURE REVIEW

Crop Residue: Source of Inoculum

Crop residue is a habitat for plant pathogens that may use it as a food base for growth, reproduction (sporulation), and survival (Boosalis et al., 1967; Cook et al. 1967 [in Oschwald, 1978], Cochrane, 1949; Janes et al., 1955). Chisel plowing and no tillage after maize culture were reported by Burns and Shurtleff (1973) to increase survival of race 0 of Helminthosporium maydis. Bacterial pathogens were reported by Schuster et al. (1972) and Weihing and Vidaver (1967) to survive in crop residues, and these bacteria could be spread by wind and could develop an epidemic where favorable conditions are found. Crop residues left on the soil surface or partially incorporated into the soil have been found to decompose slowly. Pathogens can be spread more easily from surface residue by wind and/or splashing rain (Cook et al., 1978).

The dispersal mechanisms of fungal spores have been discussed by several researchers. Some pycnidiospores of Septoria spp. released from wheat debris (as is the case of Septoria blotches of wheat) are splash dispersed, while some may be wind borne or dispersed at short distance (Shipton et al., 1971; King et al., 1983). Although the ascospores produced in residues are likely dispersed greater distances than the pycnidiospores produced at the same focus (Sanderson, 1974; Brown et al., 1978; Sanderson and Hampton, 1978), some pycnidiospores, ascospores and conidia
are weather dependent for long distance dispersal (Brown et al., 1978; Sanderson and Hampton, 1978).

The debris on and in the soil not only constitutes the source of inoculum allowing the pathogen to invade the host plant when the new crop is planted, but also is used by the antagonistic microorganisms that develop from it to destroy or suppress the pathogen saprophytically (Nyvall and Martinson, 1983). This was true for some debris-borne pathogens of maize such as *Colletotrichum graminicola* (cause of anthracnose), *Kabatiella zeae* (cause of eyespot), *Corynebacterium nebraskense* (cause of Goss' wilt), *Cercospora zeae-maydis* (cause of gray leaf spot), *Helminthosporium turcicum* and *H. maydis* (cause of Northern and Southern leaf blight, respectively), and *Phyllosticta maydis* (cause of yellow leaf blight) (Nyvall and Martinson, 1983). *Cercospora zeae-maydis*, *C. graminicola*, *H. turcicum* and *H. maydis* were reported to survive in crop residue under conservation and reduced tillage (Boosalis et al., 1967, 1981; Hilty et al., 1979; Phillips et al., 1980; Roane et al., 1974), where they constitute the primary source of inoculum.

Conservation tillage and minimum tillage may either increase, decrease, or have no impact on plant disease (Sumner et al., 1981; Boosalis et al., 1981). This may be due to overwintering of the pathogen in crop residue, to antagonistic activities of microorganisms in soil, and growth of roots. Tillage may stimulate pathogenic fungi to break dormancy and thereby starve in the absence of a host plant. Plant disease
may be stimulated under some conditions by tillage practices such as stubble mulching (Sewel, 1965; McCalla and Lavy, 1967).

Cook et al. (1978) reported that root and leaf pathogens that survive and sporulate in crop residue are reduced tillage dependent. Soil borne pathogens could be reduced or restricted when the infected debris was buried to certain soil depth. *Helminthosporium maydis* and *Septoria tritici*, which are debris-borne pathogens, were controlled in this way (Cook et al., 1978). Although some pathogens could be controlled with reduced tillage practices, Moore (1978), in his study on the influence of no tillage on take all of wheat, found that disease control could be achieved when the plots were tilled but not when they were not tilled. This finding was made previously by Cunningham (1967). Cunningham (1967) found that deep plowing had an effect on foot rot and root rot of spring wheat in Ireland. The level of take all was reduced when the plots were tilled to a 20-cm depth compared to a 10-cm depth. More pathogen growth was observed at 10 cm depth from the basal step and adjoining root of the previous wheat crop (Hornby, 1975).

Tillage practice modifies the crop susceptibility and plant microenvironment (Yarham, 1975; Palti, 1981; Yarham and Norton, 1981). Yarham and Norton (1981) found a higher level of leaf diseases caused by powdery mildew (*Erysiphe graminis*) and leaf rust (*Puccinia recondita*) on wheat in tilled plots than in no tillage plots. They attributed this to a lower rate of nitrogen mineralization in no-tillage plots leading to less vigorous plant growth, which rendered the tissue less susceptible to
development of these obligately parasitic pathogens. The reduced plant growth provided a poor microenvironment within the crop canopy for pathogen development (Palti, 1981). It was suggested by Yarham and Norton (1981) that tillage that increases nitrogen mineralization may also lead to increased levels of barley scald because the application of nitrogen in soil increases the severity of barley scald (Jenkyn and Griffiths, 1978). Sewel (1965) indicated that tillage may release carbon dioxide from the soil and increase oxygen supply, which may increase total microbial activity with a subsequent antagonistic effect on the disease organism. McCalla and Lavy (1967) reported that by mixing residues into the soil, tillage may increase growth and number of microorganism. In a study by Waksman (1952), it was observed that tillage could increase the number of microorganisms from 2 million to 12 million per gram of soil in 24 days. Gamble et al. (1952) indicated that when oxygen and more organic matter is found in the top 6 inches of soil, soil fungi may be favored. Bacteria, actinomycetes, fungi, denitrifying bacteria, earthworms, and nematodes were augmented in the surface of tilled plots with stubble mulch.

Brooks and Dawson (1968), in their study on the influence of direct drilling of winter wheat on the incidence of take all and eyespot, showed that take all could be reduced in no-tillage conditions compared to tilled plots. This was confirmed in Scotland with continuous barley, where the incidence of take all was limited (Lockhart et al., 1975).
Crop rotation and resistant varieties are used to reduce and control residue-borne pathogens. Boosalis et al. (1981) successfully controlled crop diseases by combining herbicides, crop rotation, and a fallow. Stalk rot of sorghum was reduced in ecofallow conditions when compared to no tillage. Snyder et al. (1959) obtained a reduction of bean root rot fungus by adding wheat and barley straw. It has been demonstrated that Fusarium becomes a poor competitor with a deficient nitrogen supply (Snyder et al., 1959). Kimber (1967) demonstrated that the retention of crop residue may affect the subsequent crops, because the plants are affected by lack of nitrogen fertilizer in the soil.

Elliot et al. (1978) observed a reduction in yield, which may be attributed to toxic compounds leached from crop residues or produced by microorganisms during residue decomposition. Little, however, is known about the compounds and mechanisms operating. Elliot et al. (1978) detected the presence of phytotoxins when the crop seedlings were in direct contact with residues.

Cook et al. (1978) found that when the debris remained in contact with the seed and seedling, the young plant may be affected by toxins produced from the decomposing debris and yield is reduced. A study by Cochran et al. (1977) demonstrated that by avoiding the contact of debris with seed or germinating seed, damage from residues can be reduced. Other researchers (Amin and Sequiera, 1966; Hartnett and Lorbeer, 1971; Boyd and Phillips, 1973; Patrick et al., 1963; Patrick and Toussoun, 1965) reported that the decomposing residues may affect lettuce and
other crops by producing toxins that induce stunting, root necrosis, and plant malformation. Langdale and Giddens (1967) detected four phenolic compounds (protocatechnic, ferulic, p-coumaric acids and vanilin) that may inhibit maize growth in seedling stages in minimum tillages.

The presence of crop residues on the soil surface may affect the control of weeds (William and Wicks, 1978; Purvis et al., 1985) and reduce plant establishment (McKeown and McCulloch, 1962; Macadam and Southwood, 1968). It has been reported that when crop residues are left on the soil surface and herbicide is applied to control weeds, the herbicide may be inefficient and may not reach the target weed because of the presence of residues (Williams and Wicks, 1978). This was the case with atrazine in North Carolina when 85% of crop residue covered the soil. In their study on response of weeds to crop stubbles, Purvis et al. (1985) reported the release of toxin from stubble that may reduce or limit weed growth.

McKeown and McCulloch (1962) and Macadam and Southwood (1968) found that when crop residues are left on the soil surface they affect the plant stand and therefore may affect crop yield because of reduced plant stand and root growth. Seed depth and spacing may vary considerably within the same crop and as a consequence, crop yield is affected.

Although crop residue presents a disease and pest threat to the crop, it has many advantages. Yasar and Wittmuss (1976) reported that the surface residues reduced both wind and water erosion and water loss. When large amounts of crop residues
are left on the soil surface, they may protect the soil against weed emergence and limit light needed by the weed (Felton et al., 1987). In Nebraska, up to 58% of the 231 mm precipitation could be saved when no tillage is used compared to conventional tillage where 46% of the precipitation is normally available (Yasar and Wittmuss, 1976). Harold et al. (1963) in their study on no-tillage maize found that residues limited water run off and reduced evaporation. They also observed an increase in maize yield of 570 kg/ha in no-till when compared to conventional tillage. This increase was attributed to increased soil moisture with no tillage.

The response of soybean cultivars to residue is environmentally dependent (Boerma, 1979). Little effect of crop residues on soybean yield was reported by Sanford (1982). Wischomier and Smith (1978) demonstrated that when equal amounts of surface residue are left on fields of maize and soybean to prevent soil erosion, soybean residue is likely to leave the soil 25% more susceptible to erosion than does the maize. However, Gregory (1968) indicated no difference in erosion susceptibility when maize and soybean residues were compared.

**Intercropping System: Disease Control**

Intercropping, or growing two or more crops simultaneously has been used more in developing countries than developed countries to ensure against total crop failure under unfavorable weather conditions or pest epidemics, to increase total productivity per unit of land area, and to make rational use of farming inputs (Willey and Osiru, 1972; Osiru and Willey, 1976; Monta and De, 1980; Andrews and Kassam, 1976).
Intercropping leads to a change in the microclimate of the canopy and, therefore, influences the succession and increase of insect pests. Intercropping has been considered as a disease control practice because of its effect on soil microorganisms (Kayumbo, 1976). In his study on pest control in a mixed cropping system, Kayumbo (1976) found that despite the disease pressure on mixed crops, the crops may be resistant to pathogen attack.

Gerard (1976) reported the presence of several crops on a field may possibly reduce pathogen multiplication and dissemination to infect the crop compared to disease development in a pure crop stand. The idea of intercropping is complex in that the cropping pattern provides less habitat for some major pests than in single cropping and, also, if one crop is damaged the others would provide a source of food and income.

Radke and Hagstrom (1973, 1976) indicated that rows of maize alternated with rows of soybean serve as a windbreak for the soybean. Maize rows also reduce wind speed and water evaporation; therefore, yield, total leaf area, and dry matter could be increased.

Dalal (1977), Akhandra et al. (1978), Crookston and Hill (1979), and Singh et al. (1973) reported an increase in yield of maize when grown in association with soybean, but yield of soybean is suppressed when compared to pure stands of soybean.
It was observed by several authors that yields of maize and soybean were reduced when the two crops were intercropped compared to when they were planted alone (Etheridge and Helm, 1924; Hughes, 1931; Kinney and Robert, 1924). This reduction was attributed to competition for soil moisture. In later studies, when maize and soybean were intercropped an increase in maize and soybean yield was observed. This may be due to the maximum utilization of light and natural resources (Willey and Osiru, 1972; Baker and Yursuf, 1976; Monta and De, 1980; Agboola and Fayemi, 1971).

Moreover, Thompson et al. (1976), Monta and De (1980), and Crookston and Hill (1979) attributed the soybean yield reduction to competition for light because soybeans were shaded by maize. Shading reduces soybean photosynthesis (Johnston et al., 1969; Trenbath, 1976), nodule formation and consequently N$_2$-fixation (Mann and Jaworski, 1970; Weber, 1968). Wahua and Miller (1978) increased the yield of sorghum grain by intercropping soybeans with two sorghum cultivars, but soybean yields were reduced by 17-75% depending on the soybean cultivar used.

Agboola and Fayemi (1971), Santa-Cecilia and Vieira (1978), and Monta and De (1980) observed that when a legume was intercropped with maize, grain yield of the legume was suppressed by maize due to shading. Agboola and Fayemi (1971) and Enyi (1973) stressed the importance of intercropping over pure culture.

Until 1924, it was observed by several authors that yield of maize and soybean was reduced when the two crops were mixed compared to when they were planted
alone in pure stands (Etheridge and Helm, 1924; Hughes, 1931; Kinney and Robert, 1924). This reduction was attributed in part to competition for soil moisture.

Etheridge and Helm (1924) planted rows of soybean alternating with rows of maize; yields of both were increased in contrast to an older method of broadcast sowing or drilling soybean between or within row hills. The highest yield of maize occurred when maize was bordered by a fallow area; maize bordered by soybean had greater yields than pure stands of maize. When maize was planted in 36-inch rows alternated with soybean, Alexander and Genter (1962) obtained a maize yield increase of 30% compared to yields in a pure stand of maize. Pendleton et al. (1963) increased maize yields 16% and 20% when planted in strips of four 40-inch and six 24-inch rows of maize, respectively, compared to yields in pure stands of maize. However, soybean yields were reduced 20% regardless of row width in the alternate strips. Alternating strips of maize and soybean in Illinois (Lang and Hilst, 1949) increased maize yield from 7 to 27 bushel per acre. Soybean yield was slightly depressed when soybean was bordered by maize compared to when bordered by soybean.

Radke and Hagstrom (1973) reported a 4% increase of soybean yield in 1969 and 5% in 1970 when soybeans were intercropped with maize. In their study double rows of maize were alternated with soybean rows at two locations in Minnesota. They attributed the yield increase to reduction of transpiration on the first seven or eight rows on the windbreak side.
Radke and Burrows (1970) reported that soybeans sheltered by a maize windbreak grew taller, produced more dry weight, had a larger leaf area index and produced higher grain yields. The soybean plants on the windward side of maize windbreaks were not as productive as the windbreak-sheltered soybeans. Root competition for nutrients and moisture may have contributed to lower soybean yield.

Epidemiology: Temporal and Spatial Analysis of Epidemics

Epidemiology can be described as the study of disease increase or change with time. Models of disease progression have been developed by Vanderplank (1963), Richards (1969), and Madden (1980) to quantify disease over time.

The model is simply a description of reality (Edminster, 1978). This description may be qualitative or quantitative (mathematical) (Campbell and Madden, 1990). Mathematical modeling of epidemics and plotting the disease progress curves date back to the pioneering work of Vanderplank (1960, 1963, 1968). Mathematical formula have been used to understand the events and make decisions about them (Kranz, 1990). Disease progress curves can be plotted to show disease development over time. These curves allow visualization, graphic analysis and comparison of different epidemics. Usually, disease intensity assessments are plotted versus time.

Different models were reviewed and discussed (Gilligan, 1985; Kranz and Royle, 1978; Vanderplank, 1963; Fleming and Bruhn, 1983; Jeger, 1986a,b). The choice of model depends on the objectives of the study and generally the simplest model that satisfies the objectives is the best choice.
Users of models assume that the environment is uniform in space and constant in
time, the pathogens (or organisms) are distributed uniformly in space, and finally, all
organisms are identical (Campbell and Madden, 1990). Logistic, Gompertz,
monomolecular, and exponential models are the most commonly used (Campbell and
Madden, 1990; Vanderplank, 1963). Those disease progress models can be linearized
with ln(y/1-y), - ln(-ln(y)), ln (1/1-y), and ln(y), respectively.

Analysis of variance (ANOVA) and linear regression analysis (LRA) are of
common use in epidemiology (Netter et al., 1985). LRA is used to analyze the
epidemic curves but when S-shape or other curves are found the data often are
transformed to a straight line.

An exponential model is used where there is no limitation of the disease increase.
Its linearized form is [ln(y)] indicating that the higher the disease level the higher the
disease increase (Vanderplank, 1963). A monomolecular model is used when disease
tissue does not contribute to additional disease (Vanderplank, 1963; Thresh, 1983;
Richards, 1969). Its linearized form is [-ln(-ln(y))]. A logistic model describes the
proportionality between level of disease and number of healthy plants with the rate of
disease increase (Vanderplank, 1963; Pennypacker et al., 1980); this indicates that
when the plants are more infected, with time there will be less green plant tissue
available to be infected. Its linearized form is [ln(y/1-y)]. The Gompertz model
describes a skewed curve, that the pathogen loses equal proportions of its power to
increase in an equal small interval of time (Campbell and Madden, 1990; Waggoner, 1986).

Area under the disease progress curve (AUDPC) is also used if the above models do not describe the epidemics (Shaner and Finney, 1977). The disease intensity (y) is usually plotted versus time (t) using the following formula.

\[ AUDPC = \sum_{i}^{n-1} \left( \frac{y_i + y_{i+1}}{2} \right) (t_{i+1} - t_i) \]

where \( n \) = number of sampling times

\( y \) = disease intensity

\( t \) = time

\( t_{n-ti} \) = total time duration of the epidemics.

AUDPC helps when comparing different curves from different epidemics (Fry, 1977).

Several models have been presented to describe disease spread (Stranberg, 1973; Campbell and Pennypacker, 1980; Gilligan, 1982; Madden et al., 1982; Martin et al., 1983). The disease gradient can be affected by a change in environment, soil fertility (Gregory, 1968), and incoming inoculum (Kiyosawa and Shiyomi, 1972). The dispersal gradient can be observed by looking at the shape and steepness of a gradient (Gregory, 1968; Thresh, 1976). Different gradients are compared to determine the role of inoculum in epidemics (Johnson and Powelson, 1983), to locate source of
inoculum, and to test the model of disease spread (Minogue and Fry, 1983a,b; Jeger et al., 1983).

Disease gradients are usually plotted by using disease incidence or severity (y) versus the distance (x) over which change occurs. Data are transformed when straight lines cannot be reached. There are two commonly used models to plot disease gradient. Gregory (1968) proposed the power law which was later called the inverse power law by Minogue (1986). This model assumes that the amount of disease is inversely proportional to some power of the distance from the inoculum source. The second is an exponential model called the Kiyosawa and Shiyomi model (1972) and assumes that the amount of disease (y) decreases exponentially with distance from the source. Later additional models were proposed by Berger and Luke (1972) and Minogue and Fry (1983a) using a \( \ln\left[\frac{y}{(1-y)}\right]-\log(s) \) and \( \ln\left[\frac{y}{(1-y)}\right]-\text{linear S} \) transformation, respectively (y = amount of disease and S = distance from the source).

Various methods have been proposed and described to determine the distribution or pattern of diseased plants in the field. Vanderplank (1946) proposed doublet analysis as a technique to determine how the pathogen spreads in field plots. A doublet can be understood as a succession of two adjacent symptomatic or diseased plants. This technique was modified by Converse et al. (1979) and Freeman (1953). Furthermore, ordinary run analysis was proposed by Gibbons (1971) to determine random or non-random distribution. An ordinary run was defined as a succession of
one or more diseased or healthy plants (Gibbons, 1971; Madden et al., 1982; Campbell and Madden, 1990). Run analysis was preferred over doublet analysis after Madden et al. (1986) compared doublet analysis to ordinary runs using maize dwarf mosaic virus on sweet maize as the pathosystem. Madden et al. (1986) proposed several formula that could be used to calculate the expected number of doublets and number of runs. A row of plants was considered to have "non-random" sequence of infected plants (in case of doublet) and a sequence of infected and healthy plants if $Z(u)$ or $Z(d)$ was less than -1.64 ($P = 0.05$) and 2.33 ($P = 0.001$) (Campbell and Madden, 1990).

\begin{align*}
(1) \quad E(u) &= 1 + \frac{2m(N-m)}{N} \\
(2) \quad S(u) &= \left[ \frac{2m(N-m)[2m(N-m)-N]}{N^2(N-1)} \right] \\
(3) \quad Zu &= \frac{[u-E(u)]}{S(u)}
\end{align*}

$Z =$ standardized statistic  
$n =$ number of row  
$E(u):$ Expected runs  
$S(u):$ Standard deviation of runs  
m = number of infected plants in a row  
$N =$ total number of plants in the row
SECTION I. ANALYSIS OF TEMPORAL AND SPATIAL PATTERN OF SEPTORIA BLIGHT OF OAT AS INFLUENCED BY STRIP INTERCROPPING OF MAIZE (ZEA MAYS L.), SOYBEAN (GLYCINE MAX. L.) AND OAT (AVENA SATIVA L.)
Diseases were assessed in oat strips associated with strip intercropping that involved oat, maize, soybean strips and sequences of rotation. Two multifactorial strip intercrop experiments at the McNay Research Center and two commercial strip intercrop fields near Alta Vista, Iowa, were studied in 1990. An additional site near Ames was studied in 1991. Disease was quantified in the outside rows of strips in 1990 and in five selected rows of each strip in 1991. Septoria blight (incited by *Septoria avenae* Frank) was the primary disease that appeared associated with the proximity to oat debris from the prior year.

The highest incidence and most severe symptoms of Septoria blight were in the row that was contiguous with land planted to oat the prior year. The least disease was in the row farthest from the potential source of inoculum. Disease gradients across the strips were linear and the disease progress in a row was usually linear adjacent to the debris source and monomolecular at a distance from the debris. Primary inoculum appeared to be very important for perpetuating the epidemic. Disease patterns in the strips were random. Intensive tillage was associated with higher disease incidence than with no tillage management practices. Tillage operations evidently caused or allowed the spread of oat debris and potential inoculum into adjacent strips.
INTRODUCTION

Oat is an old crop that has been widely cultivated since before the Christian era (Vanderplank, 1968). Losses from diseases are greater in oat than in other small grains because the environmental conditions favoring disease development occur when oat is planted in the spring, especially in warm and humid climates (Simons and Murphy, 1952). In 1946 and 1947 *Helminthosporium victoria* caused losses up to 25% and barley yellow dwarf virus caused losses of more than 15%.

Septoria disease of oat is a problem in oat crops worldwide (Meehan and Murphy, 1949; Stanton, 1952). The pathogen, *Septoria avenae* Frank, overwinters in the field as mycelia, micropycnidia, and pycnidia in oat debris and is disseminated by wind and rain to new plants when the same crop is replanted (Huffman, 1955). Environmental conditions such as cool temperatures and adequate moisture at the time micropycnidia are formed are critical for the increase of inoculum. Huffman (1955) found that hot and dry weather delayed pycnidial development in the field. The teleomorphic stage, *Phaeosphaeria avenaria* (G. F. Weber) O. Erikson, evidently does not function in survival and perithecia and ascospores form late in the spring (Huffman, 1955). Several researchers (Noble and Montgomerie, 1954; Lund and Shands, 1955, 1956) reported the pathogen to be seed borne. Knowledge of the type of disease distribution and spread in the field is important in analyzing yield loss. Little is known about the spread of the primary and secondary inoculum of *S. avenae*. Temporal disease development and the spatial pattern of disease could be used to
determine the spread of the fungus as described elsewhere (Berger and Luke, 1979; Gregory, 1968; Minogue, 1986; Minogue and Fry, 1983a,b; Cliff and Ord, 1981; Reynolds and Madden; 1988).

Strip intercropping consists of two, three or more crops planted in repetitive narrow strips, 4 to 5 meters wide, that will accommodate current equipment. The crops are rotated among the strips according to a prescribed sequence, sometimes with intercropping of a legume with strips of small grains. Strip intercropping requires good management, careful equipment operation, and reduced tillage practices that allow for permanent sites. The benefits of strip intercropping are increased yields compared to single cropping (Andrews and Kassam, 1976; Francis et al., 1986; Boosalis et al., 1976; Whigham, 1985) and possibly less soil erosion (Yassar and Wittmuss, 1976; Laflen et al., 1985).

Foliar diseases have become problems when infected crop debris is allowed to remain on the surface of the soil for erosion control and the same crop is replanted into the field. Many pathogens overwinter in the crop residues (Cook et al., 1978; Kimber, 1967; Brown et al., 1978), including S. avenae (Huffman, 1955), which provide large amounts of inocula for infecting the next crop. Crop rotation normally minimizes the disease threat because the pathogens die as the crop debris decomposes (Cook et al., 1978). Strip intercropping may nullify the disease control benefits of the crop rotation because one edge of every strip is contiguous with debris of the same crop from the prior year.
The objectives of this study were: 1) to identify the principal pathogens of oat associated with strip intercropping; 2) to evaluate tillage and weed control practices on disease incidence and severity; and 3) to describe spatial and temporal spread of the pathogens from the residue source across the strip.
MATERIALS AND METHODS

All data were collected from strip intercrop experimental plots and commercial fields that were managed with strip intercropping (a maize, soybean, oat sequence). At the McNay Research Center and Alta Vista, oat was planted with alfalfa.

Experimental Design

McNay Research Center

Five multifactorial split-plot-design experiments with four replications were established in 1988 at Iowa State University's McNay Research Center near Chariton in south-central Iowa on poorly drained haig series soil (fine, montmorillonitic mesoic type Argiagnoll), with less than 1% slope. The treatments in the experiments were: Experiment 1, tillage and crop; Experiment 2, tillage, weed control practice, and crop; Experiment 3, N fertilizer (on maize) and alternate rotation crops including oat; Experiment 4, weed control practices, N fertilizer (on maize), and crop; and Experiment 5, tillage, N fertilizer (on maize) and crop. Disease data were collected in all experiments in 1990 and it was found that weed control practices (herbicide, cultivation treatments, and no herbicides) and N fertilizer application rates in maize were insignificant factors in disease development, but tillage was significant. Therefore disease data are presented only for Experiments 1 and 5 in 1990 and these were the only experiments studied in 1991.
Experiment 1  The main plot factor was tillage and four replications were employed. Treatments used were conventional tillage (fall moldboard plowing and two secondary tillage operations in the spring), reduced tillage (fall chisel plowing and one secondary tillage in the spring), and no preplant tillage. Each tillage plot was 19 m wide and 12.2 m long. Across the width there were five crop strips, each 3.8 m wide. The crop sequence was a three-year rotation of maize to soybean to oat plus alfalfa and then back to maize. In one year the five strips were planted to oat plus alfalfa, soybean, maize, oat plus alfalfa, and soybean. In the next year the crops across the tillage plot were maize, oat plus alfalfa, soybean, maize, and oat plus alfalfa. The third year sequence was soybean, maize, oat plus alfalfa, soybean, maize. In 1988, each tillage plot was randomly started in one of the three cropping patterns.

Maize and soybean strips were 5 rows (0.76 cm spacing) across and oat strips had 20 rows (18 cm spacing) per strip. In 1990 rows 1 and 20 were sampled. In 1991 rows 1, 5, 10, 15, and 20 were sampled. All experiments were planted to Ogle oat cultivar on 6 April and 8 April during 1990 and 1991, respectively. Maize and soybeans were planted simultaneously, 30 and 44 days after oat planting for 1990 and 1991, respectively. The herbicide 2,4-D was applied to the maize and oats before maize was planted, and alachlor was band applied to the maize and soybeans to control weeds. Data collection started on 20 June in 1990 and proceeded biweekly. In 1991, weekly data collection started on 25 May.
Experiment 5 The main plot factor was tillage as described for Experiment 1 and four replications were employed. Each tillage plot was 48.8 m long and 19 m wide. Five strips of crops as described for Experiment 1 were planted. The maize strips were divided into four subplots (perpendicular to the rows), with each subplot 12.2 m long. Randomly selected subplots received additional N at 0, 33.7, 67.3, and 101.0 kg/ha. All other factors, including data acquisition, were the same as described for Experiment 1.

Alta Vista Farms

Data were collected from two commercial fields located in northeast Iowa near Alta Vista that had been strip intercropped since 1989. One was at the Mike Reicherts farm and the other was at the Thomas Frantzen farm. The soil type was a fine, loamy, mixed mesic type arguidoll Cresco series soil at both farms. Both used a rotation sequence of maize, soybean and oat plus alfalfa and both practiced ridge till farming.

Reicherts farm The crop strips were 4.56 m wide. Thirty rows of oat on 15.24 cm spacing were planted per strip. Rows 1 and 30 were subject to removal during cultivation of the adjacent strips of maize and soybean, thus they were not included in the data collection. Row 2 was called row 1 and row 29 was considered as row 28. Soybean and maize were planted in 6-row (76 cm spacing) strips. Oat cultivar Don was planted on 18 April in 1990 and 25 April in 1991. Maize was
planted on 6 May in 1990 and 12 May in 1991. Soybeans were planted on 30 May in 1990 and 3 June in 1991. Data were taken on 16 data strips using rows 1 and 28 (the outer rows in each strip) in 1990 and rows 1, 7, 14, 21, and 28 in 1991. Each strip was a replication. Data collection began on 30 June in 1990 (and continued biweekly) and 29 May in 1991 (and continued weekly).

**Frantzen farm**  
The crop strips were 3.76 m wide. A strip was planted to 23 rows of oat (15.24 cm spacing), or four rows of maize or soybean with 94 cm row spacing. The oats (cultivar Don) were planted on 6 April, 1990 and 6 April, 1991. Maize was planted 28 April in 1990 and 12 May in 1991. Soybean was planted 14 May in 1990 and 28 May in 1991. Data were collected on 16 strips of oats. Each strip was a replication. Data collection began on 13 June in 1990 (and continued biweekly) and 29 May in 1991 (and continued weekly).

**Ames**

**Johnson farm**  
A strip intercrop experiment was established by the Entomology Department at the Iowa State University's Johnson farm located near Ames. The experiment had six blocks (replications) of strips. Each block was 33.5 m wide and 33.5 m long. Eleven 3.05 m wide cropping strips were established in each block with a repetitive sequence of maize, soybean, and oat plus alfalfa in adjacent strips. The rotation was maize, soybean and oat plus alfalfa. The center nine strips in each block were used for data collection. The width of each strip accommodated 20 rows of oats.
(15.25 cm between rows) and four rows (each 76 cm between rows) of maize and soybean. Reduced tillage (fall chisel plowing and spring cultivation) was employed. Oat cultivar Don was planted on 8 April, 1991. Data were collected in 1991 from rows 1, 5, 10, 15, and 20. Data collection started 2 June and continued weekly.

Data Evaluations

1990

In 1990, leaf disease severity (percentage leaf area affected) was measured on ten adjacent plants per row at three arbitrary sites per row (determined by prescribed number of passes) using a manual of assessment key described by James (1971). Disease evaluations were made in the outside rows of oat in each strip. Row 1 was the row contiguous to the strip planted to oats the prior year. The disease severity was averaged for the ten adjacent plants in a row and then averaged for the three sites evaluated in a row. Samples of diseased leaves were initially and periodically gathered in the plots and returned to the laboratory for identification of pathogens. Samples were either placed in moist chambers to induce sporulation or surface sterilized and isolated onto agar.

1991

The prevalent diseases were identified by clinical processing in the laboratory (sporulation or isolation onto agar).
At the McNay Research Center, four rectangular microplots were established within each oat strip. A microplot was 21 plants in a row for rows 1, 5, 10, 15, and 20. The 21 plants in a row were delimited by flags and disease incidence for Septoria blight was visually assessed on each plant weekly. Data were taken in sequence down the row so that the distribution of infected plants could be established. This allowed a characterization of the disease spread among the plants in a row and among rows. Data were collected beginning 25 May and weekly thereafter until 29 June, when crown rust became too heavy to separate it from the Septoria lesions.

At the Alta Vista farms, disease incidence and disease severity data (percentage leaf area affected) were collected by arbitrarily selecting a 10 plant sequence in row 1 and moving perpendicularly across to the remaining rows in the strip to assess disease on 10 plants for each row selected. Oat disease incidence and disease severity were assessed on rows 1, 6, 12, 18, and 23 on the Frantzen farm and rows 1, 7, 14, 21, and 28 at the Reicherts farm. Weekly oat disease evaluations were started 29 May in 1991.

At the Johnson farm near Ames, data were collected weekly on rows 1, 5, 10, 15, and 20 beginning 2 June. The method of data collection was the same as described for Alta Vista.

Statistical Analyses

All data were analyzed by the general linear models analysis of variance (SAS Institute, 1989).
The pattern of diseased plants on each measurement date in the microplots at the McNay Research Center in 1991 was determined using ordinary "runs" (Gibbons, 1971; Campbell and Madden, 1990). Ordinary runs were calculated for each row to determine if there was significant clustering of diseased plants and the standardized statistic (Z) was calculated. A cluster pattern is indicated if Z < -1.64 at P = 0.05 and Z < -2.33 at P = 0.01 level.

Analysis of variance (ANOVA) and linear regression analysis were calculated (Netter et al., 1985).

The disease progress curve and disease gradients were determined by the best model using coefficient of determination ($R^2$), standard deviation (or error), and plot residual versus predicted value, as criteria (Campbell and Madden, 1990).

The area under the disease progress curve (AUDPC) was calculated according to the formula presented by Shaner and Finney (1977). The disease intensity ($Y$) is plotted versus time ($t$).

$$\text{AUDPC} = \sum_{i}^{n-1} \left[ \frac{y_i + y_{i+1}}{2} \right] (t_{i+1} - t_i)$$

where $n =$ number of sampling times

$y =$ disease intensity

$t =$ time

$t_{n-t_i} =$ total time of the epidemics.
Weather

Rainfall and temperature during 1990 and 1991 was measured at the McNay Research Center, Alta Vista, and the Johnson farm (USDC-NOAA 1990, 1991). Normal (average) temperatures were determined from the National Oceanic and Atmospheric Administration records for Ottumwa (for McNay Research Center), Des Moines (for Ames), and Mason City (for Alta Vista). The daily weather data are recorded in Figures A1 to A11 in the Appendix.

In 1990 the rainfall in the Alta Vista and McNay Research Center areas was above normal and rainfall was recorded on more than 30% of the days each month. At Alta Vista, 63% of the days in June had measurable rainfall. The temperatures were below normal in both areas.

The weather in 1991 was abnormal. April rainfall at McNay Research Center was 129.5 mm above normal and started soon after planting the oats. Localized flooding occurred at the McNay Research Center. The extremely wet weather was common to all three research areas and continued through May. This resulted in delayed maize and soybean planting. The wet weather ceased about mid June and thereafter extended periods of no or sparse rainfall were common to all research areas. The temperatures were above normal during the growing season.
RESULTS

Diagnoses of Leaf Diseases in 1990 and 1991

The major oat diseases encountered in the research plots and farmers fields were (in order of relative prominence): 1) Crown rust (*Puccinia coronata* Corda); 2) Septoria blight (*Septoria avenae* A. B. Frank); 3) Helminthosporium leaf blotch (*Drechslera avenacea* (Curt. ex. Cke) Shoemaker); and 4) Barley yellow dwarf virus.

Crown rust and barley yellow dwarf virus appeared late in each season and was uniformly dispersed over the strips. A crown rust epidemic developed in 1991 and interfered with late Septoria blight evaluations. The frequency of Helminthosporium leaf blotch was less than 5% of isolations compared to Septoria blight, and it had a limited distribution. Septoria blight was the prominent leaf disease that developed in the oat row contiguous to the prior year’s oat crop debris. The variation and similarity in symptoms of Septoria blight and Helminthosporium leaf blotch may have resulted in some error in identification during field evaluations; this error was minimal because *D. avenae* was rarely found in routine samplings.

McNay Research Center

Experiment 1

Septoria blight severity on plants sampled on 30 June, 1990, (when the first observations were made) was 2% in the row contiguous with debris compared to 1%
in the farthest row. The disease severity increased with time to reach 23% in the row contiguous with debris compared to 6% in the row farthest from debris. Analysis of the area under the disease progress curve (AUDPC) indicated that the most disease developed with reduced tillage and the least with no tillage (Figure 1). Disease was significantly greater in the row contiguous with debris than the row farthest from the debris except for the no tillage treatment.

In 1991, it was impossible to measure a significant effect of tillage on the incidence of Septoria blight during the season. Tillage had an insignificant effect on the disease in every row; therefore, the 1991 data were combined across tillages.

In 1991, disease incidence ranged from 2 to 12% at the first sampling date (25 May) when the first symptoms were observed (Figure 2). Incidence of Septoria blight increased with time in all rows to reach 29 to 71% on plants sampled on the last sampling date (29 June). The highest level was reached in row 1, contiguous with debris, compared to row 20, the row farthest from the debris source. Several models could explain disease progress in the 5 rows (Table 1). Row 1 and row 5 were best described by a linear model. Rows 10, 15, and 20 (farthest from the debris) were, however, best described by a monomolecular or a simple interest model as defined by Vanderplank (1963).

In 1991, tillage had no significant effect on the AUDPC. AUDPC was greater, however, in the row contiguous with debris than in the row farthest from debris (Figure 3).
Figure 1. Area under the disease progress curve for severity (percentage leaf area affected) of Septoria blight of oat in the edge rows of a 20-row strip of oat, in relation to land planted in oats in 1989, and to different tillage practices in 1990. LSD$_{(0.05)}$ = 166 for tillage and 136 for row.
Figure 2. Disease progress curve for Septoria blight (data combined for tillage) in 20-row strip planting of oats in 1991 in Experiment 1 at McNay Research Center; row 1 was contiguous with oat debris from 1990 crop. Vertical line represents the LSD$_{(0.05)}$. 
Figure 3. Area under the disease progress curve for incidence (data combined for tillage) of Septoria blight of oat in 1991 in specific rows of a 20-row strip of oats in relation to land planted to oats in 1990. Means followed by the same letter are not statistically different according to LSD (P = 0.05).
Table 1. Selected best-fit models for analysis of disease progress curve for incidence of Septoria blight (data combined for tillage) on each row of oats in Experiment 1 at McNay Research Center. Row 1 was contiguous with oat debris from 1990 crop.

<table>
<thead>
<tr>
<th>Sampled row</th>
<th>Best model</th>
<th>Intercept</th>
<th>Slope</th>
<th>Coefficient of determination (R²)</th>
<th>Standard error of Y estimate</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Linear</td>
<td>0.122</td>
<td>0.016</td>
<td>0.98</td>
<td>0.034</td>
</tr>
<tr>
<td>5</td>
<td>Linear</td>
<td>0.119</td>
<td>0.014</td>
<td>0.99</td>
<td>0.023</td>
</tr>
<tr>
<td>10</td>
<td>Monomolecular</td>
<td>0.131</td>
<td>0.018</td>
<td>0.98</td>
<td>0.037</td>
</tr>
<tr>
<td>15</td>
<td>Monomolecular</td>
<td>0.153</td>
<td>0.011</td>
<td>0.92</td>
<td>0.046</td>
</tr>
<tr>
<td>20</td>
<td>Monomolecular</td>
<td>0.060</td>
<td>0.009</td>
<td>0.90</td>
<td>0.046</td>
</tr>
</tbody>
</table>
The disease gradient across the oat strip was assessed weekly during 1991 after the first symptomatic plants were observed in the field plots (Figure 4). The percentage of infected plants was negatively correlated ($P \leq 0.01$ with the distance from the potential inoculum source ($r = -0.77$ and $r = -0.99$ on 25 May and 29 June, respectively (Figure 4). The gradient was much greater on the last sampling date (29 June) than on the first sampling date (25 May). All gradients were significantly different ($P = 0.05$) from a theoretical line where $b = 0$. Disease incidence declined linearly with increased distance from the source (Figure 4).

The spatial pattern of Septoria blight was random as indicated by the calculated standardized statistic ($Z_u$) being greater than -1.64 ($P = 0.05$ level). From 0 to 33% of the rows showed evidence of clustering (Table 2). There was a tendency for more clustering of diseased plants in rows farther from the prior year’s oat strip. When $Z_u$ was calculated for sampling time based on runs analysis of diseased plants, it was found that 10 to 25% of the rows showed evidence of clustering on the first three assessment dates, 25 May to 8 June (Table 3). The spatial pattern of diseased plants in the rows became more random with time.

**Experiment 5** The disease progress model for Septoria blight in 1990, as calculated by AUDPC, showed more disease in row 1 (contiguous with oat debris) than in row 20 (farthest from debris) (Figure 5). This difference was significant for all tillages. Septoria blight severity ranged from 0 to 2% on the first sampling date (20 June) and reached 3 to 10% on the last sampling date (20 July) with 3% in the
Figure 4. Disease incidence gradient (data combined for tillage) on different assessment dates (weeks) for Septoria blight of oat in 1991 from edge of oat planting in 1990. Slopes (b) of linear regression are significantly (P = 0.05) different from a theoretical line where b = 0.
Table 2. Percentage of rows showing evidence of clustering of diseased plants based on runs analysis (Zu) when rows were 1, 5, 10, 15, and 20 rows from inoculum source with different tillage practices.

<table>
<thead>
<tr>
<th>Row</th>
<th>No till</th>
<th>Reduced till</th>
<th>Intensive till</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>0</td>
<td>8</td>
<td>13</td>
</tr>
<tr>
<td>5</td>
<td>13</td>
<td>17</td>
<td>17</td>
</tr>
<tr>
<td>10</td>
<td>21</td>
<td>17</td>
<td>21</td>
</tr>
<tr>
<td>15</td>
<td>33</td>
<td>0</td>
<td>21</td>
</tr>
<tr>
<td>20</td>
<td>8</td>
<td>25</td>
<td>13</td>
</tr>
</tbody>
</table>

Table 3. Percentage of rows in the oat strip showing evidence of clustering of diseased plants based on runs analysis (Zu) for different assessment dates and tillage practices.

<table>
<thead>
<tr>
<th>Tillage</th>
<th>25 May</th>
<th>1 June</th>
<th>8 June</th>
<th>15 June</th>
<th>22 June</th>
<th>29 June</th>
</tr>
</thead>
<tbody>
<tr>
<td>No tillage</td>
<td>10</td>
<td>25</td>
<td>20</td>
<td>15</td>
<td>10</td>
<td>10</td>
</tr>
<tr>
<td>Reduced tillage</td>
<td>20</td>
<td>20</td>
<td>10</td>
<td>15</td>
<td>5</td>
<td>10</td>
</tr>
<tr>
<td>Intensive tillage</td>
<td>20</td>
<td>20</td>
<td>10</td>
<td>30</td>
<td>10</td>
<td>10</td>
</tr>
</tbody>
</table>
Figure 5. Area under the disease progress curve for severity (percentage leaf area affected) of Septoria blight of oat in 1990 in the edge rows of a 20 row strip of oat in relation to land planted to oat in 1989, and to different tillage practices in Experiment 5 at McNay Research Center in 1990. LSD_{0.05} = 23 for tillage; and 19 for rows.
row farthest from the debris (row 20) and 10% in the row contiguous with debris (row 1). Tillage per se had an effect on the disease severity; in row contiguous with debris there was significantly more disease with intensive tillage than with reduced and no tillage practices.

In 1991, incidence of Septoria blight increased linearly over time in row 1 of the oat strip, which was contiguous with oat debris from the prior year, and reached an average of 67% of the plants sampled (Figures 6-8 and Table 4). Disease was apparent in all rows sampled on the first assessment date (25 May). Data collection was terminated earlier in 1991 than in 1990 because of interference from crown rust. There was a very significant tillage effect when disease in all rows was considered (Figure 9). The highest Septoria blight incidence was in the row contiguous with the oat debris, the amount of disease in row 1 was not affected by tillage (Figure 10). When analyzed by sampling date, the tillage effect became significant 61 days after planting and was significant thereafter; disease was greater with intensive tillage than with no tillage (Figure 9). Incidence of Septoria blight in reduced tillage was less than with intensive tillage, but greater than with no tillage, though usually not statistically different.

There was a significant tillage x row interaction for incidence of Septoria blight that became apparent 61 days after planting (Figures 6, 7, and 8). Analysis of the AUDPC for Septoria blight incidence by row, revealed that the greater AUDPC with
Figure 6. Disease progress curve for Septoria blight in a 20-row strip planting of oat in 1991 under no tillage in Experiment 5 at McNay Research Center; row 1 was contiguous with land cropped to oat in 1990. Vertical line represents the $LSD_{(0.05)}$. 
Figure 7. Disease progress curve for Septoria blight in a 20-row strip planting of oat in 1991 under reduced tillage in Experiment 5 at McNay Research Center; row 1 was contiguous with land cropped to oat in 1990. Vertical line represents the LSD(0.05).
Figure 8. Disease progress curve for Septoria blight in a 20-row strip planting of oat in 1991 under conventional tillage in Experiment 5 at McNay Research Center; row 1 was contiguous with land cropped to oat in 1990. Vertical line represents the LSD(0.05).
Figure 9. Incidence of Septoria blight in strip plantings of oat in Experiment 5 as influenced by tillage practices at McNay Research Center during 1991.
Figure 10. Area under the disease progress curve for incidence of Septoria blight of oat in 1991 in specific rows of a 20-row strip of oat in Experiment 5 at McNay Research Center; row 1 was contiguous to land cropped to oat in 1990.
Table 4. Selected models for analysis of disease progress data on incidence of Septoria blight on each row of oat at McNay Research Center in Experiment 5.

<table>
<thead>
<tr>
<th>Tillage</th>
<th>Row</th>
<th>Best model</th>
<th>Intercept</th>
<th>Slope</th>
<th>Coefficient of determination ($r^2$)</th>
<th>SE</th>
</tr>
</thead>
<tbody>
<tr>
<td>No tillage</td>
<td>1</td>
<td>Linear</td>
<td>0.123</td>
<td>0.016</td>
<td>0.99</td>
<td>0.029</td>
</tr>
<tr>
<td></td>
<td>5</td>
<td>Linear</td>
<td>0.146</td>
<td>0.010</td>
<td>0.98</td>
<td>0.021</td>
</tr>
<tr>
<td></td>
<td>10</td>
<td>Monomolecular</td>
<td>0.092</td>
<td>0.009</td>
<td>0.91</td>
<td>0.036</td>
</tr>
<tr>
<td></td>
<td>15</td>
<td>Monomolecular</td>
<td>0.088</td>
<td>0.002</td>
<td>0.61</td>
<td>0.022</td>
</tr>
<tr>
<td></td>
<td>20</td>
<td>Monomolecular</td>
<td>0.105</td>
<td>0.002</td>
<td>0.68</td>
<td>0.027</td>
</tr>
<tr>
<td>Reduced tillage</td>
<td>1</td>
<td>Linear</td>
<td>0.105</td>
<td>0.015</td>
<td>0.99</td>
<td>0.028</td>
</tr>
<tr>
<td></td>
<td>5</td>
<td>Linear</td>
<td>0.134</td>
<td>0.010</td>
<td>0.99</td>
<td>0.014</td>
</tr>
<tr>
<td></td>
<td>10</td>
<td>Monomolecular</td>
<td>0.122</td>
<td>0.012</td>
<td>0.94</td>
<td>0.042</td>
</tr>
<tr>
<td></td>
<td>15</td>
<td>Monomolecular</td>
<td>0.139</td>
<td>0.006</td>
<td>0.89</td>
<td>0.034</td>
</tr>
<tr>
<td></td>
<td>20</td>
<td>Monomolecular</td>
<td>0.090</td>
<td>0.004</td>
<td>0.90</td>
<td>0.018</td>
</tr>
<tr>
<td>Conventional tillage</td>
<td>1</td>
<td>Linear</td>
<td>0.119</td>
<td>0.015</td>
<td>0.99</td>
<td>0.013</td>
</tr>
<tr>
<td></td>
<td>5</td>
<td>Linear</td>
<td>0.149</td>
<td>0.011</td>
<td>0.98</td>
<td>0.022</td>
</tr>
<tr>
<td></td>
<td>10</td>
<td>Monomolecular</td>
<td>0.213</td>
<td>0.013</td>
<td>0.96</td>
<td>0.039</td>
</tr>
<tr>
<td></td>
<td>15</td>
<td>Monomolecular</td>
<td>0.163</td>
<td>0.008</td>
<td>0.80</td>
<td>0.060</td>
</tr>
<tr>
<td></td>
<td>20</td>
<td>Monomolecular</td>
<td>0.096</td>
<td>0.004</td>
<td>0.72</td>
<td>0.032</td>
</tr>
</tbody>
</table>
intensive tillage in contrast to no tillage was observed in rows 5, 10 and 15 (Figure 10). In rows 10 and 15 the AUDPC for Septoria blight was greater with intensive tillage than with reduced and no tillage, and AUDPC was greater with reduced tillage than with no tillage. AUDPC for Septoria blight in rows 1 and 20 was unaffected by tillage.

Analysis of the disease progress curves for each row (Figures 6-8) showed that the best fit model for rows 1 and 5 was a linear model (Table 4) and for rows 10, 15, and 20 was a monomolecular model (Table 4). The best fit model for a row was the same for all tillages.

Row 1 of the 1991 oat crop was theoretically planted 43 cm from row 20 of the 1990 oat crop with precise driving of the tractor pulling the planter. Rows 5, 10, 15 and 20 in 1991 were theoretically 114, 203, 292 and 391 cm, respectively, from row 20 of the 1990 oat crop. The disease incidence gradient decreased linearly with distance from the edge of the inoculum source (Figures 11-13). The edge of the inoculum source was, in reality, not a straight line source because of dispersal of straw during harvest and possible spread of straw and stubble during tillage and flooding. The greater incidence of disease in the middle rows of intensively tilled plots (Figure 13) compared to other tillages (Figures 11 and 12) was apparent from the initial disease assessment and evidence of this effect remained through subsequent readings. Row 1, the row nearest to the 1990 oat residue, had the greatest increase in incidence of Septoria blight, and the rate of disease increase generally decreased with
Figure 11. Weekly assessments of the disease gradient for Septoria blight incidence across oat strip plantings in 1991 in relation to distance from land cropped to oat in 1990 and use of no tillage crop management at McNay Research Center, Experiment 5. Slopes (b) of linear regression are significantly (P = 0.05) different from a theoretical line where b = 0.
Figure 12. Weekly assessment of the disease gradient for Septoria blight incidence across oat strip plantings in 1991 in relation to distance from land cropped to oat in 1990 and use of reduced tillage crop management at McNay Research Center, Experiment 5. Slopes (b) of linear regression are significantly (P = 0.05) different from a theoretical line where b = 0.
Figure 13. Weekly assessments of the disease gradient for Septoria blight incidence across oat strip plants in 1991 in relation to distance from land cropped to oat in 1990 and use of intensive tillage crop management at McNay Research Center, Experiment 5. Slopes (b) of regression are significantly (P = 0.05) different from a theoretical line where b = 0.
distance from the primary inoculum source (Figures 11-13). With successive disease
evaluations the slope of the disease gradient increased each time. The percentage of
infected plants was negatively correlated ($P < 0.01$) with the distance from a line
source across all tillage practices. The disease gradient was much greater on the last
sampling date (29 June) than on the first sampling date (25 May) across all tillage
practices (Figures 11-13).

Analysis of the data for evidence of clustering showed that few of the rows had
clustering of diseased plants regardless of tillage or row (Table 5) and tillage or date
of sampling (Table 6). The diseased plants were distributed randomly in most of the
rows and evidence for clustering did not increase appreciably with time.

Alta Vista

In 1990, a row effect was observed for disease severity on the Reicherts and
Frantzen farms. The AUDPC showed more Septoria blight in the row contiguous to
land planted to oat in 1989 compared to the farthest row (Figure 14). Severity of
Septoria blight (average proportion of leaf area affected) at the Reicherts farm ranged
from 1 to 3% on date 1 (30 June) and increased slightly to 2 to 4% on the last
sampling date. Disease severity level was lower at the Reicherts farm compared to
the Frantzen farm. At the Frantzen farm the average proportion of leaf area affected
on the last sampling date was 18% in the row contiguous with the debris from 1989
crop and 4% in the farthest row.
Table 5. Percentage of rows in oat rows 1, 5, 10, 15, and 20 (in oat strip plantings in 1991 with different tillage practices) that showed evidence of clustering of diseased plants (Septoria blight) based on runs analysis (Zu); rows are numbered in relation to land planted to oat in 1990. Experiment 5, McNay Research Center.

<table>
<thead>
<tr>
<th>Sampled row</th>
<th>No tillage</th>
<th>Reduced till</th>
<th>Intensive till</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>0</td>
<td>11</td>
<td>0</td>
</tr>
<tr>
<td>5</td>
<td>5</td>
<td>0</td>
<td>5</td>
</tr>
<tr>
<td>10</td>
<td>5</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>15</td>
<td>0</td>
<td>22</td>
<td>0</td>
</tr>
<tr>
<td>20</td>
<td>0</td>
<td>0</td>
<td>5</td>
</tr>
</tbody>
</table>

Table 6. Percentage of rows in oat strips showing evidence of clustering of diseased plants (based on runs analysis [Zu]) on six sampling dates and with different tillage practices in Experiment 5 at McNay Research Center.

<table>
<thead>
<tr>
<th>Tillage</th>
<th>May 25</th>
<th>1 June</th>
<th>8 June</th>
<th>15 June</th>
<th>22 June</th>
<th>29 June</th>
</tr>
</thead>
<tbody>
<tr>
<td>No tillage</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>13</td>
</tr>
<tr>
<td>Reduced tillage</td>
<td>7</td>
<td>0</td>
<td>0</td>
<td>7</td>
<td>13</td>
<td>7</td>
</tr>
<tr>
<td>Intensive tillage</td>
<td>0</td>
<td>13</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
</tbody>
</table>
Figure 14. Area under the disease progress curve for severity (percentage of leaf area affected) of Septoria blight of oat in 1990 in the edge rows of a 20-row strip of oat, in relation to the land planted to oat in 1989. \( \text{LSD}_{0.05} = 35 \) and 96 for row at Reicherts and Frantzen farms, respectively.
In 1991, the AUDPC was calculated for both incidence of Septoria blight (Figure 15) and disease severity (Figure 16) at both farms. The AUDPC was greatest in row 1 which was contiguous with land planted to oat in 1990, the potential source of inoculum (Figures 15-16).

Distance from the potential source of inoculum was indirectly related to disease incidence and severity. The disease incidence in nearly every sampled row across a strip was significantly different from the nearest sampled rows (Figure 15). The severity of the disease was mild but there definitely was significantly more disease in the row contiguous with the potential source of inoculum than those rows farthest from it (Figure 16).

The average incidence of Septoria blight ranged from 6 to 23% on the first sampling date (29 May) at the Reicherts farm. On the same day the incidence of Septoria blight ranged from 31 to 43% at the Frantzen farm. The highest incidence of disease was obtained by the third sampling date and this was 48% at the Reicherts farm and 58% at the Frantzen farm in row 1, which was nearest to the land planted to oat in 1990. The highest incidence of Septoria blight in the row farthest from the debris was 31% at the Frantzen farm and 22% at the Reicherts farm. The hot dry weather in June and July stopped the epidemic early in the season. The cropping strips at the Frantzen farm were narrower than at the Reicherts farm and the slope of the land at the Frantzen farm would allow surface drainage of water to move from the strip planted to oat in 1990 into and across the 1991 oat strip.
Figure 15. Area under the disease progress curve for incidence of Septoria blight of oat in row strip planting of oat at Reicherts (A) and Frantzen (B) farms in 1991; row 1 was contiguous with land planted in oat in 1990. Columns followed by the same letter are not significantly different according to LSD (P = 0.05).
Figure 16. Area under the disease progress curve for severity (percent leaf area affected) of Septoria blight at Reicherts (A) and Frantzen (B) farms in 1991; row 1 was contiguous with land planted to oat in 1990. Means followed by the same letter are not significantly different according to LSD (P = 0.05).
Johnson Farm

From 14 to 20% of the oat plants had Septoria blight when the initial evaluations were made 2 June (Figure 17). Heavy rainfall in May (255 mm in 18 days) caused extensive surface flooding and prevented earlier access to the plots. Although the percentage of diseased plants in row 1 was initially less than in rows 5 and 10, eventually the disease incidence in row 1 was the greatest. Disease incidence in row 1 increased linearly for the next 28 days whereas the disease increase in rows 5, 10, 15, and 20 followed a monomolecular model. Further disease increase occurred only in row 1.

The AUDPC showed a significantly greater incidence of Septoria blight in rows 1 and 5 than in the other three sampled rows (Figure 18). Disease severity was low and only row 20 had significantly less disease than row 1. The low precision of estimating low disease levels contributed to large variability in the estimates.
Figure 17. Incidence of Septoria blight in different rows across strip plantings of oat at Johnson farm in 1991; row 1 was contiguous to land planted to oat in 1990. Vertical line represents the $LSD_{(0.05)}$. 
Figure 18. Area under the disease progress curve for incidence and severity (percentage leaf area affected) of Septoria blight in 20-row strip planting of oat at Johnson farm in 1991; row 1 was contiguous with land planted in 1990. Means followed by the same letter are significantly different according to LSD (P = 0.05).
DISCUSSION

The only disease of oat that was consistently observed with strip intercropping was Septoria blight, induced by *Septoria avenae*. Septoria blight was the prominent foliar disease that developed on the edge of the oat strip contiguous with the land planted to oat the prior year. Septoria blight occurred in all experiments evaluated in 1990 and 1991 and in two commercial fields that incorporated oat in a three-crop strip intercrop pattern both years. The consistent association of high incidence and/or severity of Septoria blight with the contiguous edge of each oat strip was good evidence that the primary source of inoculum was in the oat residue from the prior year's oat crop.

The evidence that *Septoria avenae* is debris borne was investigated earlier. Huffman (1955) reported that the *S. avenae* overwinters in the field as mycelia, micropycnidia, and pycnidia in oat debris and is disseminated by wind and rain to new plants when the same crop is replanted. *S. avenae* is seedborne according to Noble and Montgomerie (1954) and Lund and Shands (1955), but seed transmission seems to be rare (Neergaard, 1977).

Other diseases anticipated were halo blight incited by *Pseudomonas coronafaciens* and Helminthosporium leaf blotch, incited by *Drechslera avenacea*. Both have been reported to be commonly debris borne and infected debris is the primary source of inoculum (Gorlenko and Naydenko, 1944; Dennis, 1933; Turner and Millard, 1931;
Earhart and Shands, 1952). Neither disease was prevalent in any of the experiments or commercial fields observed.

Five multifactorial experiments were studied at the McNay Research Center in 1990. These involved various combinations of tillage, crop, weed control practices, N fertility, and different legume companion crops with oat. Only tillage had a significant effect on disease development. There was no hint of any relationship of disease with any of the other factors. Therefore tillage was the only factorial studied in 1991.

When tillage had a significant effect on Septoria blight of oat, no tillage always had the lowest amount of disease. This was unexpected because no-tillage practices are consistently associated with greater foliar disease than intensive tillage practices that bury debris with soil (Nyvall and Martinson, 1983; Cook et al., 1978).

Intensive tillage had significantly greater disease than no-tillage as shown in Experiment 5 at McNay Research Center during both years of this study (Figures 9-13). Several explanations for the higher level of disease with intensive tillage are:

1. straw or stubble may have been thrown into the adjacent plots during tillage, especially plowing;
2. tillage loosened or cut the stubble and it could have washed into the adjacent plots during localized surface flooding;
3. tillage may have placed straw or stubble in a flattened position on the soil surface where the residue could have been matted and moist for a longer period of time than when erect and subject to drying breezes;

4. the integrity of plot borders may have been lost with tillage.

In 1990 it was presumed that the reservoir of inoculum would be low because of the extended and severe droughts of 1988 and 1989. Moisture was adequate throughout the 1990 growing season and disease developed later than in 1991 but continued to develop until late in the growing season. The late season development was aided by frequent rainfall in June. Heavy disease pressure was expected for 1991. April had the highest rainfall on record for the state and heavy frequent rains persisted through much of May. Then the rains ceased and near drought conditions developed by the end of the growing season. When it was possible to enter the fields and plots for data collection and not cause soil compaction, the disease incidence was lower than expected. The localized flooding and persistent rains may have caused the inoculum source to sporulate early and deplete food reserves before seedling emergence and significant seedling growth (Imhoff et al., 1982). The potential for sporulation may have waned by the time plants developed adequately. With dry weather the increase of disease abated quickly resulting in linear or, more commonly, monomolecular disease progress curves.

Taking data in 5 rows per strip in 1991 allowed observation of the disease gradient across the strip. At the McNay Research Center, microplots were established
within the plots of both experiments and the same plants observed repeatedly. At the other sites plants were selected arbitrarily to avoid observing the same plants each time. The gradient of disease across the oat strip, from the side contiguous to oat debris from the prior crop to the farthest side, was relatively flat initially. The gradient increased with time and disease incidence on the contiguous side continued to increase faster than on the farthest side. The gradient across the strip was linear when regressed with distance from the theoretical inoculum source and compared with other models (Gregory, 1968; Berger and Luke, 1979; Campbell and Madden, 1990).

The initial flattened slope was probably due to background contamination (Gregory, 1968) from dispersed inoculum sources or inoculum that spread with surface drainage. The increase of the disease gradient slope with time may be explained by two possible mechanisms. The initial inoculum was associated with rain and rainwater dispersal and when the rains subsided the primary inoculum was airborne. As the oat crop got taller the airborne primary inoculum from the oat debris was mechanically intercepted by the plants.

There was little evidence of clustering of diseased plants which should have been expected if oat debris had washed into the adjacent oat strip and accumulated. Clustering did not increase with time which should have occurred if spread of secondary inoculum was involved (Vanderplank, 1963; Imhoff et al., 1982; Minogue and Fry, 1983a,b).
Analysis of the disease progress curves for 1991 showed that row 1 usually had a linear disease development whereas the other rows commonly exhibited monomolecular disease progress curves. This would indicate that continued production of primary inoculum near row 1 may have been more important than secondary inoculum. Huffman (1955) found that hot and dry weather delayed pycnidial development in the field. The teleomorphic stage, *Phaeosphaeria avenaria* (G. F. Weber) O. Erikson, evidently does not function in survival and perithecia and ascospores form late in the spring. No single model best described the epidemics that occurred in each row (Tables 1 and 4). Kranz (1974), Campbell et al. (1980, 1984), and Madden et al. (1987) also have used different models within one study.

AUDPC values between rows were greater in rows contiguous with debris from the prior oat crop than in rows farthest from debris. This trend was observed in all locations. These data would lead one to suggest that strip intercropping increases the likelihood of disease problems in oat, especially with Septoria blight. Unfortunately, there was no opportunity to compare disease incidence in oat strips with nearby solid stands in large oat fields. Therefore I cannot say that Septoria blight was worse with strip intercropping than with conventional solid stands, but the level of disease experienced in these studies was greater than normally expected. The disease gradient across each strip of oat is circumstantial evidence that strip intercropping increases the potential for disease problems.
At the two Alta Vista farms, the level of disease was greater at the Frantzen farm than at Reicherts farm. The same oat variety was used at both farms yet there are several explanations. The Frantzen field had been in strips much longer than the Reicherts field and the disease had a longer time to build up. Also, in the area of the Frantzen field studied the drainage of surface water would tend to be from the prior year's strip of oats into the current oat strip. Inoculum or inoculum sources could have washed into the oat crop with heavy rainfall water.

Yield losses from the diseases were impossible to determine from these trials. Although yields were taken from different areas of the strips, edge effects and effects of the neighboring maize and soybean plants confounded any disease effect and vice versa. Fungicidal control of the leaf disease would help to assess yield losses due to disease.
REFERENCES CITED


SECTION II. EFFECT OF TILLAGE AND STRIP INTERCROPPING ROTATION ON INCIDENCE AND SEVERITY OF BROWN SPOT AND BACTERIAL BLIGHT DISEASE OF SOYBEAN (GLYCINE MAX. (L) MERR.)
ABSTRACT

Diseases were assessed in soybean strips associated with strip intercropping that involved maize, oat, and soybean strips and sequences of rotations. Two multifactorial strip intercrop experiments at the McNay Research Center and two commercial strip intercrop fields near Alta Vista, Iowa, were studied in 1990. An additional site near Ames was studied in 1991. Disease was quantified in the outside rows of the strips in 1990 and in every row of each strip in 1991. Brown spot (incited by Septoria glycines) and bacterial blight (incited by Pseudomonas syringae pv. glycinea) were the primary diseases that developed. Brown spot appeared early and bacterial blight developed late in the growing season.

The highest incidence and most severe symptoms of brown spot and bacterial blight were in the row that was contiguous with land planted to soybean the prior year. The least disease was in the row farthest from the potential source of inoculum. Brown spot gradients across the strips were linear. The disease gradient for bacterial blight sometimes disappeared late in the season when nearly all plants in the strip were diseased. No single epidemiological model could be best describe the disease progress in each row. In most instances, there was no tillage x row interaction observed with either disease. When tillage had an effect on disease, the least disease was associated with no-tillage. A 1.5 to 2.3 m barrier of oats between the inoculum source and the soybean strip helped to delay and decrease the diseases.
INTRODUCTION

Brown spot (caused by *Septoria glycines* Hemmi) and bacterial blight (caused by *Pseudomonas syringae* pv. *glycinea* [Coerper] Young, Dye and Wilkie) are known to be the most prevalent soybean foliar diseases found worldwide (Sinclair and Backman, 1989). The two pathogens overwinter in seed or infected crop residues (Daft and Leben, 1972, 1973; Graham, 1953; Kennedy, 1969; MacNeill and Zalasky, 1957; Wolf, 1926). The spread of brown spot is favored by warm, moist weather and wind (Daft and Leben, 1972; Laurence and Kennedy, 1974; MacNeill and Zalasky, 1957). The disease starts in the lower leaves and spreads to the upper leaves. Brown spot severity varies depending on year and season because the disease is environmentally dependent. Soybean physiology may be important in brown spot development (Lim, 1980; Williams and Nyvall, 1980; Young and Ross, 1979). Bacterial blight spreads and multiplies during cool rainy weather when extended leaf wetness prevails (Sinclair and Backman, 1989; Mew and Kennedy, 1971, 1982; Kennedy and Ercolani, 1978).

Soybean yields were reduced when tillage depth was reduced to 12 cm or less, and when spring chisel plowing depths were less than 25-cm (Kamprath et al., 1979; Trouse, 1979). Tillage from 15 to 25 cm deep in June or July reduced not only soybean plant growth (Crabtree and Rupp, 1980; Jeffers et al., 1973; Sanford, 1982), but also yield (Tupper, 1978; Tyler and McCutchen, 1980).

The strip intercropping being studied is an experimental agronomic practice that has been adopted by some farmers. Two, three or more crops are planted in
repetitive narrow strips, 3 to 5 meters wide, that will accommodate current equipment. The crops are rotated among the strips according to a prescribed sequence, sometimes with intercropping of a legume with strips of small grains. Strip intercropping requires good management, careful equipment operation, and reduced tillage practices that allow for permanent sites. The benefits are increased yields compared to separate solid planting of the crops (Andrews and Kassam, 1976; Boosalis et al., 1981; Francis et al., 1986; Whigham, 1985) and possibly less soil erosion (Harold et al., 1963; Laflen et al., 1985; Yassar and Wittmuss, 1976).

Foliar diseases have become problems when infected crop debris is allowed to remain on the surface of the soil for erosion control and the same crop is replanted into the field (Sumner et al., 1981; Nicol et al., 1974). Many pathogens overwinter in the crop residues (Cook et al., 1978; Kimber, 1967; Brown et al., 1978) which provide large amounts of inocula for infecting the next crops. Crop rotation normally minimizes the disease threat because the pathogens die as the crop debris decomposes (Cook et al., 1978). Gerad (1976) reported the presence of debris from several crops on a field may reduce the chance of pathogens to multiply and infect the crop compared to debris from a single crop. Strip intercropping may nullify the disease control benefits of the crop rotation because one edge of every strip is contiguous with debris of the same crop from the prior year.

The purpose of this study was: 1) to identify the diseases that are prevalent in soybean with strip intercropping management; 2) to characterize the temporal and
spatial development of diseases in soybean strips; and 3) to determine whether agronomic practices may affect disease development.
MATERIALS AND METHODS

This study involved strip intercrop experiments at Iowa State University Research Centers and data collected on commercial farms with strip intercropping. A three crop system with maize, soybean, and oat was employed at all sites. Soybean followed maize and was followed by oat in the rotation. At the McNay Research Center and Alta Vista farm, oat was intercropped with alfalfa.

Experimental Design

McNay Research Center

Five multifactorial split-plot-design experiments with four replications were established in 1988 at Iowa State University's McNay Research Center near Chariton in south-central Iowa on poorly drained haig series soil (fine, montmoillonitic mesoic type Argiaquoll), with less than 1% slope. The treatments in the experiments were: Experiment 1, tillage and crop; Experiment 2, tillage, weed control practice, and crop; Experiment 3, N fertilizer (on maize) and alternate rotation crops including oat; Experiment 4, weed control practices, N fertilizer (on maize), and crop; and Experiment 5, tillage, N fertilizer (on maize) and crop. Disease data were collected in all experiments in 1990 and it was found that weed control practices (broadcast, banded, and no herbicide) and N fertilizer rate application in maize were insignificant factors in disease development, therefore disease data are presented only for
Experiments 1 and 5 in 1990 and these were the only experiments studied in 1991 at the McNay Research Center.

**Experiment 1**  The main plot factor was tillage and four replications were employed. Treatments used were conventional tillage (fall moldboard plowing and two secondary tillage operations in the spring), reduced tillage (fall chisel plowing and one secondary tillage in the spring), and no preplant tillage. Each tillage plot was 19 m wide and 12.2 m long. Across the width there were five crop strips, each 3.8 m wide and 12.2 m long. The crop sequence in a three year rotation was maize to soybean to oat plus alfalfa and then back to maize. In one year the cropping pattern across the five strips was oat plus alfalfa, soybean, maize, oat plus alfalfa, soybean. In the next year the order of crops across the five strips was maize, oat plus alfalfa, soybean, maize, and oat plus alfalfa. In the third year it was soybean, maize, oat plus alfalfa, soybean, maize. In 1988, each tillage plot was randomly started in one of the three cropping patterns. Only the three strips in the center were used for data collection. The first and last crop strips were border strips. Maize and soybean strips were 5 rows (0.76 cm spacing) across and oat strips had 20 rows (18 cm spacing) per strip. In 1990, rows 1 and 5 in the soybean strip were sampled with row 1 being the row contiguous with the strip planted to soybean in 1989. In 1991 all rows were sampled. All experiments were planted with seeds of Pella 86 soybean cultivar on 4 June in 1990 and 29 May in 1991. Maize and soybeans were planted the same day, 30 and 44 days for 1990 and 1991, respectively, after oat planting.
The herbicide 2,4-D was applied to the maize and oats before maize was planted, and alachlor was band applied to the maize and soybeans to control weeds. Data collection started on 20 June in 1990 and proceeded biweekly. In 1991, periodic data collection started on 13 July and continued at 7-14 day intervals.

**Experiment 5** The main plot factor was tillage as described for Experiment 1 and four replications were employed. Each tillage plot was 48.8 m long and 19 m wide. Five strips of crops, each 48.8 m long and 3.8 m wide, were planted in each tillage plot. The cropping sequence and patterns were as described for Experiment 1. The maize strips were divided into four subplots (perpendicular to the rows), with each subplot 12.2 m long. Randomly selected subplots of maize received additional N at 0, 33.7, 67.3, and 101.0 kg/ha. All other factors, including data acquisition, were the same as described for Experiment 1.

**Alta Vista Farms**

Data were collected from two commercial fields located in northeast Iowa near Alta Vista that had been strip intercropped since 1989. One was at the Mike Reicherts farm and the other was at the Thomas Frantzen farm. The soil type was a fine, loamy, mixed mesic type arguidoll Cresco series soil. Both used a rotation sequence of maize, soybean, and oat and both practiced ridge till farming.

**Reicherts farm** The crop strips were 4.56 m wide. Thirty rows of oat on 15.24 cm spacing were planted per strip. Then soybean and maize strips were planted
to 6 rows (76 cm spacing) of the crop per strip. Oat was planted on 18 April in 1990 and 25 April in 1991. Maize was planted on 6 May in 1990 and on 12 May in 1991. Riverside 3033 soybean was planted on 30 May in 1990 and 3 June in 1991. Data were taken on 16 soybean strips using rows 1 and 6 (the outer rows in each strip) in 1990 and all rows in 1991. Each strip was a replication. Data collection began on 30 June in 1990 (and continued biweekly) and 11 July in 1991 (and continued periodically).

Frantzen farm  The crop strips were 3.76 m wide. A strip was planted to 23 rows of oat (15.24 cm spacing), or four rows of maize or soybean with 94 cm row spacing. Oat was planted on 6 April in 1990 and 1991. Maize was planted 28 April in 1990 and 12 May in 1991. Soybean variety NKS 1312 was planted 14 May in 1990 and 28 May in 1991. Data were collected on 16 strips of soybean. Each strip was a replication. Data collection began on 30 June in 1990 (and continued biweekly) and 11 July in 1991 (and continued periodically).

Ames

At the Agricultural Engineering farm, tillage plots (24 m long x 40 rows [0.76 cm spacing] wide) that have been in continuous maize since 1977 were split and planted into alternating 10-row strips of maize (W64A x W117) and soybean (Williams) in 1990. The tillage treatments were fall plow, fall chisel plow, ridge till, and no-till. Soybean plants were inoculated with the bacterial blight pathogen. In
1991 the two soybean and two maize strips in each 40 row plot were planted on 22
May as follows:

a. one 10-row maize strip in 1990 was planted to soybean (Corsoy 79);
b. an adjacent 10-row soybean strip in 1990 was planted to maize (Pioneer
3362);
c. the remaining 20 rows from 1990 that were adjacent to either a or b were
planted as follows: Three strips of oat were planted in late April into the
land area occupied by rows 1, 2, and 3; 9, 10, 11, and 12; and 18, 19, and
20 in 1990. The two remaining 5-row strips were planted to maize (on
soybean land) and soybean (on maize land).

The planting pattern allowed the assessment of disease spread across the 10-row strips
from the debris in the adjacent 10-row strip. The 5-row strips provided either a 1.52
or 2.28 m oat barrier between the 1991 planting of maize or soybean and the debris of
the same crop in 1990.

Disease Evaluations

1990

In 1990, leaf disease severity (percentage leaf area affected) was measured on 10
adjacent plants per row at 3 arbitrary sites per row (determined by prescribed number
of paces) using a manual of assessment key described by James (1971). Disease
evaluations were made in the outside rows of soybean in each strip. Row 1 was the
row contiguous to the land planted to soybean the prior year. The disease severity
was averaged for the 10 adjacent plants in a row and then averaged for the 3 sites evaluated in a row. Samples of diseased leaves were gathered in the plots and returned to the laboratory for identification of the pathogens. Samples were either placed in moist chambers to induce sporulation, observed for bacteria in lesions by phase contrast microscopy, and/or surface sterilized and isolated onto nutrient media.

1991

The prevalent diseases were identified by clinical processing in the laboratory as in 1990.

At the McNay Research Center, disease incidence and disease severity (percentage leaf area affected) was determined for every plant in an arbitrarily selected 10 plant sequence in row 1 (the row contiguous with land planted to soybean in 1990). Sampling continued perpendicularly across the remaining rows to assess disease on 10 plants for each row in the strip.

At the Alta Vista farms, disease incidence and severity data were collected arbitrarily selecting a 10 plant sequence in row 1 and moving perpendicularly across the remaining rows to assess disease on 10 plants for each row in the strip. Data were assessed biweekly in 1990 and periodically in 1991.

Soybean disease incidence and disease severity was assessed on all 4 rows in each strip on the Frantzen farm and all 6 rows at the Reicherts farm. Weekly soybean disease evaluations were started 30 June and 11 July in 1990 and 1991,
respectively. Data was gathered for the same diseases as at the McNay Research Center.

**Ames**

At the Agricultural Engineering Farm both brown spot and bacterial blight developed in the soybeans. The method of disease assessment was the same as indicated for Alta Vista. Weekly data collection was started on 26 June.

Individual row yield data were collected for soybean at McNay Research Center, the Frantzen farm, and the Agricultural Engineering farm. The soybean yields were calculated on the basis of 13% moisture (Appendices B and C).

**Statistical Analyses**

All data were analyzed by the general linear models analysis of variance (SAS Institute, 1989). The disease progress curve and disease gradient were determined, the coefficient of determination ($R^2$), standard error and residual versus predicted value, were used as criteria to select the best model (Campbell and Madden, 1990).

**Weather**

Rainfall and temperature during 1990 and 1991 was measured at the McNay Research Center and Alta Vista (USDC-NOAA, 1990, 1991). Normal (average) temperatures were determined from the National Oceanic and Atmospheric Administration records for Ottumwa (for McNay Research Center), for Des Moines
(for Ames), and Mason City (for Alta Vista). The daily weather data are recorded in Figures A1 to A11 in the Appendix.

In 1990 the rainfall in the Alta Vista and McNay Research Center areas was above normal and rainfall was recorded on more than 30% of the days each month. At Alta Vista, 63% of the days in June had measurable rainfall. The temperatures were below normal in both areas.

The weather in 1991 was abnormal. April rainfall at McNay Research Center was 129.5 mm above normal and started soon after planting the oats. Localized periodic flooding occurred in the McNay Research Center fields because the land has very little slope. The extremely wet weather was common to all three research areas and continued through May. This resulted in delayed maize and soybean planting. The wet weather ceased about mid June and thereafter extended periods of no or sparse rainfall were common to all research areas. The temperatures were above normal during the growing season.
RESULTS

Brown spot, incited by *S. glycines*, and bacterial blight, incited by *P. syringae* pv. *glycinea*, were the major soybean diseases that developed. Bacterial blight should have been the initial disease observed because it is favored by cool and rainy weather. Data were not collected in the plantings until 30 July in 1990 and 13 July in 1991 and evidence of bacterial blight was slight during the initial observations. Brown spot was the first disease to develop significantly and bacterial blight was observed late in the growing season with cooler nights and more rains and winds. Bacterial pastule, which is incited by *Xanthomonas compestris* pv. *glycines* (Nakano) Dye, never developed into a disease of significance.

McNay Research Center

Diseases were monitored in all five experiments in 1990. The only variable that was significant was row position in relation to land planted to soybean in 1989. There were trends with tillage data that supported a decision to include experiments with this variable in 1991. The variables of weed control practice, and fertility were not important in disease development; therefore, Experiments 2, 3, and 4 are not included in this study.

Experiment 1

In 1990, brown spot was observed on the second observation date, 43 days after planting, but disease had not developed significantly. Disease severity ranged from 0
to 3% on the third observation (5 August). On the last date of evaluation, 29 August, the average disease severity for brown spot was 17% in the row contiguous with debris and 7% in the row farthest from debris (Figure 1). There was no significant effect of tillage practices. Brown spot severity was significantly greater on the last sampling date (5 September) in the soybean row contiguous to the land planted to soybean the prior year than in the farthest row. In 1990, there was a definite row effect on the development of bacterial blight of soybean (Figure 1); this was observed on the last two sampling dates. No tillage effect and no tillage x row interaction effects were observed.

In 1991, it was not possible to measure a significant effect of tillage on the incidence of brown spot and bacterial blight during the season. Tillage had an insignificant effect on the disease in every row; therefore, the 1991 data were combined across tillages.

Brown spot was present in the plots on 13 July, the first disease evaluation date in 1991 (Figure 2). The disease incidence ranged from 11 to 30% of the plants per row. The row contiguous to the land planted to soybean in 1990 had the highest incidence of brown spot (Figure 2). The incidence of brown spot increased on the second evaluation, but thereafter it remained static until 16 August when extensive defoliation made disease evaluation difficult and the incidence was less. The incidence of brown spot in the row contiguous to the land planted to soybean in 1990 was greatest at the end of data collection on 29 August.
Figure 1. Disease progress curve for brown spot (BS) and bacterial blight (BB) in a 5-row strip planting of soybean in 1990 in Experiment 1 at McNay Research Center; row 1 was contiguous to land planted to soybean in 1989.
Figure 2. Disease progress curve for brown spot incidence in a 5-row strip planting of soybean in 1991 in Experiment 1 at McNay Research Center; row 1 was contiguous with land planted to soybean in 1990. Vertical line represents the LSD\textsubscript{(0.05)}. 
Bacterial blight development prevented accurate assessment of brown spot on later dates. Bacterial blight did not become a significant disease in the soybean strips in 1991 until late in the growing season. The first data for bacterial blight was taken on 16 August, 80 days after planting. The highest incidence of bacterial blight was in the row contiguous to the land planted to soybean in 1990 (Figure 3). Incidence of bacterial blight increased rapidly and the disease was present on most of the plants in the strip on the last evaluation (6 September).

The severity of leaf diseases (percentage of leaf area affected) in the soybean strips was evaluated as the total for all leaf diseases (Figure 4). The hot dry weather that began soon after disease evaluations were started greatly reduced any disease epidemics. Initially the prevalent disease was brown spot. The severity of disease increased until early August and then decreased. Part of the decrease in disease severity was related to a lower incidence (Figure 2), but mostly it was a result of plant growth (new leaves) and abscission of diseased leaves. The development of bacterial blight in August resulted in a stable level of severity after 9 August. Severity increased greatly at the last evaluation on 6 September (data not shown), but it was difficult to separate disease from physiological chlorosis and normal maturation processes. About 50% of the leaf area was diseased or missing because of leaf shredding and abscission.

Modeling of the disease progress curves for brown spot was attempted, but the dry weather and loss of diseased tissues resulted in erroneous models with very low
Figure 3. Disease progress curve for incidence of bacterial blight in a 5-row strip planting of soybean in 1991 in Experiment 1 at McNay Research Center; row 1 was contiguous with land planted to soybean in 1990. Vertical line represents the LSD\(_{(0.05)}\).
Figure 4. Disease progress curve for total leaf diseases in a 5-row strip planting of soybean in 1991 in Experiment 1 at McNay Research Center; row 1 was contiguous with land planted to soybean in 1990. Vertical line represents the LSD\(_{(0.05)}\).
The disease progress curves for the soybean rows were analyzed and the best fit model for rows 1, 2, and 4 was logarithmic; a logistic model fit row 3, and a Gompertz model was best for row 5 (Table 1).

Plotting 1991 disease incidence (Y) versus distance (X) from land cropped to soybean in 1990 showed that brown spot incidence on week 1 (13 July) and week 8 (29 August) decreased linearly with increased distance from the potential inoculum source (Figure 5). The percentage of infected plants was negatively correlated (P ≤ 0.01) with distance from inoculum source (r = -0.84 and r = -0.99 on week 1 and week 8, respectively) (Figure 6). The gradient was much greater on the last sampling date (6 September) than on the first sampling date (13 July). The gradients for both weeks were significantly different (P = 0.05) from a theoretical line where b = 0. The percentage of infected plants at various distances from the inoculum source showed a goodness of fit (Netter et al., 1985) with coefficient of determination of $R^2 = 0.70$ and $R^2 = 0.97$ for incidence on week 1 (13 July) and week 8 (6 September), respectively.

Plotting the incidence of bacterial blight on soybean in 1991 against distance of the row from the land planted to soybean in 1990 revealed that the incidence of infected plants was indirectly related to distance from the potential inoculum source (Figure 6). The correlation coefficients for comparison of incidence of bacterial blight with distance from the inoculum source were $r = -0.87$ for week 1 (16 August)
Table 1. Model, intercept, slope, coefficient of determination $R^2$ and standard error of estimate of regression of bacterial blight on soybean plots at McNay Research Center, Experiment 1 during 1991.

<table>
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<tr>
<th>Row</th>
<th>Best model</th>
<th>Intercept</th>
<th>Slope</th>
<th>Coefficient of determination</th>
<th>Standard error</th>
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</thead>
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<td>0.96</td>
<td>0.23</td>
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<tr>
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<td>0.97</td>
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<td>0.99</td>
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<tr>
<td>4</td>
<td>Logarithmic</td>
<td>-2.836</td>
<td>0.125</td>
<td>0.99</td>
<td>0.12</td>
</tr>
<tr>
<td>5</td>
<td>Gompertz</td>
<td>-1.544</td>
<td>0.104</td>
<td>0.99</td>
<td>0.12</td>
</tr>
</tbody>
</table>
Figure 5. Incidence of brown spot of soybean in Experiment 1 at the McNay Research Center in 1991 on the first (week 1) and last (week 8) assessment dates in relation to distance from land planted to soybean in 1990. Slopes (b) of regression are significantly (P = 0.05) different from b = 0.
Figure 6. Incidence of bacterial blight of soybean in Experiment 1 at McNay Research Center in 1991 on the first (16 August) and last (6 September) assessment dates in relation to distance from land planted to soybean in 1990.
and $r = -0.73$ for week 4 (6 September). The gradient of -0.03% incidence/cm for week 1 was significantly different ($P = 0.05$) from a theoretical line where $b = 0$. The gradient across the strip for week 4 was not significantly different from $b = 0$. Therefore, no gradient was detected across the strip on the last reading. The coefficients of determination for week 1 and week 4 were $r^2 = 0.76$ and $r^2 = 0.54$, respectively.

When total disease severity (percentage of leaf area affected) values for the first and eighth weeks were regressed on distance from the land planted to soybean in 1990, the gradient of disease across the strip was insignificant on 13 July, week 1 (Figure 7). A significant gradient was observed on the last week of ratings (6 September) where percentage of leaf area affected was negatively correlated with distance from the potential inoculum source ($r = -0.93$).

**Experiment 5**

In 1990, brown spot was observed 45 days after planting but the severity was only a trace. About 1% of the leaf area was affected on 5 August, 60 days after planting (Figure 8). Brown spot continued to increase and reached a severity level of 5% in the row nearest to the inoculum source and 4% in the row farthest from the inoculum source on the last sampling date (5 September). Bacterial blight developed later than brown spot and the highest severity (though not significant at $P = 0.05$) was in the row contiguous with land planted to soybean in 1989 (Figure 8). The last
Figure 7. Total disease severity on first (13 July) and last (6 September) assessment dates on soybean strips in relation to distance (cm) from land planted to soybean in 1990 in Experiment 1 at McNay Research center in 1991.
Figure 8. Disease progress curve of brown spot (BS) and bacterial blight (BB) in a 5-row strip planting of soybean in 1990 in Experiment 5 at McNay Research Center; row 1 was contiguous with land planted to soybean in 1989.
assessment showed that 10% of the leaf area was affected on the plants in the row contiguous with the potential inoculum source. Tillage practices had no measurable effect on brown spot or bacterial blight.

In 1991, tillage had a significant effect on disease development. The average disease incidence of brown spot was 28%, 35%, and 31% with no tillage, reduced tillage and intensive tillage, respectively. The LSD\textsuperscript{q, rs} was 1.97, thus all averages were significantly different. There was no row x tillage interaction and the disease progress curves were essentially the same except for magnitude. Therefore, the data were combined across tillages for analysis and presentation. Incidence of brown spot was greatest on the row (row 1) contiguous with land planted to soybean in 1990 and least in the row (row 5) farthest from the potential source of inoculum (Figure 9). Incidence of brown spot was 25% in row 1 on the first assessment date (13 July) and it increased to 51% within two weeks. The disease was restricted to the lower leaves. The diseased leaves began to abscise after 27 July (third assessment date) and two weeks later nearly all of the diseased leaves were gone. The incidence of diseased plants decreased to about 1%. Within one week the incidence of brown spot increased to nearly the levels observed two weeks earlier. The 29 August evaluation was the last one for brown spot because bacterial blight developed intensively and had become the prominent disease, thereby confounding assessments. The brown spot incidence on 29 August was 40% on row 1 but only 14% on row 5.
Figure 9. Disease progress curve for incidence of brown spot in a 5-row strip planting of soybean in Experiment 5 at McNay Research Center in 1991; row 1 was contiguous with land planted to soybean in 1990. Vertical line represents the LSD(0.05).
Bacterial blight developed in mid to late August in 1991, much later than expected. Incidence of bacterial blight ranged from 0 to 5% on 16 August, the first date that data were recorded (Figure 10). The initial increase in disease incidence was logarithmic (Table 2) and the only dates where row position (proximity to land area in soybean in 1990) had a significant effect were the second and the third evaluations on 24 August and 29 August. The two rows nearest to the soybean debris from 1990 had a higher incidence of bacterial blight than the farthest row. On the last sampling date, 6 September, 100% of the plants in every row were infected with \( P. \text{syringae pv. glycinea} \). Spread of the pathogen was favored by rains, cooler weather, and wind in August.

Total disease severity in the soybean strips was difficult to assess. Only about 1% of the leaf area was affected (primarily with brown spot) on 13 July. This increased to about 10% of the leaf area in two weeks with greater disease in the rows nearer the potential source of inoculum than farther from it. However, many of the diseased leaves were abscised and accurate disease severity assessment was impossible. Disease severity, based upon leaves remaining on the plant on 10 August, declined to less than 1%. The rapid increase in brown spot and bacterial blight thereafter resulted in about 50% disease severity on the last assessment date, 6 September.

Characterization of the disease progress curves for brown spot (Figure 9) was not fruitful because of the bimodal nature of the curves. The coefficients of determination were low and the standard errors of the estimate of regression were
Figure 10. Disease progress curve for bacterial blight in a 5-row strip planting of soybean in Experiment 5 at McNay Research Center in 1991; row 1 was contiguous with land planted to soybean in 1990. Vertical line represents the $\text{LSD}_{0.05}$. 
Table 2. Model, intercept, slope, coefficient of determination and standard error of Y estimate (SE) of regression of bacterial blight incidence on sampled soybean rows in Experiment 5 at McNay Research Center during 1991.

<table>
<thead>
<tr>
<th>Row</th>
<th>Best model</th>
<th>Intercept</th>
<th>Slope</th>
<th>Coefficient of determination</th>
<th>SE</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Linear</td>
<td>-0.079</td>
<td>0.044</td>
<td>0.89</td>
<td>0.16</td>
</tr>
<tr>
<td>2</td>
<td>Logarithmic</td>
<td>-2.854</td>
<td>0.132</td>
<td>0.93</td>
<td>0.41</td>
</tr>
<tr>
<td>3</td>
<td>Logarithmic</td>
<td>-3.368</td>
<td>0.154</td>
<td>0.92</td>
<td>0.49</td>
</tr>
<tr>
<td>4</td>
<td>Logistic</td>
<td>-5.999</td>
<td>0.462</td>
<td>0.90</td>
<td>1.71</td>
</tr>
<tr>
<td>5</td>
<td>Logarithmic</td>
<td>-4.987</td>
<td>0.235</td>
<td>0.92</td>
<td>0.76</td>
</tr>
</tbody>
</table>
very high. Late season development of bacterial blight was characterized and the best fit models determined (Table 2). The best model to describe disease development in row 1 was linear, and a logarithmic or logistic model best described disease development in the other rows.

Plotting disease incidence versus distance from the potential inoculum source indicated that brown spot on week 1 and week 8 decreased linearly with increased distance from the land planted to soybean in 1990 and the potential inoculum source (Figure 11). The percentage of infected plants was negatively correlated ($P \leq 0.01$) with distance from the potential inoculum source ($r = -0.96$ and $r = -0.95$ on week 1 and week 8, respectively) (Figure 11). The gradient was much greater on the last sampling date (6 September) than the initial week. Both disease gradients were significantly different from a theoretical line where $b = 0$. No disease gradient across the soybean strips was observed with bacterial blight incidence when disease incidence on the first and final dates of evaluation was regressed on distance from the potential inoculum source. The disease incidence for bacterial blight on the initial assessment date was very low and all rows had 100% incidence on the last date and significant disease gradient was unexpected. Regression of the incidence of bacterial blight on 24 August (week 2) and 29 August (week 3) on distance from the land planted to soybean in 1990 revealed there was a definite disease gradient across the soybean strips on the assessment dates. The coefficients of regression were $b = -0.06\%/\text{cm}$ for both 24 August and 29 August.
Figure 11. Brown spot incidence on first and last assessment dates on soybean strip in relation to distance from land planted to soybean in 1990 in Experiment 5 at McNay Research Center in 1991. Slopes of linear regression are significantly (P < 0.01) different from a theoretical slope of b = 0.
Both disease gradients were significantly different than a theoretical line where $b = 0$. Correlations were $r = -0.99$ and $r = -0.95$ for the second and third week ratings, respectively.

**Alta Vista**

The row of soybean on the Reicherts farm that was contiguous with the land planted to soybean in 1989 (row 1) had significantly more brown spot than row 6, which was the farthest from the potential source of inoculum (Figure 12). Significant disease did not appear until 10 August when as much as 2% of the tissue had brown spot. This increased to about 9% disease severity in row 1 on 29 August, compared to about 2% in row 6.

The difference in brown spot between the two outside rows of soybean strips on the Frantzen farm in 1990 was not significant. However, the trend was for more disease in the row contiguous with the land planted to soybean in 1989 than in the farthest row (Figure 12). This same trend was seen for bacterial blight (Figure 13). Although the difference in bacterial blight between the two rows was insignificant, the trend in the data was for more bacterial blight in the row contiguous to land planted to soybean in 1989 than in the row on the opposite side of the strip (Figure 13).

The first bacterial blight was observed on both farms on 10 August. The severity increased greatly during the next 19 days, and on the last date of disease
Figure 12. Progress of brown spot in strip plantings of soybean at Reicherts (R) and Frantzen (F) farms near Alta Vista in 1991; row 1 was contiguous with land planted to soybean in 1989. Vertical line represents the $\text{LSD}_{(0.05)}$. 

$LSD = 4.5$ (Reicherts farm) 
$LSD = \text{NS}$ (Frantzen farm)
Figure 13. Progress of bacterial blight in strip plantings of soybean at Reicherts (R) and Frantzen (F) farms near Alta Vista in 1990; row 1 was contiguous with land cropped to soybean in 1989.
assessment, 29 August, row 1 in the strips on the Reicherts farm had significantly more disease than row 6, which was farthest from the potential source of inoculum.

In 1991, a high incidence of brown spot was observed on 11 July, the first day that disease assessments were made. The incidence ranged from 13 to 43% of the plants being diseased on the Reicherts farm (Figure 14) and 24 to 55% on the Frantzen farm (Figure 15). The highest disease incidence was in row 1 of the soybean strip at both farms; row 1 was contiguous with the land planted to soybean in 1990. Variation in the incidence was experienced from one week of data collection to the next because an effort was made to avoid taking data from the same area of a strip each time. The dry weather, which occurred for more than one month before the first disease assessment, was not conducive to pathogen spread and this resulted in very little increase in the incidence of brown spot during the summer (Figures 14 and 15). On 5 September, the incidence ranged from 27% to 62% of the plants diseased on the Frantzen farm strips (Figure 15). The incidence of disease on the Reicherts farm never exceeded 50% (Figure 14).

The severity of brown spot remained relatively stable during the season at the Frantzen farm (Figure 16) and increased towards the end of the season at the Reicherts farm (Figure 17). The increase was related to increased numbers of lesions and larger lesions on the diseased plants. The spread of disease on a plant evidently compensated for diseased leaves lost because of abscission and the continuous production of new disease-free leaves.
Figure 14. Progress of brown spot in a 6-row strip planting of soybean at Reicherts farm near Alta Vista, Iowa, in 1991; row 1 was contiguous with land planted to soybean in 1990. Vertical bar represents $LSD_{0.05}$. 
Figure 15. Progress of brown spot in 4-row strip plantings of soybean at the Frantzen farm near Alta Vista, Iowa, in 1991; row 1 was contiguous with land cropped to soybean in 1990. Vertical lines represent the LSD\(_{(0.05)}\).
Figure 16. Progress of brown spot severity in a 4-row strip planting of soybean at the Frantzen farm near Alta Vista, Iowa, in 1991; row 1 was contiguous with land cropped to soybean in 1990. Vertical line represents the $\text{LSD}_{0.05}$. 
Figure 17. Progress of brown spot severity in 6-row strip plantings of soybean at the Reicherts farm near Alta Vista, Iowa, in 1991; row 1 was contiguous with land cropped to soybean in 1990. Vertical line represents the LSD_{0.05}. 
Figure 18. Brown spot incidence on the first and last assessment dates in soybean strips on the Reicherts farm in relation to distance from land cropped to soybean in 1990. Slopes (b) of linear regression are significantly (P = 0.01) different from a theoretical line where b = 0.
The incidence of disease was significantly different among rows of a strip (Figures 14 and 15). The edge effect for strips was consistent throughout the season. Plotting brown spot incidence against distance from the land planted to soybean in 1990 showed that the incidence of brown spot decreased linearly across the soybean strips at the Reicherts farm in 1991 (Figure 18). The disease gradient across the strips was essentially the same, $b = -0.06\%/cm$, for the initial week of disease assessment and the final week of disease assessment (week 7). The percentage of infected plants was negatively correlated ($P = 0.01$) with distance from the inoculum source with $r = -0.96$ for week 1 and $r = -0.93$ for week 7. The gradients calculated were significantly different from a theoretical line where $b = 0$.

Regression of brown spot incidence against distance from the land planted to soybean in 1990 showed a significant linear gradient of disease across the soybean strips at the Frantzen farm in 1991 (Figure 19). The number of infected plants per row was negatively correlated with the distance from the potential inoculum source ($r = -0.95$ for week 1 and for week 7). The gradients calculated for both weeks were significantly different from a theoretical gradient where $b = 0$.

Bacterial blight developed late in the growing season at both Alta Vista sites. It was initially observed as a significant disease on 2 August when about 20% of the plants in row 1 (contiguous to land cropped to soybean in 1990) showed symptoms of bacterial blight at the Reicherts farm (Figure 20). At the Frantzen farm, in row 1 33% of the plants were diseased with bacterial blight (Figure 21). About 2-4% of the
Figure 19. Brown spot incidence at the Frantzen farm on the first (11 July) and last (5 September) assessment dates on soybean strips and in relation to distance from the land cropped to soybean in 1990. Slopes of regression lines are significantly different from a theoretical line where $b = 0$. 
Figure 20. Disease progress curve of bacterial blight in a 6-row strip planting of soybean at Reicherts farm near Alta Vista, Iowa, in 1991; row 1 was contiguous with land cropped to soybean in 1990. Vertical line represents the LSD_{0.05}. 
Figure 21. Progress of bacterial blight in a 4-row strip planting of soybean at Frantzen farm near Alta Vista, Iowa, in 1991; row 1 was contiguous with land planted to soybean in 1990. Vertical line represents the LSD$_{(0.05)}$. 
plants in the row farthest from the potential inoculum source had the disease.

Development of bacterial blight was significantly greater in row 1 than in the row farthest from the potential source of inoculum. A logarithmic increase in disease resulted and 100% of the plants observed in the strip had bacterial blight on 5 September. The severity of bacterial blight was difficult to assess because of brown spot, but it was estimated that at least 25% of the leaf area was diseased on the last day of disease assessments on both farms.

Agricultural Engineering Farm

The 1991 soybean strips were planted on 10-row strips of land cropped to maize in 1990 but bordered on each side with land cropped to soybean in 1990. The outside rows of 10-row strips of soybean in 1991 were contiguous with land planted to soybean in 1990. The outside rows of the 5-row strips of soybean in 1991 had either a 1.52 m- or a 2.28 m-strip of oat between them and the land planted to soybean in 1990. The tillage treatments had no significant effect on the incidence of foliar diseases in the soybean strips. There was a significant difference in the development of diseases in the 10-row strips versus the 5-row strips of soybean (Table 3) combined across tillages. Brown spot appeared earlier and was more severe in the 10-row strips than in the 5-row strips (Table 3). Only the lower leaves were diseased and essentially all of these abscised in late July. The disease reappeared but the frequency of brown spot diseased plants was insignificant thereafter.
Table 3. Incidence of brown spot and bacterial blight diseased plants in 5-row and 10-row strips of soybean where the 10-row strips were bordered with land planted to soybean and the 5-row strips were bordered with oat plantings.

<table>
<thead>
<tr>
<th>Date</th>
<th>Percent of plants diseased</th>
<th>Brown spot</th>
<th>Bacterial blight</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>10-row</td>
<td>5-row</td>
</tr>
<tr>
<td>26 June</td>
<td></td>
<td>3*</td>
<td>0</td>
</tr>
<tr>
<td>2 July</td>
<td></td>
<td>13*</td>
<td>9</td>
</tr>
<tr>
<td>11 July</td>
<td></td>
<td>14*</td>
<td>6</td>
</tr>
<tr>
<td>18 July</td>
<td></td>
<td>19*</td>
<td>12</td>
</tr>
<tr>
<td>26 July</td>
<td></td>
<td>24*</td>
<td>19</td>
</tr>
<tr>
<td>1 August</td>
<td></td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>7 August</td>
<td></td>
<td>8*</td>
<td>5</td>
</tr>
<tr>
<td>16 August</td>
<td></td>
<td>2</td>
<td>2</td>
</tr>
<tr>
<td>27 August</td>
<td></td>
<td>2</td>
<td>1</td>
</tr>
<tr>
<td>3 September</td>
<td></td>
<td>3</td>
<td>1</td>
</tr>
</tbody>
</table>

*Incidence of diseased plants in 10-row strips was significantly greater than in 5-row strips (p = 0.05).
Bacterial blight was rarely seen until late July and it developed more rapidly in the 10-row strips than the 5-row strips, which had oat buffers between them and the potential sources of inoculum (Table 3). By the end of August, 100% of the plants in both types of strips had some bacterial blight lesions.

The severity of brown spot (percentage of leaf area affected) was minimal and never surpassed 5%. The bacterial blight was affecting about 50% of the leaves by the end of the season.
DISCUSSION

The two diseases consistently associated with soybean grown with strip intercrop management practices were brown spot (incited by *Septoria glycines*) and bacterial blight (incited by *Pseudomonas syringae pv. glycinea*). No other soybean diseases became prevalent in any of the experiments or commercial farms studied. Lack of inoculum, the environment, and varieties employed may have influenced the disease prevalence.

The evidence collected would support an opinion that the source of inoculum for brown spot and bacterial blight was the soybean debris from the prior year (Kennedy, 1980; Kennedy and Erco, 1978; Daft and Leben, 1972, 1973; Young, 1982; Sinclair and Backman, 1989; Park and Lim, 1985). The most severe brown spot and bacterial blight epidemics in both years of this study occurred in rows contiguous with the potential source of inoculum.

In both years, it was observed that the brown spot disease developed first and the bacterial blight appeared later (Young, 1982; Sinclair and Backman, 1989; Kennedy, 1980; Kennedy and Erco, 1978; Park and Lim, 1985; Daft and Leben, 1972, 1973). Bacterial blight is reported to develop in seedlings from both seed borne and debris borne inoculum. It may have been present at a low level and missed during the initial disease assessments, which were made about one month after soybean emergence (Williams and Nyvall, 1980; Dunleavy, 1973; Daft and Leben, 1973).
Bacterial blight developed late in the season with cooler weather and more leaf wetness and rains (Sinclair and Backman, 1989).

In most instances, there was no tillage x row interaction effects observed with either disease. Tillage practice had a significant effect on disease incidence in one experiment, Experiment 5 at the McNay Research Center in 1991. The lowest incidence of disease was observed with no tillage practices. Young (1982) observed no difference in brown spot incidence when no tillage was compared to conventional tillage. He reported also that bacterial blight incidence increased with no tillage practices.

A significant gradient of disease developed across the soybean strips with greatest disease incidence and severity in the rows contiguous with the potential source of inoculum (line source). The gradients across the strips were linear with the incidence of disease decreasing linearly with greater distance from the land cropped to soybean the prior year. With brown spot the gradient remained until the end of data collection. However, the disease gradient for bacterial blight sometimes disappeared late in the season when all plants in the strip were diseased. Rapid production of secondary inoculum and numerous secondary cycles probably contributed greatly to the rapid spread of the bacterial blight pathogen. Sporadic local foci of infection in the soybean strips could have contributed to the epidemics (Cammack, 1958; Minogue and Fry, 1983a,b; Imhoff et al., 1982; Gregory, 1973, 1982).
A variation in disease progress curves was observed in all sampled rows. Those curves separated significantly with time. Redistribution and loss of inoculum due to flooding that occurred before planting in 1991 may have contributed to the variability (Hirst and Stedman, 1963). This is evidenced by the variation in disease among sampled sites at each sampling date indicating that the levels of inoculum in areas sampled were not uniform.

Several disease progress models were applied to the data. The epidemics in sampled rows were not uniform in most instances. No single model was able to explain disease progress for disease incidence. Because of defoliation that occurred during the assessments of brown spot disease and the dry weather of 1991, the characterization of the disease progress curves was not fruitful. Bacterial blight was best fit by logarithmic or logistic models.

It is misleading to present yield data because of too many interacting factors such as border effects, neighboring crops, shading, and tillage effect (Appendix B). We were not able to establish a good relationship between yield loss and disease incidence or severity per se. Although low yields in some instances were found in the row nearest to the potential inoculum source (Garcias, 1990), I was also unable to make comparative measurements of disease incidence and severity between soybean in pure stands and soybean in strip intercrop systems.

It appears that foliar plant diseases in soybean may limit the profitability of strip intercrop production practices. Disease resistant varieties must be utilized to
minimize disease losses. Fungicides would help control brown spot but not bacterial
blight. However, fungicides are not economically nor environmentally desirable
alternatives.
REFERENCES CITED


SECTION III. DISEASE INCIDENCE AND SEVERITY IN MAIZE
(ZEA MAYS L.) IN A STRIP INTERCROPPING
ROTATION
Diseases were assessed in maize strips associated with strip intercropping that involved oat, maize, soybean strips and sequences of rotation. Two multifactorial strip intercrop experiments at the McNay Research Center and commercial strip intercrop fields near Alta Vista, Iowa, were studied in 1990 and 1991. An additional site near Ames was studied in 1991. Disease was quantified in the outside rows of strips in 1990 and in every row of each strip in 1991. Eyespot (incited by Kabatiella zeae) was the prevalent disease and the only disease that was associated with proximity to maize debris from the prior year.

Highest incidence and most severe symptoms of eyespot were in the row that was contiguous with land planted to maize the prior year. The least disease was in the row farthest from the potential source of inoculum. Disease gradients across the strips were linear and negatively related to distance from the line source of inoculum. Primary inoculum appeared to be important for perpetuating the disease increase. Stalk rot of maize was less in the outer rows of the maize strips than in the center rows. In some instances the outer rows of maize contiguous with land cropped to maize in the prior year had more stalk rot than the farthest row.
INTRODUCTION

Eyespot of maize (caused by *Kabatiella zeae* Narita and Y. Hiratsuka) was first described by Narita and Hiratsuka (1959). The pathogen was found specific to maize (Cassini, 1971) and the disease is distributed worldwide. The pathogen is seedborne (Cassini, 1971), but the evidence for seed transmission is not yet established (McGee, 1988). It overwinters on crop residue. Conidia produced on maize residue may be disseminated by wind to adjacent plants (Arny et al., 1971; Boothroyd, 1977). A cool, moist environment favors eyespot development in the field; (Shurtleff, 1980). Up to a 50% loss was reported in the maize growing area in France (Cassini, 1971), 19% in Iowa (Martinson, 1981), and 9% in Minnesota (Reifschneider and Arny, 1983; Teng et al., 1982). The disease is more pronounced in reduced tillage than in conventional tillage (Boothroyd, 1977).

Stalk rot of maize is a stress regulated disease. The causes of stalk rots are soil and residue borne fungi and include several *Fusarium* spp., *Stenocarpella maydis* (Berk.) Sutton, and *Colletotrichum graminicola* (Ces.) G. Wils (Christensen and Wilcoxon, 1966; Hooker, 1976; Lipps. 1983a,b, 1985; Naylor and Leonard, 1977; White et al. 1979). Other biological and environmental factors interact with stalk rot development; these interactions may be insect feeding, virus diseases, foliar diseases, drought, cloudy weather and mineral deficiencies (Shurtleff, 1973; Halbert et al., 1935; Vanderplank, 1984). Stalk rot severity of sorghum (*Sorghum bicolor* (L)
Moench.) is associated with tillage and crop rotation in semi-arid regions (Doupnik and Boosalis, 1980). Stalk rot is greater in conventional tillage than in no tillage, regardless of rotation sequence (Byrnes and Carroll, 1986; Hartman et al., 1983).

Strip intercropping is an experimental agronomic practice that has been adopted by some farmers. Two, three, or more crops are planted in repetitive narrow strips, 3 to 5 meters wide, that will accommodate current equipment. The crops are rotated among the strips according to a prescribed sequence, sometimes with intercropping of a legume with strips of small grains. Strip intercropping requires good management, careful equipment operation, and reduced tillage practices that allow for permanent establishment of the strips. The benefits are increased yields of maize and small grains compared to separate solid planting of the crops (Andrews and Kassam, 1976; Boosalis et al., 1981) and possibly less erosion (Harold et al., 1963; Yassar and Wittmuss, 1975).

Foliar diseases have become problems when infected crop debris is allowed to remain on the surface of the soil for erosion control and the same crop is replanted into the field. Many plant pathogens overwinter in the crop residues (Cook et al., 1978; Kimber, 1967; Brown et al., 1978) which provides the inoculum for infection of the crop the next year. Crop rotation normally minimizes the disease threats because the pathogen dies as the crop debris decomposes (Cook et al., 1978). Strip intercropping with 2 or 3 crops may nullify the disease control benefits of the crop.
rotation because at least one edge of every strip is contiguous with debris of the same crop from the prior year.

The objectives of this study were to determine the pathogens or diseases that occur in maize with strip-intercropping practices and characterize the spread temporal and spatial development of the disease in strips of maize.
MATERIALS AND METHODS

All data were collected from strip intercropping experimental plots and commercial fields that were managed with strip intercropping. A maize, soybean, oat sequence was employed at all sites. At the McNay Research Center experiments and Alta Vista, oat was planted with alfalfa.

McNay Research Center

Five multifactorial split-plot-design experiments with four replications were established in 1988 at Iowa State University's McNay Research Center near Chariton in south-central Iowa on poorly drained haig series soil (fine, montmoillonitic mesoic type Argiaqunol), with less than 1% slope. The treatments in the experiments were: Experiment 1, tillage and crop; Experiment 2, tillage, weed control practice, and crop; Experiment 3, N fertilizer (on maize) and alternate rotation crops including oat; Experiment 4, weed control practices, N fertilizer (on maize), and crop; and Experiment 5, tillage, N fertilizer (on maize) and crop. Disease data were collected in all experiments in 1990 and it was found that weed control practices (broadcast, banded, and no herbicide) and N fertilizer rate application in maize were insignificant factors in disease development, therefore disease data are presented only for Experiments 1 and 5 in 1990 and these were the only experiments studied in 1991 at the McNay Research Center.
Experiment 1

The main plot factor was tillage and four replications were employed. Treatments used were conventional tillage (fall moldboard plowing and two secondary tillage operations in the spring), reduced tillage (fall chisel plowing and one secondary tillage in the spring), and no preplant tillage. Each tillage plot was 19 m wide and 12.2 m long. Across the width of a tillage plot there were five crop strips, each 3.8 m wide. The crop sequence was a three year rotation of maize to soybean to oat plus alfalfa and then back to maize. Accomplishment of this rotation was achieved by planting the five strips across the tillage plot one year to oat plus alfalfa, soybean, maize, oat plus alfalfa, and soybean. In the next year the crops across the tillage plot would be maize, oat plus alfalfa, soybean, maize, and oat plus alfalfa. In the third year it would be soybean, maize, oat plus alfalfa, soybean, maize. In 1988, each tillage plot was randomly started in one of the three cropping patterns. Data were collected from the maize strip from the center three strips. Maize and soybean strips were 5 rows (0.76 cm spacing) across and oat strips had 20 rows (18 cm spacing) per strip. In 1990 rows 1 and 5 were sampled. In 1991, all rows were sampled. All experiments were planted in maize hybrid NKS 7751 on 6 May and 12 May during 1990 and 1991, respectively. Maize and soybeans were planted the same day, 30 and 44 days for 1990 and 1991, respectively, after oat planting. The herbicide 2,4-D was applied to the maize and oats before maize was planted, and alachlor was band applied to the maize and soybeans to control weeds. Data collection started on 20
June in 1990 and proceeded biweekly. In 1991, periodic data collection started on 13 July and continued 7-14 days interval.

Experiment 5

The main plot factor was tillage as described for Experiment 1 and four replications were employed. Each tillage plot was 48.8 m long and 19 m wide. Five strips of crops as described for Experiment 1 were planted. The maize strips were divided into four subplots (perpendicular to the rows), with each subplot 12.2 m long. Randomly selected subplots received additional N at 0, 33.7, 67.3, and 101.0 kg/ha. All other factors, including data acquisition, were the same as described for Experiment 1.

Alta Vista Farms

Data were collected from two commercial fields located in northeast Iowa near Alta Vista that had been strip intercropped since 1989. One was at the Mike Reicherts farm and the other was the Thomas Frantzen farm. The soil type was a fine, loamy, mixed mesic type arguidoll Cresco series soil. Both used a rotation sequence of maize, soybean, and oat plus alfalfa and both practiced ridge till farming.

Reicherts farm

The crop strips were 4.56 m wide. Thirty rows of oat on 15.24 cm spacing were planted per strip. The soybean and maize strips were planted to 6 rows (76 cm spacing) of the crop per strip. Oat cultivar Don was planted on 18 April in 1990 and
25 April in 1991. Viking SX6000 maize was planted on 6 May in 1990 and Pioneer 3794 on 12 May in 1991. Riverside 3033 soybean was planted on 30 May in 1990 and 3 June in 1991. Data were taken on 16 maize strips across the field using rows 1 and 6 (the outer rows in each strip) in 1990 and all rows in 1991. Each strip was a replication. Data collection began on 30 June in 1990 (and continued biweekly) and 11 July in 1991 (and continued periodically).

**Frantzen farm**

The crop strips were 3.76 m wide. A strip was planted to 23 rows of oat (15.24 cm spacing), or four rows of maize or soybean with 94 cm row spacing. Oat cultivar Don was planted on 6 April in 1990 and 1991. Pioneer 3615 maize was planted 28 April in 1990 and 12 May in 1991. NK1312 soybean was planted 14 May in 1990 and 28 May in 1991. Data were collected on 16 strips of maize. Each strip was a replication. Data collection began on 30 June in 1990 (and continued biweekly) and 6 June in 1991 (and continued periodically).

**Ames**

At the Agricultural Engineering farm, tillage plots (24 meters long x 40 rows wide) that had been in continuous maize since 1977 were split and planted into alternating 10-row strips of maize (W64A x W117) and soybean (Williams) in 1990. The tillage treatments were fall plow, fall chisel plow, ridge till, and no-till. The maize was inoculated lightly in 1990 with spores of *Kabatiella zeae*, *Exserohilum*
turcicum races 1 and 2, Bipolaris zeicola races 2 and 3, and Cercospora zeae-maydis.

In 1991 the two soybean and two maize strips in each 40 row tillage plot were planted on 22 May as follows:

a. one 10-row maize strip was planted to soybean (Corsoy 79);

b. an adjacent 10-row soybean strip was planted to maize (Pioneer 3362);

c. the remaining 20 rows adjacent to either a or b were planted as follows:

Three strips of oats were planted in late April into the spaces where rows 1, 2, and 3; 9, 10, 11, and 12; and 18, 19, and 20 existed in 1990. The two remaining 5-row strips were planted to maize (on soybean land) and soybean (following maize).

Disease Data

1990

In 1990, leaf disease severity (percentage leaf area affected) was measured on 10 adjacent plants per row at 3 arbitrary sites per row (determined by prescribed number of paces) using a manual of assessment key described by James (1971). Disease evaluations were made in the outside rows of maize in each strip. Row 1 was the row contiguous to the strip planted to maize the prior year. The disease severity was averaged for the 10 adjacent plants in a row and then averaged for the 3 sites evaluated in a row. Samples of diseased leaves were initially and periodically gathered in the plots and returned to the laboratory for identification of the pathogens.
Samples were either placed in moist chambers to induce sporulation or surface sterilized and isolated into agar.

1991

The prevalent diseases were identified by clinical processing in the laboratory (sporulation and isolation onto agar).

At the McNay Research Center, disease incidence and severity (percentage leaf area affected) data were collected by arbitrarily selecting a 10 plant sequence in row 1 and moving perpendicularly across the remaining rows in the strip to assess disease on 10 plants for each row in the strip.

Alta Vista

Disease incidence and severity data were collected by arbitrarily selecting a 10 plant sequence in row 1 and moving perpendicularly across to the remaining rows in the strip to assess disease on 10 plants for each row in the strip. Data were assessed biweekly in 1990 and periodically in 1991.

Oat disease incidence and disease severity was assessed on all 4 rows in each strip on the Frantzen farm and all 6 rows at the Reicherts farm. Weekly maize disease evaluations were started 6 June and 11 July at Frantzen and Reicherts farm, respectively. Data was gathered for the same diseases as at the McNay Research Center.
Ames

At the Agricultural Engineering Farm the planting pattern allowed the assessment of disease spread across the 10-row strips from the debris in the adjacent 10-row strip. The 5-row strips provided either a 1.52 m or 2.28 m oat barrier between the 1991 planting of maize or soybean and the debris of the 1990 crop. The method of disease assessment was the same as indicated for Alta Vista in 1991. Weekly data collection was started on 6 June.

Individual maize row yield data were collected at McNay Research Center, the Frantzen farm, and the Agricultural Engineering Farm (Appendix C).

Stalk rot of maize was assessed in all rows (40 adjacent plants per row) at all of the sites. Stalk quality was determined by manual compression (pinching) of the second elongated internode. The response was categorized into the following classes:

1 = solid stalk;

2 = stalk is soft but most of pith is intact;

3 = stalk is easily compressed with most of the pith tissue rotted.

An index of the stalk rot was calculated as follows:

$$SRI = \text{sum of (class value x percentage of plants in class)}.$$  

Maximum and minimum values for SRI are 300 and 100, respectively.

Statistical Analyses

All data were analyzed by the general linear models analysis of variance (SAS Institute, 1989). The disease progress curve and disease gradient were determined.
Weather

Rainfall and temperature during 1990 and 1991 were measured at the McNay Research Center and Alta Vista (USDC-NOAA, 1990, 1991). Normal (average) temperatures were determined from the National Oceanic and Atmospheric Administration records for Ottumwa (for McNay Research Center), Des Moines (for Ames), and Mason City (for Alta Vista). The daily weather data are recorded in Figures A1 to A11 in the Appendix.

In 1990 the rainfall in the Alta Vista and McNay Research Center areas was above normal and rainfall was recorded on more than 30% of the days each month. At Alta Vista 63% of the days in June had measurable rainfall. The temperatures were below normal in both areas.

The weather in 1991 was abnormal. April rainfall at McNay Research Center was 129.5 mm above normal and started soon after planting the oats. Localized periodic flooding occurred in the McNay Research Center fields because the land has very little slope. The extremely wet weather was common to all three research areas and continued through May. This resulted in delayed maize and soybean planting. The wet weather ceased about mid June and thereafter extended periods of no or sparse rainfall were common to all research areas. The temperatures were above normal during the growing season.
RESULTS

Foliar Diseases

The foliar maize diseases encountered in the research plots and farmer fields were (in descending order of frequency): 1) eyespot (*Kabatiella zeae* Narita & Hiratsuka); 2) common rust (*Puccinia sorghi* Schwein); 3) Northern leaf blight (*Exserohilum turcicum* [Pass.] K. J. Leonard and E. G. Suggs); 4) gray leaf spot (*Cercospora zeae-maydis* Tehon and E. Y. Daniels); 5) Northern leaf spot (*Bipolaris zeicola* [G. L. Stout] Shoemaker), and 6) yellow leaf blight (*Phyllosticta maydis* D. C. Arny and R. R. Nelson).

Although these diseases were observed during data collection, the frequency of all but eyespot and common rust was insufficient to relate them to a row effect. Eyespot was observed in the field plots most frequently and incidence appeared related to particular rows. The pattern of distribution of common rust was generally related to random foci and not particular rows, therefore data are not presented. The incidence of the other diseases was too low to warrant data collection.

McNay Research Center

**Experiment 1** In 1990, eyespot was observed 62 days after planting but the severity was less than 5% (Figure 1). Eyespot continued to increase and reached a severity level of 23% in the row nearest to the land cropped to maize in 1989 and 4%
Figure 1. Disease progress curve for eyespot in a 5-row strip planting of maize in 1990 in Experiment 1 at McNay Research Center; row 1 was contiguous with land planted to maize in 1989.
in the row farthest from the potential inoculum source on the last sampling date (6 September). No tillage effect was apparent.

In 1991, tillage had an insignificant effect on disease development. There was no row x tillage interaction and the disease progress curves were essentially the same except for an insignificant magnitude, therefore the disease data were combined across tillages for analysis and presentation (Figure 2). Incidence of eyespot was greatest in the row (row 1) contiguous with land planted to maize in 1990 and least in the row (row 5) farthest from the potential source of inoculum (Figure 2). Eyespot incidence was 13% in row 1 on the first assessment date (16 August) and this increased to 42% on the last sampling date, 6 September. The severity of eyespot (percentage of leaf area affected) was minimal and never surpassed 5%.

Plotting disease incidence (y) versus distance (s) from the potential inoculum source (the land planted to maize in 1990) indicated that eyespot on week 1 (16 August) and week 3 (6 September) decreased linearly with increased distance from the land planted to maize in 1990, the potential inoculum source (Figure 3). The percentage of infected plants was negatively correlated ($P = 0.01$) with distance ($r = -0.96$ and $r = -0.98$ on week 1 and week 3, respectively) (Figure 3). The disease gradient was much greater on the last sampling date (6 September) than at the initial sampling. Both disease gradients were significantly different from the theoretical line where $b = 0$. 
Figure 2. Disease progress curve for eyespot in a 5-row strip planting of maize in 1991 in Experiment 1 at McNay Research Center; row 1 was contiguous with land planted to maize in 1990. Vertical bars represent LSD$_{(0.05)}$. 
Figure 3. Incidence of eyespot of maize in Experiment 1 at McNay Research Center in 1991 on the first (week 1) and last week (week 3) assessment dates in relation to distance from land planted to maize in 1990. Slopes (b) of regression are significantly (P = 0.05) different from b = 0.
Experiment 5

In 1990, eyespot was observed 62 days after planting but the severity was less than 5% (Figure 4). Eyespot continued to increase and reached a severity of 18% in the row nearest to the potential inoculum source (row 1, which was contiguous with land planted to maize in 1989) and 13% in the row farthest from the potential inoculum source on the last sampling date (5 September). Differences among tillages were insignificant and therefore the data were combined for presentation.

In 1991, tillage had a significant effect on disease incidence. The average disease incidences were 16%, 11% and 12% for no tillage, reduced tillage, and intensive tillage treatments, respectively. The no tillage treatment had a significantly higher disease incidence than the other two tillage treatments. A row x tillage interaction was observed, thus, eyespot incidence is presented for each tillage practice (Figures 5, 6, and 7).

The incidence of eyespot was greatest on the row (row 1) contiguous with land planted to maize in 1990 and least in the row (row 5) farthest from the potential source of inoculum (Figures 5, 6, and 7). Eyespot incidence in row 5 was 3%, 0%, and 0% in no tillage, reduced tillage and intensive tillage, respectively, on the first sampling date (16 August) and this increased to 33%, 13%, and 17%, respectively, on the last sampling date (6 September). The severity of eyespot on a plant was minimal and never surpassed 5% of the leaf area affected.
Figure 4. Disease progress curve for eyespot in a 5-row strip planting of maize in 1990 in Experiment 5 at McNay Research Center; row 1 was contiguous with land planted to maize in 1989.
Figure 5. Disease progress curve for eyespot in a 5-row strip planting of maize in 1991 under no tillage management in Experiment at McNay Research Center; row 1 was contiguous with land cropped to maize in 1990. Vertical line represents the $\text{LSD}_{0.05}$. 
Figure 6. Disease progress curve for eyespot in a 5-row strip planting of maize in 1991 under reduced tillage management in Experiment 5 at McNay Research Center; row 1 was contiguous with land cropped to maize in 1990. Vertical line represents the LSD(0.05).
Figure 7. Disease progress curve for eyespot in a 5-row strip planting of maize in 1991 under intensive tillage management in Experiment 5 at McNay Research Center; row 1 was contiguous with land cropped to maize in 1990. Vertical line represents the LSD_{0.05}.
Plotting disease incidence versus distance from the potential inoculum source (the land planted to maize in 1990) indicated that eyespot on 16 August and 6 September decreased linearly with increased distance from the land planted to maize in 1990, and the potential inoculum source with reduced tillage (Figure 8) and intensive tillage (Figure 9). The percentage of infected plants was negatively correlated ($P = 0.01$) with distance ($r = -0.94$ and $r = -0.98$ on week 1 and week 3, respectively, for reduced tillage, and $r = -0.95$ and $r = -0.94$ on 16 August and 6 September, respectively, for intensive tillage). The disease gradient was greater on the last sampling than the initial sampling as previously stated. Both disease gradients were significantly different from a theoretical line where $b = 0$.

The disease gradients for no tillage were not significantly different from a theoretical line where $b = 0$. However, the incidence of disease in row 1 was significantly greater than the incidence in row 5 with no tillage treatment.

**Alta Vista**

The rows of maize on the Reicherts farm that were contiguous with the land planted to maize in 1989 had significantly more eyespot than row 6, which was farthest from the potential source of inoculum (Figure 10). Significant disease did not appear until 10 August when as much as 1-3% of the tissue had eyespot. This increased to about 15% disease severity in row 1 on 29 August, compared to about
Figure 8. Incidence of eyespot of maize in Experiment 5 at McNay Research Center in 1991 on the first (16 August) and last week (6 September) assessment date in relation to distance from land cropped to maize in 1990 under reduced tillage. Slopes (b) of regression are significantly (P = 0.05) different from b = 0.
Figure 9. Incidence of eyespot of maize in Experiment 5 at McNay Research Center on the first (16 August) and last week (6 September) assessment dates in relation to distance from land planted to maize in 1990 under intensive tillage. Slopes (b) of regression are significantly (P = 0.05) different from b = 0.
5% in row 6, the farthest row from the potential source of inoculum (Figure 10). At the Frantzen farm no row effect was observed in 1990 although there was a trend for more disease in the row (row 1) nearest to the potential inoculum source compared to row 4, the row farthest from the inoculum source (Figure 10).

In 1991, eyespot was observed 59 days after planting maize at the Reicherts farm (11 July) (Figure 11). The eyespot incidence continued to increase and reached 59% in the row (row 1) nearest to the potential inoculum source and 34% in the row farthest from the potential inoculum source on the last sampling date (5 September) (Figure 11). At the Frantzen farm, incidence of eyespot was greatest in the row (row 1) contiguous with land planted to maize in 1990 and least in the row (row 4) farthest from the potential inoculum source (Figure 12). Eyespot incidence was 43% in row 1 on the first assessment date (6 June) and this increased to 98% on the last sampling date (5 September).

A plot of disease incidence versus distance from the edge of the inoculum source (the land planted to the maize in 1990) indicated that eyespot decreased linearly with increased distance from the land planted to maize in 1990 and the potential inoculum source (line source) Figures 13-14). The percentage of infected plants was negatively correlated (P = 0.01) with distance (r = -0.83 and r = -0.95) the first and last disease assessment dates, respectively at the Reicherts farm (Figure 13) and r = -0.96 and r = -0.99 on 6 June and 5 September, respectively, at the Frantzen farm (Figure
Figure 10. Severity of eyespot development in the outer rows of strip plantings of maize at Frantzen and Reicherts farms in 1990; row 1 was contiguous with land planted to maize in 1989. Severity of disease in row 6 on the Reicherts farm was significantly less than in row 1 on 29 August. All other comparisons were not significant (P = 0.05).
Figure 11. Progress of disease incidence for eyespot in a 6-row strip planting of maize in 1991 at Reicherts farm; row 1 was contiguous with land cropped to maize in 1990. Vertical line represents the LSD$_{(0.05)}$. 

Days after planting

Percent incidence

ROW 1
ROW 2
ROW 3
ROW 4
ROW 5
ROW 6
Figure 12. Disease incidence for eyespot in a 4-row strip planting of maize in 1991 at Frantzen farm; row 1 was contiguous with land cropped in maize in 1990. Vertical line represents the LSD\(_{(0.05)}\).
Figure 13. Incidence of eyespot of maize at Reicherts farm on the first (11 July) and last (5 September) assessment dates in 1991 in relation to distance from land planted to maize in 1990. Disease gradient on last observation was significantly (P = 0.05) different from b = 0.
Figure 14. Incidence of eyespot of maize in strip plantings on the Frantzen farm on the first (6 June) and last (5 September) assessment dates in relation to distance from land planted to maize in 1990. Disease gradient (b) was significantly different from a line where b = 0.
14). Except for the initial assessment on the Reicherts farm, the disease gradients were significantly greater than a theoretical line where $b = 0$.

**Agricultural Engineering Farm**

In 1991, maize was planted on 10-row strips of land cropped to soybean in 1990 but bordered on each side with land cropped to maize in 1990. The outside rows of the 10-row strips of maize in 1991 were contiguous with land planted to maize in 1990, but cropped to soybean in 1991. The 5-row strips of maize in 1991 were planted on rows 3, 4, 5, 6, and 7 of the soybean strip from 1990 and oats were planted on the two edges of the strip (rows 1, 2, 8, 9, and 10 from the prior year of soybean). The outside rows of the 5-row strips of maize in 1991 had either a 1.52 or a 2.28 m strip of oat between them and the land planted to maize in 1990.

Eyespot was the only disease of any consequence that developed in 1991, although all of the inoculated pathogens developed in 1990. The tillage treatments had no significant effect on the incidence of foliar diseases in the maize strips. There was a significant difference in the incidence of eyespot in the 10-row strips versus the 5-row strips of maize combined across tillages (Table 1).

Eyespot appeared earlier and more frequently in the 10-row strips than in the 5-row strips (Table 1). Eyespot was observed initially on the lower leaves and thereafter spread to the upper leaves as the plants matured. The lower leaves died
Table 1. Incidence and severity of eyespot diseased maize plants in 5-row and 10-row strips of maize where the 10-row strips were bordered with land planted to maize and the 5-row strips were bordered with oat plantings.

<table>
<thead>
<tr>
<th>Dates</th>
<th>Percent of plant diseased</th>
<th>Incidence</th>
<th>10-row</th>
<th>5-row</th>
<th>10-row</th>
<th>5-row</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>Severity</td>
<td></td>
<td></td>
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<td></td>
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</tr>
<tr>
<td>26 June</td>
<td></td>
<td></td>
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<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>2 July</td>
<td></td>
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<td></td>
</tr>
<tr>
<td>11 July</td>
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<td></td>
<td></td>
</tr>
<tr>
<td>18 July</td>
<td></td>
<td></td>
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<td></td>
<td></td>
</tr>
<tr>
<td>26 July</td>
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<tr>
<td>1 August</td>
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<tr>
<td>7 August</td>
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<tr>
<td>16 August</td>
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<tr>
<td>27 August</td>
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<td></td>
<td></td>
</tr>
<tr>
<td>3 September</td>
<td></td>
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</tbody>
</table>

*Incidence and severity of diseased plants in the 10-row strips was significantly greater than in the 5-row strips (P = 0.05).
because of the drought starting in late July. In most instances, the disease severity was much greater in the 10-row strips than the 5-row strips, which had oat buffers between them and the potential source of inoculum. However, percentage of leaf area affected was biased by the drought symptoms. The highest eyespot incidence was observed on 1 to 7 August and thereafter the incidence decreased because diseased leaves died. The severity of eyespot (percentage of leaf area affected) ranged from 1 to 33% in the 10-row strips and 1 to 19% in the 5-row strips (Table 1).

Stalk Rot

Stalk rot was observed in both years of this study but no isolations were made to sample the microflora of the rotted and healthy stalks.

McNay Research Center

Experiment 1 Severity of stalk rot infection in both years of this study was about the same (Table 2). It was observed that the outer rows of the strips had less stalk rot than the center rows, especially less than the rows adjacent to the outer rows (Table 2). The row effect was much greater in 1991 compared to 1990. There was no significant difference among the tillages for stalk rot severity in 1990. In 1991, the highest stalk rot severity was observed with intensive tillage (SRI = 155) which was greater than the level of stalk rot with no tillage (SRI = 140) (LSD = 8.1; P = 0.05).
Table 2. Stalk rot of maize in individual rows of a 5-row strip planting of maize with several tillages in Experiment 1 at McNay Research Center in 1990 and 1991.

<table>
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</thead>
<tbody>
<tr>
<td>Row 1⁺</td>
<td>152</td>
<td>131</td>
<td>156</td>
<td>148</td>
<td>154</td>
<td>154</td>
</tr>
<tr>
<td>Row 2</td>
<td>149</td>
<td>143</td>
<td>145</td>
<td>156</td>
<td>147</td>
<td>161</td>
</tr>
<tr>
<td>Row 3</td>
<td>149</td>
<td>141</td>
<td>151</td>
<td>147</td>
<td>146</td>
<td>149</td>
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<tr>
<td>Row 4</td>
<td>154</td>
<td>148</td>
<td>147</td>
<td>159</td>
<td>144</td>
<td>158</td>
</tr>
<tr>
<td>Row 5</td>
<td>145</td>
<td>139</td>
<td>141</td>
<td>143</td>
<td>141</td>
<td>152</td>
</tr>
<tr>
<td>Mean</td>
<td>149</td>
<td>140</td>
<td>148</td>
<td>149</td>
<td>146</td>
<td>155</td>
</tr>
</tbody>
</table>

LSD\(_{0.05}\) = NS for 1990 and 8.1 for 1991

\(^{a}\)SRI = Sum of (class value x percentage of plants in class).

\(^{+}\)Contiguous with debris source.
Experiment 5  The severity of stalk rot differed more among individual rows in 1991 than in 1990. The outer rows had significantly less stalk rot than the rows adjacent to them. The maize plants were more stressed late in the season in 1991 than in 1990 because of the drought. The outside row adjacent to oat (row 5) tended to have less stalk rot than the row adjacent to soybean (row 1) (Table 3). Stalk rot severity was correlated neither with distance from nor nearness to the land planted to maize in 1990, a potential source of inoculum, for either year ($r = -0.51$ and $r = -0.54$ in 1990 and 1991, respectively). Tillage had no significant effect on stalk severity either year (Table 3).

Alta Vista

Stalk rot severity at the Frantzen farm and the Reicherts farm in 1990 was not affected by row position. Variability was large and differences in stalk rot severity were not measurable (Table 4). In 1991 there was a difference in stalk rot severity among rows. The outer row adjacent to the oat strip (row 6 at the Reicherts farm and row 4 at the Frantzen farm) had less stalk rot than many of the other rows, including the outer row adjacent to the soybeans (row 1). Row 1 was contiguous with the land planted to maize the prior year.
Table 3. Stalk rot of maize in individual rows of a 5-row strip planting of maize with several tillages in Experiment 5 at McNay Research Center in 1990 and 1991.

<table>
<thead>
<tr>
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</thead>
<tbody>
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<td>Row 1 ^</td>
<td>154</td>
<td>144</td>
<td>155</td>
<td>150</td>
<td>145</td>
<td>155</td>
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<tr>
<td>Row 2</td>
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<td>190</td>
<td>159</td>
<td>177</td>
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<td>176</td>
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<td>Row 3</td>
<td>158</td>
<td>151</td>
<td>151</td>
<td>144</td>
<td>154</td>
<td>152</td>
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<tr>
<td>Row 4</td>
<td>163</td>
<td>157</td>
<td>149</td>
<td>169</td>
<td>152</td>
<td>171</td>
</tr>
<tr>
<td>Row 5</td>
<td>149</td>
<td>126</td>
<td>139</td>
<td>151</td>
<td>150</td>
<td>146</td>
</tr>
<tr>
<td>Mean</td>
<td>155</td>
<td>154</td>
<td>151</td>
<td>158</td>
<td>150</td>
<td>160</td>
</tr>
</tbody>
</table>

LSD(0.05) = (Row) = 9.4 and 20.6 in 1990 and 1991, respectively.

^SRI = Sum of (class value x percentage of plants in class).

^Contiguous with the debris for 1990.
Table 4. Stalk rot of maize by individual row in strip on the Frantzen and Reicherts farms in 1990 and 1991.

<table>
<thead>
<tr>
<th>Row</th>
<th>Reicherts farm</th>
<th>Frantzen farm</th>
</tr>
</thead>
<tbody>
<tr>
<td>Row 1\textsuperscript{b}</td>
<td>146</td>
<td>200</td>
</tr>
<tr>
<td>Row 2</td>
<td>138</td>
<td>196</td>
</tr>
<tr>
<td>Row 3</td>
<td>147</td>
<td>214</td>
</tr>
<tr>
<td>Row 4</td>
<td>144</td>
<td>184</td>
</tr>
<tr>
<td>Row 5</td>
<td>138</td>
<td>—</td>
</tr>
<tr>
<td>Row 6</td>
<td>135</td>
<td>—</td>
</tr>
<tr>
<td>\text{LSD}_{(0.05)}</td>
<td>9</td>
<td>NS</td>
</tr>
</tbody>
</table>

\textsuperscript{a}Means represent the averages from 16 observations (4 replications x 4 strips).

\textsuperscript{b}Contiguous with debris.

\textsuperscript{c}Farthest from debris.

\textsuperscript{a}Stalk rot index (SRI) = sum of (class value x percentage of plants in class).
Agricultural Engineering Farm

Stalk rot severity was observed in maize strips planted on 5- and 10-row strips of land cropped to soybean in 1990. Stalk rot severity was not significantly different between the 10-row and 5-row strips (SRI = 140 and 130 in 10-row and 5-row strips, respectively).
DISCUSSION

The major disease consistently associated with maize grown with strip intercrop management practices was eyespot, which was incited by Kabatiella zeae. No other maize diseases became prevalent in any of the experiments or commercial farms studied. Eyespot, gray leaf spot (C. zeae-maydis), Northern leaf blight (E. turricum), and yellow leaf blight (P. maydis) are the major leaf diseases of maize in Iowa where the pathogen survives in crop debris (Nyvall and Martinson, 1983), and they were seen occasionally. Lack of inoculum, the environment, and varieties employed may have influenced the disease prevalence.

The evidence collected would support an opinion that the source of inoculum for eyespot was infected maize plant residues from the prior year (Young, 1982; McGee, 1988; Arny et al., 1971; Boothroyd, 1977). The most severe eyespot development in both years of this study occurred in rows contiguous with land planted to maize the prior year and the debris of that prior crop. Tillage practices that bury crop debris will lessen the inoculum potential of debris borne pathogens (Martinson, 1981; Nyvall and Martinson, 1983) and a tillage effect was expected. Tillage practice had a significant effect on disease incidence one time, Experiment 5 at McNay Research Center in 1991. The highest eyespot incidence was observed with no-tillage. This was as expected according to Martinson (1981). Young (1982), however, observed no difference on eyespot incidence when no tillage was compared to conventional tillage.
A variation in disease progress curves was observed in all sampled rows. Those curves departed significantly with time. This could have been the result of redistribution and loss of inoculum due to flooding that occurred at the onset of the experiment (Hirst and Stedman, 1963). This is evidenced by different sample sites at each sampling date, indicating that the level of inoculum at areas sampled were not uniform as exemplified by the variation in data shown in various figures of this study.

Because of low level of eyespot severity and incidence, no attempt was made to fit the data to specific epidemiological models (Gompertz, monomolecular, logistic, or logarithmic).

A significant gradient of disease developed across every maize strip with greatest disease incidence and severity in the row contiguous with the potential source of inoculum (line source). The gradients across the strip were linear with incidence of disease increasing linearly with greater distance from the land cropped to maize the prior year (Vanderplank, 1963; Minogue, 1986; Gregory, 1968, 1982).

Stalk rot infection was observed in both years of this study. No pathogens were isolated for this study, because it is a stress regulated disease (Dodd, 1980; Nyvall and Martinson, 1983; Schneider and Pendery, 1983). The most common causes of stalk rot are fungi and include several Fusarium spp., Stenocarpella maydis, and Colletotrichum graminicola (Lipps, 1988, 1991; Schneider and Pendery, 1983; Martinson, 1981). These are soil- and debris-borne pathogens. The strip intercropping system could influence stalk rot by providing debris-borne inoculum,
and rows within the strip with different level of leaf disease, insect exposure, plant
crowding, and light exposure (Boothroyd et al., 1955; Byrnes and Carroll, 1986;
The results indicated that stalk rot severity was affected by row position, and less
often by tillage. More stalk rot was observed with intensive tillage compared to no-
tillage at the McNay Research Center. This has been reported previously (Lipps,
observe any difference in stalk rot between no tillage and intensive tillage.

It is misleading to present yield data because there were too many interacting
factors such as border effects, neighboring crops, competition for moisture, shading,
and tillage effects (Appendix C). Yield data were taken in most experiments but were
not discussed. Low yields in some instances were found in the row nearest to the
potential inoculum source (Garcias, 1990). Comparative measurements of disease
incidence and severity between maize in pure stands and maize in strip intercrop
systems were not made.

Foliar plant diseases in maize may limit the profitability of strip intercrop
production practices. Disease resistant varieties must be utilized to minimize disease
losses (Arny et al., 1971; Reifsneider and Arny, 1983). Fungicides would help to
control eyespot (Reifsneider and Arny, 1983). However, fungicides are neither
economically nor environmentally desirable alternatives.
REFERENCES CITED


The major foliar diseases of oat, maize, and soybean associated with strip intercrop management practices were Septoria blight of oat incited by *Septoria avenae*, brown spot (incited by *Septoria glycines*) and bacterial blight (incited by *Pseudomonas syringae pv. glycinea*) of soybean, and eyespot of maize incited by *Kabatiella zeae*. No other oat, maize and soybean diseases became prevalent in any of the experiments or commercial farms studied. Lack of inoculum, the environment, and varieties employed probably influenced the disease prevalence. Evidence collected at the numerous sites would support an opinion that the source of inoculum for these diseases was the maize, soybean, and oat residues from the prior year.

In 1990 it was presumed that the reservoir of inoculum would be low because of the extended and severe droughts of 1988 and 1989. Moisture was adequate throughout the 1990 growing season and diseases developed eventually and continued to develop until late in the growing season.

High inoculum potentials were expected for 1991. But, then April had the highest rainfall on record for the state and heavy frequent rains persisted through much of May. The rains ceased about mid June and near drought conditions developed for most of the growing season (USDC-NOAA, 1990, 1991).

The low levels of diseases from residue-borne pathogens was unexpected and probably due to localized flooding and persistent rains that may have caused the early sporulation from the inoculum source and depletion of food reserves before seedling
emergence and significant seedling growth (Imhoff et al., 1982). The potential for sporulation may have waned by the time plants developed adequately.

Infected crop debris has been demonstrated to be an important source of primary inoculum for all of the diseases that were demonstrated to develop unevenly across a strip. The evidence that Septoria avenae is debris borne was investigated earlier. Huffman (1955) reported that S. avenae overwinters in the field as mycelia, micropycindia, and pycindia in oat debris and is disseminated by wind and rain to new plants when the same crop is replanted. S. avenae is seedborne according to Noble and Montgomerie (1954) and Lund and Shands (1955), but seed transmission seems to be rare (Neerggard, 1977). The evidence collected from soybean strip experiments supports an opinion that the source of inoculum for brown spot (incited by Septoria glycines) and bacterial blight (incited by Pseudomonas syringae pv. glycinea) is the debris from the prior crop. The importance of soybean debris in the epidemiology of these diseases was shown earlier (Kennedy, 1980; Kennedy and Ercolani, 1978; Daft and Leben, 1972, 1973; Sinclair and Backman, 1989; Young, 1982; Park and Lim, 1985). Eyespot of maize has been associated with maize debris in many studies (Young, 1982; Arny et al., 1971; Boothroyd, 1977; Martinson, 1981). The present study supports the prior research. The earliest development, highest incidence, and greatest severity of a disease always occurred in the row contiguous with the potential source of inoculum (land planted to the same crop the
prior year). The least disease was in the row that was farthest from the potential inoculum source.

Tillage was the only crop management factor that had a significant effect on disease progress. Septoria blight of oat was much greater with intensive tillage than in no tillage in experiments at the McNay Research Center. This was unexpected but may be explained. Straw or stubble was possibly thrown into the adjacent strips during tillage, especially plowing. Loose stubble could have moved with localized flooding whereas the intact stubble with no tillage did not move. Tillage may have placed straw or stubble in a flattened position on the soil surface where the residue could have been matted and moist for a longer period of time than when erect and subject to drying breezes. Similar explanations may be advanced for soybean residues and the higher disease incidence with intensive culture.

There was little evidence of clustering of diseased oat plants, which should have occurred if fugitive debris was moved into the adjacent oat strips. Clustering did not increase with time, which should have occurred if spread of secondary inoculum was involved (Vanderplank, 1963).

Significant gradients of the diseases developed across the strips with greatest disease incidence and severity in the row contiguous with the potential source of inoculum (line source). The gradients across the strips were negative and linear when disease incidence was regressed with distance from the land cropped to the same crop the prior year (Vanderplank, 1963; Minogue, 1986; Gregory, 1968, 1982). The
disease gradient for bacterial blight of soybean sometimes disappeared late in the season when all plants in the strip were diseased. Rapid production of inoculum and numerous secondary cycles probably contributed greatly to the rapid spread of bacterial blight. Sporadic local foci of infection within soybean strips could have contributed to the epidemics (Cammack, 1958; Minogue and Fry, 1983b; Imhoff et al., 1982; Gregory, 1973).

Several disease progress models were applied to the data. The epidemics in sampled rows were not uniform in most instances. No single model was able to explain disease progress curves.

Strip intercropping has increased the yield of maize and oat compared to solid plantings of the crops (Francis et al., 1986; Whigham, 1985; Kayumbo, 1978; Radke and Burrow, 1970; Osiru and Willey, 1976; Baker and Yursuf, 1976; Monta and De, 1980). Strip intercropping requires good management, careful equipment operation, and reduced tillage that allow for permanent establishment and recognition of the strips.

Crop rotation is a well recognized control measure for the diseases that developed in the strips in this study (Cook et al., 1978; Gerad, 1978; Sumner, 1981). Strip intercropping with a three-crop rotation scheme may nullify the disease control benefit of crop rotation because one edge of every strip is contiguous with the debris of the same crop from the prior year. Alternative management schemes that physically separate the source of inoculum from the susceptible crop may be needed.
Moldboard plowing is not a feasible alternative because it obliterates the established strips and was of questionable benefit for disease control in this study.

The current research would support an opinion that foliar diseases in oat, maize, and soybean may limit the profitability of strip intercrop production practices. Although yield data are not discussed here, the levels of disease are consistent with disease loss models for some of the diseases (Martinson, 1981). Disease resistant varieties (Arny et al., 1971; Reifschneider and Arny, 1983; Sinclair and Backman, 1989) must be utilized to minimize yield losses. Fungicides, if economically and environmentally accepted, would help to control fungal diseases (Reifschneider and Arny, 1983; Sinclair and Backman, 1989), but not bacterial blight (Sinclair and Backman, 1989).

Finding that stalk rot of maize was less in the outer rows than the central rows of a strip crop planting may help to explain some of the higher yields of maize in the outer rows. This also indicates that the outer rows are probably less stressed by the environment than the central rows. This would fit the photosynthetic sink-translocation stress model for stalk rot proposed by Dodd (1980a,b). There was some indication, however, that stalk rot was more severe in the row contiguous with the land cropped to maize in the prior year than in the farthest row. This would indicate that the debris may be an important source of inoculum for stalk rot pathogens and also that the higher eyespot incidence in that row predisposed the maize plants to stalk
rot. This too would support the concepts of stalk rot based upon stress phenomena (Dodd, 1980ab; Nyvall and Martinson, 1983; Martinson, 1981).
ADDITIONAL LITERATURE CITED


ACKNOWLEDGMENTS

I would particularly like to thank Dr. Martinson for helping and guiding me in every phase of my graduate program at Iowa State University. Without his financial support, the completion of this dissertation could have been only a dream. Special acknowledgment is given to Leopold Center for Sustainable Agriculture for financial support of this research. I would also like to express my appreciation to the members of my committee, Drs. A. R. Hallauer, D. C. McGee, N. G. Vakili, and R. Cruse, for their support and assistance in editing this dissertation. Further acknowledgment is extended to Dr. F. W. Nutter for his suggestions in data collection and analysis, to Drs. A. Epstein and T. C. Harrington for financial and moral support, and to Mrs. Linda Edson for her assistance in typing this manuscript.

Finally, special appreciation is given to my wife, Mathilda Lusamba, to my sons, Thierry Mbolela Tubajika, Claude Francois Mukendi Tubajika, and to my daughter, Carina Kalanga Tubajika, for their sacrifice and pains endured for the accomplishment of my PhD degree, and to my parents for their encouragement.

"Do not let your hearts be troubled. Trust in God; trust also in me. In my father's house are many rooms; if it were not so, I would have told you. I am going there to prepare a place for you."

John 14:1-2
Figure A1. Mean daily temperatures (sum of the daily maximum and minimum temperatures divided by two) during 1991 at the Agricultural Engineering Farm (near Ames) compared with mean temperatures for 30 years (1951-1980) at Des Moines.
Figure A2. Mean daily temperatures (sum of the daily maximum and minimum temperatures divided by two) during 1990 at Chariton (near McNay Research Center) compared with mean temperatures for 30 years (1951-1980) at Ottumwa.
Figure A3. Mean daily air temperatures (sum of the daily maximum and minimum temperatures divided by two) during 1991 at Chariton (near McNay Research Center) compared with mean temperatures for 30 years (1951-1980) at Ottumwa.
Figure A4. Mean daily air temperatures (the sum of the daily maximum and minimum temperatures divided by two) during 1990 at Charles City (near Alta Vista) compared with mean temperatures for 30 years (1951-1980) at Mason City.
Figure A5. Mean daily air temperatures (the sum of the daily maximum and minimum temperatures divided by two) during 1991 at Charles City (near Alta Vista) compared with mean temperatures for 30 years (1951-1980) at Mason City.
Figure A6. Daily rainfall (mm) during the 1991 growing season at Ames.
Figure A7. Daily rainfall (mm) during the 1991 growing season at Johnson farm near Ames.
Figure A8. Daily rainfall (mm) during the 1990 growing season at Chariton, near McNay Research Center.
Figure A9. Daily rainfall (mm) during the 1991 growing season at Chariton, near McNay Research Center.
Figure A10. Daily rainfall (mm) during the 1990 growing season at Charles City, near Alta Vista.
Figure A11. Daily rainfall (mm) during the 1991 growing season at Charles City, near Alta Vista.
APPENDIX B: ADDITIONAL TABLES TO SECTION II
Table B1. Grain yield of soybean by individual row in soybean strips in Experiment 1 at McNay Research Center during 1990 and 1991 growing seasons.

<table>
<thead>
<tr>
<th>Effect</th>
<th>Yield Mg/ha</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>No tillage</td>
</tr>
<tr>
<td>Mean (tillage)</td>
<td>1.92</td>
</tr>
<tr>
<td>LSD(^a)</td>
<td>---</td>
</tr>
<tr>
<td>SD(^b)</td>
<td>0.3</td>
</tr>
<tr>
<td>Row</td>
<td></td>
</tr>
<tr>
<td>Row 1(^c)</td>
<td>1.50</td>
</tr>
<tr>
<td>Row 2</td>
<td>1.81</td>
</tr>
<tr>
<td>Row 3</td>
<td>2.03</td>
</tr>
<tr>
<td>Row 4</td>
<td>2.03</td>
</tr>
<tr>
<td>Row 5(^d)</td>
<td>2.24</td>
</tr>
<tr>
<td>LSD</td>
<td>--</td>
</tr>
<tr>
<td>SD</td>
<td>0.05</td>
</tr>
</tbody>
</table>

\(^a\)P = 0.05 (1991 data).
\(^b\)Standard deviation (1990 data).
\(^c\)Contiguous with debris.
\(^d\)Farthest from debris.
\(^e\)1990 Data kindly provided by Drs. Cruse and Garciás, ISU Dept. of Agronomy.
Table B2. Grain yields of soybean at individual row in soybean strips in Experiment 5 at McNay Research Center during 1991 growing season.

<table>
<thead>
<tr>
<th></th>
<th>Yield Mg/ha</th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Mean</td>
<td>1.33</td>
<td>1.50</td>
<td>1.38</td>
</tr>
<tr>
<td>LSD</td>
<td>0.13</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Row</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Row 1</td>
<td>1.29</td>
<td>1.25</td>
<td>1.05</td>
</tr>
<tr>
<td>Row 2</td>
<td>1.27</td>
<td>1.92</td>
<td>1.41</td>
</tr>
<tr>
<td>Row 3</td>
<td>1.47</td>
<td>1.53</td>
<td>1.43</td>
</tr>
<tr>
<td>Row 4</td>
<td>1.34</td>
<td>1.44</td>
<td>1.56</td>
</tr>
<tr>
<td>Row 5</td>
<td>1.24</td>
<td>1.37</td>
<td>1.43</td>
</tr>
<tr>
<td>LSD</td>
<td>0.17</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

^aP = 0.05.

^bContiguous with debris.

^cFarthest from debris.
Table B3. Grain yield of soybean by individual row in soybean strips at Frantzen farm during 1990 and 1991 growing seasons.

<table>
<thead>
<tr>
<th>Row</th>
<th>Yield (Mg/ha)</th>
<th>1990</th>
<th>1991</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>Yield (Mg/ha)</td>
<td></td>
</tr>
<tr>
<td>Row 1&lt;sup&gt;a&lt;/sup&gt;</td>
<td>3.13</td>
<td>2.41</td>
<td></td>
</tr>
<tr>
<td>Row 2</td>
<td>3.30</td>
<td>2.88</td>
<td></td>
</tr>
<tr>
<td>Row 3</td>
<td>3.21</td>
<td>2.96</td>
<td></td>
</tr>
<tr>
<td>Row 4&lt;sup&gt;b&lt;/sup&gt;</td>
<td>3.13</td>
<td>3.34</td>
<td></td>
</tr>
<tr>
<td>LSD&lt;sup&gt;c&lt;/sup&gt;</td>
<td>0.41</td>
<td>0.41</td>
<td></td>
</tr>
<tr>
<td>SD&lt;sup&gt;d&lt;/sup&gt;</td>
<td>0.08</td>
<td>--</td>
<td></td>
</tr>
</tbody>
</table>

<sup>a</sup> Contiguous with debris.

<sup>b</sup> Farthest from debris.

<sup>c</sup> P = 0.05.

<sup>d</sup> Standard deviation.

<sup>e</sup> 1990 Data kindly provided by Drs. Cruse and Garcias, ISU Department of Agronomy.
APPENDIX C: ADDITIONAL TABLES TO SECTION III
Table C1. Grain yield of maize by individual row in maize strips in Experiment 1 at McNay Research Center, during 1990 and 1991 growing seasons.

<table>
<thead>
<tr>
<th>Effect</th>
<th>No tillage</th>
<th></th>
<th>Yield (Mg/ha)</th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>Mean (tillage)</td>
<td>LSD</td>
<td>SD</td>
<td>Inc. tillage</td>
</tr>
<tr>
<td></td>
<td></td>
<td>8.35</td>
<td>4.06</td>
<td>9.76</td>
<td>6.17</td>
</tr>
<tr>
<td>Mean (tillage)</td>
<td></td>
<td>8.35</td>
<td>4.06</td>
<td>9.76</td>
<td>6.17</td>
</tr>
<tr>
<td>LSDb</td>
<td></td>
<td>--</td>
<td>0.36</td>
<td></td>
<td></td>
</tr>
<tr>
<td>SDc</td>
<td></td>
<td>1.03</td>
<td>--</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Row</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Row 1d</td>
<td></td>
<td>8.48</td>
<td>3.78</td>
<td>11.53</td>
<td>5.55</td>
</tr>
<tr>
<td>Row 2</td>
<td></td>
<td>7.80</td>
<td>3.72</td>
<td>8.86</td>
<td>6.44</td>
</tr>
<tr>
<td>Row 3</td>
<td></td>
<td>7.91</td>
<td>4.90</td>
<td>8.70</td>
<td>6.37</td>
</tr>
<tr>
<td>Row 4</td>
<td></td>
<td>8.00</td>
<td>4.08</td>
<td>9.11</td>
<td>6.23</td>
</tr>
<tr>
<td>Row 5e</td>
<td></td>
<td>9.55</td>
<td>3.84</td>
<td>10.61</td>
<td>6.26</td>
</tr>
<tr>
<td>LSD</td>
<td></td>
<td>--</td>
<td>0.47</td>
<td></td>
<td></td>
</tr>
<tr>
<td>SD</td>
<td></td>
<td>0.18</td>
<td>--</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

a 1990 Data kindly provided by Drs. Cruse and Garcias, ISU Department of Agronomy.
b P = 0.05.
c Standard deviation.
d Contiguous with debris.
e Farthest from debris.
-- Not determined.
Table C2. Grain yield of maize by individual row in maize strips in Experiment 5 at McNay Research Center during 1991 growing season.

<table>
<thead>
<tr>
<th></th>
<th>No tillage 1991</th>
<th>Yield Mg/ha 1991</th>
<th>Intensive tillage 1991</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mean (tillage)</td>
<td>4.15</td>
<td>5.49</td>
<td>5.93</td>
</tr>
<tr>
<td>LSD</td>
<td>0.53</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Row</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Row 1&lt;sup&gt;a&lt;/sup&gt;</td>
<td>3.12</td>
<td>4.34</td>
<td>4.41</td>
</tr>
<tr>
<td>Row 2</td>
<td>3.94</td>
<td>5.28</td>
<td>6.20</td>
</tr>
<tr>
<td>Row 3</td>
<td>4.09</td>
<td>6.19</td>
<td>6.36</td>
</tr>
<tr>
<td>Row 4</td>
<td>5.02</td>
<td>5.81</td>
<td>6.31</td>
</tr>
<tr>
<td>Row 5&lt;sup&gt;b&lt;/sup&gt;</td>
<td>4.61</td>
<td>5.83</td>
<td>6.37</td>
</tr>
<tr>
<td>LSD&lt;sup&gt;c&lt;/sup&gt;</td>
<td>0.68</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

<sup>a</sup>Contiguous with debris.

<sup>b</sup>Farthest from debris.

<sup>c</sup>P = 0.05.
Table C3. Grain yield of maize by individual row in maize strips at Frantzen farm during 1990 and 1991 growing seasons.

<table>
<thead>
<tr>
<th>Row</th>
<th>1990 Yield (Mg/ha)</th>
<th>1991 Yield (Mg/ha)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Row 1&lt;sup&gt;a&lt;/sup&gt;</td>
<td>11.77</td>
<td>7.60</td>
</tr>
<tr>
<td>Row 2</td>
<td>9.79</td>
<td>7.58</td>
</tr>
<tr>
<td>Row 3</td>
<td>10.19</td>
<td>8.97</td>
</tr>
<tr>
<td>Row 4&lt;sup&gt;b&lt;/sup&gt;</td>
<td>11.23</td>
<td>7.25</td>
</tr>
<tr>
<td>LSD&lt;sup&gt;c&lt;/sup&gt;</td>
<td>--</td>
<td>0.91</td>
</tr>
<tr>
<td>SD&lt;sup&gt;d&lt;/sup&gt;</td>
<td>0.64</td>
<td>--</td>
</tr>
</tbody>
</table>

<sup>a</sup>Contiguous with debris.

<sup>b</sup>Farthest from debris.

<sup>c</sup>P = 0.05.

<sup>d</sup>Standard deviation.

<sup>e</sup>1990 Data kindly provided by Drs. Cruse and Garcias, ISU Department of Agronomy.