ABSTRACT

DUFFIE, LAURA ELIZABETH. Effects of intercropping corn and peanut on peanut leaf spot management and the spatial and temporal epidemiology of *Cercospora arachidicola*. (Under the direction of Dr. Turner B. Sutton and Dr. Barbara B. Shew)

Peanut-corn intercropping was evaluated for its potential to reduce early leaf spot (ELS) of peanut, caused by *Cercospora arachidicola*. Peanut (*Arachis hypogaea*) was intercropped in field plots with corn (*Zea mays*), and the effects of intercropping on dispersal of *C. arachidicola* and spatial dynamics of disease were examined over time. In 2000, the experiment consisted of five replicate blocks of square plots 16 rows wide and 14.6 m long. Treatments included unsprayed peanut (p) monoculture (Monocrop), sprayed peanut monoculture (Monocrop Sprayed), alternating rows of peanut and corn (c) (Intimate Intercrop), and four-row strip intercrops (2c, 4p, 4c, 4p, 2c) (Strip Old). In 2001, a second strip intercrop treatment was added (4c, 4p, 4c, 4p) (Strip New) and plots were 15.4 m long. Corn and peanut (VA 98R) were planted on May 9, 2000 at the Horticultural Crops Research Station near Castle Hayne, NC, and May 10, 2001 at the Umstead Farm Unit near Butner, NC. Both locations are outside of normal peanut production areas but are suitable for peanut culture. In late July (2000) and in mid August (2001), focal epidemics were initiated by placing infected peanut stems centrally in each plot. Leaf spot incidence and defoliation were determined weekly in a stratified sampling routine that allowed estimation of disease gradients in four directions. Airborne conidia were trapped with a Rotorod spore sampler in three blocks of all treatments except the Monocrop Sprayed. Peanuts were dug at maturity, and yield data were taken. Corn yield was estimated in 2001.
Early leaf spot symptoms were first observed near the inoculation site 22 days after inoculation in 2000 and 23 days after inoculation in 2001. In 2000, Intimate Intercrop and Monocrop reached the highest mean level (averaged across distance and direction) of disease incidence at around 41% by 63 days after inoculation. Disease incidence AUDPCs for Intimate Intercrop and Monocrop were significantly greater than the AUDPC for Strip Old intercrop, which was significantly greater than the AUDPC for Monocrop Sprayed. Natural populations of *Cercosporidium personatum* caused a non-point source late leaf spot (LLS) epidemic on the peanuts. In 2001, Monocrop again reached the highest level of disease incidence at 15%, 62 days after inoculation. Disease incidence AUDPCs for Monocrop were significantly higher than the other four treatments. Intimate Intercrop had an intermediate AUDPC, which was significantly greater than the two Strip treatments and the Monocrop Sprayed. The two Strip intercrop treatments also had intermediate AUDPCs. The Strip New treatment had significantly greater AUDPC values than the spray treatment. The Strip Old treatment had a statistically similar AUDPC to the sprayed treatment. Another non-point source late leaf spot epidemic occurred, but was much less severe than in 2000. Peanut yield was not significantly affected by either disease or intercropping during 2000. In 2001, peanut yield patterns were similar to those in 2000, but the Intimate Intercrop was significantly depressed. No benefit or reduction was observed in corn yield due to intercropping.

Apparent infection rates \( (r) \) were calculated as the slopes of the linearized logistic model applied to the disease progress curves. A repeated measures analysis revealed a treatment x distance interaction \( (p<0.05) \) during the last four sampling days of each ELS epidemic. This likely was due to the Monocrop Sprayed treatment, which maintained
low disease levels at all distances from the inoculum. Disease gradients were plotted for each treatment/day combination, and log-linear, log-log, logit-linear, and logit-log models were applied to gradients. In both years, best fits were obtained when log distance was used. The logit-log model was selected in 2001 due to higher disease levels and the increased likelihood of multiple infection. In 2001, the log-log model was used because multiple infections probably did not limit disease spread. Linearized daily slopes within each treatment were analyzed with ANOVA and those that were significantly different from the majority of others in that treatment were discarded. Averaged individual plot slopes from the remaining days were used to estimate gradient parameters \((b)\) for that treatment. Velocity \((V)\) then was calculated according to the formula \(V_s = rs/b\) using the averaged values of \(b\) obtained previously and arbitrarily chosen distances \((s)\). Velocity increased with increasing distance from the inoculum source. At \(s=1\), velocities during both years ranged from 0.40 to 0.96 over all treatments with the exception of Strip New, where \(V=0.222\). In 2000, Strip Old had the highest velocity at all distances, followed by Monocrop. Intimate Intercrop had the lowest velocity at all distances. In 2001, Strip New had the highest velocity at all distances from the source of inoculum. Velocities were comparable to those previously reported for LLS of peanut.
EFFECTS OF INTERCROPING CORN AND PEANUT ON PEANUT LEAF SPOT MANAGEMENT AND THE SPATIAL AND TEMPORAL EPIDEMIOLOGY OF _Cercospora arachidicola_.

by

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BIOGRAPHY

Laura Elizabeth Duffie was born on July 6th, 1978 in Kingston, New York. She attended primary and secondary schools in Raleigh, North Carolina. She attended Warren Wilson College in Asheville, North Carolina from 1996 until 2000, where she obtained her B.A. in Environmental Studies with a concentration in Sustainable Agriculture. Laura began work on a Master of Science degree in Plant Pathology in July, 2000, at North Carolina State University. She completed her degree in the Spring of 2003 under the direction of Drs. Turner B. Sutton and Barbara B. Shew.
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INTRODUCTION

ARACHIS HYPOGAEA: CHARACTERISTICS AND BACKGROUND

Peanut (*Arachis hypogaea* L.) is native to South America, originating in the central part of Brazil or northeastern Paraguay (Simpson et al. 2001). Six centers of genetic diversity have been recognized in South America (Wynne et al. 1991). Spanish explorers most likely carried peanuts to the western Pacific, Indonesia, and China in the 16th century and Portuguese explorers probably introduced peanut to India, Africa, and the Far East (Bunting et al. 1985). Africa is recognized as a secondary center of genetic variation (Hammons 1982). Peanut was probably introduced to the United States and reintroduced to South America from Africa (Moss and Rao 1995). The genus *Arachis* contains 70 species (Coffelt and Simpson 1997) and is within the subtribe Stylosanthisinae, the tribe Aeschynomeneae, and the family Fabaceae. *A. hypogaea* is the only economically important species within the genus *Arachis* (Moss and Rao 1995); however, *A. villosulicarpa* Hoehne and *A. stenosperma* Krapov. and W.C. Gregory are grown for nutritional and medicinal purposes in Brazil (Simpson et al. 2001). *A. hypogaea* is divided into two subspecies, *hypogaea* and *fastigiata*. Subspecies *hypogaea* is further subdivided into the botanical varieties *hypogaea* and *hirsuta* (Moss and Rao 1995). Virginia and runner market type peanuts belong to subspecies *hypogaea* var. *hypogaea* (Coffelt and Simpson 1997). Subspecies *fastigiata* is further divided into the botanical varieties *fastigiata, peruviana, vulgaris, and aequatoriana* (Moss and Rao 1995). Spanish and valencia peanuts are in the subspecies *fastigiata* var. *vulgaris* and var. *fastigiata*, respectively.
Peanut is grown globally; the three largest producers are India, China, and the United States, in decreasing order. The United States, however, leads the world in production of peanut per unit area (Coffelt and Simpson 1997). In 2000, total United States production was 1.5 million metric tons on 0.53 million ha. Within the United States, Georgia, Texas, North Carolina, Florida, Virginia, Oklahoma, New Mexico, and South Carolina led in production in 2001. North Carolina ranked third, having produced 15,300 metric tons on 49,800 ha (USDA-NASS 2001).

**BIOLOGY AND EPIDEMIOLOGY OF CERCOSPORA ARACHIDICOLA AND CERCOSPORIDIUM PERSONATUM**

**Introduction and significance.** The foliar peanut diseases early leaf spot (ELS) and late leaf spot (LLS) are caused by the globally significant pathogens, *Cercospora arachidicola* S. Hori and *Cercosporidium personatum* (Berk. and M.A. Curtis) Deighton [syn. *Phaeosariopsis personata* (Berk. & Curt.) V.Arx.], respectively. The teleomorphic stages of both organisms have been placed in the class Loculoascomycetes under the order Dothideales in the Ascomycotina. The anamorphs of both organisms are classified as Hyphomycetes according to the Saccardo system of classification of the imperfect fungi. The host range of *C. arachidicola* and *C. personatum* is confined to the genus *Arachis* (Stalker and Simpson 1995). Epidemics of ELS have frequently led to yield losses of 50% on unsprayed peanuts (Melouk and Shokes 1995). In the United States, yield losses can be up to 10% on sprayed crops. During the 1980’s, late leaf spot was the predominant foliar pathogen in runner production areas, and yield losses were similar to those discussed for early leaf spot (Knauft 1988). Currently, LLS causes losses sporadically in the United States, but it remains a serious problem in tropical and
subtropical production areas (Gibbons 1979). The two pathogens are similar in that they often form necrotic lesions on leaves and petioles and less frequently on stems, stipules and pegs. However, there are notable differences in the biology of these pathogens.

**Biology of *Cercospora arachidicola***. Conidia are subhyaline, olivaceous, and curved. Each conidium has 3 to 12 septa, a truncate base, and a subacute tip. The conidia form on short conidiophores in clusters of 5 to many, which are borne in dense fascicles on dark brown stromata. Conidia measure 37$\mu$m to 108$\mu$m x 2.7$\mu$m to 5.4$\mu$m, and leave a definite scar, as they are abjointed from the conidiophore. *C. arachidicola* commonly sporulates within the lesion on the adaxial leaflet surface with conidiophores becoming amphigenous only at maturity. The teleomorphic stage of the ELS pathogen, *Mycosphaerella arachidis* Deighton, is rarely observed in leaf lesions, and when formed, occurs on the previous years’ leaf litter (Jenkins 1938).

**Biology of *Cercosporidium personatum***. *Cercosporidium personatum* conidia have 1 to 9 septa and are obclavate. Conidia vary in size from 18$\mu$m to 60$\mu$m x 5$\mu$m to 11$\mu$m. Conidiophores form in dense clusters giving rise to medium olivaceous, straight to slightly curved, cylindrical conidia. Sporulation often occurs in concentric rings on the abaxial leaflet surface, but may also occur on the adaxial surface (Jenkins 1938). The teleomorph of the LLS pathogen, *Mycosphaerella berkeleyi* Jenk., rarely exists on peanut (Shokes and Culbreath 1997).

**Leaf spot disease cycles**. Peanut leaf spot pathogens have similar disease cycles. Primary inoculum is comprised of conidia or mycelia that have overwintered on crop residue such as pods, stems or petioles. Ascospores potentially serve as other sources. Wind, splashing raindrops, and insects disseminate conidia. Multi-celled conidia land on
peanut tissue, and germinate with one to several germ tubes (Shokes and Culbreath 1997). Infection may occur on both abaxial and adaxial leaf surfaces, and penetration pegs enter the plant through lateral surfaces of epidermal cells, or through natural openings, such as stomata (Jenkins 1938). *C. arachidicola* is a necrotroph, as intracellular hyphae are only found in cells that have been killed by the pathogen. *C. personatum*, however, remains intercellular, and is known to produce haustoria in living cells (Jenkins 1938, Abdou et al. 1974, Mims et al. 1989). Under ideal conditions, visible ELS symptoms develop 6 to 8 days after infection in favorable conditions, and LLS symptoms can be seen 10 to 14 days after infection (Shokes and Culbreath 1997).

ELS symptoms include the presence of necrotic lesions, which often are surrounded by a chlorotic halo. Individual lesions have a tan to reddish brown center and are 1 to 10 mm in diameter (Shokes and Culbreath 1997). Lesions caused by LLS are similar to those caused by ELS in size and shape. However, LLS lesions commonly have a less conspicuous or absent chlorotic halo and a dark brown to black color in the necrotic region.

Optimal environmental conditions for *C. arachidicola* are variable depending on the developmental process. Waliyar et al. (1994) found that sporulation occurred from 10 to 32° C, with the shortest latent period occurring at 25° C, under intermittent 16-h periods of leaf wetness. However, Alderman and Beute (1987) found that, when relative humidity was near 100%, the greatest amount of sporulation occurred at and increased within a narrower temperature range of 16 to 24° C. Furthermore, they reported a sharp decrease in sporulation from 28 to 32° C. Temperatures of 20 to 24° C with relative humidity >90% favor spore release (Shokes and Culbreath 1997). In India, Mallaiah and
Rao (1980) associated peak numbers of airborne conidia with temperatures between 29 and 31°C and relative humidity between 75 and 85%. Spores have been trapped as high as 2.7 m above the peanut canopy (Smith and Crosby 1973). Spores are released in a diurnal rhythm with peak release at 1000 hours (Sreeramulu 1970, Mallaiah and Rao 1980) and 1200 hours (Smith and Crosby 1973, Alderman et al. 1987). Conidial germination is favored by high relative humidities, near 100% (Oso 1972, Alderman and Beute 1986, Alderman and Beute 1987), and temperatures ranging from 19 to 25°C (Alderman and Beute 1986). Germ tube elongation, however, occurs over a broad range of temperatures (Alderman and Beute 1986).

Optimal environmental conditions for stages in the *C. personatum* life cycle are similar to those of *C. arachidicola*. Leaf wetness is an important limiting factor for infection (Butler et al. 1995). Shew et al. (1988) reported that maximum infection occurred at 20°C and it diminished at 28 to 32°C. Spore production is favored by temperatures between 15 and 20°C and relative humidity >95%. However, the correlation between relative humidity and sporulation is less significant than for *C. arachidicola* (Alderman and Nutter 1994). Alderman and Nutter (1994) suggest that this could be due to the sub-epidermal stroma, which rarely ruptures the epidermis. They conclude that this pathogen would therefore be more dependent on the water status of the host for conidial production. *C. personatum* also exhibits a diurnal pattern of spore release with maximum release reported at 1200 hours (Mallaiah and Rao 1980). Optimum temperatures for conidial germination are 16 to 20°C (Sommartya and Beute 1986).
DISEASE MANAGEMENT OF EARLY LEAF SPOT AND LATE LEAF SPOT

**Cultural practices.** Most cultural practices used to control either pathogen are aimed at reducing initial inoculum. Growers are encouraged to rotate peanut fields on a 3-year cycle, typically with cotton, corn, and soybean (Shokes and Culbreath 1997). Growers should also follow sanitary measures, including removal of volunteer peanuts and burial of peanut residue using a moldboard plow (Shokes and Culbreath 1997). Recent research indicates that planting peanut in residues of previous rotational or cover crops can suppress early leaf spot development (Monfort et al. 2001). Early planting dates in Florida shortened the time the crop was exposed to both ELS and LLS pathogens, thus significantly reducing severity and defoliation, and resulting in higher yields (Shokes et al. 1982). However, at early digging dates (105 days after planting) pod quality was reduced, with a lower overall proportion of total sound mature kernels than at later digging dates (118 days after planting) (Knauf et al. 1986).

**Host resistance.** Commercial cultivars vary somewhat in their susceptibility to ELS and LLS (Waliyar et al. 1995, Bailey 2002), but one resistant cultivar, Southern Runner, has been released (Gorbet et al. 1987). The highest levels of partial resistance are found in unadapted germplasm lines and in wild species-derived breeding lines (Wynne et al. 1991). Holbrook and Isleib (2001) found that Bolivia provided the most sources of ELS- and LLS-resistant peanut germplasm. Furthermore, India, Nigeria, and Sudan provided important sources of ELS-resistant germplasm, while germplasm originating in Ecuador were an unexpected source for LLS resistance. Incompatibility and ploidy barriers, along with market standards, make resistance via sexual hybridization transfer difficult (Ozias-Akins and Gill 2001).
Several biological rate-reducing components of partial resistance have been proposed for both pathogens, including decreased number of lesions per leaf, small lesion diameter, long latent period, small amounts of damaged leaf area, and decreased maximum percentage of lesions sporulating (MPLS) (Nevill 1981, Ricker et al. 1985). LLS resistance has been correlated with longer latent periods, reduced capacity for sporulation, and less defoliation. However, components of resistance for ELS were not clearly correlated (Nevill 1981). Quantifying resistance based on rate reducing components is important. Ricker et al. (1985) suggested that latent period could be used as an effective tool in the evaluation of ELS resistance in peanut lines, but number of lesions proved to be an unreliable measure. Johnson et al. (1986) found that components of resistance are best correlated with individual AUDPCs for defoliation and ELS disease incidence in field plots.

Mechanisms of resistance have not been characterized in detail. Conidia of *C. arachidicola* on susceptible cultivars germinate more quickly and in greater numbers than those on resistant cultivars (Waliyar et al. 1995). Abdou et al. (1974) found that pre-infection resistance to both ELS and LLS could be attributed to non-directional germ tube growth; conversely germ tubes on susceptible plants display directed growth toward open stomata. In addition, they suggested that post-infection resistance to both ELS and LLS is related to thickening of cell walls and deposition of pectic substances around the site of infection. Ketring and Melouk (1982) correlated ethylene production in ELS-susceptible plants with enhanced leaflet abscission.

Expression of resistance to ELS and LLS can, at times, be significantly influenced by various environmental factors, including temperature and relative humidity, thus
resulting in unstable resistance expression (Subrahmanyam et al. 1982, Waliyar et al. 1994, Waliyar et al. 1995). However, Shew et al. (1988) found that genotype rankings across different temperature and humidity regimes remained similar, based on LLS lesion numbers. Genotype x environment interactions may or may not be encountered across diverse environments, but should remain a consideration when breeding for resistance. Although attempts have been made to exploit low to moderate resistance present in released cultivars, this alone has proved inadequate to achieve optimal yields during most years (Bailey et al. 1994).

**Chemical control.** Control is currently achieved primarily through fungicidal sprays, which should be applied beginning approximately 30 to 40 days after planting and continuing at 10 to 14 day intervals (Smith and Littrell 1980, Melouk and Shokes1995). Shokes et al. (1982) report earlier initiation of fungicide applications on a calendar schedule in Florida reduces severity and defoliation, and results in higher yield. They further suggest that delaying spray initiation may increase amounts of initial inoculum available for the following year. Although chlorothalonil and tebuconazole are the most commonly used compounds, many other fungicides also are used to control ELS (Melouk and Shokes 1995, Bailey 2002). Chlorothalonil use is discouraged in fields with a history of Sclerotinia blight caused by *Sclerotinia minor* Jagger, as it often increases blight severity (Hau and Beute 1983).

Fungicide applications currently account for 20% of the variable costs needed to produce peanuts (Hagan et al. 2000). Techniques for predicting ELS development are historically based on minimum temperature and leaf wetness (predicted by relative humidity) and date back to 1966 (Jensen and Boyle 1966). Several forecasting systems
have been developed to reduce spray frequencies. The Parvin, Smith, and Crosby (PSC) program (Parvin et al. 1974) adapted and computerized the Jensen and Boyle model (1966). PLAM (peanut leaf spot advisory) in North Carolina (Bailey et al. 1994) and the early leaf spot advisory in Virginia (Cu and Phipps 1993) are based on algorithms that use records of temperature and relative humidity to predict effective spray dates. However, the Virginia program attempts to relate the aforementioned components to pathogen sporulation, conidial germination, stomatal penetration, and germ tube elongation (Cu and Phipps 1993). AU-Pnut considers daily rain events (Hagan et al. 2000). Although AU-Pnut was originally developed for use on LLS, it also has application to ELS (Jacobi et al. 1995). In Oklahoma, Wu et al. (1996) reported that PSC, the VA program, AU-Pnut, and modified versions of all these models all reduced number of spray applications during 2 years, but varied in yield maintenance, disease control, and total spray savings. Typically, use of a disease forecaster will save 1 to 3 sprays in the Virginia-Carolina area compared to a calendar spray program (Hagan et al. 2000). Implementation of these forecasters varies, but adaption is widespread in North Carolina and Virginia. In 1990, 80% of surveyed North Carolina farmers said they were using weather-based advisories (Bailey et al. 1994).

In areas of the world where economic constraints limit the use of fungicides and communication technology is limited, neither a calendar schedule nor a weather-based spray plan is feasible. Waliyar et al. (2000) have developed decision trees for farmers in Niger and Benin based on the cultivar, the number of sprays, and combinations of spray dates and resulting net gains from those combinations. These help farmers achieve effective control while limiting uneconomical sprays (Waliyar et al. 2000).
OTHER PEANUT PATHOGENS

**TSWV.** Tomato spotted wilt virus (TSWV) is a tospovirus in the family Bunyaviridae, and is prevalent in Europe, Australia, Africa, and the Americas. Virions are 80-100 nm and have a lipid-protein coat (Buchen-Osmond 1998). TSWV is transmitted to a wide host range by eight species of thrips and can only be acquired by larval thrips. Adult thrips cannot acquire the virus. Both second-instar larvae and adults can transmit the virus after a latent period within the host (Chatzivassiliou 1999, Adkins 2000). TSWV is probably not transmitted by seed (Demski and Reddy 1997).

Symptoms of TSWV include mottling or chlorotic spots that mature into streaks or rings of chlorosis or necrosis. Temperatures greater than 30° C are correlated with the wilting of fully expanded leaflets, and afterwards terminal buds die. The plant is killed as necrosis spreads toward the base. Plants that are infected early in the season often exhibit secondary infection, such as stunting, mosaic mottling, proliferation of axillary shoots, general chlorosis, and a bushy appearance (Demski and Reddy 1997).

Control of TSWV includes cultural controls and breeding for resistance. Good growing practices that result in a closed canopy disrupt thrips that are attracted to patchy crops. Intercropping in a 1:3 ratio with a fast growing grain may also decrease incidence. Good sources of resistance to TSWV have been identified and are the focus of breeding efforts (Demski and Reddy 1997). For example, Georgia Green is a partially resistant cultivar (Branch 1996).

**Sclerotium rolfsii.** *Sclerotium rolfsii* Sacc. is the causal agent of stem rot, also known as Sclerotium rot, southern stem rot, southern blight, or white mold. The
economically unimportant teleomorph is a Basidiomycete. *S. rolfsii* has a world wide
distribution and causes yield losses of 7 to 10% in the United States (Taber 1997)

The first noticeable symptoms of stem rot are general chlorosis of the entire plant,
the main stem, or a lateral branch. Light brown lesions develop on stems and pegs near
the soil surface and turn dark brown over time. The lesions may be wet and spongy in
texture, or have a shredded appearance. Signs of the disease include white mycelial
sheathes around the soil line and base of the plant, as well as abundant round sclerotia.
Sclerotia are initially pale in color, but become dark brown with maturity (Taber 1997).

Cultural controls for stem rot include deep plowing, cultivation for weed control,
and 3 to 4 year crop rotations. Some *Trichoderma* spp. have been shown to have
biocontrol activity against *S. rolfsii*. Formulation difficulties pose problems for
commercialization of these biocontrol agents. Some pesticides may increase the
incidence of stem rot by killing resident populations of *Trichoderma* spp. Chemical
compounds, such as tebuconazole, flutalonil, and azoxystrobin provide good to excellent
control of stem rot, but correct timing and application methods are critical for success
(Jackson and Damicone 1996). Finally, resistance has been identified in genotypes with
erect growth habits as compared to those with spreading growth habits. Resistance
unrelated to growth habit has also been identified, and is integral in breeding programs
(Taber 1997).

**INTERCROPPING**

Intercropping is defined as the simultaneous cultivation of two or more species
that are grown in close association (Andrews and Kassam 1976). Both species may be
economic (e.g. maize and beans, rubber and pineapple) or a production crop may be grown with a “living mulch” primarily for weed and erosion control (e.g. cabbage with white clover). Intimate (row) intercropping involves growing multiple crops simultaneously when one or more crops are planted in rows, and strip intercropping involves the cultivation of multiple crops in variably wide strips such that the crops are independent, but may still interact agronomically (Andrews and Kassam 1976). Intercropping has been studied largely from an agronomic and yield-based perspective, and particularly with reference to small-scale farming in less-developed countries, where it is practiced extensively (Papendick et al. 1976, Francis 1985, Vandermeer 1989, Thurston 1992). Intercropping was a common practice before mechanization. Crop diversity through intercropping helped maintain healthy diets, and guarded against famine if one crop failed due to pests, disease, or unfavorable conditions. However, the transition to homogeneous, single-species stands was favored by the advent of modern machinery, breeding for high yielding cultivars, and other modern cropping strategies (Vilich-Meller 1992). Intercropping is occasionally suggested in extension publications for production systems in the United States (Davis 1997). The overall yield of an intercrop relative to its components grown in monocrop are quantified by the parameters “Relative Yield Total” (RYT) or “Land Equivalence Ratio” (LER). Legumes are commonly grown in intercrops because of the nitrogen they make available to other species, and have been shown to increase corn yields in Kenya (Rao and Mathuva 2000).

Although the motivation for intercropping has been connected with and advocated for disease control (Browning 1974, Burdon 1978, Altieri and Liebman 1986), few manipulative studies have been designed explicitly to evaluate its efficacy for this
purpose. Observational studies reveal the absence of “crowd” diseases where native grain populations co-evolved with their parasitic fungal counterparts, and subsequently, developed only isolated pockets of mild disease, which are known to be devastating to cultivated crops (Browning 1974).

A review of studies on intercropping and disease indicates that the general trend is toward less disease in intercrops compared to monocrops. For example, powdery mildew on barley was reduced when grown with wheat or oats (Burdon and Chilvers 1977, Vilich-Meller 1992), bean rust declined under intercropping with corn (Moreno and Mora 1984, Boudreau and Mundt 1992), soybean mosaic virus was lessened when soybeans were intercropped with sorghum (Bottenburg and Irwin 1992), and plantain suffered less from black Sigatoka when cultivated with cassava (Emebiri and Obiefuna 1992). Disease prevention through intercropping has not been studied extensively for peanut, but peanut rosette virus was less intense when peanuts were alternately sewn with common bean (Farrell 1976), and intercropping peanut with pigeonpea has been demonstrated to reduce ELS as well as LLS in India (Ghewande et al. 1993). Some diseases are favored by intercropping, such as white mold on beans grown with corn (Van Rheenen et al. 1981). In fact, a given crop combination may not always affect a pathogen in the same way. Examples exist in which disease has been decreased, unaffected, and increased by the same intercrop, depending on the site and/or season, for several fungi [e.g. *Phaeoisariopsis griseola*, causing angular leaf spot of bean (Sengooba 1990, Boudreau 1993)], a virus [African mosaic virus on cassava (Fargette and Faquet 1988)], and a nematode [*Pratylenchus brachyurus* on corn (Egunjobi 1984)]. For this reason, Coolman and Hoyt (1993) stated that every crop combination must be evaluated separately for
intercropping efficacy on disease control. Alternatively, Boudreau and Mundt (1992, 1997) emphasized the need to understand mechanisms of disease-intercrop interaction in order to predict how mixtures perform in specific settings.

Several mechanisms have been proposed for intercrop-disease interactions (Trenbath 1977, Burdon 1978, Boudreau and Mundt 1992). These potential mechanisms may be divided into those that affect dispersal of the pathogen and those that affect non-dispersal aspects, such as germination, growth on/in the host, and reproduction. The former might include spore interception by the non-host (trapping) (Burdon and Chilvers 1977), alteration in the velocity and quality of the wind, raindrop attenuation, competition effects on the host which in turn influence dispersal, and so on. The latter include microclimate alterations (changes in temperature and humidity), competitive effects that alter host susceptibility, and resistance induced by products or pathogens associated with the non-host. Clearly, these mechanisms may be interrelated.

Little research has been done to evaluate the contribution of these mechanisms to disease alterations in intercrops. Researchers have observed that barrier rows of corn or cotton between peanut test plots are very effective at preventing spread of ELS and LLS between adjacent plots (Johnson et al. 1986). Wind speed above a corn-bean intercrop was reduced by 55-63% of that above a bean monocrop in one study (Boudreau 1993), which could reduce spore removal and lateral displacement. Raindrops may be intercepted by corn leaves; sudangrass located between strawberry plants significantly reduced deposition of spores of *Colletotrichum acutatum* due to simulated rain (Boudreau and Madden 1995). Disease reduction has been associated with changes in insect vector
movement due to intercropping in peanut-bean and corn-bean associations (Farrell 1976, Power 1987).

Density is an important complicating factor that always must be considered when intercropping studies are evaluated and compared. “Replacement” experiments substitute some of the host population with another species, thus lowering host density. “Additive” experiments insert the new species among the host plants and change overall density, but not the density of the host. These density changes may be responsible for disease alterations independent of the presence of a new species.

Generally, it is thought that decreased host density decreases disease incidence due to a lesser probability that a propagule will contact the host (Burdon and Chilvers 1977). Burdon and Chilvers (1976a, 1976b, 1977) were able to ascribe reductions in powdery mildew under wheat-barley intercrops, and reductions in damping off in ryegrass-cress mixtures, to concomitant reductions in host density, and not the presence of the non-host species *per se*. However, there were also instances in which increased host density lead to lesser or no change in disease levels. Mann and Scarborough (Power 1987) found that fusiform rust was reduced when trees were spaced closely (1948), and insect-vectored virus disease often diminishes as host density is increased.

Boudreau and Mundt (1992) eliminated host density as a factor by using an “additive” design to study rust of common bean as affected by intercropping with corn. Dispersal parameters (dispersal gradient slope and spore escape from plots) were affected not by physical interference of corn with spore movement, but instead by changes in the bean canopy due to competition with corn. This competition factor consistently steepened dispersal gradients, but effects on spore escape changed as the crop grew.
Corn reduced disease severity due to non-dispersal effects by 96% late in one season, but not in a second, with microclimate changes implicated as the cause. In another pathosystem, *Colletotrichum acutatum* spore deposition was diminished when strawberry plants were grown at 75 and 100% of maximum density, increased at 50%, and diminished slightly at 25% (Boudreau and Madden 1995). Computer simulations (Boudreau and Mundt 1993) indicated that dispersal gradient changes such as those observed in the field may have little effect on the overall disease progress, but spore escape and non-dispersal changes due to species mixing can lead to pronounced reductions in area under disease progress curves.

**LITERATURE CITED**


EFFECTS OF INTERCROPPING CORN AND PEANUT ON EPIDEMICS OF PEANUT LEAF SPOT

ABSTRACT

Peanut-corn intercropping was evaluated for its potential to reduce early leaf spot (ELS) of peanut, caused by Cercospora arachidicola. Peanut (Arachis hypogaea) was intercropped in field plots with corn (Zea mays), and the effects of intercropping on dispersal of C. arachidicola and spatial dynamics of disease were examined over time. In 2000, the experiment consisted of five replicate blocks of square plots 16 rows wide and 14.6 m long. Treatments included unsprayed peanut (p) monoculture (Monocrop), sprayed peanut monoculture (Monocrop Sprayed), alternating rows of peanut and corn (c) (Intimate Intercrop), and four-row strip intercrops (2c, 4p, 4c, 4p, 2c) (Strip Old). In 2001, a second strip intercrop treatment was added (4c, 4p, 4c, 4p) (Strip New) and plots were 15.4 m long. Corn and peanut (VA 98R) were planted on 9 May 2000 at the Horticultural Crops Research Station near Castle Hayne, NC, and 10 May 2001 at the Umstead Farm Unit near Butner, NC. Both locations are outside of normal peanut production areas but are suitable for peanut culture. In late July (2000) and in mid August (2001), focal epidemics were initiated by placing infected peanut cuttings centrally in each plot. Leaf spot incidence and defoliation were determined weekly in a stratified sampling routine that allowed estimation of disease gradients in four directions. Airborne conidia were trapped with a Rotorod spore sampler in three blocks of all treatments except the Monocrop Sprayed. Peanuts were dug at maturity, and yield data were taken. Corn yield was estimated in 2001. ELS symptoms were first observed near the inoculation site 22 days after inoculation in 2000 and 23 days after inoculation in 2001. In 2000, Intimate Intercrop and Monocrop reached the highest mean level of disease incidence (averaged across distance and direction) at around 41% by 63 days.
after inoculation. Disease incidence AUDPCs for Intimate Intercrop and Monocrop were significantly greater than the AUDPC for Strip Old intercrop, which was significantly greater than the AUDPC for Monocrop Sprayed. Natural populations of *Cercosporidium personatum* caused a non-point source late leaf spot (LLS) epidemic on the peanuts. In 2001, Monocrop again reached the highest level of disease incidence at 15%, 62 days after inoculation. Disease incidence AUDPCs for Monocrop were significantly higher than the other four treatments. Intimate Intercrop had an intermediate AUDPC, which was significantly greater than the two Strip treatments and the Monocrop Sprayed. The two Strip intercrop treatments also had intermediate AUDPCs. The Strip New treatment had a significantly greater AUDPC value than the Monocrop Sprayed treatment. The Strip Old treatment had a statistically similar AUDPC to the Monocrop Sprayed treatment. Another non-point source LLS epidemic occurred, but was much less severe than in 2000. Peanut yield was not significantly affected by either disease or intercropping during 2000. In 2001, peanut yield patterns were similar to those in 2000, but the Intimate Intercrop was significantly depressed. No increase or reduction in corn yield was observed due to intercropping.

The peanut diseases early leaf spot (ELS) and late leaf spot (LLS) are caused by the globally significant pathogens, *Cercospora arachidicola* S. Hori and *Cercosporidium personatum* (Berk. and M.A. Curtis) Deighton [syn. *Phaeosariopsis personata* (Berk. & Curt.) V. Arx.], respectively. Yield losses have been reported to exceed 50% on unsprayed fields (Melouk and Shokes 1995), and can exceed 10% on sprayed fields in the United States. Combinations of several control strategies are recommended. Reduction of initial inoculum is achieved through cultural measures such as crop rotation, volunteer
peanut removal, and burial of peanut residue (Shokes and Culbreath 1997). Reduction in temporal exposure may be achieved by early digging or planting dates (Knauff et al. 1986, Shokes et al. 1982). Spray applications with fungicides such as chlorothalonil or tebuconazole (Melouk and Shokes 1995, Bailey 2002) are required to achieve optimal yields during most years (Bailey et al. 1994). Several forecasting systems have been developed (Jensen and Boyle 1966, Parvin et al. 1974, Cu and Phipps 1993, Bailey et al. 1994, Jacobi et al. 1995) and use is widespread in North Carolina and Virginia (Bailey et al. 1994). Use of a forecast will generally save from 1 to 3 sprays in the Virginia-Carolina area compared to a calendar spray program (Hagan et al. 2000). In addition, low to moderate resistance is present in some released cultivars and much effort has been directed at developing cultivars with high levels of leaf spot resistance. Components of resistance include decreased number of lesions per leaf, small lesion diameter, long latent period, small amounts of damaged leaf area, and decreased maximum percentage of lesions sporulating (MPLS) (Nevill 1981, Ricker et al. 1985).

Intercropping is defined as the simultaneous cultivation of two or more species that are grown in close association (Andrews and Kassam 1976). Both species may be economic (e.g. maize and beans, rubber and pineapple) or a production crop may be grown with a “living mulch” primarily for weed and erosion control (e.g. cabbage with white clover). Intercropping has been studied largely from an agronomic and yield-based perspective, and particularly with reference to small-scale farming in less-developed countries, where it is practiced extensively (Papendick et al. 1976, Francis 1985, Vandermeer 1989, Thurston 1992). Although the motivation for intercropping has been connected with and advocated for disease control (Browning 1974, Burdon 1978, Altieri
and Liebman 1986), few manipulative studies have been designed explicitly to evaluate its efficacy for this purpose. A review of studies on intercropping and fungal diseases indicates that the general trend is toward less disease in intercrops compared to monocrops (Burdon and Chilvers 1977, Moreno and Mora 1984, Boudreau and Mundt 1992, Emebiri and Obiefuna 1992, Vilich-Meller 1992). However, some fungal diseases are favored by intercropping, such as white mold on beans grown with maize (Van Rheenen et al. 1981). Others are inconsistently affected, due to site-to-site and season-to-season variability (Sengooba 1990, Boudreau 1993).

Increased sustainability in peanut cultivation must include strategies for controlling peanut leaf spot. Furthermore, strategies must be amenable to the diverse growing practices utilized by peanut farmers worldwide. The goal of this research was to evaluate the efficacy of intercropping peanut with corn as a disease management tool for ELS in commercial-mechanized and small-scale production systems. Strip cropping corn and peanut (row ratio 4:4) was tested in the former case, and alternating single rows of corn and peanuts (row ratio 1:1) was used in the latter. The Strip intercrop is more amenable to mechanization and was designed to accommodate commercial production as in the United States. Our goal was specifically addressed through the following objectives: to understand the effects of corn on peanut leaf spot epidemics and disease dynamics over time and to determine what, if any, disease or yield advantages accrue from growing peanuts with corn.
MATERIALS AND METHODS

Peanut and corn were grown in the summer of 2000 at the Horticultural Crops Research Station in Castle Hayne, NC, and in 2001 at the Umstead Farm Unit in Butner, NC. Both locations are suitable for peanut production, but are outside the normal commercial production region, in order to eliminate background inoculum. Field plots grown in Castle Hayne were irrigated during dry periods, and those grown in Butner were not. The ELS susceptible peanut cultivar VA 98R was planted in both years. Corn cultivars Pioneer 33V08 and Pioneer 32K61 were planted in 2000, and the high biomass tropical cultivar Garst 8315 IT was planted in 2001.

Intercrops were established on 9 May in 2000 and on 10 May in 2001. Rows were 91 cm apart in 2000 and 102 cm apart in 2001. Peanut and corn spacing was one seed per 0.8 cm and 0.6 cm deep. Corn was later thinned to one plant per 15 cm. Treatments in 2000 consisted of peanut Monocrop, peanut Monocrop Sprayed with chlorothalonil applications at two week intervals for leaf spot control (standard production practice), alternating rows of peanut and corn (Intimate Intercrop) and peanut and corn grown in four-row strips (Strip Old). The strips of corn (c) and peanut (p) were planted as follows: c-c-p-p-p-c-c-c-c-p-p-p-p-c-c. For 2001, another arrangement of corn and peanut (Strip New) was added to the experiment. In this treatment, rows of corn and peanut (Strip New) was added to the experiment. In this treatment, rows of peanut and corn were arranged as follows: c-c-c-c-p-p-p-c-c-c-c-p-p-p-p-p-p-p-p (Fig. 2.1). The square plots were 16 rows wide and approx 15m or 15.5m long in 2000 or 2001. The experimental design in both years was a randomized complete block with five replications.
Peanut and corn were grown using standard recommendations for pesticide, herbicide, and fertilizer applications. The soil insecticide aldicarb was applied in furrow to peanut rows during both years at 11.2 kg formulation/ha. The insecticide chloropyrifos was applied at a rate of 15.7 kg/ha, 4 weeks after planting in 2000 and 8 weeks after planting in 2001. In each year, the herbicide 5-metolachlor was applied in furrow to both corn and peanut rows at a rate of 1.75 L/ha. Imazethapyr and sethoxydim were applied as needed to control weeds throughout the growing season. Fertilizer was applied as a split application based on North Carolina Department of Agriculture recommendations for N, P, and K from soil tests. A commercial inoculant of *Bradyrhizobium* was added in furrow to the peanut rows during both years. Landplaster was applied by hand, at a rate of 673 kg/hectare, to all peanut rows, 4 weeks after planting.

Focal epidemics of ELS were established by placing an inoculum source in the center of each plot on 28 July 2000 and on 10 August 2001. Inoculum sources were prepared by placing infected peanut cuttings, collected from unsprayed plots grown in Lewiston, NC, in moist sand in 0.6 L-capacity plastic cups. At the time of collection, only ELS was observed in these plots and no other disease was ever observed on the cuttings during the course of the experiment. The cuttings were placed in a moist chamber at 100% RH for 48 hours to induce sporulation. The cups with inoculum were placed in the center of all plots. Due to the planting pattern, the inoculum was placed between two non-host corn rows in the center of the Strip Old treatment. In the Strip New treatment, the center of the plot was between a row of corn and a row of peanut; thus the inoculum source was placed in proximity to a peanut row (Fig. 2.1). A second set
of fresh cuttings were used to replace the initial cuttings approximately 7 days after introduction into the plots. All sources of inoculum, during both years, were heavily watered with a watering can at the time of placement in the field, to ensure a moist and humid environment for spore development and dispersal.

The Monocrop Sprayed treatment was sprayed with chlorothalonil, using a carbon dioxide backpack sprayer. The fungicide was applied at 1.26 kg a.i./ha and was delivered in 190 L water/ha. In 2000, sprays occurred on 3 August and 29 August. In 2001, sprays occurred on 14 August, 29 August, and 20 September. These sprays were adequate to control disease under conditions present in each year (Fig. 2.2).

Disease progress data was taken once a week after inoculation until harvest. ELS disease incidence was estimated as the percentage of leaflets in the sample area with at least one spot. Percentage defoliation, or percent leaflets lost, was determined by counting numbers of defoliated leaflets and nodes on two arbitrarily selected lateral stems within each sampling area (Johnson et al. 1986). To characterize disease gradients, data were taken at the inoculum source and in four perpendicular arms, with adjustments for each treatment, as depicted in Fig. 2.1. Each arm was divided into four sections, except the vertical arms in the Strip treatments, which were divided into two sections. In the treatments Monocrop, Monocrop Sprayed, Strip Old, and Strip New, each of the sections was 91 cm x 91 cm and encompassed two rows. In the Intimate Intercrop treatment, each of the sections was 91 cm x 46 cm, and encompassed one row. In the Intimate Intercrop and both Monocrop treatments, the center-most of the four sections of each arm overlapped at the point of inoculum. In the Strip Old treatment, the center-most section of the horizontal arms, and the center-most section of one of the vertical arms overlapped
two rows upwind of the inoculum, which was between two non-host corn rows. The Strip New treatment had the same pattern of overlap, but the point of origin occurred at the inoculum (Fig. 2.1).

Eight subsamples were taken from each arm. This was done to ensure the same level of precision in each plot (Boudreau and Mundt 1992). Sections within the four arms of the Intimate treatment were divided into two equal parts, and one subsample was taken from each half. Sections within the four arms in the Monocrop and Monocrop Sprayed treatments were divided into four equal quadrants. Two of the four quadrants were randomly subsampled within each section. The four sections of the two horizontal arms within the Strip Old and Strip New treatments were divided and subsampled according to the same methodology of those in both Monocrop treatments. The two sections within the two vertical arms of the Strip treatments were divided into four equal quadrants, and one subsample was taken from each quadrant. In all treatments, a total of 32 samples were taken from each plot.

In addition to ELS assessment, natural epidemics of late leaf spot were observed in each year and disease incidence was evaluated at each sampling location. For disease progress curves and for calculation of Areas Under the Disease Progress Curves (AUDPC) for both diseases, data were averaged from all sample locations in each plot.

Peanuts were dug on 19 October in 2000 and 16 October in 2001. Yield was taken in each row and data were pooled for the plot total. Subsamples from each row within each plot were collected, pooled, and weighed. The subsamples from each plot were dried, weighed, and percent moisture was calculated. Plot yield totals were then adjusted for water content.
Corn yield was estimated in 2001 by hand harvesting arbitrarily-selected 2 m sections in each of four rows of the intercrops. Corn was hand harvested because a mechanical harvester was not available on the research station. In the Strip Old treatment, yields from interior rows were recorded separately from rows bordering peanut. The interior row yields were used to estimate Corn Monocrop yields and the combined bordering and interior row yields were used to estimate the Strip New yields (Lesoing and Francis 1999).

Aerial conidial concentrations were determined using Rotorod Aerobiological Samplers (Sampling Technologies, Inc., Minnetonka, MN). Samplers were placed in three of the five blocks, in all treatments except the Monocrop Sprayed, at one half the distance from the plot center to the northeast plot edge (3.75 m in 2000 and 3.88 m in 2001). This was the predominant wind direction at both locations during both years. Samplers were fixed on poles 1.0 m from ground level. All samplers were controlled by a CR 21 micrologger, which was programmed to run for 1 minute every 10 minutes, 5 days a week. Occasionally, the hardware malfunctioned, but a minimum of 3 samples were obtained each week. Rods were changed every 24 hours and stored at 4°C until analysis. *C. arachidicola* conidia on each rod were counted, and daily aerial concentrations were determined.

Treatment effects on ELS AUDPCs, LLS AUDPCs, defoliation AUCs, peanut yield, and corn yield were evaluated by analysis of variance conducted with SAS version 8 software for personal computers (SAS Institute, Inc., Cary North Carolina).
RESULTS

**Early leaf spot.** Conditions were highly conducive for ELS development in 2000. Averaged final incidence in the Monocrop and Intimate Intercrop was 40.1% (Fig. 2.2a). Individual observations of disease incidence near 100% were made near the inoculation site in many plots. Disease progress curves showed clear differentiation among treatments. Disease in the Monocrop and Intimate Intercrop followed similar curves and displayed the highest levels of ELS. Monocrop Sprayed had a low, flat curve, and displayed the lowest levels of ELS. Strip Old intercrop had an intermediate curve with maximum average disease levels reaching 24.6%.

In 2001, conditions were somewhat less favorable for leaf spot development in our plots due to a drier growing season. The resulting epidemic was scaled considerably lower than the previous year. Averaged final incidence in the Monocrop was 15.4% (Fig. 2.2b). Individual observations of disease incidence near 80% were made near the inoculation site in many plots. Disease progression in the Monocrop Sprayed treatment was similar to the previous year and disease progress curves remained low and flat. All intercropping treatments, including Intimate Intercrop, had lower rates of disease progress than the Monocrop. Intimate Intercrop and Strip New treatments averaged 9.1% and 8.3% final ELS incidence. Final average disease incidence for Strip Old was 5.1%.

AUDPCs for treatments during the 2000 epidemic reflected trends similar to disease progress curves and showed significant reductions in ELS for the Strip Old intercrop relative to the Monocrop and Intimate Intercrop (Fig 2.3a). Very little disease developed in the Monocrop Sprayed plots. AUDPCs for Monocrop, Intimate Intercrop and Strip Old intercrop plots were significantly greater than AUDPCs of the Monocrop.
Sprayed plots. AUDPCs for Intimate Intercrop and Monocrop were not significantly different.

AUDPCs in 2001 reflected trends from the disease progress curve as well as the diminished scale of the epidemic. All intercrops showed significant reductions in ELS incidence relative to the Monocrop (Fig. 2.3b). The Intimate Intercrop and Strip New treatments did not differ significantly, nor did the Strip New and Strip Old treatments. AUDPC for the Strip Old treatment was not significantly different from that of the Monocrop Sprayed treatment.

**Late leaf spot.** Symptoms of naturally occurring LLS were observed 35 days after inoculation with ELS (31 August) in 2000 and 42 days after inoculation (26 September) in 2001. In 2000, the disease progress curves of the Monocrop, Intimate Intercrop, and Strip Old intercrop treatments were similar, and overall, were not similar to ELS disease progress curves (Fig. 2.4a). LLS incidence in Strip Old began to rapidly rise around day 35, remained higher than the other treatments on days 40 and 55, and reached a final incidence of 33.5%. Incidence of LLS in the Monocrop and Intimate Intercrops began to rise rapidly around day 43, yet they remained lower than Strip Old on days 40 and 55. The final incidence of Monocrop and Intimate Intercrop treatments reached 36.7% and 40.2%, respectively, by the end of the season and were higher than that of the Strip Old intercrop. Monocrop Sprayed had a low flat disease progress curve.

The LLS epidemic was strikingly diminished in 2001 (Fig. 2.4b). The overall patterns of the LLS disease progress curves were similar to those of ELS disease progress curves during 2001, and all curves began to rapidly rise around day 43. Monocrop reached the highest average level of LLS disease incidence at 3.3%. Final disease
incidence in the Intimate Intercrop, Strip New, and Strip Old intercrops averaged 1.5%, 1.4%, and 1.1% respectively. Monocrop Sprayed averaged low levels of LLS on all days of the epidemic.

AUDPCs for treatments during the 2000 LLS epidemic reflected trends similar to the disease progress curves. AUDPC for Monocrop was significantly lower than that of Strip Old, significantly higher than that of Monocrop Sprayed, and not significantly different from that of Intimate Intercrop (Fig. 2.5a). The Intimate Intercrop had an AUDPC that did not significantly differ from that of Monocrop or Strip Old. Very little disease developed in the Monocrop Sprayed.

AUDPCs in 2001 reflected trends from the LLS disease progress curve and treatment differences were similar to those for ELS. All intercrops showed significant reductions in LLS incidence relative to the Monocrop (Fig. 2.5b). The Intimate Intercrop and Strip New treatments did not significantly differ; the Strip New and Strip Old treatments did not differ from each other. AUDPC for the Strip Old treatment was not significantly different from that of the Monocrop Sprayed.

**Defoliation.** The defoliation AUCs increased slowly until the end of the 2000 growing season in all treatments (Fig. 2.6a). The Intimate Intercrop and Strip Old treatments have the highest average percentage defoliation at 33%. The Monocrop treatment was comparatively lower, and reached 27% average defoliation. Average defoliation in the Monocrop Sprayed treatment reached only 15%.

In 2001, the defoliation AUCs were more erratic than in 2000, and overall average defoliation was diminished (Fig. 2.6b). The Intimate Intercrop reached the
highest average percentage defoliation followed by Monocrop, Strip New, Strip Old, and Monocrop Sprayed with respective averages of 5.0%, 4.2%, 4.0%, 3.2%, and 2.0%.

Defoliation AUCs in 2000 revealed that defoliation in the intercropped treatments averaged significantly higher than the monocrop treatments (Fig. 2.7a). The AUC for the Intimate Intercrop was not significantly different from that of Strip Old and the Monocrop AUC was not significantly different from the AUC for the Monocrop Sprayed. In 2001, there was no significant difference among areas under the defoliation progress curves of the five treatments (Fig. 2.7b).

**Yield:** Peanut yields averaged 3077 kg/ha in 2000, and did not differ among treatments (Fig. 2.8a). Monocrop and Monocrop Sprayed had similar yields of 3410 kg/ha and 3,512 kg/ha respectively. Intimate Intercrop and Strip Old also had similar yields of 2692 kg/ha and 2696 kg/ha. Although differences were not significant, yields in intercropped treatments were depressed. Peanut yields averaged 2337 kg/ha in 2001. Yields in strip intercrop treatments were reduced again in 2001, and the difference were significant in the Strip Old treatment (Fig. 2.9b). The Monocrop, Strip New, and Monocrop Sprayed yields were not significantly different. The Intimate Intercrop yields were severely depressed and significantly lower than those of all other treatments at 723 kg/ha. Averaged across treatments, corn yields were 8166 kg/ha in 2001. Corn yields were not significantly different across treatments (Fig. 2.9).

**Aerial Conidial Concentration:** Concentrations of *C. arachidicola* conidia were plotted against time in 2000 (Fig 2.10a). There were no clear differences among treatments in spore concentrations, and major peaks occurred at similar times in all treatments. The first peak in conidial catch occurred around 15 days after inoculation.
The most notable peak for all treatments occurred at 63 days after inoculation. The highest concentration of conidia occurred in the Strip Old treatment on day 62 at 80 conidia/m³ air.

Environmental data were monitored in 2000 by a weather station present at the research station, which was located about 100 m from the southeast corner of the plots. Daily measurements for temperature range, precipitation, relative humidity range, and maximum wind speed were graphed over the course of the epidemic (Fig. 2.11). Average daily temperatures fluctuated within optimal disease progression ranges with no obvious extremes. Relative humidity reached values near 100% on nearly every day of the epidemic. Minimum relative humidity was quite variable, but reached values near 80% at two times during the epidemic. There were two notable rain events. On day 28, 0.99 cm accumulated rapidly on a single day. The second rain event occurred between 30 and 43 days after inoculation with maximum daily accumulation reaching 0.31 cm. Daily maximum wind speed was quite variable over the course of the epidemic.

**DISCUSSION**

Strip intercropping inhibited temporal progression of ELS epidemics during both years, and temporal progression of LLS during 2001. During both years of this study, Strip (New and Old) intercrop AUDPCs (ELS) were significantly lower than those of the Monocrop (p<0.05). This indicates a relative stability in single focus ELS epidemic reduction despite any site-to-site variability that may have been present. Similar stability was observed when beans were present with maize in comparable row ratios of 2:2 and 1:2. Halo blight, anthracnose, common blight, scab, *Phoma*, and mildew were all
significantly reduced over several years and sites when natural inoculum was present (Van Rheenen et al. 1981). Effects of intercropping on LLS were inconsistent between the two years, possibly due to the large differences in total amount of the disease. Inconsistent results were also reported for bean rust incidence when beans and maize were grown in ratios of 2:2 and 1:2 over several years and sites when natural inoculum was present (Van Rheenen et al. 1981). The Strip intercrops in the present study were in corn-peanut row ratios of 4:4 and relevant literature to fungal disease progress in comparable row ratios is lacking.

Intimate Intercropping did not significantly reduce temporal increase of ELS or LLS during 2000, but AUDPCs (ELS and LLS) were significantly lower (P<0.05) than the Monocrop in the 2001 epidemic. This suggests that disease suppression in the Intimate cropping system depended on environment, inoculum availability, or combinations of these and other factors. Site-to-site variability has been reported for intercrop pathosystems of similar ratios (Van Rheenen et al. 1981, Boudreau 1993). Boudreau (1993) specifically examined bean-maize intercrops in 2:1 row ratios. He found reductions in angular leaf spot of bean in two site-season combinations, but not in a third. Site-to-site environmental variability may be particularly important for the performance of the Intimate planting pattern in the present study, as the field plots in 2000 were irrigated, and those in the 2001 season were not. Corn in intercrops has been associated with increases in humidity (Van Rheenen et al. 1981), which could enhance development of both leaf spots. Corn was more evenly dispersed throughout the plots in Intimate Intercrops than in Strip intercrops, where rows were clumped. This coupled with irrigation might have raised relative humidity such that any other mechanisms of control
could not compensate during the 2000 season. Van Rheenen et al. (1981) have suggested that additional mechanisms for disease changes in corn intercrops might additionally include a decrease in temperature, light interception, raindrop attenuation, and physical windbreak. Boudreau and Mundt (1992) also examined bean rust in a 2:1 bean-maize intercrop, and found stable reductions in incidence that were attributed to intercropping mechanisms related to competition and spore escape, and not a reduction in bean density, interference from the non-host crop, or microclimate changes brought about by the non-host crop.

During both years of our study, there was no significant difference between the yields of Monocrop Sprayed and Monocrop treatments, despite the fact that ELS and LLS AUDPCs differed significantly for these two treatments. Therefore, neither early nor late leaf spot had an effect on yield based on disease incidence. Natural ELS epidemics tend to arise from several foci within a peanut population (Shew, personal communication). However, in our study, in order to observe disease progression in time and space, a single focus was used to initiate the epidemics. Dispersal gradients (See Chapter 3) from the central inoculum source were steep, and high levels of disease did not develop in distal portions of the plots until near harvest. This likely accounts for the lack of leaf spot effects on yield. Thus, ELS suppression by fungicide did not affect yield.

Yield in the intercropped treatments were not consistent over the course of the study. In 2000, no significant difference was observed between the monocrop treatments and the intercropped treatments, but a non-significant yield depression was seen in the intercropped plots. This may have been a reflection of competition from corn that was
not compensated for by ELS control, or the significant increase in defoliation in intercropped peanuts compared to monocropped peanuts.

In the 2001 epidemic, the yield of the Intimate Intercrop treatment was significantly lower than all treatments (P<0.05), whereas yield in Strip Old was significantly lower than in the Monocrop Sprayed treatment. It is unlikely that yield suppression occurred as a response to differences in disease incidence. Strip Old and Intimate Intercrop treatments had significantly lower ELS and LLS AUDPCs (P<0.05) than the Monocrop, but yield differences were not noted between sprayed Monocrop and Monocrop treatments. It is likely that yield reductions were attributed to field and climatic conditions, or competition with corn. In addition, the corn cultivar was a tropical hybrid used for biomass production for silage and may have given a competitive advantage compared to the corn cultivars grown during the previous year. Thus, ELS suppression as a result of Strip intercropping did not affect peanut yield. However, it is likely that competition was a contributing factor to yield reductions in intercrops during both years.

Stability of intercrop yields is important, especially to small-scale farmers. Tonye and Titi-Newl (1995) reported that peanut was unstable in peanut-maize intercrops (3:1 row ratio) when no fertilizer was added, and yields steadily decreased over a 3-year period in the same field. This should be taken into account when using intercropping as a management strategy in peanut cultivation.

Intercropping did not significantly affect corn yield during the 2001 epidemic, but yield in the Intimate Intercrop was notably higher, compared to the other treatments. Corn rows were probably exposed to less shading in intercropped rows than in corn
monocrops, because they were adjacent to ground-level plants, instead of plants of similar height. Lesoing and Francis (1999) found that in soybean-corn intercrops (8:8 row ratio), rows of corn bordering soybeans yielded higher than rows of corn bordering other rows of corn and attributed this to increased light interception. In addition, non-leguminous crops such as corn may benefit from planting associations with leguminous, nitrogen fixing crops such as peanuts. Rao and Mathuva (2000) reported that a pigeonpea-maize intercrop (1:2 row ratio) resulted in 24% higher maize yields than maize grown alone. However, they warn that intercropping with the perennial legume gliricidia resulted in competition for water, and reduced maize yields. Increased light interception and benefits of nitrogen may have compensated for competition that resulted from intercropping with peanuts. Furthermore, Tonye and Titi-Newl (1995) reported that maize was unstable in peanut-maize intercrops (3:1 row ratio) when no fertilizer was added, and yields steadily decreased over a 3 year period in the same field. It seems that fertilizer applications will be a fundamental component of corn cultivation in peanut intercrops over time.

Conidial concentrations during the 2000 epidemic are not representative of inoculum levels found in peanut production areas, where concentrations commonly reach 300 conidia/m³ air (Smith and Crosby 1973). This is likely due to the single focus initiation of the epidemic. Aerial conidia concentrations are likely to be higher in areas where *C. arachidicola* occurs naturally, simply because more disease is present. The first real peak in conidia concentration for all treatments occurred between days 15 and 18 days after inoculation. Since the latent period for *C. arachidicola* can reach 10 to 14 days, this indicates that the epidemics were most likely initiated by our inoculation
techniques rather than by natural inoculum. Conidial concentrations were variable throughout the season, but the Monocrop appeared to have the highest concentrations in at least nine of the smaller peaks. The most notable peak occurred for all treatments around day 63. Because concentrations rose in all treatments at the same time, environmental effects could have been important; however weather data prior to the peak provide no clear explanation for this rise. There were no major rain events in the days prior to the peak conidial concentrations. Temperature increased to optimal values after a cool period, and the range of relative humidity decreased with minimum values exceeding 75%. It is possible that the extreme increase in conidial concentration is due to the nature of multiple disease cycles. Perhaps there were simply more sporulating lesions resulting from secondary and tertiary inoculum by this time of the season.

Based on overall disease levels observed in this study, we conclude that strip intercropping has potential as a viable approach to peanut leaf spot reduction in mechanized farming systems. More research must be conducted to determine the reaction of disease progress in peanut-corn strip intercrops when abundant natural inoculum is present. Furthermore, yield depression should be addressed in future research.
Figure 2.1: Planting and sampling patterns for A) Intimate Intercrop of corn and peanut, B) peanut Monocrop and Monocrop Sprayed, C) Strip Old intercrop, and D) Strip New intercrop. Corn rows are represented by [Corn symbol] and peanut rows are represented by [Peanut symbol]. The star represents the location of inoculum source for each planting treatment. Sampling areas are represented by the gray boxes.
Figure 2.2 Progress of early leaf spot incidence on peanut over time in days after inoculation. Treatments were alternating rows of corn and peanut (Intimate), unsprayed peanut monocrop (Monocrop), four-row strips of corn and peanut (Strip Old and Strip New) and sprayed peanut monocrop (Monocrop Spray).
Figure 2.3 Area under the early leaf spot disease progress curves. Treatments were alternating rows of corn and peanut (Intimate), unsprayed peanut monocrop (Monocrop), four-row strips of corn and peanut (Strip Old and Strip New) and sprayed peanut monocrop (Monocrop Sprayed). Bars with the same letter are not significantly different based on ANOVA.
Figure 2.4 Progress of late leaf spot incidence on peanut over time in days after inoculation. Treatments were alternating rows of corn and peanut (Intimate), unsprayed peanut monocrop (Monocrop), four-row strips of corn and peanut (Strip Old and Strip New) and sprayed peanut monocrop (Monocrop Spray).
Figure 2.5 Area under the late leaf spot disease progress curves. Treatments were alternating rows of corn and peanut (Intimate), unsprayed peanut monocrop (Monocrop), four-row strips of corn and peanut (Strip Old and Strip New) and sprayed peanut monocrop (Monocrop Sprayed). Bars with the same letter are not significantly different based on ANOVA.
Figure 2.6 Progress of peanut leaflet defoliation over time in days after inoculation. Treatments were alternating rows of corn and peanut (Intimate), unsprayed peanut monocrop (Monocrop), four-row strips of corn and peanut (Strip Old and Strip New) and sprayed peanut monocrop (Monocrop Sprayed).
Figure 2.7 Area under the defoliation progress curve for all treatments. Treatments were alternating rows of corn and peanut (Intimate), unsprayed peanut monocrop (Monocrop), four-row strips of corn and peanut (Strip Old and Strip New) and sprayed peanut monocrop (Monocrop Sprayed). Bars with the same letter are not significantly different based on ANOVA.
Figure 2.8 Peanut yield as pounds per acre □ and kilograms per hectare ◆. Treatments were alternating rows of corn and peanut (Intimate), unsprayed peanut monocrop (Monocrop), four-row strips of corn and peanut (Strip Old and Strip New) and sprayed peanut monocrop (Monocrop Sprayed). Bars with the same letter are not significantly different based on ANOVA.
Figure 2.9 Corn yield as bushels per acre † and kilograms per hectare ‡ for all treatments during the 2001 epidemic. Treatments were alternating rows of corn and peanut (Intimate), four-row strips of corn and peanut (Strip Old and Strip New) and estimated corn monocrop (Monocrop Sprayed). Bars with the same letter are not significantly different based on ANOVA.
Figure 10 Average daily aerial conidial concentrations in 2000 obtained using Rotorod spore samplers. Treatments were alternating rows of corn and peanut (Intimate), four-row strips of corn and peanut (Strip Old) and uniform peanut (Monocrop).
Figure 2.11 Environmental parameters over the course of the epidemic in Castle Hayne, NC in 2000, including A) Maximum, minimum, and average daily temperatures, B) Total Daily precipitation, C) Maximum and minimum daily relative humidity, and D) Maximum daily wind speed.
LITERATURE CITED


The effects of intercropping corn and peanut on the temporal and spatial dynamics of early leaf spot (ELS) of peanut were examined. In 2000, the experiment consisted of five replicate blocks of square plots 16 rows wide and 14.6 m long. Treatments included unsprayed peanut (p) monoculture (Monocrop), sprayed peanut monoculture (Monocrop Sprayed), alternating rows of peanut and corn (c) (Intimate Intercrop), and four-row strip intercrops (2c, 4p, 4c, 4p, 2c) (Strip Old). In 2001, a second strip intercrop treatment was added (4c, 4p, 4c, 4p) (Strip New) and plots were 15.4 m long. Corn and peanut (VA 98R) were planted on 9 May 2000 at the Horticultural Crops Research Station near Castle Hayne, NC and 10 May 2001 at the Umstead Farm Unit near Butner, NC. Both locations are outside of normal peanut production areas, but are suitable for peanut culture. Focal epidemics were initiated by placing infected peanut stems centrally in each plot. ELS incidence was determined weekly in a stratified sampling routine that allowed estimation of disease gradients in four directions. Apparent infection rates ($r$) were calculated as the slopes of the linearized logistic model applied to the disease progress curves. A repeated measures analysis revealed a treatment x distance interaction (p<0.05) during the last four sampling days of each ELS epidemic. This likely is due to the Monocrop Sprayed treatment, in which low disease levels were maintained all distances from the inoculum. Disease gradients were plotted for each treatment/day combination, and log-linear, log-log, logit-linear, and logit-log models were applied to gradients. In both years, best fits were obtained when log distance was used. The logit-log model was selected in 2001 due to higher disease levels and in 2001, the log-log model was used because levels of disease
were lower. Linearized daily slopes within each treatment were analyzed with ANOVA and those that were significantly different from the majority of others in that treatment were discarded. Averaged individual plot slopes from the remaining days were used to estimate gradient parameters ($b$) for that treatment. Velocity ($V$) then was calculated according to the formula $V=rs/b$ using the averaged values of $b$ obtained previously and arbitrarily chosen distances ($s$). Velocity increased with increasing distance from the inoculum source. At $s=1$, velocities during both years ranged from 0.40 to 0.96 over all treatments with the exception of Strip New, where $V=0.222$. In 2000, Strip Old had the highest velocity at all distances, followed by Monocrop. Intimate Intercrop had the lowest velocity at all distances. In 2001, Strip New had the highest velocity at all distances from the source of inoculum. Velocities were comparable to those previously reported for LLS of peanut.

The peanut diseases early leaf spot (ELS) and late leaf spot (LLS) are caused by the globally distributed pathogens, *Cercospora arachidicola* S. Hori and *Cercosporidium personatum* (Berk. and M.A. Curtis) Deighton [syn. *Phaeosariopsis personata* (Berk. & Curt.) V. Arx.], respectively. Yield losses have been reported to exceed 50% on unsprayed fields (Melouk and Shokes 1995), and can exceed 10% on sprayed fields in the United States. Combinations of several control strategies are recommended to manage the diseases. Reduction of initial inoculum is achieved through cultural measures such as crop rotation, volunteer peanut removal, and burial of peanut residue (Shokes and Culbreath 1997). Reduction in temporal exposure may be achieved by early digging or planting dates (Knauf et al. 1986, Shokes et al. 1982) but applications of fungicides such
as chlorothalonil or tebuconazole (Melouk and Shokes 1995, Bailey 2002) are required to achieve optimal yields during most years (Bailey et al. 1994). Several forecasting systems have been developed (Jensen and Boyle 1966, Parvin et al. 1974, Cu and Phipps 1993, Bailey et al. 1994, Jacobi et al. 1995) and are widely used in North Carolina and Virginia (Bailey et al. 1994). Use of a forecast will generally save from 1 to 3 sprays in the Virginia-Carolina area compared to a calendar spray program (Hagan et al. 2000). In addition, low to moderate resistance is present in some released cultivars and much effort has been directed at developing cultivars with high levels of leaf spot resistance.

Intercropping is defined as the simultaneous cultivation of two or more species that are grown in close association (Andrews and Kassam 1976). Both species may be economic (e.g. maize and beans, rubber and pineapple) or a production crop may be grown with a “living mulch” primarily for weed and erosion control (e.g. cabbage with white clover). Intercropping has been studied largely from an agronomic and yield-based perspective, and particularly with reference to small-scale farming in less-developed countries, where it is practiced extensively (Papendick et al. 1976, Francis 1985, Vandermeer 1989, Thurston 1992). Although the motivation for intercropping has been connected with and advocated for disease control (Browning 1974, Burdon 1978, Altieri and Liebman 1986), few manipulative studies have been designed explicitly to evaluate its efficacy for this purpose. A review of studies on intercropping and fungal disease indicates that the general trend is toward less disease in intercrops compared to monocrops (Burdon and Chilvers 1977, Moreno and Mora 1984, Boudreau and Mundt 1992, Emebiri and Obiefuna 1992, Vilich-Meller 1992). However, some fungal diseases are favored by intercropping (Van Rheenen et al. 1981), and others are inconsistently
affected, due to site-to-site and season-to-season variability (Sengooba 1990, Boudreau 1993).

Boudreau and Mundt (1992, 1997) emphasized the need to understand mechanisms of disease-intercrop interaction in order to predict how mixtures perform in specific settings. Potential mechanisms may be divided into those that affect dispersal of the pathogen and those that affect non-dispersal aspects, such as germination, colonization, and reproduction. Examples of the former might include spore interception by the non-host (trapping) (Burdon and Chilvers 1977), alteration in the velocity and quality of the wind, raindrop attenuation, and competition effects on the host which in turn influence dispersal. The latter include microclimate alterations (changes in temperature and humidity), competitive effects that alter host susceptibility, and resistance induced by products or pathogens associated with the non-host.

Epidemiological models are commonly used to evaluate both temporal and spatial progression of an epidemic in various cropping systems. Vanderplank (1963) recommends use of logistic (logit-linear) models to describe the temporal progress of polycyclic diseases. The logistic model is expressed as \( \ln[y/(1-y)] = a - rt \) where \( y \) = disease incidence, \( a \) = y-intercept, \( r \) = slope, and \( t \) = days after inoculation. Logistic models are often applied to disease progress curves and have been used to determine apparent infection rates (r) for Septoria leaf spot of tomato (Parker et al. 1997), late leaf spot of peanut (Alderman et al. 1989), stem canker of soybean (Subbaro et al. 1992), and late blight of potato (Minogue and Fry 1983b) and many others (Campbell and Madden 1990).

Many models have been proposed to describe the relationship between disease intensity and distance from a focus of disease or inoculum. The most common models
use the slope of the linearized disease gradient \((b)\) to describe rate of change in disease with distance. These models include Kiyosawa and Shiyomi’s log-linear model (exponential law) expressed as \(\ln(y)=a-bs\) and Gregory’s log-log model (power law) expressed as \(\ln(y)=\ln(a)-b\ln(s)\) where \(y=\text{disease incidence}, a=\text{y-intercept}, b=\text{slope},\) and \(s=\text{distance from inoculum}.\) In cases where multiple infection is limiting the spread of disease, logit transformations are recommended for disease incidence (Berger and Luke 1979). These models include Minogue and Fry’s logit-linear model (logistic) expressed as \(\ln[y/(1-y)]=a-bs\) and Jeger’s logit-log model expressed as \(\ln[y/(1-y)]=\ln a-b\ln s\) where the variables are the same as those aforementioned. In models where disease is related to the log of distance, the gradient parameter \((b)\) is dimensionless (Jeger 1983). The logistic-linear model best described disease gradients for Septoria leaf spot (Parker et al. 1997), late leaf spot of peanut (Alderman et al. 1989), gray leaf spot of corn (Nutter et al 1992), stem canker of soybean (Subbaro et al. 1992), and late blight of potato (Minogue and Fry 1983b). The exponential model best fit disease gradients for Fusarium head blight of wheat (Fernando et al. 1997). However, in reviewing the literature, Ferrandino (1996) found that 80\% of the disease or dispersal gradients were fit equally well by the power law as the exponential law.

Velocity can be used to relate disease incidence at a given time to disease incidence at a given distance from the inoculum focus. The calculation of velocity requires the aforementioned parameters apparent infection rate \((r)\) and gradient parameter \((b)\). Distance from the source of inoculum may also be a required parameter, depending on the model used to obtain the gradient parameter. The equation \(V=r/b\), where \(V=\text{velocity}, r=\text{apparent infection rate},\) and \(b=\text{gradient parameter},\) is used when gradient
parameter models do not relate disease to the log of distance. The equation \( V = \frac{rs}{b} \), where \( s \) = distance from the source of inoculum and other variables are the same as those aforementioned, is used when gradient parameter models do relate disease to the log of distance (Jeger 1983).

The specific objective of this study was to indirectly evaluate the role of dispersal mechanisms in the leaf spot intercropping interaction by examining the spatial and temporal progress of ELS in a corn-peanut intercrop. We accomplished these goals by developing disease progress curves, gradient models, and estimating velocity for the intercropping treatments.

**MATERIALS AND METHODS**

Peanut and corn were grown in the summer of 2000 at the Horticultural Crops Research Station in Castle Hayne, NC and in 2001 at the Umstead Farm Unit in Butner, NC. Both locations were suitable for peanut production, but were outside the normal commercial production region in order to eliminate background inoculum. Treatments in 2000 consisted of peanut Monocrop, peanut Monocrop Sprayed with chlorothalonil applications at 2-week intervals for leaf spot control (standard production practice), alternating rows of peanut and corn (Intimate Intercrop) and peanut and corn grown in four-row strips (Strip Old). The strips of corn (c) and peanut (p) were planted as follows: c-c-p-p-p-p-c-c-c-c-p-p-p-p-c-c. For 2001, a new arrangement of corn and peanut (Strip New) was added to the experiment. In this treatment, rows of peanut and corn were arranged as follows: c-c-c-c-p-p-p-p-c-c-c-c-p-p-p-p (Fig. 3.1). The square plots were 16 rows wide and approx 15m in 2000 and 15.5m long in 2001. Planting details, cultivars, cultural practices, and harvesting procedures were as previously described (Chapter 1).
The experimental design in both years was a randomized complete block with five replications.

Focal epidemics of ELS were established by placing an inoculum source in the center of each plot on 28 July 2000 and on 10 August 2001. Inoculum sources were prepared by placing infected peanut cuttings, collected from unsprayed plots grown in Lewiston, NC, in moist sand in 0.6 L-capacity plastic cups. At the time of collection, only ELS was observed in these plots and no other disease was ever observed on the cuttings during the course of the experiment. The cuttings were placed in a moist chamber at 100% RH for 48 hours to induce sporulation. The cups with inoculum were placed in the centers of all plots. Due to the planting pattern, the inoculum was placed between two non-host corn rows in the center of the Strip Old treatment. In the Strip New treatment, the center of the plot was between a row of corn and a row of peanut; thus the inoculum source was placed in proximity to a peanut row (Fig. 3.1). Fresh cuttings were used to replace the initial cuttings approximately 7 days after introduction into the plots. All sources of inoculum, during both years, were heavily watered with a watering can at the time of placement in the field, to ensure a moist and humid environment for spore development and dispersal.

The Monocrop Sprayed treatment was sprayed with chlorothalonil, using a carbon dioxide backpack sprayer. The fungicide was applied at 1.26 kg a.i./ha and was delivered in 190 L water/ha. In 2000, sprays occurred on 3 August and 29 August. In 2001, sprays occurred on August 14 and 29, and 20 September. These sprays adequately controlled ELS and LLS in each year (Fig. 3.2).
Disease progress data were taken approximately once a week after inoculation until harvest. To characterize disease gradients, ELS disease incidence and percentage defoliation data were taken at the inoculum source and in four perpendicular arms. Each arm was divided into four sections, except the vertical arms in the Strip treatments, which were divided into 2 sections. In the treatments Monocrop, Monocrop Sprayed, Strip Old, and Strip New, each of the sections was 91 cm x 91 cm and encompassed two rows. In the Intimate treatment, each of the sections was 91 cm x 46 cm, and encompassed one row. In the Intimate and both Monocrop treatments, the center-most of the four sections of each arm overlapped at the point of inoculum. In the Strip Old treatment, the center-most section of the horizontal arms, and the center-most section of one of the vertical arms overlapped two rows upwind of the inoculum, which was between two non-host corn rows. The Strip New treatment had the same pattern of overlap, but the point of origin occurred at the inoculum (Fig. 3.1).

Eight subsamples were taken from each arm, which ensured the same level of precision in each plot (Boudreau and Mundt 1992). Sections within the four arms of the Intimate treatment were divided into two equal parts, and one subsample was taken from each half. Sections within the four arms in the Monocrop and Monocrop Sprayed treatments were divided into four equal quadrants. Two of the four quadrants were randomly subsampled within each section. The four sections of the two horizontal arms within the Strip Old and Strip New treatments were divided and subsampled according to the same methodology of those in both Monocrop treatments. The two sections within the two vertical arms of the Strip treatments were divided into four equal quadrants, and
one subsample was taken from each quadrant. In all treatments, a total of 32 samples were taken from each plot.

During both years, some individual or contiguous peanut plants within plots did not survive until maturity. In cases where this occurred on a quadrant level, an adjacent quadrant was substituted. In cases where this occurred across sections or arms, missing data were noted. In 2001, due to a herbicide application error, all plants in an Intimate Intercrop plot died, and missing data points were noted in the data set. Minimal corn mortality was observed during both seasons.

Disease incidence data were estimated as the percentage of leaflets with symptomatic lesions within the sampling area according to Johnson et al. (1986). Because a natural epidemic of LLS occurred in the field plots during both years of the study, ELS and LLS were estimated separately.

Disease data were averaged from all sample locations in each plot to construct disease progress curves. Linearized logistic models were expressed as \( \ln(y / 1-y) = a - rt \) where \( y \)= disease incidence, \( a \)= y-intercept, \( r \)=slope, and \( t \)=days after inoculation. Logistic models were applied to disease progress data on a whole plot basis for Intimate, Monocrop, and Strip Old and New treatments for both years. Values equal to zero were omitted when fitting the model. Apparent infection rates \( (r) \) for these treatments were calculated as the slopes of the linearized logistic models. Standard deviations, coefficients of determination, and y-intercepts also were calculated and residual plots were examined to assess model fit.

A visual representation of the spread of disease from the focus within each treatment over time was created for the 2000 and 2001 epidemics. Treatment averages for
each distance and direction combination on each day were expressed as a hue gradations imposed on a schematic of the actual sampling pattern. The effects of treatment, direction and distance were then analyzed on the last five sampling days of the ELS epidemic, and on the last three sampling days of the LLS epidemic using a repeated measures analysis of variance for each year. The analysis was performed using the PROC MIXED procedure available in SAS version 8 software for personal computers (SAS Institute, Inc., Cary North Carolina). Distance was the repeated measure and block was a random effect. The spatial covariance structure was specified as a power function of the grid coordinates of the centers of each of the sampling areas. The specific model was \[ Y_{ijkl} = \mu + B_i + T_j + D_k + M_l + (TD)_{jk} + (TM)_{jl} + (DM)_{kl} + (TDM)_{jkl} + \varepsilon_{ijkl}, \] where \((i,j)\) identifies the plot, \(i=1,\ldots,5\) blocks, \(j=1,\ldots,4\) treatments (2000) and 1,\ldots,5 treatments (2001), \(k=1,\ldots,4\) directions, and \(l=1,\ldots,4\) distances. The covariance structure within plots was expressed as \[ \text{cov}(\varepsilon_{ijkl}, \varepsilon_{ijkl'}) = \sigma^2 \rho^d \] where \(d=\text{distance between locations } (j,k) \text{ and } (j',k') \) within the same plot. The covariance structure between plots was expressed as \( \text{cov}(\varepsilon_{ijkl}, \varepsilon_{ijkl'}) = 0. \) Errors within the same plots were assumed to be correlated, whereas errors in different plots were not (Dr. Marcia Gumpertz, personal communication).

Disease gradients were examined by fitting disease versus distance data to standard models: a linearized log-linear (\(\ln(y) = a - bs\)), a log-log (\(\ln(y) = \ln a - b \ln s\)), a log-loglinear (\(\ln[y/(1-y)] = a - bs\)), and a logit-log (\(\ln[y/(1-y)] = \ln a - b \ln s\)) model where \(y=\text{disease incidence}, a=\text{Y-intercept}, b=\text{slope}, \) and \(s=\text{distance from the source of inoculum}\).

Goodness-of-fit was determined by examining the plots of the disease gradients, the coefficient of determination for regression, and plots of the standardized residuals for the linear regression. Linearized disease gradient slopes within each treatment and year were
subjected to ANOVA to compare days, and minimum significant differences (P<0.05) were calculated.

For each treatment, velocity was calculated to relate disease incidence at a given time to disease incidence at a given distance using the equation: $V = rs/b$, where $V=$velocity, $r=$ apparent infection rate, $s=$distance, and $b=$gradient parameter (Jeger 1983). For this calculation of velocity a constant value of $b$ is assumed (Jeger 1983). Therefore, when the values of the daily treatment slopes ($b$) of the gradient models were significantly different for days within a treatment (based on MSDs from the ANOVA above), they were omitted, and only statistically equal slopes were included in each calculation. These slopes then were averaged to form a gradient parameter ($b$) for each treatment (Parker et al. 1997). Because gradient parameters were derived from log-log and logit-log models, velocity was dependent on distance ($s$) (Minogue 1986.).

RESULTS

Disease progress curves and apparent infection rates: Conditions were highly conducive for ELS development in 2000. Averaged final ELS incidence in the Monocrop and Intimate Intercrop was approximately 40% (Fig. 3.2a). Disease incidence was almost 100% near the inoculation site in many plots. ELS disease progress curves showed clear differentiation among treatments. Disease in Monocrop and Intimate Intercrop followed similar curves and displayed the highest levels of ELS. Disease progress in Monocrop Sprayed was low and flat, and displayed the lowest final levels of ELS. Strip Old intercrop had an intermediate disease progress curve with maximum average disease levels reaching 27%. The logistic model explained approximately 41% of the variation in disease progress in the Intimate Intercrop and Monocrop treatments (Table 3.1). The
logistic model did not fit the Strip Old disease progress curve as well, with an $R^2 = 0.235$. The apparent infection rates were similar for the Intimate Intercrop and Monocrop at 0.091 and 0.095, respectively. The apparent infection rate of the Strip Old treatment was lower at 0.066. Y-intercepts were similar across treatments.

In 2001, conditions were somewhat less favorable for leaf spot development in our plots due to a drier growing season. The resulting ELS epidemic was scaled considerably lower than the previous year and disease did not increase smoothly in 2001 (Fig. 3.2b). Averaged final ELS incidence in the Monocrop was approximately 15% (Fig. 3.2b). The Monocrop Sprayed treatment was similar to the previous year and the disease progress curve remained low and flat. All intercropping treatments, including Intimate Intercrop, appeared to have lower rates of disease progress than the Monocrop. Intimate Intercrop and Strip New treatments averaged approximately 10% final ELS incidence. Final average ELS disease incidence for Strip Old was around 5%. The coefficient of determination for the logistic model was highest (0.437) for the Strip Old treatments; coefficients of determination were considerably lower for all other treatments. The slopes derived from the linearized logistic model for all treatments reveal that apparent infection rates were lower overall than for the previous year. The Monocrop treatment had the highest apparent infection rate of 0.052. Intimate, Strip New, and Strip Old had lower rates of 0.045, 0.041, and 0.042 respectively. Y-intercepts were similar across treatments.

Symptoms of naturally occurring late leaf spot (LLS), caused by the pathogen *Cercosporidium personatum*, were observed 35 DAI (31 August) in 2000 and 42 days after inoculation (26 September) in 2001. Patterns in disease progress curves were
variable across years and are described in detail in Chapter 1. AUDPCs for treatments during the 2000 LLS epidemic showed that incidence in Monocrop was significantly lower than in Strip Old, significantly higher than in Monocrop Sprayed, and not different from that in Intimate Intercrop. The Intimate Intercrop had an AUDPC that did not significantly differ from that of Monocrop or Strip Old. Very little disease developed in the Monocrop Sprayed. AUDPCs in 2001 showed that LLS was lower in all intercrops than the Monocrop (P<0.05). The Intimate and Strip New treatments did not differ; the Strip New and Strip Old treatments did not differ from each other. The AUDPC for the Strip Old treatment was not different from that of the Monocrop Sprayed.

**Spatial epidemiology.** During the 2000 epidemic, there was clear disease development radiating out from the focus over time (Fig. 3.3). A pattern was observed at 43 DAI, 49 DAI, and 63 DAI in which the Intimate Intercrop and Monocrop had the highest overall levels of disease, Strip Old had intermediate levels of disease, and Monocrop Sprayed had low levels of disease. This pattern was consistent with those patterns observed in the ELS disease progress curves during 2000.

In 2001, the diminished scale of the ELS epidemic was apparent (Fig. 3.4). However, there was still a recognizable pattern of disease originating at, and spreading over time, from a focus. This was especially recognizable at 46 DAI, 55 DAI, and 62 DAI. In addition, the disease pattern mentioned above (Monocrop, Intimate > Strip > Monocrop Sprayed) was again evident. Again, the pattern was consistent with those observed in the ELS disease progress curves during 2001.

During 2000, LLS incidence increased in all treatments over time (Fig. 3.5). Unlike the case with ELS, no clear gradient was apparent in the pattern of LLS incidence
and there was no apparent relationship of LLS with the ELS foci. On the final sampling
day of the epidemic, LLS distribution appeared to be more or less uniform across
Monocrop, Intimate Intercrop, and Strip Old treatments. There was little disease
development in the Monocrop Sprayed treatment.

**Repeated measures analysis.** A repeated measures analysis of ELS data revealed an
effect of cropping pattern (P<0.05) and distance (P<0.05) beginning on day 29 in the
2000 epidemic and day 32 in the 2001 epidemic (Table 3.2). The main effect of direction
was not significant except on day 62 in 2001. As disease progressed, a treatment x
distance interaction (P<0.05) was observed on days 35, 43, 49, and 63 of the 2000
epidemic and days 41, 46, 55, and 62 of the 2001 epidemic. In addition, there was a
distance x direction interaction (P<0.05) on day 49 in 2000 and day 46 in 2001.

For late leaf spot, no effects were observed on days 43 and 49 in 2000 or days 46 and 55 in 2001 (P>0.05). In 2000, a distance x direction (P<0.05) interaction occurred on
day 63. In 2001, treatment and distance affected disease (P<0.05) on day 62.

**Disease gradients and gradient parameters:** Dispersal models were fit to disease
gradients that were determined for each day x treatment combination. Goodness of fit
criteria determined that log-log and logit-log models fit well during both years. The
logit-log model was chosen for the 2000 gradients because disease levels approached the
asymptotic limit of 100% in some plots. The log-log model was chosen for the 2001
gradients because the spread of disease probably was not limited by an upper bound in
these plots (Berger and Luke 1979).

Strip intercrop treatments generally exhibited little to no relationship between
disease progress and distance from the source of inoculum, as expressed by low
coefficients of determination during both years (Table 3.3). As a result, linearized
disease gradients are shown only on days, within the Strip intercrops, where the slope
was significantly different from zero (P<0.05) (Fig. 3.6; Fig. 3.7). In 2001, no disease
gradients within the Strip Old treatment had a slope that was different from zero
(P<0.05).

Linearized logit-log models for disease gradients during 2000 showed a general
trend in which slopes remained constant over time and within treatments (Fig. 3.6; Table
3.3). Slopes that obviously deviated from the others include day 49 within the Monocrop
and day 63 within the Strip Old intercrop. Linearized log-log models for disease gradients
during 2001 also showed a general trend for slopes to remain constant over time and
within treatments (Fig. 3.7; Table 3.3). However, the slopes appeared to become steeper
in the monocrop treatment over time.

Further analysis of daily slopes within treatments by ANOVA revealed
differences (P<0.05) during both years (Table 3.3). During 2000, slopes from days 43
and 49 were steeper than slopes from other days in the Monocrop treatment, and the slope
from day 63 was steeper than remaining slopes within the Strip Old treatment. During
2001, the slope from day 46 within the Intimate Intercrop was steeper from remaining
slopes, and the slope from day 62 within the Strip Old treatment was steeper than
remaining slopes. Within the Monocrop, gradients on days 25 through 46 were shallower
than on days 55 and 62. Furthermore, slopes from days 41 through 62 were steeper than
slopes from days 25 and 32.

Mean gradient parameters (b) were calculated as the average of the daily slopes
within a treatment in a given year. If a daily slope was significantly different from the
others, it was not included in the calculation of the mean. Two gradient parameters were calculated for the Monocrop during 2001, because significance tests divided slopes for the days into two groups. In 2000, the Strip Old treatment was omitted from analysis because no daily slope was significantly different from zero (P<0.05). Intimate Intercrop had the highest mean value of \( b \) (1.78) followed by Monocrop (1.42). Strip Old had the lowest mean value of the gradient (\( b \)) parameter at 0.86. In 2001, Intimate Intercrop had a mean gradient parameter of 0.54. Monocrop had a comparable gradient parameter during the early stages of the epidemic (0.43), but a notably higher parameter during the late stages of the epidemic (1.01). Strip New had the lowest gradient parameter at 0.19.

**Velocity:** Velocity was calculated at three arbitrarily chosen distances from the source of inoculum during both years. In all treatments that were calculated, velocity increased with increasing distance from the inoculum source because of our choice of the velocity model \( V=rs/b \). At \( s=1 \), velocities during both years ranged from 0.40 to 0.96 over all treatments with the exception of Strip New, where \( V=0.222 \). In 2000, Strip Old had the highest velocity at all distances, followed by Monocrop. Intimate Intercrop had the lowest velocity at all distances. In 2001, Strip New had the highest velocity at all distances from the source of inoculum. This calculated velocity was greater, in all instances, by an order of magnitude. Intimate Intercrop and early season Monocrop had intermediate velocities at all distances, and late season Monocrop had the lowest velocity at all distances.

**DISCUSSION.**

We have found multiple lines of evidence that indicate that disease levels are reduced in peanut corn Strip intercrops, and are likely due to factors affecting dispersal
processes of *C. arachidicola* within the strip intercrops. Year-to-year observations reveal that apparent infection rates for all treatments in 2001 were generally lower than treatments in 2000. An unfavorable environment in 2001 slowed disease progress. In 2000, apparent infection rates (*r*) were highest in Monocrop and Intimate Intercrop treatments, and the Strip Old intercrop had an (*r*) value that was diminished by nearly 1/3 of the aforementioned treatments. This observation follows trends observed in disease progress curves and AUDPCs for ELS in 2000 (See Chapter 1). Furthermore, in 2001, the Monocrop had the highest apparent infection rate, with Intimate, Strip New, and Strip Old having lower rates that were similar in value. This observation also reinforces trends observed in disease progress curves and AUDPCs for ELS in 2001 (See Chapter 1).

The variability accounted for by the logistic model was less than 41%. The relatively low coefficients of determination generated by the logistic model in this study are likely due to the fact that the model does not take into account variability due to spatial gradients. In 2001, poor model fit may also be associated with the prominent concave bend in the Monocrop, Intimate, and Strip Old treatments that occurred 55 DAI. Coefficients of determination were lower in these treatments.

Year to year variability in Y-intercepts was expected, since location and environmental effects, independent of treatments, may have affected the number and viability of infective propagules at the focus. Furthermore, Y-intercepts were relatively similar across treatments within each year, suggesting that the inoculum source strength was uniform across treatments.

Visual analysis of mean ELS disease incidence at distance and direction locations illustrated the spread of disease from a focus over time during both years. This
confirmed that the inoculum of *Cercospora arachidicola* introduced at the center of the plots was responsible for the disease we observed throughout the epidemic. It also suggests that there was limited interplot interference, since high levels of disease were not detected at the perimeters of plots until the end of the growing season. Furthermore, it refutes the idea that disease spreads more rapidly along rows rather than across rows in intercrops. In 2000, disease patterns observed in the plot diagrams revealed trends that were congruous with ELS disease progress curves, AUDPCs, and apparent infection rates. Not only was there more disease in the Monocrop and Intimate Intercrop, but overall incidence also seemed to increase earlier in these treatments. In 2001, plot diagrams also agreed with previous data. The scale of the epidemic was notably diminished in all plots, and disease did not begin to intensify until the end of the season.

In 2000, foci of LLS tended to vary across treatments and days, and were not associated with ELS foci. In 2001, LLS incidence was very low with no apparent pattern. This suggests that the epidemic was not the result of latent infection contamination in the symptomatic ELS infected cuttings used in the inoculation process. Furthermore, it suggests that the LLS epidemic resulted from inoculum dispersed from production areas. It is possible that the darker, melanized spores of *C. personatum* were able to withstand environmental pressures, and disseminate to our plots from a peanut production area.

The ELS repeated measures analysis showed nearly identical trends during both years of the study. The significant distance x direction interaction noted on 49 DAI in 2000 and on 46 DAI in 2001 occurred at the temporal point where disease began to increase rapidly during both years. This would be expected near the middle of the growing season, because disease progress had expanded from the focus, but had not
reached the perimeters of the plots. Perhaps this effect would have been noted later in the epidemic if disease spread had not been limited by the size of the plots. The significant cropping pattern and distance effects noted early in the season, on 29 and 32 DAI in respective years, were probably due to the lack of disease progress (<5%) at those points in time. More disease was found near the focus at the start of an epidemic across all plots. Furthermore, production of secondary inoculum would have been severely inhibited in the Monocrop Sprayed plots as compared to the other treatments, resulting in lower disease incidence.

The cropping pattern x distance interaction noted on the last four sampling days of both years was expected, because disease spread from the focus was slower in the Monocrop Sprayed than in the other treatments. However, this interaction inspired further examination of disease gradients.

The LLS repeated measures analysis did not reflect trends that were similar across years of the study, or similar to those for ELS. In 2000, a distance x direction (P<0.05) interaction was observed 63 DAI. This interaction corresponded with high disease levels that appeared to extend well into the perimeters of the plots, and may have arisen because inoculum was not dispersed from uniform foci. In 2001, main effects of treatment (P<0.05) and distance (P<0.05) were observed 62 DAI. These effects corresponded to the temporal point that LLS disease incidence reached maximum levels within the plots during that year. Treatment effects were likely due to depressed levels of disease observed in the Monocrop Sprayed treatments. Distance effects may reflect isolated “hot spots” present in some treatments.
Plots of linearized disease gradients and resulting slopes \((b)\) reveal generally steeper slopes in 2000 than in 2001. Although differences in average treatment slopes within years were not analyzed for significance, patterns of treatment relationships were similar for both years; intimate and Monocrop had highest average slopes, whereas Strip intercrops had the lowest. These trends were consistent with trends observed in disease progress curves and suggest that higher overall disease levels may be correlated with steeper disease gradient slopes.

The validity of using a constant gradient parameter \((b)\) as a variable in determining velocity is questionable. The velocity equation assumes that gradient parameters remain constant over time (Campbell and Madden 1990). Although we observed that a majority of slopes were statistically equal within most treatments during both years, there did not seem to be a relationship between DAI and significant differences among slopes, either across treatments or across years. Furthermore, in the 2001 Monocrop treatment, slopes were divided into two significantly different groups. The resulting averaged parameters were quite variable between early and late season in this treatment.

The use of gradient parameters is also based on the assumption that there is a relationship between progression of disease and distance from the focus. Many factors could lead to a scenario in which the relationship is absent, including lack of disease, copious disease levels, and random events. Gradient parameters resulting from all Strip intercrop treatments were notably flat. They were also characterized by low coefficients of determination, and in all cases were calculated with slopes that were not significantly different from zero.
Treatment velocities calculated from apparent infection rates \((r)\) and gradient parameters \((b)\) increase with increasing distance from inoculum. This was due to the distance multiplier that was included in the velocity equation \(V=rs/b\). Velocity values obtained at \(s=1\) were comparative to constant velocities calculated using the logistic model for the gradient parameter. The usefulness of this equation was validated in cases where spatial and temporal increase of disease is close to the source of inoculum. Velocities calculated at \(s=1\) were similar in magnitude to those reported by Alderman et al. (1989) when using the logistic model for the gradient parameter \((b)\) for LLS of peanut. This pathogen has similar epidemiology to \(C.\ arachidicola\), and validates the use of this velocity equation for ELS at distances near the source of inoculum.

The idea that velocity should increase as distance from the source of inoculum approaches infinity may seem counterintuitive and is limited by constraints of host population dimensions and abundance or availability of inoculum. However, it is not unreasonable that velocity should increase with increasing distance as long as there are susceptible hosts, virulent pathogens, and a favorable environment. Furthermore, gradient models in this study that did not use log transformations for distance were far inferior to those that did. Therefore, an increase in velocity over distance may have occurred, and does not seem to be a mathematical artifact.

Significance tests could not be performed for treatment velocities within years, because values were based on means. However, at \(s=1\), patterns of treatment relationships were similar for both years; intimate and Monocrop had lowest velocities, whereas Strip intercrops had the highest velocities. The interpretation of velocity within the Strip intercrop treatments is questionable, since the relationship of disease across
distance is uncertain. Mathematics dictates that velocity will increase as the value of the gradient parameter approaches zero. The results of this study suggest that spatial and temporal increase in disease in Strip intercrops may not be accurately quantified by velocity.

If we do assume that Strip intercrop gradient parameters are an accurate expression of the relationship between disease and distance, then it may also be assumed that the spatial and temporal increase of disease was faster in the Strip intercrops. This combined with lower overall Strip intercrop disease levels, compared to the Monocrop, suggests that spore escape might be a mechanism affecting disease levels in Strip intercrops. Poor sporulation or inadequate spore removal from the lesions could also be an important factor. In these scenarios, propagules would likely move faster in Strip plots, with unique environmental parameters, such as wind speed, quality, and velocity affecting their end destination.

Reduction in disease levels in peanut corn Strip intercrops are likely due to factors affecting dispersal processes of *C. arachidicola*. Gradient calculations suggest that the pathogen moved faster over time within the Strip intercrops. Related research has been conducted at Warren Wilson College, in Asheville, North Carolina to evaluate non-dispersal effects of intercropping on ELS disease incidence (M.A. Boudreau, unpublished data). Further research must be conducted to determine the exact mechanism(s) responsible for these factors, and to evaluate the spatial and temporal spread of the disease in intercrops where natural ELS inoculum foci are present.
Table 3.1 Y-Intercepts, apparent infection rates (r), standard deviations, and coefficients of determination calculated from a logistic model of early leaf spot disease progress.

<table>
<thead>
<tr>
<th>Year</th>
<th>Treatment a</th>
<th>Y-Intercept</th>
<th>Apparent Infection Rate(days⁻¹)</th>
<th>R²</th>
</tr>
</thead>
<tbody>
<tr>
<td>2000</td>
<td>Intimate</td>
<td>-6.574 ± 0.223</td>
<td>0.091 ± 0.005</td>
<td>0.405</td>
</tr>
<tr>
<td></td>
<td>Monocrop</td>
<td>-6.878 ± 0.233</td>
<td>0.095 ± 0.005</td>
<td>0.412</td>
</tr>
<tr>
<td></td>
<td>Strip Old</td>
<td>-6.236 ± 0.300</td>
<td>0.066 ± 0.007</td>
<td>0.235</td>
</tr>
<tr>
<td></td>
<td>Strip New</td>
<td>-5.688 ± 0.133</td>
<td>0.042 ± 0.003</td>
<td>0.352</td>
</tr>
<tr>
<td>2001</td>
<td>Intimate</td>
<td>-5.765 ± 0.201</td>
<td>0.045 ± 0.004</td>
<td>0.242</td>
</tr>
<tr>
<td></td>
<td>Monocrop</td>
<td>-5.632 ± 0.132</td>
<td>0.052 ± 0.003</td>
<td>0.354</td>
</tr>
<tr>
<td></td>
<td>Strip Old</td>
<td>-6.035 ± 0.126</td>
<td>0.041 ± 0.003</td>
<td>0.437</td>
</tr>
<tr>
<td></td>
<td>Strip New</td>
<td>-5.688 ± 0.133</td>
<td>0.042 ± 0.003</td>
<td>0.352</td>
</tr>
</tbody>
</table>

a) Treatments were alternating rows of corn and peanut (Intimate), unsprayed peanut monocrop (Monocrop), four-row strips of corn and peanut (Strip Old and Strip New) and sprayed peanut monocrop (Monocrop Sprayed).

b) The logistic model was expressed as ln(y/1-y) = ln(a) - rt, where y=disease incidence, a=Y intercept, r=slope, and t=days after inoculation.
Table 3.2 P-values for treatment, distance, and direction effects on early leaf spot disease incidence repeated measures analysis of variance of data from intercropped treatments five sampling days during 2000 and 2001.

<table>
<thead>
<tr>
<th>Effect</th>
<th>2000 Days after inoculation</th>
<th>2001 Days after inoculation</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>29</td>
<td>35</td>
</tr>
<tr>
<td>Trt</td>
<td>0.0403</td>
<td>0.0944</td>
</tr>
<tr>
<td>Direction (Dir)</td>
<td>0.6846</td>
<td>0.2900</td>
</tr>
<tr>
<td>Distance (Dis)</td>
<td>0.0001</td>
<td>0.0001</td>
</tr>
<tr>
<td>Trt*Dir</td>
<td>0.3956</td>
<td>0.0453</td>
</tr>
<tr>
<td>Trt*Dis</td>
<td>0.2721</td>
<td>0.0001</td>
</tr>
<tr>
<td>Dis*Dir</td>
<td>0.4985</td>
<td>0.1655</td>
</tr>
<tr>
<td>Trt<em>Dis</em>Dir</td>
<td>0.8947</td>
<td>0.9756</td>
</tr>
</tbody>
</table>

a) Treatments were alternating rows of corn and peanut (Intimate), unsprayed peanut monocrop (Monocrop), four-row strips of corn and peanut (Strip Old and Strip New) and sprayed peanut monocrop (Monocrop Sprayed).

b) Distances from inoculum and Directions were expressed as grid coordinates of the centers of the sampling areas where the origin was the south west corner of the plot.

c) P-value is the probability of falsely rejecting \( H_0 \): no difference between treatment or interaction means.
Table 3.3: Daily slopes of linearized curves derived by fitting the logit-log model in 2000 and the log-log model in 2001 to early leaf spot dispersal gradient data in intercropped corn and peanut with corresponding coefficients of determination for each treatment x sampling day combination on six observation days.

<table>
<thead>
<tr>
<th></th>
<th>Intimatea</th>
<th>Monocropa</th>
<th>Strip Olda</th>
<th>Intimateb</th>
<th>Monocropb</th>
<th>Strip Newb</th>
</tr>
</thead>
<tbody>
<tr>
<td>Day</td>
<td>R²</td>
<td>Slopeb</td>
<td>R²</td>
<td>Slopeb</td>
<td>R²</td>
<td>Slopeb</td>
</tr>
<tr>
<td>22</td>
<td>0.445</td>
<td>-1.796 A</td>
<td>0.510</td>
<td>-1.487 AB</td>
<td>0.113</td>
<td>-1.596 AB</td>
</tr>
<tr>
<td>29</td>
<td>0.421</td>
<td>-1.576 A</td>
<td>0.371</td>
<td>-1.248 A</td>
<td>0.083</td>
<td>-1.516 AB</td>
</tr>
<tr>
<td>35</td>
<td>0.457</td>
<td>-1.801 A</td>
<td>0.469</td>
<td>-1.314 A</td>
<td>0.010</td>
<td>-0.641 A</td>
</tr>
<tr>
<td>43</td>
<td>0.300</td>
<td>-1.751 A</td>
<td>0.476</td>
<td>-1.844 B</td>
<td>0.000</td>
<td>-0.184 A</td>
</tr>
<tr>
<td>49</td>
<td>0.336</td>
<td>-2.429 A</td>
<td>0.471</td>
<td>-2.402 B</td>
<td>0.002</td>
<td>-0.358 A</td>
</tr>
<tr>
<td>63</td>
<td>0.240</td>
<td>-1.632 A</td>
<td>0.265</td>
<td>-1.623 AB</td>
<td>0.096</td>
<td>-2.782 B</td>
</tr>
<tr>
<td></td>
<td>MSD=0.789</td>
<td>MSD=0.468</td>
<td>MSD=1.788</td>
<td>MSD=0.538</td>
<td>MSD=0.318</td>
<td>MSD=0.402</td>
</tr>
</tbody>
</table>

a) Treatments were: Intimate, alternate rows of corn and peanut; Monocrop, peanut only; Strip New and Strip Old, 4 rows of peanut, 4 rows of corn.

b) Results of ANOVA comparing slopes (unitless) of linearized early leaf spot dispersal gradients within each treatment are indicated as letters within treatment columns. MSD is indicated for each ANOVA.

c) The logit-log model was expressed as ln(y/(1-y)) = ln(a) – b ln(s) and the log-log model was expressed as ln(y) = ln(a) – b ln(s) in 2001 where y=disease incidence, a=Y intercept, b=slope, and s=distance from inoculum.

d) MSD=minimum significant difference.
Table 3.4 Apparent infection rates, gradient parameters and velocities at three distances from the source of inoculum in peanut corn intercropping for each treatment and during 2000 and 2001.

<table>
<thead>
<tr>
<th>Year</th>
<th>Treatment</th>
<th>Apparent Infection Rate(^b) (r) (day(^{-1}))</th>
<th>Gradient Parameter(^c) (b)</th>
<th>Velocity(^d) (m*day(^{-1}))</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>(unitless)</td>
<td>s=1(m)</td>
<td>s=4(m)</td>
</tr>
<tr>
<td>2000</td>
<td>Intimate(^a)</td>
<td>0.091</td>
<td>1.78</td>
<td>0.052</td>
</tr>
<tr>
<td></td>
<td>Monocrop(^a)</td>
<td>0.095</td>
<td>1.42</td>
<td>0.066</td>
</tr>
<tr>
<td></td>
<td>Strip Old(^a)</td>
<td>0.066</td>
<td>0.86</td>
<td>0.076</td>
</tr>
<tr>
<td>2001</td>
<td>Intimate(^a)</td>
<td>0.045</td>
<td>0.54</td>
<td>0.084</td>
</tr>
<tr>
<td></td>
<td>Monocrop(^a)</td>
<td>0.041</td>
<td>0.43</td>
<td>0.096</td>
</tr>
<tr>
<td></td>
<td>Strip New(^a)</td>
<td>0.042</td>
<td>0.19</td>
<td>0.222</td>
</tr>
</tbody>
</table>

a) Treatments were: Intimate, alternate rows of corn and peanut; Monocrop, peanut only; Strip New and Strip Old, 4 rows of peanut, 4 rows of corn.

b) Apparent infection rates \((r)\) were calculated during both years by applying the logistic model expressed as \(\ln(y/(1-y)) = \ln(a) - rt\), where \(y\)=disease incidence, \(a\)=Y intercept, \(r\)=slope, and \(t\)=days after inoculation, to disease progress curves.

c) Gradient parameters were calculated by applying the logit-log model (2000) expressed as \(\ln(y/(1-y)) = \ln(a) - b \ln(s)\), and the log-log model (2001) expressed as \(\ln(y) = \ln(a) - b \ln(s)\), where \(y\)=disease incidence, \(a\)=Y intercept, \(b\)=slope, and \(s\)=distance from inoculum, to dispersal gradients for each treatment x sampling day combination and determining the slope of the linearized model curve.

d) Velocity was calculated using the equation \(V=rs/b\), where \(V\)=velocity, \(r\)=apparent infection rate, \(s\)=distance from inoculum, and \(b\)=gradient parameter.
Figure 3.1: Planting and sampling patterns for A) Intimate Intercrop of corn and peanut, B) peanut Monocrop and Monocrop Sprayed, C) Strip Old intercrop, and D) Strip New intercrop. Corn rows are represented by \[\text{\textbullet\textbullet\textbullet\textbullet} \] and peanut rows are represented by \[\text{LLLLLLLLLLLLLLLLLLLLLLL} \]. The star represents the location of inoculum source for each planting treatment. Sampling areas are represented by the gray boxes.
Figure 3.2 Progress of early leaf spot incidence on peanut over time in days after inoculation. Treatments were alternating rows of corn and peanut (Intimate), unsprayed peanut monocrop (Monocrop), four-row strips of corn and peanut (Strip Old and Strip New) and sprayed peanut monocrop (Monocrop Spray).
Figure 3.3 Mean levels of early leaf spot disease incidence during 2000 on peanut at four sampling distances and four directions from a central focus of inoculation. Disease incidence is indicated at approximate weekly intervals beginning two weeks after the focus was established. Treatments were: Intimate, alternate rows of corn and peanut; Monocrop, peanut only; Strip Old, 4 rows of peanut, 4 rows of corn; Monocrop Sprayed, peanut treated with fungicide for leaf spot control.
Figure 3.4 Mean levels of early leaf spot disease incidence during 2001 on peanut at four sampling distances and four directions from a central focus of inoculation. Disease incidence is indicated at approximate weekly intervals beginning two weeks after the focus was established. Treatments were: Intimate, alternate rows of corn and peanut; Monocrop, peanut only; Strip Old and Strip New, 4 rows of peanut, 4 rows of corn; Monocrop Sprayed, peanut treated with fungicide for leaf spot control.
Figure 3.5 Mean levels of naturally occurring late leaf spot disease incidence during 2000 on peanut at four sampling distances and four directions from a central focus of early leaf spot inoculation. Disease incidence is indicated at approximate weekly intervals beginning at the point that late leaf spot symptomatic leaves were first observed. Treatments were: Intimate, alternate rows of corn and peanut; Monocrop, peanut only; Strip Old, 4 rows of peanut, 4 rows of corn; Monocrop Sprayed, peanut treated with fungicide for leaf spot control.
Figure 3.6 Linearized curves derived by fitting the logit-log model expressed as \( \ln(y/(1-y)) = \ln(a) - b \ln(s) \) where \( y \) = disease incidence, \( a \) = Y intercept, \( b \) = slope, and \( s \) = distance from inoculum to early leaf spot dispersal gradient data. Linearized curves were derived for each treatment x sampling day combination during the 2000 epidemic where treatments were: Intimate, alternate rows of corn and peanut; Monocrop, peanut only; Strip Old, 4 rows of peanut, 4 rows of corn. Four sampling days were omitted from Strip Old intercrops because gradient slopes were not significantly different from zero (\( P<0.05 \)).
Figure 3.7 Linearized curves derived by fitting the log-log model expressed as $\ln(y) = \ln(a) - b \ln(s)$ where $y=$disease incidence, $a=$Y intercept, $b=$slope, and $s=$distance from inoculum to early leaf spot dispersal gradient data. Linearized curves were derived for each treatment x sampling day combination during the 2001 epidemic where treatments were: Intimate, alternate rows of corn and peanut; Monocrop, peanut only; Strip New, 4 rows of peanut, 4 rows of corn. Five sampling days were omitted from Strip New intercrops because gradient slopes were not significantly different from zero ($P<0.05$). All Strip Old slopes were omitted, because all were statistically equal to zero ($P<0.05$).
Literature Cited.


MULTIPLE CONTROL STRATEGIES FOR MANAGEMENT OF EARLY LEAF SPOT IN PEANUT

Taken together, the results of the previous experiments suggest that intercrops are effective at reducing ELS, and that this reduction is most likely due to dispersal processes. Strip intercropping shows real promise as an additional tool to reduce severity of ELS on peanuts. The current study aimed to assess the overall effect of intercropping in an otherwise typical production system on disease incidence and yield. This protocol also incorporated characterizations of some mechanisms by which the intercrop influences disease dynamics, thereby providing a theoretical foundation for future research and application to diverse large-scale mechanized agroecosystems.

The presence of TSWV is a growing cause of concern to North Carolina peanut farmers. The cultivar NC-V11 has field resistance to tomato spotted wilt, which appears to be mediated through effects on the thrips vector rather than the virus (Garcia et al. 2000). There is extensive evidence that tomato spotted wilt severity is closely related to planting pattern, with widely spaced plantings having highest rates of disease (Culbreath et al. 1998). Conversely, stubble or crop residues appear to confuse thrips and suppress disease (Monfort et al. 2001). Experience in Georgia suggests that intercropping may suppress tomato spotted wilt development in peanut (A.K. Culbreath, U. Georgia, personal communication).

The objectives of this study were first, to evaluate the efficacy of corn-peanut intercropping alone and with a reduced spray schedule as a disease management tool for leaf spot diseases and TSWV in commercial mechanized production systems; and second,
to collect yield data to determine the effects of intercropping and spray management practices.

**MATERIALS AND METHODS**

The experiment consisted of research plots at the Peanut Belt Research Station near Lewiston, NC during the 2002 season. This site is located in a peanut production area and has several projects involving peanut (p), and cotton (c) production research annually. Twenty-eight 15 x 15 m plots were established on 14 May. Each plot was an experimental unit and consisted of 16 rows. Borders equivalent to eight rows surrounded plots on all sides. The following treatments were replicated four times each in a split plot design: a) Unsprayed peanut monocrop; b) Reduced spray peanut monocrop; c) Sprayed peanut monocrop; d) Unsprayed strip intercrop representing alternating sets of four cotton and four peanut rows (C-C-C-P-P-P-C-C-C-P-P-P-P); e) Reduced spray strip intercrop; f) Sprayed strip intercrop; and g) Cotton monocrop. Sprayed treatments received full or half rate chlorothalonil applications at approximate 2 week intervals for leaf spot control (See chapter 1).

The Virginia-type peanut cultivar NC-V 11 was used in this experiment due to the presence of TSWV in Lewiston, NC. This cultivar accounted for almost 28 % of all seed produced for use in NC in 2000 (Jordan et al 2001). It has moderate field resistance to TSWV and is susceptible to leaf spots. The Sure Grow 105 field cotton was used. All crops were managed in accordance with recommended practices for North Carolina (http://ipmwww.ncsu.edu/Production_Guides/), but with certain choices made to account for a mixture of two crop species. Specifically, plots were established in an area
following a non-peanut crop. Cotton received a split application of nitrogen fertilizer as recommended by soil testing performed by NCDA&CS. Nutrients recommended by soil testing were applied. All crops were planted simultaneously at the standard peanut planting date to optimize labor input and herbicide application, and particularly to prevent excessive competition from cotton. There was ample time for maturity of all crops. Weed control was achieved with compatible herbicides.

In contrast to earlier experiments, these studies relied on the presence of natural inoculum in production areas. Assessment began in late July. Incidence of ELS and LLS was estimated in one randomly selected 91 cm sector in all eight peanut rows of each strip intercrop, and in eight randomly selected peanut rows in each monocrop, for a total of 8 samples per plot. The number of defoliated leaflets was also recorded from two arbitrarily selected stems in each sector (Johnson et al., 1986) on three of the sampling dates. Other peanut diseases, including Sclerotium rolfsii, TSWV, Sclerotinia minor, and web blotch were monitored for incidence on all of the evaluation dates. Sclerotium rolfsii and TSWV incidence were determined by counting the number of infected plants in a 1.0 m sampling area, and was not based on percent. Web blotch was estimated using the same methodology as ELS and LLS. Disease ratings were preformed on 97, 111, 118, and 125 days after planting (DAP). Plots were mechanically harvested and gross yields were determined for each crop.

Disease data were analyzed by taking the simple mean of all data from each plot. Disease progress curves and yield data was determined for each plot.

RESULTS AND DISCUSSION
ELS was first detected in field plots on 19 August 2002, 97 Days after planting (DAP), and reached levels near 50% by 125 DAP in Strip and Monocrop treatments that did not receive a spray application (Fig. 4.1). Monocrop and Unsprayed Strip intercrop had highest levels of disease across the epidemic. Disease progress curves were similar across cropping patterns in terms of full rate spray applications, half rate spray applications, and treatments that were not sprayed. But, Strip intercrop treatments were lower that their Monocrop counterparts under all spray application regimes.

Symptoms of stem rot and TSWV were first observed 97 DAP (Fig. 4.2; Fig. 4.3). Monocrop treatments had higher final levels of stem rot compared to Strip intercrop treatments. Stem rot disease progress curves for all treatments did not advance smoothly, and displayed concave humps at 118 DAI. Levels of TSWV were not clearly associated with either cropping pattern or fungicide regime. Disease incidence in the Strip Unspray treatment failed to increase over time, and the disease progress curve remained flat throughout the season.

Symptoms of LLS (Fig. 4.4) and web blotch (Fig. 4.5) were not observed until 125 DAP. Monocrop Unspray had highest levels of LLS and web blotch. All other treatments had LLS levels, 0.5% at 125 DAP. Web blotch disease incidence levels were variable, but treatments sprayed with the full rate of chlorothalonil application had lowest disease incidence levels.
Table 4.1  Mean cotton yields within individual plots for treatments during 2002.

<table>
<thead>
<tr>
<th>Block</th>
<th>Cropping Pattern</th>
<th>Spray Treatment</th>
<th>Yield (lbs/15 m row)</th>
</tr>
</thead>
<tbody>
<tr>
<td>2</td>
<td>Monocrop</td>
<td>NA</td>
<td>5.041666667</td>
</tr>
<tr>
<td>4</td>
<td>Monocrop</td>
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<td>5.5</td>
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<tr>
<td>2</td>
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<td>4.458333333</td>
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<td>3</td>
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<td>4.291666667</td>
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<td>4.28125</td>
</tr>
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</tr>
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<tr>
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<td>Strip</td>
<td>Unspray</td>
<td>4.6875</td>
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a) Treatments were: cotton monocrop (Monocrop), and four-row strips of cotton and peanut with full rates of spray application (Strip/Full), half rates of spray application (Strip/Reduced), and no spray applications (Strip/Unspray).
Table 4.2  Mean peanut yields for individual plots during the 2002 growing season.

<table>
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<tr>
<th>Block</th>
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<th>Moisture (%)</th>
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<td>12.25</td>
<td>8.75</td>
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<td>Full</td>
<td>10.40625</td>
<td>7.142857143</td>
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<td>Full</td>
<td>12.6875</td>
<td>9.459459459</td>
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<td>9.333333333</td>
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<td>7.042253521</td>
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<td>Reduced</td>
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</tr>
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<td>Unspray</td>
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</table>

a) Treatments were, peanut monocrops with full rates of spray application (Mono/Full), half rates of spray application (Mono/Reduced), and no spray applications (Mono/Unspray). In addition there were four-row strips of cotton and peanut with full rates of spray application (Strip/Full), half rates of spray application (Strip/Reduced), and no spray applications (Strip/Unspray).
Figure 4.1  Progress of early leaf spot incidence on peanut over time in days after planting in 2002. Treatments were, peanut monocrops with full rates of spray application (Mono/Full), half rates of spray application (Mono/Reduced), and no spray applications (Mono/Unspray). In addition there were four-row strips of cotton and peanut with full rates of spray application (Strip/Full), half rates of spray application (Strip/Reduced), and no spray applications (Strip/Unspray).
Figure 4.2 Progress of stem rot incidence on peanut over time in days after planting in 2002. Treatments were, peanut monocrops with full rates of spray application (Mono/Full), half rates of spray application (Mono/Reduced), and no spray applications (Mono/Unspray). In addition there were four-row strips of cotton and peanut with full rates of spray application (Strip/Full), half rates of spray application (Strip/Reduced), and no spray applications (Strip/Unspray).
Figure 4.3 Progress of TSWV incidence on peanut over time in days after planting in 2002. Treatments were, peanut monocrops with full rates of spray application (Mono/Full), half rates of spray application (Mono/Reduced), and no spray applications (Mono/Unspray). In addition there were four-row strips of cotton and peanut with full rates of spray application (Strip/Full), half rates of spray application (Strip/Reduced), and no spray applications (Strip/Unspray).
Figure 4.4  LLS disease incidence at 125 DAP in 2002. Treatments were, peanut monocrops with full rates of spray application (Mono/Full), half rates of spray application (Mono/Reduced), and no spray applications (Mono/Unspray). In addition there were four-row strips of cotton and peanut with full rates of spray application (Strip/Full), half rates of spray application (Strip/Reduced), and no spray applications (Strip/Unspray).
Figure 4.5 Web blotch disease incidence at 125 DAP in 2002. Treatments were, peanut monocrops with full rates of spray application (Mono/Full), half rates of spray application (Mono/Reduced), and no spray applications (Mono/Unspray). In addition there were four-row strips of cotton and peanut with full rates of spray application (Strip/Full), half rates of spray application (Strip/Reduced), and no spray applications (Strip/Unspray).
LITERATURE CITED

