DIFFERENTIATION, PREDICTION AND MANAGEMENT OF EARLY AND LATE LEAF SPOT OF PEANUT IN THE SOUTHEASTERN UNITED STATES AND HAITI

by

ABRAHAM M. FULMER

(Under the Direction of Robert C. Kemerait)

ABSTRACT

Early (ELS) and late (LLS) leaf spot, caused by the fungi *Cercospora arachidicola* (Ca) and *Cercosporidium personatum* (Cp), respectively, are distinct diseases of peanut. Although similar in appearance, disease cycle and threat to the crop, the predominance and onset of the two diseases can differ greatly, and the underlying mechanisms are still not clear. The primary objective was to discern the epidemics of ELS and LLS, to identify factors responsible for their differential development, and to determine whether risk predicted via Peanut Rx and management with reduced input fungicide programs were equally applicable.

Field trials were conducted in Georgia and Florida from 2010 to 2016 to evaluate the effect of fungicide treatment, inoculum source (infested peanut residue), planting date (April, May or June), and peanut cultivar. Components of resistance and disease associations were evaluated in greenhouse trials via separate or co-inoculations. Prediction via Peanut Rx was evaluated based on 168 unique combinations of risk factors.

An inoculum source of each pathogen was identified as the primary factor regulating the onset and prevalence of ELS and LLS. Planting date and cultivar, dependent upon inoculum level, also had significant additive effects on disease development. The development of LLS tended to be more suppressed than the development of ELS when plots or leaflets were co-inoculated with Ca + Cp. Peanut Rx risk points had a strong relationship with leaf spot onset, and were a better predictor for ELS than

LLS. Delayed fungicide applications in prescription programs provided statistically better LLS control than normal programs, and did not result in a significant yield loss. Overall results from these studies demonstrate that predominance and onset of ELS and LLS can be predicted, and that fungicide treatments can be enhanced by timing applications based on expected disease development.

Fungicide trials were conducted from 2015 to 2017 in Haiti. Leaf spot and rust (*Puccinia arachidis*) severity and yield loss generally increased with decreasing fungicide applications. Absolute yield loss was 64 and 43%, for the local Haitian runner and Georgia-06G, respectively, and ranged from 28 to 34% for the local Haitian Valencia and New Mexico Valencia A.

INDEX WORDS: Early leaf spot, Late leaf spot, Cercospora arachidicola, Cercosporidium personatum, Disease onset, Predominance, Ecological association, Inoculum source, Planting date, Cultivar, Peanut Rx, Prescription fungicide programs, Haiti

DIFFERENTIATION, PREDICTION AND MANAGEMENT OF EARLY AND LATE LEAF SPOT OF PEANUT IN THE SOUTHEASTERN UNITED STATES AND HAITI

by

ABRAHAM M. FULMER

A.A., Abraham Baldwin Agricultural College, 2007

B.S.A., University of Georgia, 2009

A Dissertation Submitted to the Graduate Faculty of the University of Georgia in Partial Fulfillment of the Requirements for the Degree

DOCTOR OF PHILOSOPHY

ATHENS, GEORGIA

2017

DIFFERENTIATION, PREDICTION AND MANAGEMENT OF EARLY AND LATE LEAF SPOT OF PEANUT IN THE SOUTHEASTERN UNITED STATES AND HAITI

by

ABRAHAM M. FULMER

Major Professor:	Robert C. Kemerait, Jr.
Committee:	Timothy B. Brenneman Katherine L. Stevenson Albert K. Culbreath Emily G. Cantonwine Harald Scherm

Electronic Version Approved:

Suzanne Barbour
Dean of the Graduate School
The University of Georgia
August 2017

ACKNOWLEDGEMENTS

One of the most rewarding aspects of my time spent at the University of Georgia has been the great privilege to meet and work with so many wonderful folks. To each and every person who has assisted me along the way, I express my heartfelt appreciation for all your hours of service spent on my behalf. Truly, without your assistance, I would not have been able to complete this project. I would especially like to express my deep gratitude to Dr. Bob Kemerait for providing me with the opportunity to study in his program, for his unrelenting faith in my potential to succeed in graduate school and for his financial support that allowed me to pursue my curiosity. He has proven to be an exceptional advisor and mentor whose desire for excellence and passion for plant pathology challenged me to become a better scientist and enabled me to realize that I am capable of moving past my comfort zone. Through his guidance, patience and friendship, I also learned to have a greater love for literature, history, folklore, pre-historic artifacts and to never forget the human aspect of plant pathology. I would also like to express my great appreciation to Dr. David Langston for introducing me to the idea of pursuing a graduate degree in plant pathology as well as for being a unique mentor, friend and an ever willing participant in the philosophical exchange of ideas.

I am very appreciative to all of the faculty members who agreed to serve on my graduate committee. I thank Dr. Albert Culbreath for his open door policy, guidance, friendship, and the many stimulating conversations ranging from the intricacies of the peanut leaf spot pathosystem to our mutual admiration of the Sackett family saga. I also thank Dr. Tim Brenneman for his advice, guidance, and friendship and for his full support of me to pursuing research in Haiti. In addition to our conversations related to eternal realities, his love for God, family, plant pathology, nature and humanity have helped inspire me to pursue life to the fullest. I am very appreciative to Dr. Katherine Stevenson, Dr. Harald Scherm and Dr. Emily Cantonwine for their patience, advice, critiques, assistance and encouragement throughout this project. I am especially grateful for them taking the time to carefully review my

dissertation and clearly express helpful suggestions. I also express my appreciation to the all the faculty members at the University of Georgia who have broadened my knowledge of plant pathology and production agriculture.

I express my sincere gratitude to all of the research personnel at the University of Georgia and Florida who assisted me with field trials and long hours of lab work. I am indebted to the wonderful ladies in Dr. Kemerait's lab who spent hundreds of hours rating samples and entering data into Excel files. I especially thank Lina Flor Young, Jhen Bennett and Edna Shannon for their friendship, and imparting to me a great love and admiration for the Filipino culture. I very much appreciate all of the folks who have worked in Dr. Kemerait's lab and assisted me in field trials. I particularly thank Jared Walls, Bobby Wade, Caleb Clements, Ty Cook, Kory Herrington and Avery Olds for their invaluable assistance. I would also like to express my deep gratitude to John Paulk, Billy Mills, Mark Gomillion, Stan Jones, Hunt Sanders, Randy Hill, Lee Hitson, Dewayne Dales, Andy Carter, Dan Everts, Mike Heath, Eric Smith, Ed Troxel, Maynard Douglas, Jim Boyer, Dusty Dunn, Anthony Black, Neil Robertson, Lewis Mullis, Calvin Perry, Bobby Washington, Michael Foster and Michael Donahoo for their technical assistance, friendship and advice. My sincere gratitude to John Paulk and Mark Gomillion for the many pleasant conversations, and the countless hours spent mentoring me in the applied aspects of peanut production. I am grateful to Jason Brock for his friendship and taking the time to mentor me in the laboratory diagnostic techniques of plant diseases. In addition, I thank Matt Seader for the many hours spent helping plan and maintain greenhouse studies, and Xuelin Luo for her statistical assistance.

My educational journey was greatly enriched by my fellow graduate students at the University of Georgia who shared the road with me. I am greatly appreciative to Matt Roberts, Kelly Morris, Jared Walls, Blake Williams, Peter Eure, Rand Merchant, Jeff Standish, Lelani Sumabat, Marian Luis, Suzette Arcibal, Imana Power, Clarence Codod, Jake Fountain, Eddie Beasley, Brian Jordan and Sara Beth Pelham for their friendship, rousing conversations, encouragement and advice that they have provided along the way.

I am indebted to all the folks who contributed to the research project in Haiti. I would like to express my gratitude to the Peanut Mycotoxin Innovation Lab for partial funding of my graduate assistantship during the time of my involvement in Haiti. I thank Jamie Rhoads for his friendship, advice and thought-provoking conversations, as well as Dr. Greg MacDonald for his friendship and willingness to support my research in Haiti. Special thanks are also extended to Dr. David Hoisington for his advice and support during my time in Haiti. I express my gratitude to Dr. Barry Tillman for his friendship and advice provided during the project in Haiti as well as for being ever willing to support my research efforts at Marianna. I very much appreciate all the personnel at Meds and Foods for Kids and the Acceso Peanut Enterprise Corporation for providing assistance in research activities. I especially thank Will Sheard, Patrick Dorzan, Alex Carroll, Georgy Faroutine, Rick Macajoux, Luc Simprevil, and Dominique Laforest for their friendship, technical support and research assistance. I would also like to acknowledge the valuable assistance of the student interns from the Université Roi Henri Christophe and the Université Chrétienne du Nord d'Haïti who were responsible for implementing, managing, rating and harvesting many of the trials at Meds and Foods for Kids.

Finally, I am very grateful to all my family for their endless patience, love and encouragement during my stay at the University of Georgia. Each one in their own special way helped provide me with strength needed during the more difficult times, and the expectation of spending the weekend at the grey submarine was always one of the great delights of my soul. Special thanks go to all my friends and church family who supported me along the way and for understanding the many hours I was not able to spend with them. También quiero agradecer a todos mis colegas hispanohablantes que me animaron durante mi proyecto. Particularmente quiero agradecer a los maniseros de Argentina que me han enseñado las alegrías de la hospitalidad y me han permitido aprender más sobre la producción mundial de maní. It is extremely humbling to know that I have so many folks that care about my life and that I can call my friends. Truly, I have been blessed with a wonderful life – indeed, one that will forever be a monument to mercy. Above all, I thank God in heaven for His unmerited favor toward me as well as the gift of life and breath and sustaining grace.

Soli Deo Gloria

TABLE OF CONTENTS

Page

CHAPTER 1

INTRODUCTION

Peanut (*Arachis hypogaea* L.) is an important crop in many parts of the world, and is primarily valued for its high oil seed content as well as a flavorful and nutritionally dense food source (Davis and Dean, 2016; Flecher and Zhaolin, 2016). India and China are the world's largest producers of peanut, but Nicaragua and the United States have the highest yields per acre (USDA-FAS, 2016). In 2016, Georgia was the largest producing state, with 709,000 of the total 1,547,000 acres harvested in the United States representing a crop value of $502,823,000 (USDA-NASS, 2016). In Haiti, peanut represents less than 3 % of the total agricultural land use (FAOSTAT, 2016), but is an important cash and food crop for farmers (Nelson et al., 2003).

The peanut plant is susceptible to a wide array of foliar and soilborne diseases, caused by fungi, bacteria, viruses and nematodes (Porter et al., 1982). Among these, early and late leaf spot are two of the most important foliar diseases wherever peanut is grown, and are caused by the fungi *Cercospora arachidicola* Hori and *Cercosporidium personatum* (Berk & M.A. Curtis) Deighton syn. *Passalora personata*, respectively (Shokes and Culbreath, 1997). In untreated, high risk fields, both diseases can cause complete defoliation of the peanut plant resulting in yield losses as high as 70 % (Backman and Crawford, 1984; McDonald et al., 1985; Shokes and Culbreath, 1997). Disease intensity varies each season and across geographies, but wherever peanut is grown, a 10 to 50 % yield loss in untreated fields is the norm (McDonald et al., 1985). In Georgia, the annual average damage plus cost of control from 2012 to 2014 was estimated at $37.9 million (Kemerait, 2012, 2013, 2014). In Haiti, yields are often limited by environmental and agronomic issues, but the combination of leaf spot and peanut rust (caused by *Puccinia arachidis* Speg) often result in complete defoliation of the peanut plant (Fulmer et al., 2014;

Fulmer et al., 2012). Due to limited studies, exact yield losses in Haiti caused by these diseases is unknown, but in other tropical areas, 50 to 86 % yield losses have been reported (Subrahmanyam, 1997).

In Georgia, management of early and late leaf spot has historically been accomplished by applying fungicides on a 14-day calendar-based schedule (Culbreath et al., 2006). A more recent option for growers to move away from the calendar-based program is through an integrated approach that includes the use of more resistant cultivars, cultural practices and extended spray intervals (Cantonwine et al., 2006; Monfort et al., 2004; Woodward et al., 2014; Woodward et al., 2010). The integration of these factors has resulted in Peanut Rx – a risk index that acts as an educational tool for peanut producers in the southeastern US to estimate the relative intensity of the most important foliar and soilborne diseases in a given field prior to planting (Kemerait et al., 2017). For leaf spot, pre-plant risk factors include variety, planting date, rotation, field history, and irrigated vs dryland production. Risk points are assigned to each factor and after calculation, fields are assigned a high, moderate or low level of risk (Woodward et al., 2010). Agrichemical companies have developed prescription fungicide programs that are recommended for each risk level (Kemerait et al., 2017). These programs feature spray intervals that are proportional to the level of risk, i.e., intervals become greater as risk decreases. High risk fields are treated according to the standard 14-day calendar based program (7 applications), whereas moderate and low-risk fields are sprayed at extended intervals, totaling 5 and 4 applications, respectively (Kemerait et al., 2017). While the efficacy of prescription programs for leafspot management has been documented in numerous field trials (Woodward et al., 2014; Woodward et al., 2010), the use of Peanut Rx to predict the risk of each specific disease within the leaf spot complex has yet to be demonstrated.

In Haiti, management options for early and late leaf spot and peanut rust are limited (Kostandini et al., 2017). Resistant varieties have been developed by the International Crops Research Institute for the Semi-Arid-Tropics (ICRISAT) and screened in preliminary variety trials in Haiti (Fulmer et al., 2012). The results suggest that the high levels of resistance to foliar diseases in these lines offered greater yield potential than improved cultivars from the United States when grown without fungicides (Fulmer et al.,

2012). Unfortunately, none of these varieties have been successful in Haiti due to poor roasting or flavor quality. The use of the protective fungicide, chlorothalonil, has been investigated in a few preliminary field trials in Haiti, and the results indicate that one or two applications could prove economically beneficial for growers (J. Rhoads, unpublished data). Other studies in developing tropical countries have demonstrated that two to four fungicides applications can significantly increase yield, but the economic gains are often dependent on specific application timings and intervals, market type and seasonal rainfall (Naab et al., 2009; Waliyar et al., 2000).

Because of similarities in appearance, disease cycle and threat to the crop, both leaf spots are generally managed as single disease (Garren and Wilson, 1951). However, the relative abundance of each disease varies in space and time. In Georgia, late leaf spot was predominant from 1976 to the early 1990's; otherwise early leaf spot has generally been the most prevalent (Cantonwine et al., 2008; Smith and Littrell, 1980). In recent years the prevalence of late leaf spot has increased, and predominance varies for each field (Cantonwine et al., 2008). Furthermore, these diseases generally differ in relation to onset and rate of progress. When the inoculum of both pathogens is present, the onset of early leaf spot generally occurs 30 to 50 days after planting (DAP), followed 2 to 4 weeks later by late leaf spot (Shokes and Culbreath, 1997; Smith and Littrell, 1980). However, in some situations, early leaf spot has been observed later than late leaf spot (R. Kemerait, personal communication). Notwithstanding the delay in onset, late leaf spot is thought to be the more destructive of the two due to a greater rate of increase (Backman and Crawford, 1984; Hemingway, 1955; Jenkins, 1938).

The mechanisms governing the differences in the temporal dynamics of early and late leaf spot are not clear. Smith and Littrell (1980) listed several possible explanations for late leaf spot resurgence in the late 1970's, e.g., shifting to later harvest dates, introduction of new fungicide chemistries, timing of fungicide applications, the expansion of acres planted to a single genotype (Florunner), crop nutrition, drought stress, previous cropping history, irrigation practices and fungicide regimes. Others have linked increased late leaf spot to cooler, wet temperatures (Sommartya and Beute, 1986), although direct

evidence to support this hypothesis seems to be lacking. As plausible as these potential causes may be, Smith and Littrell (1980) concluded that more knowledge concerning the ecology and epidemiology of the causal pathogens would be needed in order to offer a more satisfactory explanation of temporal dynamics. While advancements have been made in the area of epidemiology in the last 30 years (Alderman and Beute, 1986, 1987; Alderman et al., 1987; Alderman and Nutter, 1994; Alderman et al., 1989; Butler et al., 1994; Butler et al., 1995; Cantonwine et al., 2008; Cantonwine et al., 2007; Cu and Phipps, 1993; Shew et al., 1988; Sommartya and Beute, 1986; Wadia et al., 1998; Wu et al., 1999), none of the studies have specifically addressed the differences in the behavior of early and late leaf spot.

Detailed knowledge of disease dynamics is requisite for recommending the most effective integrated disease management programs and disease forecasting systems (Fry, 1982). Given the recent increase in the prevalence of late leaf spot in Georgia, and the fact that neither relative abundance nor onset of either leaf spot disease is completely understood, it follows that the continued advancement of integrated management options depends on filling this knowledge gap. Therefore, the main objective of the studies reported in this dissertation was to characterize the epidemics of early and late leaf spot in relation to a range of pre-plant risk factors and fungicide programs in order to i) elucidate possible mechanisms responsible for the differences and similarities in their behavior, ii) determine whether the development of each disease is equally predicted with Peanut Rx, and iii) determine whether an improved understanding of the differential development of these diseases can lead to improved timing of fungicide applications or novel management tactics. Specific objectives were to:

1) determine the effect of (i) prescription fungicide programs, (ii) inoculum level and fall tillage, (iii) inoculum level and planting date, and (iv) variety (under differing planting dates and inoculum pressures) on prevalence, onset, rate and final intensity of early and late leaf spot;
2) compare the components of resistance to *C. arachidicola* and *C. personatum* when inoculated alone or co-inoculated on three primary cultivars grown in the southeastern United States;

3) characterize the within-plant distribution and ecological association of early and late leaf spot in fields with differing inoculum levels and planting dates;
4) evaluate the accuracy and precision of Peanut Rx to predict the prevalence, onset and final intensity of early and late leaf spot;
5) determine the appropriate number and timing of fungicide applications for managing leaf spot and rust in Haiti.

LITERATURE CITED

Alderman, S., and Beute, M. 1986. Influence of temperature and moisture on germination and germ tube elongation of *Cercospora arachidicola*. Phytopathology 76:715-719.

Alderman, S., and Beute, M. 1987. Influence of temperature, lesion water potential, and cyclic wet-dry periods on sporulation *Cercospora arachidicola* on peanut. Phytopathology 77:960-963.

Alderman, S., and Nutter, F. 1994. Effect of temperature and relative humidity on development of *Cercosporidium personatum* on peanut in Georgia. Plant Dis. 78:690-694.

Alderman, S., Nutter Jr, F., and Labrinos, J. 1989. Spatial and temporal analysis of spread of late leaf spot of peanut. Phytopathology 79:837-844.

Alderman, S., Matyac, C., Bailey, J., and Beute, M. 1987. Aeromycology of *Cercospora arachidicola* on peanut. Transactions of the British Mycological Society 89:97-103.

Backman, P., and Crawford, M. 1984. Relationship between yield loss and severity of early and late leafspot diseases of peanut. Phytopathology 74:1101-1103.

Butler, D., Wadia, K., and Jadhav, D. 1994. Effects of leaf wetness and temperature on late leaf-spot infection of groundnut. Plant Pathol. 43:112-120.

Butler, D., Wadia, K., and Reddy, R. 1995. Effects of humidity, leaf wetness, temperature and light on conidial production by *Phaeoisariopsis personata* on groundnut. Plant Pathol. 44:662-674.

Cantonwine, E., Culbreath, A., and Stevenson, K. 2007. Characterization of early leaf spot suppression by strip tillage in peanut. Phytopathology 97:187-194.

Cantonwine, E., Culbreath, A., Holbrook, C., and Gorbet, D. 2008. Disease progress of early leaf spot and components of resistance to *Cercospora arachidicola* and *Cercosporidium personatum* in runner-type peanut cultivars. Peanut Sci. 35:1-10.

Cantonwine, E. G., Culbreath, A. K., Stevenson, K. L., Kemerait, R. C., Brenneman, T. B., Smith, N. B., and Mullinix, B. G. 2006. Integrated disease management of leaf spot and spotted wilt of peanut. Plant Dis. 90:493-500.

Cu, R., and Phipps, P. 1993. Development of a pathogen growth response model for the Virginia peanut leaf spot advisory program. Phytopathology 83:195-201.

Culbreath, A., Kemerait Jr, R., and Brenneman, T. 2006. Management of early leaf spot of peanut as affected by fungicide and date of spray program initiation. Plant Health Progress doi:10.1094/PHP-2006-0214-01-RS.

Davis, J., and Dean, L. 2016. Peanut composition, flavor and nutrition. Pages 289-345 in: Peanuts: Genetics, Processing, and Utilization. H. T. Stalker and R. F. Wilson, eds. AOCS Press, Urbana, IL.

FAOSTAT. 2016. Food and Agriculture Organization of the United Nations. Statistics Division. http://www.fao.org/faostat.

Flecher, S., and Zhaolin, S. 2016. An overwiew of world peanut markets. Pages 267-287 in: Peanuts: Genetics, Processing, and Utilization. H. T. Stalker and R. F. Wilson, eds. AOCS Press, Urbana, IL.

Fulmer, A., Kemerait, R., and Brenneman, T. 2014. Mountains beyond mountains: Challenges and opportunities for managing peanut diseases in Haiti. Phytopathology 104 (Suppl.)S:155.

Fulmer, A., Kemerait, R., Sherwood, J., Jordan, D., Rhoads, J., and Brenneman, T. 2012. Evaluation of ICRISAT varieties for resistance to foliar peanut diseases in Haiti. Phytopathology 102 (Suppl.)S2:4.

Garren, K., and Wilson, C. 1951. Peanut diseases. Pages 262-324 in: The Peanut, the Unpredictable Legume. Nat. Fertilizer Assoc., Washington, D.C.

Hemingway, J. 1955. The prevalence of two species of Cercospora on groundnuts. Trans. Brit. Mycol. Soc. 38:243-246.

Jenkins, W. A. 1938. Two fungi causing leaf spot of peanut. J. Agr. Res. 56:317-332.

Kemerait, R. C., Jr. 2012. Georgia Plant Disease Loss Estimates. Coop. Ext. Ser. Bull. 102-5, University of Georgia, Athens.

Kemerait, R. C., Jr. 2013. Georgia Plant Disease Loss Estimates. Coop. Ext. Ser. Bull. 102-6, University of Georgia, Athens.

Kemerait, R. C., Jr. 2014. Georgia Plant Disease Loss Estimates. Coop. Ext. Ser. Bull. 102-7, University of Georgia, Athens.

Kemerait, R. C., Jr., Brenneman, T. B., and Culbreath, A. K. 2017. Peanut disease update. Pages 23-62 in: 2017 peanut update. W. Monfort, ed. Coop. Ext. Ser. College of Agric. Environ. Sci. University of Georgia., Athens.

Kostandini, G., Rhoads, J., Johnson, R., and Schwartzbord, J. R. 2017. Gender differences in peanut productivity and post-harvest practices: Evidence from Haiti. *In Review*.

McDonald, D., Subrahmanyam, P., Gibbons, R., and Smith, D. 1985. Early and Late Leaf Spots of Groundnut. Information Bull. No. 21. ICRISAT, Patancheru.

Monfort, W., Culbreath, A., Stevenson, K., Brenneman, T., Gorbet, D., and Phatak, S. 2004. Effects of reduced tillage, resistant cultivars, and reduced fungicide inputs on progress of early leaf spot of peanut *(Arachis hypogaea*). Plant Dis. 88:858-864.

Naab, J., Prasad, P., Boote, K., and Jones, J. 2009. Response of peanut to fungicide and phosphorus in on-station and on-farm tests in Ghana. Peanut Sci. 36:157-164.

Nelson, R., Jolly, C., Hinds, M., Donis, Y., and Prophete, E. 2003. Consumer preferences for peanut butter (mamba) products in Haiti: A conjoint analysis. Peanut Sci. 30:99-103.

Porter, D. M., Smith, D. H., and Rodriguez-Kabana, R. 1982. Peanut plant diseases. Pages 326-410 in: Peanut Science and Technology. H. Pattee and C. Young, eds. American Peanut Research and Education Society, Yoakum, TX.

Shew, B., Beute, M., and Wynne, J. 1988. Effects of temperature and relative humidity on expression of resistance to *Cercosporidium personatum* in peanut. Phytopathology 78:493-498.

Shokes, F., and Culbreath, A. K. 1997. Early and late leaf spots. Pages 17-20 in: Compendium of Peanut Diseases, 2nd Ed. N. Kokalis-Burelle, D. Porter, R. Rodriguez-Kabana, D. Smith and P. Subrahmanyam, eds. American Phytopathological Society Press, St. Paul, MN.

Smith, D. H., and Littrell, R. H. 1980. Management of peanut foliar diseases with fungicides. Plant Dis. 64:356-361.

Sommartya, T., and Beute, M. 1986. Temperature effects on germination and comparative morphology of conidia for Thai and USA isolates of *Cercosporidium personatum*. Peanut Sci. 13:67-70.

Subrahmanyam, P. 1997. Rust. Pages 31-33 in: Compendium of Peanut Diseases, 2nd Ed. N. Kokalis-Burelle, D. Porter, R. Rodriguez-Kabana, D. Smith and P. Subrahmanyam, eds. American Phytopathological Society Press, St. Paul, MN.

USDA-FAS. 2016. Foreign Agricultural Service. https://apps.fas.usda.gov/psdonline.

USDA-NASS. 2016. Agricultural Statistics Database. https://www.nass.usda.gov.

Wadia, K., McCartney, H., and Butler, D. 1998. Dispersal of *Passalora personata* conidia from groundnut by wind and rain. Mycol. Res. 102:355-360.

Waliyar, F., Adamou, M., and Traoré, A. 2000. Rational use of fungicide applications to maximize peanut yield under foliar disease pressure in West Africa. Plant Dis. 84:1203-1211.

Woodward, J., Brenneman, T., Kemerait, R., Culbreath, A., and Smith, N. 2010. Management of peanut diseases with reduced input fungicide programs in fields with varying levels of disease risk. Crop Protect. 29:222-229.

Woodward, J., Brenneman, T., Kemerait Jr, R., Culbreath, A., and Smith, N. 2014. On-farm evaluations of reduced input fungicide programs in peanut fields with low, moderate, or high levels of disease risk. Peanut Sci. 41:50-57.

Wu, L., Damicone, J., Duthie, J., and Melouk, H. 1999. Effects of temperature and wetness duration on infection of peanut cultivars by *Cercospora arachidicola*. Phytopathology 89:653-659.

CHAPTER 2

LITERATURE REVIEW

Origin, distribution and botanical classification of the peanut. Peanut (*Arachis hypogaea* L.) cultivation is an ancient practice that dates back to at least 1200 B.C. (Hammons et al., 2016). The origin of the peanut has been traced to the landscapes of South America, and archeological evidence suggests that it was a highly valued natural resource of the Amerindians (Hammons et al., 2016). At the time of discovery of the New World, Spaniard chroniclers found the peanut was already widely cultivated throughout the Americas (Hammons et al., 2016). Local trade routes between indigenous peoples, and the subsequent discovery of the West Indies eventually led to the regional and global distribution of peanut (Hammons et al., 2016). Peanut is now cultivated worldwide, and is primarily valued for its high seed oil content and dietary contribution to human and livestock nutrition (Stalker, 1997). Cultivation ranges from tropical to warm temperate regions between the latitudes 40º N to 40º S (Rao and Murty, 1994).

Peanut is part of the Fabaceae (previously Leguminosae) family sharing many common features such as flower structure and a synergistic relationship with rhizobia that results in root nodulation and nitrogen fixation (Stalker, 1997). Peanut is unlike most other domesticated plants, in that flowering occurs above ground, but fruit is developed below ground (Stalker, 1997). Botanical classification separates the cultivated peanut into two subspecies, *hypogaea* and *fastigiata*. The growth habit of subspecies *hypogaea* is mostly prostrate (runner), while the growth habit of the subspecies *fastigiata* is always erect (bunch) (Singh and Simpson, 1994). Varieties within the subspecies are marketed as four distinct types: runner, Virginia, Spanish and Valencia (Stalker, 1997). Runner and Virginia market types generally require 120 to 150 days to reach full maturity, whereas Spanish and Valencia types often only require 80 to 110 days to reach full maturity (J. Beasley, personal communication).

Peanut production in the southeastern United States. In the United States, peanut has traditionally been grown in the Southeast (Georgia, Alabama, Florida and Mississippi), Southwest (Texas, Oklahoma and New Mexico) and the Virginia-Carolinas (Virginia, North Carolina and South Carolina) regions of the nation (Archer, 2016). Since the reforms made in the 2002 farm bill, which abolished the quota system, production has increased in Mississippi and new acreage has expanded into Arkansas (Archer, 2016). In 2016, a of total 1,547,000 acres of peanuts were harvested in 11 different states: Alabama, Arkansas, Florida, Georgia, Mississippi, New Mexico, North Carolina, South Carolina, Oklahoma, Texas and Virginia (USDA-NASS, 2016). Among these, Georgia, Florida and Alabama accounted for 46, 9.5 and 11 % of the total area harvested with average yields of 4,416, 4,371 and 4,035 kg/ha, respectively (USDA-NASS, 2016). The runner market type has historically been planted in the southeastern United States, and continues to dominate the acreage to the present day (Stalker, 1997). Valencia types are only grown in small quantities for the fresh, boiled peanut market (J. Beasley, personal communication).

Peanut production in Haiti. Bartonlomé Las Casas first described the peanut on the island of Hispaniola in 1527 (Hammons et al., 2016), and the plant continues to be cultivated in both the Dominican Republic and Haiti up to the present day. In Haiti, peanut represents less than 3 % of the total agricultural land use (FAOSTAT, 2016), but is an important cash and food crop for many Haitian farmers (Nelson et al., 2003). There are two main peanut growing regions in Haiti: the Northeast and the Central Plateau. From a production standpoint, these two regions are characterized by different rainfall patterns and market types. The main rainy season in both regions occurs in the spring (April to June). However, the Northeast often has a second, more sporadic, rainy season that generally occurs from October to November but can also extend into December, January and February (Kostandini et al., 2017). Most peanuts are planted in the spring to align with the rainy season; however growers in the Northeast are often able to plant a second crop during the winter months. The local Haitian runner type peanut is mainly planted in the Northeast and the local Haitian Valencia is planted in the Central Plateau. The

runner requires 120 to 130 days to harvest and the Valencia requires 80 to 90 days. Both types are local landraces with unknown origin (Kostandini et al., 2017).

Similar to other agroecosystems in the tropics (Subrahmanyam et al., 1997), peanut production in Haiti is characterized by smallholder farmers in rain-fed systems with very limited inputs (Nelson et al., 2003). Soil preparation, planting, weeding and harvesting is almost always done by hand. Yields are extremely low (448 to 897 kg/ha) (FAOSTAT, 2016; Nelson et al., 2003) due to soil fertility issues (Bargout and Raizada, 2013), variable seed quality (Nelson et al., 2003) and defoliating fungi such as *Puccinia arachidis, Cercospora arachidicola* and *Cercosporidium personatum* (Fulmer et al., 2014). Soils in Haiti are mostly alkaline with a pH that ranges from 7 to 8 (Bargout and Raizada, 2013), and generally are deficient in phosphorus but high in calcium (PMIL, unpublished data). Quality is also a problem and aflatoxin contamination from *Aspergillus flavus* is widespread, often resulting in aflatoxin concentrations ranging from 100 to over a 1000 ppb in local Haitian peanut butter (Filbert and Brown, 2012; Schwartzbord and Brown, 2015). Growers generally lack the knowledge and resources needed to combat these issues.

Haiti has been selected as part of the USAID Feed the Future program to reduce hunger, poverty and malnutrition in developing countries (PMIL, 2017). Plumpy'nut, a peanut-based Ready-to-Use Therapeutic Food, has demonstrated significant effects in reversing the health of malnourished children in Haiti (Iannotti et al., 2015). Meds and Foods for Kids (MFK), a non-profit organization, has been making Plumpy'nut (previously Medika Mamba) in Haiti for over 20 years (MFK, 2017). A new factory was constructed in 2012, with the capacity to produce large volumes of Plumy’nut (J. Rhoads, personal communication). This has created a substantial demand for high quality, locally grown peanuts. However, the low quality and quantity of peanuts and high market prices limit the quantity of peanuts that MFK is able to source locally. Recent funding from the USAID-supported Peanut and Mycotoxin Innovation Lab (Project UF-204) has allowed researchers from the University of Georgia and University of Florida to continue to address these production constraints.

Epidemiology of early and late leaf spot. Woodroof (1933) conclusively showed that there are two similar, yet distinct leaf spot diseases of peanut. These have since been referred to as early leaf spot (caused by *Cercospora arachidicola* Hori) and late leaf spot (caused *Cercosporidium personatum* (Berk and M. A. Curtis) Deighton), syn. *Passalora personata*) (Shokes and Culbreath, 1997). The sexual stages of these fungi have rarely been observed since first reported by Jenkins (1938), and are not thought to play a major role in disease development (Shokes and Culbreath, 1997). Both are polycyclic diseases that cause lesions on leaflets, petioles, stems and pegs, and epidemics are highly dependent on the prevailing environmental conditions (Shokes and Culbreath, 1997). Broadly speaking, both fungi require a prolonged duration of temperatures greater than 19° C and prolonged periods (> 12 h) of leaf wetness, e.g., rainfall, irrigation, or relative humidity (RH) greater than 95%, in order for infections to occur (Jensen and Boyle, 1965; Shokes and Culbreath, 1997)

Conidia arising from stromata that overwinter in the peanut residue are considered the main source of initial inoculum, with initial infections starting by rain-splashed conidia landing on the lower leaves of the plant (Shokes and Culbreath, 1997). Primary and secondary inoculum of both leaf spot pathogens are disseminated by wind, rain and insects (Shokes and Culbreath, 1997). Conidia of both fungi have been detected at heights > 2.5 m above the peanut canopy (Mallaiah and Rao, 1980; Smith and Crosby, 1973). Alderman et al. (1989) found a steep dispersal gradient for late leaf spot (within 1 m) for the first few weeks after the introduction of an inoculum source, but after the fifth week of detection, a shallow gradient (within 9 m) was observed. Little is known about the long distance dispersal of either leaf spot pathogen (Shokes and Culbreath, 1997). Smith and Littrell (1980) proposed that most of the initial inoculum outside of the field comes from a source within a 1.6-km radius. However, longer-distance dispersal may occur through tropical storms or insects. Wolf (1916) collected insects in five counties in Alabama and found conidia on the bodies (or within feces of) 54 out of 75 insects, representing species within the orders Orthoptera, Lepidoptera, Coleóptera, and Hemiptera.

Upon contact with the leaflet, conidia produce germ tubes which are capable of direct penetration through the epidermis or indirectly through the stomata. The sub-epidermal infection process differs

between the two fungi: *C. personatum* derives nutrients by forming intercellular haustoria while *C. arachidicola* forms intra- and inter-cellular mycelium (Shokes and Culbreath, 1997). The incubation period is typically 6-8 days for *C. arachidicola* and 10-14 days for *C. personatum* (Shokes and Culbreath, 1997). However, the latent period varies for a given variety. Nevill (1981) reported 21.7 and 14.6 days for *C. arachidicola* and *C. personatum*, on the cultivar TMV-2, respectively, whereas Cantonwine et al. (2008) reported 22.1 and 24.7, respectively, on the cultivar Georgia Green . Researchers have consistently stated that *C. personatum* has more abundant sporulation than *C. arachidicola* (Backman and Crawford, 1984; Shokes and Culbreath, 1997).

Lesions caused by *C. arachidicola* infections are light tan to reddish brown, and those caused by *C. personatum* are typically dark brown to black (Shokes and Culbreath, 1997). Although both diseases can result in a conspicuous chlorotic halo encircling the edges, it is generally more associated with early leaf spot (Shokes and Culbreath, 1997). Conidia produced by *C. arachidicola* are typically curved, long and narrow (35 to110µm x 3 to 6 µm) with more than five septa (Jenkins, 1938). Conidia produced by *C. personatum* tend to be straight, short and broad (20 to 70 µm x 4 to 9 µm) with less than 5 septa (Jenkins, 1938). Within the lesion, sporulation patterns are unique for both fungi. *Cercospora arachidicola* produces sparse clusters of conidia on conidiophores embedded in dark brown stromata with appear randomly distributed (Porter et al., 1982). In contrast, *C. personatum* produces numerous, dense clusters of conidiophores embedded in pseudoparenchymatous stromata which often exhibit a concentric ring pattern (Porter et al., 1982). Another distinguishing factor is the location of the sporulation in relation to the leaflet: *C. arachidicola* mainly sporulates on the upper side of the leaflet; in contrast, *C. personatum* mainly sporulates on the bottom side of the leaflet (Shokes and Culbreath, 1997).

The epidemiological behavior of these diseases can differ greatly. The most characteristic difference is the time of disease onset. A previous report suggested that, when inoculum of both pathogens is present in a field, the onset of early leaf spot typically occurs 30 to 50 days after planting (DAP) and is generally 3 to 4 weeks prior to that of late leaf spot (Smith and Littrell, 1980). However, there are reports of both diseases initiating at 21 to 35 DAP in the tropics (McDonald et al., 1985), and at

times neither disease may not appear until the end of the season or not at all (R. Kemerait, personal communication). However, the specific time of disease onset for either disease has not generally been reported in the literature, thus, it is difficult to give a full range of the variability that can occur among fields and seasons. Regardless of differences in onset, late leaf spot is thought to be the more destructive of the two due to a greater amount of sporulation per lesion and thus a higher rate of development toward the end of the season (Backman and Crawford, 1984; Hemingway, 1955; Jenkins, 1938).

Additionally, it has long been observed that the prevalence of the two leaf spot diseases varies in time and space (Jenkins, 1938; Miller, 1953; Woodroof, 1933). Maps published by the Commonwealth Mycological Institute in 1966 and 1967 indicated that early leaf spot was more widely distributed worldwide than late leaf spot (CABI-EPPO, 1985, 1987). Hemingway (1955) observed that late leaf spot was more prevalent in Tanzania, and it is commonly thought that this disease is more prevalent in the tropics such as southern India, West Africa, Thailand and the Philippines (Butler et al., 1994; Hemingway, 1955; Jackson and Bell, 1969; Paningbatan and Opina, 1995; Sommartya and Beute, 1986). However, additional reports have stated that early leaf spot has been the predominant disease in Malawi, Zambia, Mozambique and surrounding areas (Subrahmanyam et al., 1997).

In the late 1930s, Jenkins stated that late leaf spot only occurred 2 out of every 5 years in Georgia, and Miller (1953) reported that early leaf spot was the most wide spread of the two diseases in the southeastern United States. Florida became almost exclusively dominated by late leaf spot in the late 1970s (Jackson, 1981; Smith and Littrell, 1980) and has remained so to the present (Kucharek, personal communication). On the other extreme, Virginia has remained dominated almost exclusively by early leaf spot (Phipps et al., 1997). Similar shifts have taken place across the peanut growing regions of the United States. One particularly well documented example is from long-term observations in Tifton, Georgia. From the early 1930s to the mid-1960s, early leaf spot was prevalent in most years (Jenkins, 1938; Smith and Crosby, 1973; Woodroof, 1933). A sudden shift took place in 1976 in which late leaf spot gained prevalence over early leaf spot (Smith and Littrell, 1980). Beginning in 1992, early leaf spot resurfaced and soon became the prevalent disease throughout the 1990s (A. Culbreath, unpublished data).

Since 2003, late leaf spot has experienced resurgence, but early leaf spot still dominates in most fields (Cantonwine et al., 2008).

Management. Historically, growers in the southeastern US have suppressed early and late leaf spot epidemics by combining several disease management principles: crop rotation, conventional tillage with moldboard plow, and chemical protection (Shokes and Culbreath, 1997). Since the early 1970's, however, the use of fungicides applied on a 14-day calendar schedule has been the primary method of control relied on by growers (Culbreath et al., 2006; Smith and Littrell, 1980). Over the years, researchers have investigated strategies for reducing fungicide inputs, e.g., extended application intervals, strip-tillage, host resistance and weather-based spray advisories (Cantonwine et al., 2007; Cantonwine et al., 2006; Monfort et al., 2004; Woodward et al., 2014; Woodward et al., 2010). Recently, a risk index (Peanut Rx) was developed, and offers hope of a successful integrated management program. This system provides growers the opportunity to reduce fungicide inputs with prescription programs intended for low and moderate-risk fields (Kemerait et al., 2017b; Woodward et al., 2010).

Peanut Rx acts as an educational tool for peanut producers in the southeastern US to estimate the relative intensity of foliar and soilborne diseases prior to planting. Risk is calculated based on the different management factors that represent the practices of individual growers (Kemerait et al., 2017b). Specific factors of Peanut Rx that influence the overall leaf spot epidemic include peanut cultivar, planting date, crop rotation, tillage, field history and irrigation (Woodward et al., 2008). By incorporating all of these factors, future disease severity is predicted, and a risk assessment of high, moderate or low disease threat is calculated according to the specific management practices selected for individual fields. Once the disease risk for each field has been determined, prescription fungicide programs can be developed (Kemerait et al., 2017b). Prescription fungicide programs are based on the level of risk calculated by Peanut Rx, i.e., high, moderate or low. Accordingly, spray intervals are proportional to the level of risk, i.e., intervals become greater as risk decreases and vice versa. High-risk fields are treated according to the standard calendar-based program (7 applications), whereas moderate and low-risk fields are sprayed on extended intervals (5 and 4 applications, respectively) (Kemerait et al., 2017b).

Management of leaf spot in tropical, developing countries is often limited due to economic constraints (Subrahmanyam et al., 1997). Cultural practices recommended to reduce the initial inoculum include destruction of volunteers, removal of infested peanut residue, and crop rotation, but the efficacy of these tactics is often questioned due to the proximity of neighboring fields in small landholding systems (McDonald et al., 1985; Subrahmanyam et al., 1997). Later planting dates are also recommended, but most growers in rain-fed systems do not have flexible planting dates (Subrahmanyam et al., 1997). Resistant varieties have been developed by the International Crops Research Institute for the Semi-Arid Tropics (ICRISAT) and offer promise for foliar disease management and higher yields (McDonald et al., 1985; Sudini et al., 2015). These varieties mainly have resistance to late leaf spot and rust (Subrahmanyam et al., 1985), but others have been developed that have higher resistance to early leaf spot (Subrahmanyam et al., 1997).

Studies have demonstrated that limited fungicide applications can significantly increase yield and potentially prove economically advantageous in some areas in the tropics (Naab et al., 2009a; Naab et al., 2009b; Waliyar et al., 2000). Studies in Malawi have shown that one or two applications of chlorothalonil can be economically advantageous for controlling early leaf spot and increasing yield in situations with normal rainfall (Subrahmanyam et al., 1997). In West Africa, a wide range of fungicide timings and intervals was evaluated for control of early and late leaf spot and rust for short (<100 days) and long (>120) maturing cultivars (Waliyar et al., 2000). While results showed that 2 to 3 fungicide applications were able to significantly reduce disease and increase net gains for all cultivars, not all timing and intervals were effective (Waliyar et al., 2000).

Factors that may affect the differing epidemiological behavior of early and late leaf spot

Inoculum level. Most reports agree that the primary form of initial inoculum for both *C. arachidicola* and *C. personatum* comes from conidia produced on stromata that overwinter in the peanut residue (Hemingway, 1954; Jackson and Bell, 1969; Shanta, 1960). It has been proposed that conidia, mycelial fragments, ascospores and chlamydospores residing in the soil could also serve as potential inoculum sources, but since the time of Jenkins (1938) and Woodroof (1933), the latter two have rarely, if ever, been observed (Shokes and Culbreath, 1997). In the tropics, volunteer peanuts play a major role in continual inoculum perpetuation between seasons (McDonald et al., 1985). In the southeastern United States, volunteer peanuts likely do not serve as continual inoculum source since most plants die due to freezing weather. However, if not controlled, volunteer peanuts can serve as a bridge for the local inoculum source to perpetuate between rotational crops (Shokes and Culbreath, 1997).

The survival time of the leaf spot fungi within peanut residue has proven difficult to fully elucidate. Nuesry (1981) found that sporulation of *C. arachidicola* on stromata occurred after infested leaflets, petioles and stems had been buried in the field at a depth of 10 and 20 cm for 6 months. The same author found that stromata could sporulate after being stored in a cool, closed environment for more than 1.5 years. In a similar study, (Wolf, 1914) showed that inoculum from leaves infested with *C. personatum* left to overwinter out in the field in October, could still cause infections in May. In contrast, separate studies in India reported that the late leaf spot pathogen within infested leaves and/or stems remained viable for 30 to 60 days (Rao et al., 1993) and at most up to 20 weeks (Shanta, 1960).

The survival and/or reduction of the leaf spot fungi can also be inferred from studies linked to cultural practices. In one study, a 1-year rotation away from peanut significantly reduced late leaf spot severity (Kucharek, 1975). Brenneman et al. (1995) found that leaf spot severity decreased progressively when peanuts were rotated to bahiagrass for 0, 1, 2 or 3 years. In Georgia, peanuts are generally rotated with at least 1 year of cotton or corn, but a rotation longer than 3 years is rare (S. Monfort, personal communication). Thus, it is possible that a reduced level of the inoculum of each leaf spot pathogen could survive in the soil during the normal rotational period. Taken together, it is

probable that inoculum levels of both leaf spot fungi decrease with each year rotated away from peanut, and after 3 years would be mostly eliminated.

Tillage practices also have a direct or indirect impact on the level of initial inoculum. In a conventional system, a mold board plow is used to bury any remaining peanut residue at a depth of at least 15 to 30 cm prior to planting peanut (Jackson and Bell, 1969). In a conservation tillage system, a cover crop of wheat or rye is planted in the winter and killed at least 4 weeks prior to planting the peanuts. Immediately prior to planting, a strip-till rig is used to press down the cover crop, and sub soil shanks spaced ~0.91 m apart followed by a combination of fluted coulters and press wheels break up larger clods on the surface to create a narrow seeding bed ~ 30 cm wide (Paulk, personal communication). Interestingly, conservation tillage has proven more efficacious than conventional tillage in reducing leaf spot epidemics, and this is thought to be due to the barrier created by the cover crop which hinders splash dispersal of the conidia in the soilborne residue from reaching the plant (Cantonwine et al., 2007; Porter and Wright, 1991).

The question of how long the inoculum of each leaf spot pathogen can survive remains a critical gap in our understanding of the effect these cultural practices have on the temporal dynamics of early and late leaf spot. Although the above studies clearly indicate that a reduction in the initial inoculum of each leaf spot pathogen reduces the severity of both diseases, it is difficult to predict how rotation and tillage would affect the separate carryover of *C. arachidicola* and *C. personatum*. However, it is reasonable to assume that fields with differing levels of initial inoculum of each pathogen would drastically alter the predominance, onset and final severity of each disease.

Planting date. As part of the management principle of avoidance, adjusting the planting date is a tactic that allows the host to evade contact with the pathogen in some pathosystems. This is an important cultural practice that is recommended in Georgia as part of an integrated disease management program to manage *Tomato spotted wilt virus* (Culbreath et al., 2010) and leaf spot (Kemerait et al., 2017b). In Georgia, peanuts are planted from early April to mid-June, thus providing a large window of opportunity to avoid certain diseases. Early plantings are exposed to cooler temperatures early in the

season (April and May) whereas late-planted peanuts are exposed to cooler temperatures late in the season (September and October). Therefore, such differences could lead to a differential effect for more than one disease.

The effect of planting date on leaf spot has been reported in several studies. Hemingway (1954) evaluated the effect of five different planning dates ranging from mid-January to mid-March on the development of late leaf spot in Tanzania, and found that time to defoliation increased linearly with each consecutive planting date. In Senegal, Chevaugeon (1952) reported a similar trend for late leaf spot across five planting dates ranging from 15 June to 25 July. In contrast, Farrell et al. (1967) reported that the development of early leaf spot in Malawi decreased linearly when planted during the first week of December, January and February. In the southeastern United States where both leaf spots are present, (Shokes et al., 1982) reported a linear increase of severity in peanuts planted in late April, early May and mid-May. In a more recent study in Georgia, Jordan and Culbreath (2016) evaluated six planting dates from early April to late May and found that late leaf spot increased progressively with each of the later planting dates. Together, these data demonstrate the effect of plating date to reduce the overall leaf spot epidemic. However, the comparative effect on the temporal development of each leaf spot is still unknown.

Host resistance. Smith and Littrell (1980) suggested that the shift from early leaf spot to late leaf spot in Georgia during 1976 could have been linked to the wide spread introduction of a new cultivar (Florunner), and thus indicated that differences in host resistance could influence the behavior of early and late leaf spot. Part of this hypothesis is reasonable because studies indicate that genes for partial resistance to ELS and LLS are inherited independently (Abdou et al., 1974; Cantonwine et al., 2008). Yet, with most research emphasis directed toward managing the predominant leaf spot in a given region, few studies have directly compared the relative susceptibility of these cultivars between the two leaf spot pathogens, nor shown how such differences would affect the temporal dynamics of each leaf spot disease. However, with a careful review of the literature, there is circumstantial evidence to suggest that a single genotype can indeed have differential or equal levels of resistance/susceptibility to early and

late leaf spot. For example, in one study, the variety NC3303 was shown to have high levels of resistance to early leaf spot (Ricker et al., 1985), but in another study, it was highly susceptible to late leaf spot (Walls et al., 1985). In contrast, the cultivar Florigiant was found to be highly susceptible to both leaf spot diseases (Ricker et al., 1985; Walls et al., 1985).

From the 1970s, there have been three predominant peanut cultivars grown in southeastern US: Florunner (1970-1996), Georgia Green (1996-2008), and Georgia-06G (2006-present) (J. Beasley, personal communication). All of these cultivars are considered susceptible to early and late leaf spot; however, evidence to suggest differences exist between the leaf spot diseases is limited and, at times, inconsistent. Monasterios de La Torre (1980) screened multiple genotypes, including Florunner, for resistance to early and late leaf spot in field and greenhouse studies. In the more controlled environment, he found that Florunner seemed more susceptible to early leaf spot than late leaf spot. Backman and Crawford (1984) attributed similar yield losses to both pathogens. In contrast, while not the main part of their study, Branch and Culbreath (1995) observed that Florunner appeared to have more resistance to early and late leaf spot. Anderson et al. (1993) considered lower levels of early leaf spot, compared with higher levels of late leaf spot, more a function of a negative interaction between pathogens rather than a difference in host susceptibility on Florunner. Georgia Green was known to have slightly more resistance to both leaf spot diseases than Florunner (Cantonwine et al., 2008). Alhough not specifically compared in the same study, results from detached leaf assays indicated that Georgia Green could have slightly higher levels of susceptibility to early leaf spot (Cantonwine et al., 2008). In this study, the infection frequency was high for both diseases, but it appeared that the late leaf spot pathogen had much lower levels of sporulation on Georgia Green than the early leaf spot pathogen (Cantonwine et al., 2008). Because Georgia-06G is a newer cultivar, components of resistance have not been quantified, but based on field evaluations, it is thought to have similar levels of resistance as Georgia Green (A. Culbreath, personal communication). In short, there is little evidence to determine how shifts in these cultivars may have affected the population dynamics and development of early and late leaf spot.

Fungicide chemistry. Chlorothalonil has been one of the primary active ingredient used for leaf spot suppression for the last 50 years, and was almost exclusively relied upon during the 1970s and 1980s (Culbreath et al., 2006). The benzimidazole fungicide benomyl was briefly used in the early 1970s, but within a few years, resistance by both leaf spot pathogens was detected (Culbreath et al., 2002; Smith and Littrell, 1980). In 1994, tebuconazole and propiconazole, sterol biosynthesis inhibitor (SBI) fungicides, were labeled for peanut. Since then, an increasing number of new active ingredients within and among chemical classes (as grouped by the Fungicide Resistance Action Committee) have become labeled for peanut. These include the SBI fungicide prothioconazole, the quinone outside inhibitors azoxystrobin and pyraclostrobin, and the pyrazole carboxamide penthiopyrad (Culbreath et al., 2008; Culbreath et al., 2009; Kemerait et al., 2017a).

Differences in fungicide sensitives were reported to shift the proportion of closely related fungi *Leptosphaeria maculans* and *L. biglobosa* that cause phoma stem canker, a disease complex on oilseed rape (Huang et al., 2011). In this example, an application of a triazole plus a benzamidazole increased the frequency of *L. maculans* compared to the untreated check, but the same treatment decreased the frequency of *L. biglobosa*. While both the early and late leaf spot pathogens of peanut have been reported to have populations with reduced sensitivity to tebuconazole (Stevenson and Culbreath, 2006) most fungicides generally offer equal levels of control in the field (Culbreath et al., 2006, 2008; Culbreath et al., 2009; Woodward et al., 2014; Woodward et al., 2010). Propiconzole is the only fungicide known to have more efficacy against the early leaf spot pathogen compared with the late leaf spot pathogen (A. Culbreath, unpublished data). It is unknown whether any other chemistries would have a differential effect on the development of early and late leaf spot when used in a normal spray program (for examples see (Kemerait et al., 2017a)).

Fungicide program. Historically, management of early and late leaf spot has relied on seven to eight fungicide applications initiated at 30 DAP and repeated on a 14-day calendar-based schedule. However, growers have a number of options to vary the timing of these applications. Weather-based advisories were first developed in the 1960s (Jensen and Boyle, 1966), and since that time a number of

more effective and biologically advanced models have been developed (Cu and Phipps, 1993; Damicone et al., 1994; Jacobi et al., 1995). The AU-Pnut advisory is currently recommended for growers in Alabama, Georgia, and Florida, but has not been widely accepted (Kemerait et al., 2017b; Woodward et al., 2010).

Currently, growers still use the calendar spray program or use prescription fungicide programs based upon Peanut Rx (Kemerait et al., 2017b; Woodward et al., 2010). These programs feature different initiation dates and spray intervals for each level of risk. Applications for high risk programs begin at 30 DAP and are sprayed at 14-day intervals; moderate-risk programs begin at 37 DAP and are followed by applications made at 21, 14, 14 and 21-day intervals; low-risk programs begin at 44 DAP and are sprayed on 21-day intervals (Kemerait et al., 2017b). Given the different times of disease onset of early and late leaf spot, it may be that different initiation dates or spray intervals could affect the temporal dynamics of each disease differently.

Coexistence and ecological association. As there are multiple diseases that affect cultivated crops (Agrios, 2005), it is often the case that plants will be infected with multiple pathogens simultaneously (Fitt et al., 2006). Coexistence is often associated with related and nonrelated species which require similar habitats (Begon et al., 1996). From a plant pathology perspective, the tendency in such cases is for one pathogen to predominate, or eventually exclude co-occurring species from a region (Fitt et al., 2006). Examples of closely or distantly related fungi coexisting, but with one species predominating, include: ascochyta blight complex of pea (*Pisum sativum*), caused by *Ascochyta pisi*, *Mycosphaerella pinodes*, and *Phoma medicaginis* var. *pinodella* (Le May et al., 2009); phoma stem canker (blackleg) of canola (*Brassica napus*), caused by *Leptosphaeria maculans* and *L. biglobosa*; eyespot of wheat caused by *Oculimacula yallundae* and *O. acuformis* (Fitt et al., 2006); and septoria leaf complex of wheat, caused by *Parastagonospora nodorum* and *Zymoseptoria tritici* (Eyal, 1999).

In ecological theory, there are three basic interactions between pathogens occupying the same resource: negative, neutral or synergistic effects on neighboring species (Le May et al., 2009). Negative interactions are a result of interspecific competition between pathogens when resources are in short

supply, and according to the competitive exclusion principle, species sharing the exact same niche will eventually drive competitors to extinction - or exclusion from a given area (Begon et al., 1996). A niche is a multidimensional concept characterizing the sum of the biotic and abiotic factors that determine the ability of a pathogen to survive and reproduce (Fitt et al., 2006). Crucial to this concept is the difference between the fundamental and realized niche: the range of variables in which a species could potentially exist versus the conditions in which it actually exists due to the presence of competitors (Begon et al., 1996). Thus, for coexistence to occur between competing pathogens, each species must have a realized niche within its fundamental niche that does not overlap with its rival (Begon et al., 1996; Fitt et al., 2006).

Of particular interest are complexes among *Mycosphaerella* spp., such as yellow and black sigatoka of banana (*Musa* sp.) caused by *M. musicola*, and *M. fijiensis*, respectively (Marin et al., 2003), Prior to the 1960s, the yellow sigatoka pathogen was the predominant species worldwide (Marin et al., 2003). In the early 1960s, black sigatoka was discovered in Fiji, and it soon spread to the other banana producing regions. Upon the its arrival to a new area, the two sigatoka pathogens coexisted for a time, but eventually *M. fijiensis* became the predominant cause of disease – but only in the lowland regions (Marin et al., 2003). Studies attempting to understand this phenomenon discovered that the relative abundance of each species was mainly a function of temperature. Mouliom-Pefoura et al. (1996) determined that the fundamental niche of the yellow sigatoka pathogen includes low and high altitudes, but its realized niche is in cooler temperatures found at higher elevations; whereas both the fundamental and realized niche of the black sigatoka pathogen was found at warmer temperatures at low and sometimes mid-range altitudes (Marin et al., 2003). In addition to the realized environmental niches between the two sigatoka pathogens, Mouliom-Pefoura et al. (1996) also connected the shift in abundance to differences in host resistance – all know cultivars with resistance to *M. musicola* were found to be susceptible to *M. fijiensis*. The same authors also suggested that *M. fijiensis* was more aggressive than *M. musicola*.

In field studies, Anderson et al. (1993) alluded to a negative interaction between *C. arachidicola* and *C. personatum* due to competition for limited resources. Accordingly, low levels of *C. arachidicola* were attributed to high levels of *C. personatum*. Despite this interpretation, a review of the literature indicates that interspecific competition between these pathogens has not been studied in depth. Monasterios de La Torre (1980) reported a negative interaction when the leaf spot pathogens were co-inoculated on the Florunner cultivar in greenhouse studies. Accordingly, compared with *C. personatum* alone, total late leaf spot lesions were reduced when both fungi were inoculated together. The author contributed this reduction to three possibilities: a negative interaction between species, differences in spore concentrations, or different levels of susceptibility in Florunner.

Environment. It has long been proposed that late leaf spot epidemics are more favored by cool, wet weather (Sommartya and Beute, 1986). While it appears that this hypothesis has not been tested directly, separate studies concerning the effects of temperature on conidial germination, infection and lesion sporulation for *C. arachidicola* and *C. personatum* offer circumstantial evidence that the latter pathogen is favored by cooler temperatures in some, but not all, respects. Sommartya and Beute (1986) reported that conidial germination from the United States and Thai isolates of *C. personatum* were highest between 16-20° C. In contrast, Alderman and Beute (1986) reported that optimum temperatures for *C. arachidicola* germination ranged from 19-25° C after 12 and 24 h; however, after 48h, germination at16° C was almost as high. Other studies have demonstrated that temperatures favoring infection are similar for both pathogens: 22.3°C for *C. arachidicola* (Wu et al., 1999) and 20-24°C for *C. personatum* (Shew et al., 1988); however, the latter study did not include temperatures < 20°C. Butler et al. (1994) found that *C. personatum* infection for Indian isolates ranged from 15-26°C. Concerning optimum temperatures for sporulation, Alderman and Beute (1987) found *C. arachidicola* sporulation to be greatest at 24-28°C, moderate at 20° C and negligible at 16 and 32°C. In contrast, Shew et al. (1988) reported that conidial production for *C. personatum* was highest at 24°C and progressively lower at 20°C and 28°C. A similar study by Alderman and Nutter (1994) found sporulation for *C. personatum* was highest at 20°C, and decreased by roughly a half and a third at 15°C an 28°C, respectively. Conidial production for Indian *C.*

personatum isolates was highest at 26°C and significantly lower at 15°C (Butler et al., 1995), suggesting that there may be different optima based on geography.

The evidence that *C. personatum* is favored by higher relative humidity (RH) than *C. arachidicola* is less conclusive. In fact, Alderman and Nutter (1994) suggested that *C. arachidicola* could be more dependent on prolonged moisture due its stromata being less embedded in the leaf tissue, and the lack of haustoria. Oso (1972)reported that *C. arachidicola* germination required 100% RH. However, Alderman and Beute (1987) reported 97 ± 5% and 35±30% germination percentages when RH ≥ 95% and 93-97%, respectively. Wu et al. (1999) reported heavy infection by *C. arachidicola* at a RH ≥ 95%. In contrast, *C. personatum* infection was obtained when RH was > 93% (Shew et al., 1988). For sporulation of US isolates, *C. personatum* was only studied at RH ≥95% (Alderman and Nutter, 1994). Butler et al. (1995) reported that no sporulation occurred below 94.5% RH for Indian *C. personatum* isolates.

Because it is unrealistic to assume that leaf moisture remains continuous in nature, a few studies have also investigated the effects of interrupted leaf wetness. Shew et al. (1988) found that infection was minimal when high RH < 3 h/day, and that infection was significantly higher after 12 h/day; maximum lesions occurred with 24 h of high RH/day. In contrast, Butler et al. (1994) reported that wet/dry cycles, with at least 2 h of dry time, caused higher levels of *C. personatum* infection when compared with continuous leaf wetness. For *C. arachidicola*, while minimum germ tube elongation and sporulation occurred at 8 h of high RH, both variables increased with increasing wetness periods (Alderman and Beute, 1986, 1987).

LITERATURE CITED

Abdou, Y. A.-M., Gregory, W. C., and Cooper, W. E. 1974. Sources and nature of resistance to *Cercospora arachidicola* Hori and *Cercosporidium personatum* (Beck & Curtis) Deighton in *Arachis* species. Peanut Sci. 1:6-11.

Agrios, G. 2005. Plant Pathology 5th Edition: Elsevier Academic Press. Burlington, Ma. USA.

Alderman, S., and Beute, M. 1986. Influence of temperature and moisture on germination and germ tube elongation of *Cercospora arachidicola*. Phytopathology 76:715-719.

Alderman, S., and Beute, M. 1987. Influence of temperature, lesion water potential, and cyclic wet-dry periods on sporulation *Cercospora arachidicola* on peanut. Phytopathology 77:960-963.

Alderman, S., and Nutter, F. 1994. Effect of temperature and relative humidity on development of *Cercosporidium personatum* on peanut in Georgia. Plant Dis. 78:690-694.

Alderman, S., Nutter Jr, F., and Labrinos, J. 1989. Spatial and temporal analysis of spread of late leaf spot of peanut. Phytopathology 79:837-844.

Anderson, W., Holbrook, C., and Brenneman, T. 1993. Resistance to *Cercosporidium personatum* within peanut germplasm. Peanut Sci. 20:53-57.

Archer, P. 2016. Overview of the peanut industry supply chain. Pages 253-266 in: Peanuts: Genetics, Processing, and Utilization. H. T. Stalker and R. F. Wilson, eds. AOCS Press.

Backman, P., and Crawford, M. 1984. Relationship between yield loss and severity of early and late leafspot diseases of peanut. Phytopathology 74:1101-1103.

Bargout, R. N., and Raizada, M. N. 2013. Soil nutrient management in Haiti, pre-Columbus to the present day: lessons for future agricultural interventions. Agriculture & Food Security 2:11.

Begon, M., Harper, J. L., and Townsend, C. R. 1996. Ecology: Individuals, Populations and Communities. Blackwell Science.

Branch, W., and Culbreath, A. 1995. Combination of early maturity and leaf spot tolerance within an advanced Georgia peanut breeding line. Peanut Sci. 22:106-108.

Brenneman, T., Sumner, D., Baird, R., Burton, G., and Minton, N. 1995. Suppression of foliar and soilborne peanut diseases in bahiagrass rotations. Phytopathology 85:948-952.

Butler, D., Wadia, K., and Jadhav, D. 1994. Effects of leaf wetness and temperature on late leaf-spot infection of groundnut. Plant Pathol. 43:112-120.

Butler, D., Wadia, K., and Reddy, R. 1995. Effects of humidity, leaf wetness, temperature and light on conidial production by *Phaeoisariopsis personata* on groundnut. Plant Pathol. 44:662-674.

CABI-EPPO. 1985. *Mycosphaerella arachidis*. Distribution Maps of Plant Diseases No. 166. CABI Head Office, Wallingford, UK.

CABI-EPPO. 1987. *Mycosphaerella berkeleyi*. Distribution Maps of Plant Diseases No. 152. CABI Head Office, Wallingford, UK.

Cantonwine, E., Culbreath, A., and Stevenson, K. 2007. Characterization of early leaf spot suppression by strip tillage in peanut. Phytopathology 97:187-194.

Cantonwine, E., Culbreath, A., Holbrook, C., and Gorbet, D. 2008. Disease progress of early leaf spot and components of resistance to *Cercospora arachidicola* and *Cercosporidium personatum* in runner-type peanut cultivars. Peanut Sci. 35:1-10.

Cantonwine, E. G., Culbreath, A. K., Stevenson, K. L., Kemerait, R. C., Brenneman, T. B., Smith, N. B., and Mullinix, B. G. 2006. Integrated disease management of leaf spot and spotted wilt of peanut. Plant Dis. 90:493-500.

Chevaugeon, J. 1952. Recherches sur la Cercosporiose de l'arachide en Moyenne Casamance. Ann. Inst. Nat. Rech. Agron. Ser. C 3:489-510.

Cu, R., and Phipps, P. 1993. Development of a pathogen growth response model for the Virginia peanut leaf spot advisory program. Phytopathology 83:195-201.

Culbreath, A., Stevenson, K., and Brenneman, T. 2002. Management of late leaf spot of peanut with benomyl and chlorothalonil: A study in preserving fungicide utility. Plant Dis. 86:349-355.

Culbreath, A., Kemerait Jr, R., and Brenneman, T. 2006. Management of early leaf spot of peanut as affected by fungicide and date of spray program initiation. Plant Health Progress doi:10.1094/PHP-2006-0214-01-RS.

Culbreath, A., Kemerait Jr, R., and Brenneman, T. 2008. Management of leaf spot diseases of peanut with prothioconazole applied alone or in combination with tebuconazole or trifloxystrobin. Peanut Sci. 35:149-158.

Culbreath, A. K., Brenneman, T. B., Kemerait, R. C., and Hammes, G. G. 2009. Effect of the new pyrazole carboxamide fungicide penthiopyrad on late leaf spot and stem rot of peanut. Pest Manage. Sci. 65:66-73.

Culbreath, A. K., Tillman, B. L., Tubbs, R. S., Beasley, J. P., Kemerait, R. C., and Brenneman, T. B. 2010. Interactive effects of planting date and cultivar on tomato spotted wilt of peanut. Plant Dis. 94:898-904.

Damicone, J., Jackson, K., Sholar, J., and Gregory, M. 1994. Evaluation of a weather-based spray advisory for management of early leaf spot of peanut in Oklahoma. Peanut Sci. 21:115-121.

Eyal, Z. 1999. The *Septoria tritici* and *Stagonospora nodorum* blotch diseases of wheat. Eur. J. Plant Pathol. 105:629-641.

FAOSTAT. 2016. Food and Agriculture Organization of the United Nations. Statistics Division. http://www.fao.org/faostat.

Farrell, J., Bailey, B., and Mills, W. 1967. The effects of time of planting, spacing and fungicide on Cercospora leaf spots of groundnuts in Malawi. Rhod. Zambia Malawi J. Agric. Res 5:241-247.

Filbert, M. E., and Brown, D. L. 2012. Aflatoxin contamination in Haitian and Kenyan peanut butter and two solutions for reducing such contamination. Journal of Hunger & Environmental Nutrition 7:321-332.

Fitt, B. D., Huang, Y.-J., van den Bosch, F., and West, J. S. 2006. Coexistence of related pathogen species on arable crops in space and time. Annu. Rev. Phytopathol. 44:163-182.

Fulmer, A., Kemerait, R., and Brenneman, T. 2014. Mountains beyond mountains: Challenges and opportunities for managing peanut diseases in Haiti. Phytopathology 104 (Suppl.)S:155.

Hammons, R., Herman, D., and Stalker, H. T. 2016. Origin and early history of the peanut. Pages 1-26 in: Peanuts: Genetics, Processing, and Utilization. H. T. Stalker and R. F. Wilson, eds. AOCS Press.

Hemingway, J. 1954. Cercospora leafspots of groundnuts in Tanganyika. E. Afr. Agricultural Journal 19:263-271.

Hemingway, J. 1955. The prevalence of two species of Cercospora on groundnuts. Trans. Brit. Mycol. Soc. 38:243-246.

Huang, Y.-J., Hood, J., Eckert, M., Stonard, J., Cools, H., King, G. J., Rossall, S., Ashworth, M., and Fitt, B. D. 2011. Effects of fungicide on growth of *Leptosphaeria maculans* and *L. biglobosa* in

relation to development of phoma stem canker on oilseed rape (*Brassica napus*). Plant Pathol. 60:607-620.

Iannotti, L. L., Henretty, N. M., Delnatus, J. R., Previl, W., Stehl, T., Vorkoper, S., Bodden, J., Maust, A., Smidt, R., and Nash, M. L. 2015. Ready-to-use supplementary food increases fat mass and BMI in haitian school-aged children. The Journal of nutrition 145:813-822.

Jackson, C. R., and Bell, D. K. 1969. Diseases of Peanut (Groundnut) Caused by Fungi. Research Bull. 56, College of Agric. Exp. Stations University of Georgia., Athens.

Jackson, L. 1981. Distribution and severity of peanut leafspot in Florida. Phytopathology 71:324-328.

Jacobi, J., Backman, P., Davis, D., and Brannen, P. 1995. AU-Pnuts advisory I: Development of a rule-based system for scheduling peanut leaf spot fungicide applications. Plant Dis. 79:666-671.

Jenkins, W. A. 1938. Two fungi causing leaf spot of peanut. J. Agr. Res. 56:317-332.

Jensen, R., and Boyle, L. 1965. The effect of temperature, relative humidity and precipitation on peanut leafspot. Plant Dis. Rep 49:975-978.

Jensen, R. E., and Boyle, L. W. 1966. A technique for forecasting leafspot on peanuts. Plant Disease Reporter 50:810-814.

Jordan, B., and Culbreath, A. 2016. Effect of planting date and peanut cultivar on leaf spot epidemics and pod yield with implications for organic production. (Abstr.). Phytopathology 106:S2.6. http://dx.doi.org/10.1094/PHYTO-106-4-S2.6.

Kemerait, R. C., Jr., Brenneman, T. B., and Culbreath, A. K. 2017a. Peanut disease control. Pages 201-205 in: *2017 Georgia Pest Management Handbook*. D. Horton, ed. Special Bull. 28, Univeristy of Georgia Coop. Ext. College of Agric. Environ. Sci. University of Georgia., Athens.

Kemerait, R. C., Jr., Brenneman, T. B., and Culbreath, A. K. 2017b. Peanut disease update. Pages 23-62 in: 2017 peanut update. W. Monfort, ed. Coop. Ext. Ser. College of Agric. Environ. Sci. University of Georgia., Athens.

Kostandini, G., Rhoads, J., Johnson, R., and Schwartzbord, J. R. 2017. Gender differences in peanut productivity and post-harvest practices: Evidence from Haiti. *In Review*.

Kucharek, T. 1975. Reduction of Cercospora leafspots of peanut with crop rotation. Plant Disease Reporter 59:822-823.

Le May, C., Potage, G., Andrivon, D., Tivoli, B., and Outreman, Y. 2009. Plant disease complex: antagonism and synergism between pathogens of the Ascochyta blight complex on pea. J. Phytopathol. 157:715-721.

Mallaiah, K., and Rao, A. 1980. Aerobiology of two species of Cercospora pathogenic to groundnut. Proceedings of the Indian National Science Academy, B 46:215-222.

Marin, D. H., Romero, R. A., Guzman, M., and Sutton, T. B. 2003. Black Sigatoka: an increasing threat to banana cultivation. Plant Dis. 87:208-222.

McDonald, D., Subrahmanyam, P., Gibbons, R., and Smith, D. 1985. Early and Late Leaf Spots of Groundnut. Information Bull. No. 21. ICRISAT, Patancheru.

MFK. 2017. Meds and Food for Kids. https://mfkhaiti.org.

Miller, L. I. 1953. Studies of the parasitism of *Cercospora arachidicola* Hori and *Cercospora personata* (Berk. & M.A. Curtis). Ph.D. Dissertation. University of Minnesota, Minneapolis.

Monasterios de La Torre, T. 1980. Genetic resistance to Cercospora leafspot diseases in peanut (*Arachis hypogaea* L.). Ph.D. Dissertation. University of Florida, Gainesville.

Monfort, W., Culbreath, A., Stevenson, K., Brenneman, T., Gorbet, D., and Phatak, S. 2004. Effects of reduced tillage, resistant cultivars, and reduced fungicide inputs on progress of early leaf spot of peanut *(Arachis hypogaea*). Plant Dis. 88:858-864.

Mouliom-Pefoura, A., Lassoudière, A., Foko, J., and Fontem, D. 1996. Comparison of development of *Mycosphaerella fijiensis* and *Mycosphaerella musicola* on banana and plantain in the various ecological zones in Cameroon. Plant Dis. 80:950-954.

Naab, J., Prasad, P., Boote, K., and Jones, J. 2009a. Response of peanut to fungicide and phosphorus in on-station and on-farm tests in Ghana. Peanut Sci. 36:157-164.

Naab, J., Seini, S., Gyasi, K., Mahama, G., Prasad, P., Boote, K., and Jones, J. 2009b. Groundnut yield response and economic benefits of fungicide and phosphorus application in farmer-managed trials in Northern Ghana. Exp. Agric. 45:385-399.

Nelson, R., Jolly, C., Hinds, M., Donis, Y., and Prophete, E. 2003. Consumer preferences for peanut butter (mamba) products in Haiti: A conjoint analysis. Peanut Sci. 30:99-103.

Nevill, D. 1981. Components of resistance to *Cercospora arachidicola* and *Cercosporidium personatum* in groundnuts. Ann. Appl. Biol. 99:77-86.

Nuesry, S. M. 1981. Survival of *Cercospora arachidicola*, *Cercosporidium personatum*, and primary infection of peanuts in Oklahoma. M.S. thesis. Oklahoma State University, Stillwater.

Oso, B. 1972. Conidial germination in *Cercospora arachidicola* Hori. Transactions of the British Mycological Society 59:169-IN126.

Paningbatan, R., and Opina, O. 1995. Infectiousness of abscissed peanut leaves infected with *Cercospora arachidicola* Hori and *Cercosporidium personatum* (Berk. and Curt.) Deighton. Philippine Phytopathology.

Phipps, P. M., Deck, S. H., and Walker, D. R. 1997. Weather-based crop and disease advisories for peanuts in Virginia. Plant Dis. 81:236-244.

PMIL. 2017. Peanut Mycotoxin Innovation Lab. http://www.caes.uga.edu/global/feed-the-future-innovation-labs/peanut-mycotoxin-innovation-lab.html.

Porter, D., and Wright, F. 1991. Early leafspot of peanuts: effect of conservational tillage practices on disease development. Peanut Sci. 18:76-79.

Porter, D. M., Smith, D. H., and Rodriguez-Kabana, R. 1982. Peanut plant diseases. Pages 326-410 in: Peanut Science and Technology. H. Pattee and C. Young, eds. American Peanut Research and Education Society, Yoakum, TX.

Rao, A., McDonald, D., and Reddy, K. 1993. Perpetuation of peanut leaf spots pathogens. Oléagineux (Paris) 48:77-82.

Rao, V. R., and Murty, U. R. 1994. Botany — morphology and anatomy. Pages 43-95 in: The Groundnut Crop: A scientific basis for improvement. J. Smartt, ed. Springer Netherlands, Dordrecht.

Ricker, M., Beute, M., and Campbell, C. 1985. Components of resistance in peanut to *Cercospora arachidicola*. Plant Dis. 69:1059-1064.

Schwartzbord, J. R., and Brown, D. L. 2015. Aflatoxin contamination in Haitian peanut products and maize and the safety of oil processed from contaminated peanuts. Food Control 56:114-118.

Shanta, P. 1960. Studies on Cercospora leaf spot of Groundnuts (*Arachis hypogaea*). I. Effect of the environmental factors on disease incidence and on survival of the pathogen. J. Madras Univ. 30:167-177.

Shew, B., Beute, M., and Wynne, J. 1988. Effects of temperature and relative humidity on expression of resistance to *Cercosporidium personatum* in peanut. Phytopathology 78:493-498.

Shokes, F., and Culbreath, A. K. 1997. Early and late leaf spots. Pages 17-20 in: Compendium of Peanut Diseases, 2nd Ed. N. Kokalis-Burelle, D. Porter, R. Rodriguez-Kabana, D. Smith and P. Subrahmanyam, eds. American Phytopathological Society Press, St. Paul, MN.

Shokes, F., Gorbet, D., and Sanden, G. 1982. Effect of planting date and date of spray initiation on control of peanut leaf spots in Florida. Plant Dis. 66:574-575.

Singh, A. K., and Simpson, C. E. 1994. Biosystematics and genetic resources. Pages 96-137 in: The Groundnut Crop: A scientific basis for improvement. J. Smartt, ed. Springer Netherlands, Dordrecht.

Smith, D., and Crosby, F. 1973. Aerobiology of two peanut leafspot fungi. Phytopathology 63:703-707.

Smith, D. H., and Littrell, R. H. 1980. Management of peanut foliar diseases with fungicides. Plant Dis. 64:356-361.

Sommartya, T., and Beute, M. 1986. Temperature effects on germination and comparative morphology of conidia for Thai and USA isolates of *Cercosporidium personatum*. Peanut Sci. 13:67-70.

Stalker, H. T. 1997. Peanut (*Arachis hypogaea* L.). Field Crops Res. 53:205-217.

Stevenson, K. L., and Culbreath, A. 2006. Evidence for reduced sensitivity to tebuconazole in leaf spot pathogens. Proc. Amer. Peanut Res. Ed. Soc. 38:62 (Abstr.).

Subrahmanyam, P., Reddy, L., Gibbons, R., and McDonald, D. 1985. Peanut rust: a major threat to peanut production in the semiarid tropics. Plant Dis. 69:813-819.

Subrahmanyam, P., Van Wyk, P., Kisyombe, C., Cole, D., Hildebrand, G., Chiyembekeza, A., and Van der Merwe, P. 1997. Diseases of groundnut in the Southern African Development Community (SADC) region and their management. Int. J. Pest Manage. 43:261-273.

Sudini, H., Upadhyaya, H. D., Reddy, S., Mangala, U. N., Rathore, A., and Kumar, K. V. K. 2015. Resistance to late leaf spot and rust diseases in ICRISAT's mini core collection of peanut (*Arachis hypogaea* L.). Australas. Plant Pathol. 44:557-566.

USDA-NASS. 2016. Agricultural Statistics Database. https://www.nass.usda.gov.

Waliyar, F., Adamou, M., and Traoré, A. 2000. Rational use of fungicide applications to maximize peanut yield under foliar disease pressure in West Africa. Plant Dis. 84:1203-1211.

Walls, S. B., Wynne, J., and Beute, M. 1985. Resistance to late leafspot peanut of progenies selected for resistance to early leafspot. Peanut Sci. 12:17-22.

Wolf, F. A. 1914. Leaf spot and some fruit rots of peanut. Ala. Agr. Exp. Sta. Bul. 180, Alabama Polytechnic Institute Opelika.

Wolf, F. A. 1916. Further studies on peanut leaf spot. Jour. Agr. Res 19:891-902.

Woodroof, N. C. 1933. Two leaf spots of the peanut (Arachis hypogaea L.). Phytopathology 23:627-640.

Woodward, J., Brenneman, T., Kemerait, R., Culbreath, A., and Smith, N. 2010. Management of peanut diseases with reduced input fungicide programs in fields with varying levels of disease risk. Crop Protect. 29:222-229.

Woodward, J., Brenneman, T., Kemerait Jr, R., Culbreath, A., and Smith, N. 2014. On-farm evaluations of reduced input fungicide programs in peanut fields with low, moderate, or high levels of disease risk. Peanut Sci. 41:50-57.

Woodward, J. E., Brenneman, T. B., Kemerait, R. C., Smith, N. B., Culbreath, A. K., and Stevenson, K. L. 2008. Use of resistant cultivars and reduced fungicide programs to manage peanut diseases in irrigated and nonirrigated fields. Plant Dis. 92:896-902.

Wu, L., Damicone, J., Duthie, J., and Melouk, H. 1999. Effects of temperature and wetness duration on infection of peanut cultivars by *Cercospora arachidicola*. Phytopathology 89:653-659.

CHAPTER 3

DIFFERENCES BETWEEN PEANUT EARLY AND LATE LEAF SPOT EPIDEMICS IN RESPONSE TO FIELD RISK, MARKET TYPE AND FUNGICIDE PROGRAM[1]

[1]Fulmer, A.M., Kemerait, R.C., Brenneman, T.B., Scherm, H., Cantonwine, E.G., Culbreath, A.K., and Stevenson, K.L. 2017. To be submitted to *Peanut Science*.

ABSTRACT

Field trials were conducted at multiple locations in Georgia and Florida during 2011 and 2012 to determine the effect of disease risk (Peanut Rx modified to account for previous leaf spot predominance and precipitation anomalies), cultivar and fungicide on the temporal dynamics of early (ELS) and late (LLS) leaf spot of peanut, caused by *Cercospora arachidicola* and *Cercosporidium personatum*, respectively. A secondary objective was to validate the efficacy of prescription fungicide programs on two Valencia market types. Plots of peanut cultivars Georgia-06G, New Mexico Valencia C and Georgia Valencia were treated with azoxystrobin or tebuconazole-based fungicides at timings appropriate for AU-Pnut or high, moderate and low prescription programs. Epidemics of ELS and LLS were differentiated by sampling five main stems on a bi-weekly basis and counting the number of lesions of each leaf spot. Risk, market type and fungicide all had a significant effect on the development of each disease. ELS was more severe than LLS in fields with a history of ELS, generally greater on Valencia cultivars, and tended to increase with decreasing fungicide applications. Across fungicide treatments, onset of ELS epidemics ranged from 70 to 131 DAP, and was 14 to 45 days prior to the onset of LLS epidemics in fields with a history of ELS; LLS onset ranged from 109 to 131 DAP, and was equal to or just prior to ELS in fields with a history of LLS. Symptoms of both diseases first appeared on untreated Valencia type cultivars and disease onset was generally delayed with increasing fungicide inputs. The highest rate of disease progress was similar for each leaf spot and was highest on the untreated runner type, Georgia-06G. Severity of each leaf spot was significantly lower on Valencia type cultivars than Georgia-06G, but increased with decreasing fungicide inputs for all cultivars. Compared to the high risk and Au-Pnut programs, the moderate risk programs offered comparable levels of control and yields were not significantly different across risk groups.

INTRODUCTION

Early leaf spot (ELS), caused by *Cercospora arachidicola* S. Hori, and late leaf spot (LLS), caused by *Cercosporidium personatum* (Berk & M.A. Curtis) Deighton, are two of the most important foliar diseases of peanut (*Arachis hypogaea* L.) worldwide (Shokes and Culbreath, 1997). The two leaf spot diseases share many similarities in that they both have a similar ecological niche, and often coexist in the same field and on the same leaflet. While leaflets are the primary infection sites, each leaf spot pathogen can also cause severe lesions on petioles and stems, and occasionally on the pegs. In untreated situations, this can result in complete defoliation/death of the peanut plant (Shokes and Culbreath, 1997). Together or separate, each has been reported to cause yield losses as high as 70% (Backman and Crawford, 1984; McDonald et al., 1985; Shokes and Culbreath, 1997), and costs to control these diseases represents one of the highest inputs for peanut growers. In Georgia, the annual average damage plus cost of control from 2012 to 2014 was estimated at $37.9 million (Kemerait, 2012, 2013, 2014)

Although ELS and LLS are often considered one disease from a management standpoint, they are two separate diseases with a number of distinct differences from both a mycological and an epidemiological standpoint (Shokes and Culbreath, 1997). Firstly, ELS often appears 30-50 days after planting, whereas LLS normally appears 2 to 5 weeks later (Shokes and Culbreath, 1997; Smith and Littrell, 1980). However, there have been instances when ELS appears much later in the season, and LLS appears much earlier than the times previously stated (R. Kemerait, personal communication). Secondly, the relative abundance of each leaf spot tends to fluctuate within or between seasons and geographical locations (Cantonwine et al., 2008; Hemingway, 1955; Jackson, 1981; Miller, 1953; Smith and Littrell, 1980). In Georgia, ELS was prevalent in most fields from the early 1930's until a shift took place in 1976 (Littrell, 1981). In that year, LLS gained and held prevalence over ELS until 1991 (Littrell, unpublished data; Smith and Littrell, 1980). Beginning in 1992, ELS resurfaced and soon became the prevalent disease throughout the 1990s and into early 2000s. Since the early 2000s, there has been an increasing resurgence of LLS, which has escalated in the most recent years (Cantonwine et al., 2008).

The reasons for the differences in the occurrence of these two diseases is still unknown. Smith and Littrell (1980) suggested that one reason for shifts in predominance could be due altered inoculum levels due to changes in fungicide chemistries, timing of fungicide applications, crop rotation, varieties, irrigation regimes, among others. However, to the best of our knowledge, their hypotheses have not been thoroughly tested.

Chlorothalonil has been one of the primary fungicide active ingredient used for leaf spot suppression for the last 50 years, and was almost exclusively relied upon during the 1970's and 1980's (Culbreath et al., 2006). The benzimidazole fungicide benomyl was briefly used in the early 1970's, but within a few years, resistance to both leaf spot pathogens was detected (Culbreath et al., 2002). In 1994, tebuconazole and propiconazole, sterol biosynthesis inhibitor (SBI) fungicides, were labeled for peanut. Since then, an increasing number of new active ingredients within and among chemical classes (as grouped by the Fungicide Resistance Action Committee) have been labeled for peanut. These include the SBI fungicide prothioconazole, the quinone outside inhibitors azoxystrobin and pyraclostrobin, and the pyrazole carboxamide penthiopyrad (Culbreath et al., 2008; Culbreath et al., 2009; Kemerait et al., 2017b). Propiconazole is known to have more efficacy on the ELS pathogen compared with the LLS pathogen (A. Culbreath, unpublished data), and both pathogens have been reported to have populations with reduced sensitivity to tebuconazole (Stevenson and Culbreath, 2006). However, it is unknown whether any of these active ingredients would have a differential effect on the development of ELS and LLS when used in a normal spray program (for examples see Kemerait et al. (2017a)).

Historically, fungicides have primarily been applied on a 14-day calendar based schedule. However, growers have a number of options to vary the timing of these applications. Weather-based advisories were first developed in the 1960s (Jensen and Boyle, 1965; Jensen and Boyle, 1966), and since that time a number of more effective and biologically advanced models have been developed (Cu and Phipps, 1993; Damicone et al., 1994; Jacobi et al., 1995). The AU-Pnut advisory is currently recommended for growers in Alabama, Georgia, and Florida, but has not been widely accepted (Kemerait et al., 2017a; Woodward et al., 2010).

A more recent option for growers to modify the calendar-based program is through the integrated approach that includes the use of more resistant cultivars, cultural practices and extended spray intervals (Cantonwine et al., 2006; Monfort et al., 2004; Woodward et al., 2014; Woodward et al., 2008). The integration of these factors has resulted in Peanut Rx – a risk index that acts as an educational tool for peanut producers in the southeastern US to estimate the relative intensity of the most important foliar and soilborne diseases in a given field prior to planting (Kemerait et al., 2017a). For leaf spot, pre-plant risk factors include variety, planting date, rotation, field history, and whether the field is irrigated or non-irrigated. Risk points are assigned to each factor and after calculation, fields are assigned a high, moderate or low level of risk. Agrichemical companies have developed prescription fungicide programs that are recommended for each risk level. These programs feature spray intervals that are proportional to the level of risk, i.e., intervals become longer as risk decreases. High-risk fields are treated according to the standard 14-day calendar-based program (seven applications), whereas moderate and low-risk fields are sprayed on extended intervals, totaling to five and four applications, respectively (Kemerait et al., 2017a).

Peanut cultivars are classified as four distinct types: runner, Virginia, Spanish and Valencia (Stalker, 1997). In the southeastern United States, the runner market type dominates most of the acreage, although small-scale production of Valencia is grown for the fresh market (Monfort, 2016; Stalker, 1997). Valencia varieties are considered more susceptible to ELS and LLS, but there is little actual data on the development of these diseases on this market type. Furthermore, while the efficacy of prescription fungicide programs have previously been validated on runner market types (Woodward et al., 2014; Woodward et al., 2010), it is uncertain how these fungicide programs would perform on Valencia market types.

One of the key principles of disease management is a thorough understanding of the temporal dynamics of the yield-limiting diseases in question (Fry, 1982). Given the uncertainty concerning the predominance, onset and rate of ELS relative to that of LLS, it follows that the continued advancement of integrated management options depends on filling this knowledge gap. Therefore, the main objective of

these studies was to differentiate the epidemics of ELS and LLS in order to characterize the prevalence, onset and rate of each disease in response to different fungicide programs and market types in fields with varying levels of risk and differing levels of historic leaf spot predominance. A secondary objective was to further validate the efficacy of prescription fungicide programs by comparing their performance on runner and Valencia market types.

MATERIALS AND METHODS

Field sites. Field trials were conducted at the RDC Pivot and the Black Shank farm located on the Coastal Plain Experiment Station in Tifton, Georgia, and the Attapulgus Research and Education Center in Attapulgus, Georgia, during 2011 and 2012. An additional experiment was conducted at the North Florida Research and Education Center in Marianna, Florida in 2012.

Tifton (RDC Pivot). The field site was a Tifton loamy sand and was divided into four quadrants under a center pivot irrigation system. In 2010, the peanut quadrant was planted to cotton (*Gossypium hirsutum*) and prior to that had been planted to soybean (*Glycine max*) for 10 of the previous 12 years. In 2011, the field had a history of cotton, soybean, and corn (*Zea mays)* in 2009, 2010, and 2011, respectively. An adjacent quadrant of the same field was planted in 2012 and had a history of cotton, soybean, and corn in 2010, 2011, and 2012, respectively. A winter cover crop of wheat (*Triticum aestivum*) was planted each year, and killed with an application of glyphosate (Roundup 4 EC, Monsanto, Kansas City, MO) at 1.2 kg a.i./ha approximately 2 weeks prior to planting.

Tifton (Black Shank farm). All field sites have a Tifton loamy sand. The irrigated sites had a history of peanut/peanut, peanut/peanut and peanut/cotton rotation in 2010, 2011 and 2012, respectively. The same dryland field was used in all years and had a history of vegetables, corn, peanut, and peanut in 2009, 2010, 2011, and 2012, respectively.

Attapulgus. The soil-type in irrigated fields include a mixture of a Dothan and Orangeburg loamy sand and Lucy loamy sand. The dryland fields include a Faceville sandy loam and Dothan and Orangeburg loamy sand. The irrigated site in 2011 had a history of cotton, peanut, cotton in 2008, 2009 and 2010, respectively, and in 2012 had a history of soybean, soybean, cotton in 2009, 2010 and 2011,

respectively. In 2011 the dryland field had a history of cotton, peanut, and peanut in 2008, 2009, 2010, respectively, and in 2012 had a history of peanut, corn and cotton in 2009, 2010 and 2011, respectively.

Marianna. The soil-type is an Orangeburg loamy sand and the field had a history of peanut, cotton and corn in 2009, 2010 and 2011, respectively.

Field plots were prepared with conventional tillage at Attapulgus, Black Shank, Plains and Marianna. At the RDC Pivot, plots were prepared for planting using a strip-till implement (as described by Monfort et al. (2004)). All plots were 1.8 m wide and the length was 4.6 m at Marianna, 7.6 m at Attapulgus and Black Shank, and 12.2 m at Plains and the RDC Pivot. Peanuts were planted at 19.2 seeds/m in two single rows spaced 91-cm apart for all trials. All herbicides, insecticides and fertilizers were applied following recommendations of the University of Georgia Cooperative Extension Service.

Field risk. Peanut Rx (Kemerait et al., 2016) was used to calculate the risk to leaf spot in each field for each year and location. These risk levels were further modified by 1) partitioning groups based on the historical predominance of ELS or LLS at each location, and 2) adjusting risk in dryland fields based on precipitation departures from average. Briefly, for each location with a dryland field, monthly rainfall averages were compared with the 30-year average at each location (see Fig. S3.1 for rainfall data). If at least one month in the critical growing season (July, August or September) was < ½ the 30 year rainfall average, the dryland fields were reduced to the low risk category. All modifications resulted in five risk categories: ELS Low risk, ELS Mod risk, ELS High risk, LLS Mod risk and LLS High risk. Table 3.1 provides detailed risk factors and modifications for each field. For the rest of the paper, we will refer to the modified risk groups as ‘risk’.

Experimental design. For all tests conducted in 2011 and 2012, we used a factorial design (market type [2] × fungicide treatment [9]) laid out in randomized complete blocks with four or five replications. The two levels for market type included i) runner and ii) Valencia. For runner, the cultivar Georgia-06G was planted in the all trials. For Valencia, the cultivar New Mexico Valencia C was planted at Attapulgus, Black Shank and Marianna; the cultivar Georgia Valencia was planted in Tifton at the RDC Pivot. The nine levels for fungicide treatment included an azoxystrobin or tebuconazole-based

prescription program for all three risk levels as well as an AU-Pnuts advisory program. Specific details for each treatment are provided in Table 3.2.

Fungicide treatments. High-risk, moderate and low-risk fungicide programs were initiated at approximately 30, 37 and 44 days after planting (DAP), and subsequent sprays were made every 14 or 21-day intervals, depending on the program (see Table 3.2 for details). Azoxystrobin was applied for stem rot (*Sclerotium rolfsii*) control at approximately 60 and 90 DAP, whereas tebuconazole was applied on a four-block schedule with applications beginning approximately 60 DAP.

For the weather-based advisory AU-Pnut, a modification was made with regards to the first application, which was initiated at approximately 30 days after planting (DAP). Normally, the first application is based on the number of rain events or precipitation probabilities to trigger the first application. Following each application, a 10-day protection period was assumed before resuming each advisory. The first application of azoxystrobin was made when the advisory was triggered after reaching 60 DAP. If AU-Pnut triggered an application between 60-110 DAP, azoxystrobin was applied between 60-110 DAP. If either advisory triggered before 21 days post-application, an additional propiconazole plus chlorothalonil spray was applied. Following the 21 days, another application of azoxystrobin was made if the advisory triggered before 110 DAP; all sprays after 110 DAP consisted of chlorothalonil alone. Similar criteria were established for the tebuconazole stem-rot based program as well. When advisories triggered in the 60-110 DAP interval, tebuconazole was tank mixed with chlorothalonil and applied accordingly; applications following 110 DAP consisted of chlorothalonil alone (see Supplementary Table S3.3 for the actual timings (DAP) of each application).

At Attapulgus, fungicide applications were made with a Lee Spider sprayer (LeeAgra, Inc., Lubbock, TX) calibrated at 140 liters per hectare. For all other locations applications were made with a CO_2-pressurized backpack sprayer with 4 8002VS flat tip nozzles (Teejet Technologies, Springfield, IL) calibrated to apply spray volume of 188 liters per hectare.

Data collection. Beginning approximately 30 days after planting (DAP), plots were visually monitored for initial leaf spot symptoms. Upon detecting first evidence of leaf spot occurrence, a more

intensive evaluation was initiated. Five main stems per plot were arbitrarily selected for destructive sampling and returned to the lab where the number of present and missing leaflets, and the number of ELS and LLS lesions per leaflet were counted. ELS and LLS were differentiated by symptoms and signs associated with each causal pathogen. Samples were collected approximately every other week up to approximately 1 to 2 weeks prior to inverting the peanuts. In 2011 and 2012 main stems were sampled at the following days after planting (DAP), respectively: Attapulgus – 63, 73, 88, 106, 133, 144; Black Shank – 43, 65, 82, 96, 112, 126; RDC Pivot – 68, 82, 96, 110, 129, 145; Plains – 125, 141. In 2012, locations were sampled at the following DAP: Attapulgus – 70, 84, 98, 112, 126, 140; Black Shank – 61, 75, 89, 104, 117, 132; Marianna - 84, 97, 112, 125, 139; RDC Pivot – 86, 103, 117, 131, 145.

Based on the data collected, incidence of each disease for each rating date was calculated by dividing the number of leaflets with at least one lesion by the total number of leaflets counted across all five main stems. Area under the disease progress curves (AUDPC) were calculated for each leaf spot based on the number of lesions per leaf. AUDPC values were standardized (stAUDPC) by dividing AUDPC values by the duration of the epidemic (Campbell and Madden, 1990). Predominance was estimated as the proportion of the total leaf spot epidemic attributed to LLS, and was calculated by dividing the standardized AUDPC of LLS by the total standardized AUDPC of ELS and LLS. Time of disease onset (onset) was defined as the number of DAP for ELS or LLS to reach 1% incidence. Overall leaf spot severity was estimated as the total leaf spot epidemic (stAUDPC) based on the combined number of ELS and LLS lesions. Leaf spot intensity was further assessed approximately every other week with the Florida 1-10 visual rating scale, where 1 = no spots, and 10 = a completely defoliated plant (Chiteka et al., 1988). Final ratings were usually made < 1 week prior to inverting peanuts.

To model the rate of the temporal progress ELS and LLS incidence, linear regression was used to evaluate the fit of linearized forms of the exponential [log(y)], logistic [ln(y/1-y)] and Gompertz [-ln(-ln y)] models on days after planting. Recalculated R^2 values from the regression of backtransformed predicted on observed values and resulting mean square errors were then compared. Residual plots (observed – predicted values) were also examined for each model. The model with the highest R^2, lowest

mean square error and the best residual distribution was selected as the best fit for the data. If a model failed to fit the data satisfactorily, it was excluded from the analysis.

Immediately after inverting the peanuts, stem rot incidence was estimated by counting the number of 30.5-cm sections ("hits") with at least one disease locus. Incidence was calculated for each plot by dividing the number of hits by the total possible 30.5-cm sections of linear row per plot; this was expressed as a percentage by multiplying by 100. After drying to approximately 10% moisture, pods from each plot were weighed.

Statistical analysis. Data were subjected to linear mixed model of analysis of variance using PROC GLIMMIX (SAS 9.4 Institute, Cary, NC). The model was analyzed as a split-split plot design with modified risk, market type and fungicide program as fixed effects, and replication, year, and location as random effects. When testing for a three-way interaction between all fixed effects, replication was the only random effect we considered. When significant three-way interaction effects were found, market type and fungicide and market type × fungicide were tested separately by each modified risk group. Since groups were different by year and location, each group had a different random statement according to the availability of year and location. The following describes the random effects included in each risk group: 'High ELS' -- replication, year and location; 'ELS Mod' -- replication and location; ELS Low -- replication and location; 'High LLS'- replication; and 'Mod LLS' -year. The SLICE option was used to explore risk × market type interactions. When there was no significant three-way interaction and no significant interaction between market type and fungicide, the simple effect of risk was sliced by market type and vice versa. The SLICEDIFF option was used in the LS-means statement to analyze differences in the least square means for simple effects of market type for each level of risk. In all analyses the Kenward-Roger option was used to adjust the denominator degrees of freedom due to repeated measures, and differences in the least square means were tested by Tukey's multiple comparisons test.

When data violated the assumptions of normality, transformations were used. As such, total AUDPC, FL 1-10 rating scale, and percent stem rot were log transformed, proportion of lesions that were LLS (ProLLS) was arcsin-square root transformed, and the square root transformation was used for the

maximum number of ELS and LLS lesions per leaf. Back-transformed means of all transformed variables are presented in the results.

Supplementary data from 2010 and 2011. Preliminary trials were conducted in 2010, and an additional trial was also conducted at the Southwest Georgia Research and Education Center, Plains, Georgia, in 2011. These trials differed in that they included a wider range of fungicide active ingredients, were only planted to a single cultivar (the runner market type Georgia-06G) or had a different sampling method. Due to these confounding factors, the results from these trials are included only as a supplementary feature (Table S3.4). However, it should be noted that in most respects, the results from these trials reflect what was seen in the main trials 2011 and 2012.

All studies were laid out in a randomized complete block design (RCBD) with four replications. Specific details for each field and associated risk can be found in Table 3.1. Details of fungicide treatments are provided in the supplementary Tables S3.1 and S3.2. Beginning approximately 50-70 DAP, 10 leaves per plot were arbitrarily selected from lower and middle parts of the canopy for destructive sampling. Samples were returned to the lab where the number of present and missing leaflets, and the number of ELS and LLS lesions per leaf were counted. ELS and LLS were differentiated by symptoms and signs associated with each causal pathogen. Samples were collected approximately every other week up to ~1 to 2 weeks prior to inverting the peanuts. Data were subjected to linear mixed model analysis of variance using PROC GLIMMIX (SAS 9.4). Modified risk and fungicide program were included as fixed effects in the model. Replication, year, and location were considered random effects. Due to differences in fungicide active ingredients between risk groups, treatment effects were analyzed by modified risk group.

RESULTS

Predominance. For the 2011 to 2012 seasons, risk, market type and risk × market type had highly significant effects on the proportion of LLS (Table 3.3). Generally, fields with a history of LLS were predominantly LLS and vice versa (Fig. 3.1). The difference in risk groups ranged from less than 10 % LLS in the ELS High-risk fields, to greater than 90 % LLS in the LLS Mod-risk group (Table 3.4).

One exception was the LLS-High risk field, where the Valencia type was only about 40% LLS (i.e., 60% of the lesions were attributed to ELS). The effect of market type was less consistent, but in three out of the five risk groups, the Valencia type had 4 to 10 % less LLS than the runner market type (Table 3.5). Fungicide was significant at the $P = 0.05$ level (Table 3.3). As such, the proportion of LLS lesions tended to decrease with fewer fungicide applications, and was generally lowest in plots not treated with fungicide (Table 3.5). Numerically, the azoxystrobin-based program often had a higher proportion of LLS than the tebuconazole-based program, but based on contrast statements there was no difference between fungicide chemistries except for the LLS High group (not shown).

Disease onset. Risk, market type and fungicide had significant effects on the onset of ELS (Table 3.3). Risk, market type and risk × market type had significant effects on LLS onset (Table 3.3). ELS appeared prior to LLS in all fields with a history of ELS (Fig. 3.3). However, LLS onset was equal or prior to the onset of ELS in fields with a previous history of LLS. The difference between the onset of LLS and ELS was as much as 38 days between risk groups, but only 14 days between market types (Table 3.4 and Table 3.5). For both leaf spots, onset was numerically earlier on Valencia compared with the runner type cultivars, and statistically different in all risk groups except LLS High (Table 3.5). The effect of fungicide was less consistent. In fields with a history of ELS, ELS was usually first observed in untreated and low-risk prescription fungicide programs, and tended to be more delayed with increasing fungicide applications. However, with the exception of the LLS High-risk group, there was no fungicide effect on ELS onset in fields with a history of LLS (Table 3.6). The opposite was observed for LLS (Table 3.6) in that onset did not seem to be affected by fungicide in fields that had a history of ELS.

Rate of disease progress. The Gompertz model provided the best fit for the majority of disease progress curves for incidence of both ELS and LLS. Across risk groups, market types, and fungicide programs, the estimated rate parameter (r_G) of ELS incidence ranged from 0.007 to 0.065 (1/day), and the estimate rate parameter of LLS (r_G) incidence ranged from 0.001 to 0.78 (1/day). The highest and the lowest rate for both diseases for both market types occurred in the LLS High and ELS Low-risk groups, respectively (Fig. 3.3, Table 3.4). Risk, risk × market type and fungicide were significant for the rate of

ELS and LLS spot progress. Market type and the risk × fungicide interaction were also significant for the rate of LLS (Table 3.3). Apart from the LLS High-risk group, rate of ELS was greater than that of LLS in fields with a previous history of ELS, and slowest in fields with a history of LLS (Fig 3.3, Table 3.4). The rate of LLS had a similar, but reversed pattern. For both leaf spots, the rate of progress was almost always greater on runner than on Valencia cultivars (Table 3.4). Fungicide applications reduced the rate of ELS and LLS significantly in fields where each disease had a prior history. Numerically, the rate of the predominant leaf spot tended to increase with decreasing fungicide applications, but there was no consistent treatment difference between fungicide chemistries or programs across risk groups.

Leaf spot severity. Risk, market type, risk × market type, fungicide and risk × fungicide all had significant effects on final severity based on the Florida 1 to 10 scale (Table 3.3). Final severity was similar in ELS fields to that of LLS fields, and the two Valencia market types were less diseased than the runner type, Georgia-06G (Fig. 3.2). Lack of leaf spot pressure in the ELS Low-risk group resulted in no difference between the market types. There was a risk × market type × fungicide interaction for severity based upon total standardized AUDPC (Table 3.3). However, when evaluated by risk group, differences between treatments followed a similar trend to that of severity measured by the Florida 1 to 10 scale (Table 3.6). In both cases, for both ELS and LLS risk groups, severity tended to increase with decreasing fungicide inputs regardless of fungicide and market type, but moderate risk programs were not significantly different from the high-risk or AU-Pnut program (Table 3.6).

Stem rot severity. All factors and their interactions were statistically significant for stem rot (Table 3.3). In most cases, Valencia had numerically higher levels of stem rot than the runner type (Table 3.6). Except in the ELS High risk group, there was no significant treatment effect compared with the control (Table 3.6).

Yield. Risk, market type, risk × market type, fungicide had a significant effect on pod yield. In all cases, yield of the runner cultivar was significantly higher than that of two Valencia types (Table 3.6). The fungicide effect was inconsistent in relation to field risk (Table 3.6). In ELS High-risk group, the highest numerical yields were not always observed in the plots with the highest number of fungicide applications

(Table 3.6). However, for each risk group and market type, there were no significant differences between high-risk, moderate-risk or AU-Pnut programs (Table 3.6).

DISCUSSION

Results from these studies illustrate the wide range of differences and similarities that can occur between the development of ELS and LLS of peanut. However, we found that much of the variability related to predominance, onset and disease progress rate between these two diseases was explained by a modified risk system that accounts for the historical predominance of each leaf spot. This was supported by the observation that leaf spots in fields with a history of ELS were predominantly ELS, and leaf spots in fields with a history of LLS were predominantly LLS (Fig. 3.1). Additionally, these same fields have been continually monitored up to 2016 and the predominance of ELS or LLS at each location has, with few exceptions, remained the same (Fulmer, unpublished data). To emphasize this point, we draw attention to two of the fields in these studies (Black Shank and RDC Pivot) located in Tifton, Georgia. These fields are within 2 km of each other, and for the last 6 years Black Shank has remained predominantly ELS, and RDC Pivot has remained predominantly LLS (A. Fulmer, unpublished data).

These data agree with previous suggestions that initial inoculum is most often derived from local sources (Porter et al., 1982). Both leaf spot fungi overwinter in infested peanut residues (Shokes and Culbreath, 1997), and survival decreases with increasing years of rotation away from peanut (Kucharek, 1975). Local inoculum can also be perpetuated on volunteer peanut plants (Shokes and Culbreath, 1997), but efforts to characterize the differences in the survivability of these two fungi have not been successful (Nuesry, 1981). In our trials, most of the fields had at least 1 year of rotation away from peanut, and in many cases at least 2 years away from peanut (Table 3.1), but were generally within 30 to 150 m of the previous year's field study. Though not noted for every single field trial, volunteer peanuts with leaf spot lesions were observed in the field of the previous year's field trial. For example, at the RDC Pivot (LLS Mod), volunteer peanuts with heavy LLS disease levels were found in the previous year's field trial just prior to the onset of LLS in the 2012 season (personal observation). Clearly, more research is needed to better understand the survival and dispersal of each of these fungi.

Previous studies have established that peanut genotypes may have differing levels of resistance to each leaf spot pathogen (Abdou et al., 1974; Walls et al., 1985). Similar reports have been found in other pathosystems that feature the coexistence of closely related species. For example, in the sigatoka complex of banana, *Mycosphaerella fijiensis,* the cause of black sigatoka, is highly virulent on all of the cultivars that are resistant to *Mycosphaerella musicola,* the cause of yellow sigatoka (Ploetz et al., 2003). In our studies, the cultivar New Mexico Valencia C was planted at all locations except at LLS Mod-risk group which was planted to the cultivar Georgia Valencia. The New Mexico Valencia type had less LLS and earlier disease onset of ELS compared with the runner type. Subsequent studies (by the authors) provided field evidence that suggests that New Mexico Valencia C is highly susceptible to ELS and only moderately susceptible to LLS (Chapter 6). In contrast, the Georgia Valencia cultivar appears to be highly susceptible to both leaf spot pathogens (Chapters 6 and 7). We hypothesize that this could partially explain why there was more ELS in the LLS High-risk field than at the LLS Mod-risk field.

The greatest differences in the epidemiological behavior of ELS and LLS were due to risk and market type; however, these data also suggest there is a direct and/or indirect competitive advantage for each leaf spot pathogen depending on the fungicide program. Because the onset of ELS was 20 to 40 days prior to that of LLS, severe epidemics of ELS would have been able to cause heavy defoliation in the lower canopy and a greater abundance of secondary inoculum prior to the initiation of LLS. Because the onset of ELS was delayed with increasing fungicide applications, we therefore propose that more available tissue would have been available for the LLS pathogen to infect upon its initiation.

Differences in fungicide sensitivities were reported to shift the proportion of closely related fungi *Leptosphaeria maculans* and *L. biglobosa* that cause phoma stem canker, a disease complex on oilseed rape (Huang et al., 2011). In this example, an application of a triazole plus a benzamidazole increased the frequency of *L. maculans* compared with the untreated check, but the same treatment decreased the frequency of *L. biglobosa*. Although not significantly different for all risk groups, our results suggest that the LLS pathogen was more prevalent in the azoxystrobin program versus the tebuconazole program. One explanation for this response is that the azoxystrobin program included multiple applications of

propiconazole, which is thought to have less activity on the LLS pathogen (Culbreath, unpublished data). Otherwise, the efficacy of the azoxystrobin and tebuconazole programs appeared similar for both leaf spots across risk groups, suggesting that there was not a differential level of fungicide sensitivity between leaf spot pathogen populations. It should be noted, however, that our studies were conducted prior to more recent concerns of resistance to strobilurin fungicides for the LLS pathogen (Elwakil and Dufault, 2015). Therefore, the results presented here may not reflect the current status of fungicide sensitivities among ELS and LLS pathogen populations.

The effect of moisture on the development of ELS and LLS has been the focus of much research (Alderman and Beute, 1986, 1987; Alderman and Nutter, 1994; Alderman et al., 1989; Davis et al., 1993; Jacobi et al., 1995; Phipps et al., 1997). By default, Peanut Rx does not account for precipitation other than by designating fields as either irrigated or non-irrigated (Table 3.1). Based upon the revised criteria in our study, precipitation anomalies in 2010 and 2011 in the two dryland fields with a prior history of ELS resulted in a modification of high risk to low risk (Table 3.1). Predictably, the general development and final severity of both leaf spots were reduced (Fig. 3.2). Interestingly, however, the predominance of LLS was higher in this risk group compared with the other ELS risk groups with a previous history of ELS. One possible explanation is that although the onset of ELS was still fairly early in this risk group (70 to 80 DAP), severe drought occurred for almost the entire month of August (~70 to 100 DAP), thus delaying further development of ELS. We hypothesize that this provided more available tissue for LLS toward the end of the season.

Differences in disease onset of both leaf spots were mostly associated with risk level, but were also affected by market type and fungicide. These results demonstrate that onset of ELS can vary widely (70 to 131 DAP), and can even be later than LLS, such as in the case of the LLS Mod (Fig. 3.2). In contrast, the range of LLS onset (109 to 131 DAP) was much more limited (Fig. 3.2. and Table 3.4). Subsequent studies demonstrated that both ELS and LLS onset can have a much wider range (~30 to 140 DAP for both diseases), but is dependent on inoculum availability, planting date and genotype (Chapters 4, 5 and 6). In these trials, the effect of market type was more consistent than that of fungicide. In most

cases, onset of both diseases on Valencia types preceded that of the runner types by at least 10 days, and was delayed with increasing fungicide inputs. However, the effect of both market type and fungicide was greatest on the predominant leaf spot of the given risk group.

Similarly, rate of disease progress of each disease was generally highest in fields with a previous history of the same disease (Fig. 3.2). Reports have generally suggested that the rate of disease progress is higher for LLS than ELS (Backman and Crawford, 1984; Hemingway, 1955). In studies reported here, the fastest rate of ELS (0.066 day^{-1}) was slightly lower than that of LLS (0.078 day^{-1}) (Table 3.4). Interestingly, the highest rate of ELS was observed in the High-risk LLS field (Table 3.4). One explanation for this was the result of planting the Valencia market type New Mexico Valencia C, which had higher levels of ELS in the High-risk LLS field (Fig. 3.1). This may have created more secondary inoculum of the ELS pathogen than normal. In terms of comparing ELS to LLS, it should be noted that these epidemics were modeled based on disease incidence and not on the number of lesions per leaf. In our studies, we found that the maximum possible number of lesions in the highest risk situation for each leaf spot, was highest for LLS (Supplementary Table S3.5). Therefore, it is possible that the development of a model that could account for lesion counts would provide different results than what we report here.

Results from these trials offer further validation of the efficacy of prescription fungicide programs. Similar to previous trials, the severity of leaf spot tended to increase with decreasing fungicide applications, particularly in high risk fields (Woodward et al., 2010). But in moderate and low-risk fields, this did not result in a yield loss between moderate and high-risk fungicide programs. The direct effect of ELS and LLS on yield loss is assumed to be similar (Backman and Crawford, 1984). Although we observed less yield loss in LLS fields compared with ELS fields, no direct comparisons can be made due to confounding factors such as differing levels of stem rot (*Sclerotium rolfsii*) severity between risk groups (Table 3.7). Future research is needed that addresses the yield loss relationship to each leaf spot in order to better increase our understanding of risk.

More importantly for Peanut Rx, we found that the efficacy of prescription programs was as high, or higher, for Valencia market types compared with the runner type. Interestingly, although Valencia

market types are considered more susceptible, a shorter maturity often led to disease escape. In these studies, onset of disease was earlier on Valencia types (Table 3.4), but harvest occurred 20 to 30 days prior to that of the runner varieties. Furthermore, only six fungicide applications were made in the high risk programs due to the early harvest. The evidence suggests that fewer applications are possible for Valencia market types.

In conclusion, few studies have attempted to characterize the epidemics of ELS and LLS (Gorbet et al., 1982; Jackson, 1981; Shokes et al., 1983), and these were generally limited to comparing percent frequency of lesion counts. By differentiating the epidemics of these coexisting diseases, we have been able to quantify aspects of their temporal dynamics, and determine some of the factors responsible for their differences and similarities. These studies confirm that fields were primarily ELS or LLS, and that this was linked, in every case, to the historical prevalence of these diseases at each location. Furthermore, we found that predominance was influenced through the additive effect of genotype and fungicide program. The cultivar New Mexico Valencia C, in addition to fewer fungicide applications, tended to result in a more ELS than LLS. Historical disease prevalence was also associated with the onset of ELS and LLS. In these studies, ELS onset was prior to LLS onset except in situations where LLS was historically predominant. Knowledge of field history and other factors such as early-maturating cultivars in relation to disease onset are potentially new factors to be considered for risk systems such as Peanut Rx. Furthermore, the consistently delayed onset of LLS across risk groups suggests that application timings could continue to be refined for fields that are predominantly LLS.

ACKNOWLEDGMENTS

We express our appreciation to the personnel at the University of Georgia and University of Florida research stations for all of the assistance provided. Particularly, we thank Billy Mills, Lee Hitson, John Paulk, Hunt Sanders, Mark Gomillion, Mike Heath, Stan Jones, Joy Ouano, Cruz Olalde, Lina Flor Young, Jeannette Mixon, Jhen Bennett and Jared Walls for technical support; and Xuelin Luo for statistical assistance. Weather data was kindly provided by University of Georgia Automated Environmental Monitoring Network.

LITERATURE CITED

Abdou, Y. A.-M., Gregory, W. C., and Cooper, W. E. 1974. Sources and nature of resistance to *Cercospora arachidicola* Hori and *Cercosporidium personatum* (Beck & Curtis) Deighton in *Arachis* species. Peanut Sci. 1:6-11.

Alderman, S., and Beute, M. 1986. Influence of temperature and moisture on germination and germ tube elongation of *Cercospora arachidicola*. Phytopathology 76:715-719.

Alderman, S., and Beute, M. 1987. Influence of temperature, lesion water potential, and cyclic wet-dry periods on sporulation *Cercospora arachidicola* on peanut. Phytopathology 77:960-963.

Alderman, S., and Nutter, F. 1994. Effect of temperature and relative humidity on development of *Cercosporidium personatum* on peanut in Georgia. Plant Dis. 78:690-694.

Alderman, S., Nutter Jr, F., and Labrinos, J. 1989. Spatial and temporal analysis of spread of late leaf spot of peanut. Phytopathology 79:837-844.

Backman, P., and Crawford, M. 1984. Relationship between yield loss and severity of early and late leafspot diseases of peanut. Phytopathology 74:1101-1103.

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

Cantonwine, E., Culbreath, A., Holbrook, C., and Gorbet, D. 2008. Disease progress of early leaf spot and components of resistance to *Cercospora arachidicola* and *Cercosporidium personatum* in runner-type peanut cultivars. Peanut Sci. 35:1-10.

Cantonwine, E. G., Culbreath, A. K., Stevenson, K. L., Kemerait, R. C., Brenneman, T. B., Smith, N. B., and Mullinix, B. G. 2006. Integrated disease management of leaf spot and spotted wilt of peanut. Plant Dis. 90:493-500.

Chiteka, Z., Gorbet, D., Shokes, F., Kucharek, T., and Knauft, D. 1988. Components of resistance to late leafspot in peanut. I. Levels and variability-implications for selection. Peanut Sci. 15:25-30.

Cu, R., and Phipps, P. 1993. Development of a pathogen growth response model for the Virginia peanut leaf spot advisory program. Phytopathology 83:195-201.

Culbreath, A., Stevenson, K., and Brenneman, T. 2002. Management of late leaf spot of peanut with benomyl and chlorothalonil: A study in preserving fungicide utility. Plant Dis. 86:349-355.

Culbreath, A., Kemerait Jr, R., and Brenneman, T. 2006. Management of early leaf spot of peanut as affected by fungicide and date of spray program initiation. Plant Health Progress doi:10.1094/PHP-2006-0214-01-RS.

Culbreath, A., Kemerait Jr, R., and Brenneman, T. 2008. Management of leaf spot diseases of peanut with prothioconazole applied alone or in combination with tebuconazole or trifloxystrobin. Peanut Sci. 35:149-158.

Culbreath, A. K., Brenneman, T. B., Kemerait, R. C., and Hammes, G. G. 2009. Effect of the new pyrazole carboxamide fungicide penthiopyrad on late leaf spot and stem rot of peanut. Pest Manage. Sci. 65:66-73.

Damicone, J., Jackson, K., Sholar, J., and Gregory, M. 1994. Evaluation of a weather-based spray advisory for management of early leaf spot of peanut in Oklahoma. Peanut Sci. 21:115-121.

Davis, D., Jacobi, J., and Backman, P. 1993. Twenty-four-hour rainfall, a simple environmental variable for predicting peanut leaf spot epidemics. Plant Dis. 77:722-725.

Elwakil, W., and Dufault, N. 2015. Preliminary examination of the potential risk for Qol fungicide resistance in *Cercosporidium personatum* the late leaf spot pathogen of peanut. Proc. Amer. Peanut Res. Ed. Soc. 38:62 (Abstr.).

Fry, W. E. 1982. Principles of Plant Disease Management. Academic Press, Orlando.

Gorbet, D., Shokes, F., and Jackson, L. 1982. Control of peanut leafspot with a combination of resistance and fungicide treatment. Peanut Sci. 9:87-90.

Hemingway, J. 1955. The prevalence of two species of Cercospora on groundnuts. Trans. Brit. Mycol. Soc. 38:243-246.

Huang, Y.-J., Hood, J., Eckert, M., Stonard, J., Cools, H., King, G. J., Rossall, S., Ashworth, M., and Fitt, B. D. 2011. Effects of fungicide on growth of *Leptosphaeria maculans* and *L. biglobosa* in

relation to development of phoma stem canker on oilseed rape (*Brassica napus*). Plant Pathol. 60:607-620.

Jackson, L. 1981. Distribution and severity of peanut leafspot in Florida. Phytopathology 71:324-328.

Jacobi, J., Backman, P., Davis, D., and Brannen, P. 1995. AU-Pnuts advisory I: Development of a rule-based system for scheduling peanut leaf spot fungicide applications. Plant Dis. 79:666-671.

Jensen, R., and Boyle, L. 1965. The effect of temperature, relative humidity and precipitation on peanut leafspot. Plant Dis. Rep 49:975-978.

Jensen, R. E., and Boyle, L. W. 1966. A technique for forecasting leafspot on peanuts. Plant Disease Reporter 50:810-814.

Kemerait, R. C., Jr. 2012. Georgia Plant Disease Loss Estimates. Coop. Ext. Ser. Bull. 102-5, University of Georgia, Athens.

Kemerait, R. C., Jr. 2013. Georgia Plant Disease Loss Estimates. Coop. Ext. Ser. Bull. 102-6, University of Georgia, Athens.

Kemerait, R. C., Jr. 2014. Georgia Plant Disease Loss Estimates. Coop. Ext. Ser. Bull. 102-7, University of Georgia, Athens.

Kemerait, R. C., Jr., Brenneman, T. B., and Culbreath, A. K. 2017a. Peanut disease update. Pages 23-62 in: 2017 peanut update. W. Monfort, ed. Coop. Ext. Ser. College of Agric. Environ. Sci. University of Georgia., Athens.

Kemerait, R. C., Jr., Brenneman, T. B., and Culbreath, A. K. 2017b. Peanut disease control. Pages 201-205 in: *2017 Georgia Pest Management Handbook*. D. Horton, ed. Special Bull. 28, Univeristy of Georgia Coop. Ext. College of Agric. Environ. Sci. University of Georgia., Athens.

Kucharek, T. 1975. Reduction of Cercospora leafspots of peanut with crop rotation. Plant Disease Reporter 59:822-823.

Littrell, R. H. 1981. Late leaf spot needs more research emphasis. Page 15a in: Southeastern Peanut Farmer.

McDonald, D., Subrahmanyam, P., Gibbons, R., and Smith, D. 1985. Early and Late Leaf Spots of Groundnut. Information Bull. No. 21. ICRISAT, Patancheru.

Miller, L. I. 1953. Studies of the parasitism of *Cercospora arachidicola* Hori and *Cercospora personata* (Berk. & M.A. Curtis). Ph.D. Dissertation. University of Minnesota, Minneapolis.

Monfort, W. 2016. Peanut cultivar options for 2016. Pages 8-9 in: *2016 peanut update*. W. Monfort, ed. Coop. Ext. Ser. College of Agric. Environ. Sci. University of Georgia., Athens.

Monfort, W., Culbreath, A., Stevenson, K., Brenneman, T., Gorbet, D., and Phatak, S. 2004. Effects of reduced tillage, resistant cultivars, and reduced fungicide inputs on progress of early leaf spot of peanut *(Arachis hypogaea*). Plant Dis. 88:858-864.

Nuesry, S. M. 1981. Survival of *Cercospora arachidicola*, *Cercosporidium personatum*, and primary infection of peanuts in Oklahoma. M.S. thesis. Oklahoma State University, Stillwater.

Phipps, P. M., Deck, S. H., and Walker, D. R. 1997. Weather-based crop and disease advisories for peanuts in Virginia. Plant Dis. 81:236-244.

Ploetz, R., Thomas, J., and Slabaugh, W. 2003. Diseases of banana and plantain. Pages 73-134 in: Diseases of Tropical Fruit Crops. R. Ploetz, ed. CABI Publishing, Wallingford, UK.

Porter, D. M., Smith, D. H., and Rodriguez-Kabana, R. 1982. Peanut plant diseases. Pages 326-410 in: Peanut Science and Technology. H. Pattee and C. Young, eds. American Peanut Research and Education Society, Yoakum, TX.

Shokes, F., and Culbreath, A. K. 1997. Early and late leaf spots. Pages 17-20 in: Compendium of Peanut Diseases, 2nd Ed. N. Kokalis-Burelle, D. Porter, R. Rodriguez-Kabana, D. Smith and P. Subrahmanyam, eds. American Phytopathological Society Press, St. Paul, MN.

Shokes, F., Gorbet, D. W., and Jackson, L. 1983. Control of early and late leafspot on two peanut cultivars. Peanut Sci. 10:17-21.

Smith, D. H., and Littrell, R. H. 1980. Management of peanut foliar diseases with fungicides. Plant Dis. 64:356-361.

Stalker, H. T. 1997. Peanut (*Arachis hypogaea* L.). Field Crops Res. 53:205-217.

Stevenson, K. L., and Culbreath, A. 2006. Evidence for reduced sensitivity to tebuconazole in leaf spot pathogens. Proc. Amer. Peanut Res. Ed. Soc. 38:62 (Abstr.).

Walls, S. B., Wynne, J., and Beute, M. 1985. Resistance to late leafspot peanut of progenies selected for resistance to early leafspot. Peanut Sci. 12:17-22.

Woodward, J., Brenneman, T., Kemerait, R., Culbreath, A., and Smith, N. 2010. Management of peanut diseases with reduced input fungicide programs in fields with varying levels of disease risk. Crop Protect. 29:222-229.

Woodward, J., Brenneman, T., Kemerait Jr, R., Culbreath, A., and Smith, N. 2014. On-farm evaluations of reduced input fungicide programs in peanut fields with low, moderate, or high levels of disease risk. Peanut Sci. 41:50-57.

Woodward, J. E., Brenneman, T. B., Kemerait, R. C., Smith, N. B., Culbreath, A. K., and Stevenson, K. L. 2008. Use of resistant cultivars and reduced fungicide programs to manage peanut diseases in irrigated and nonirrigated fields. Plant Dis. 92:896-902.

Table 3.1. Factors used to calculate the level of risk to early (ELS) and late leaf spot (LLS) in field trials planted to runner and Valencia peanut market types in 2010, 2011 and 2012.

	Peanut Rx risk factors							Modified risk		
Year, location[u]	Planting date	Tillage	Rotation[v]	History[w]	Irrigation	Total	Risk[x]	Leaf spot history[y]	Monthly rainfall[z] < ½ 30 yr avg	Final grouping
2010										
BsfIrr	20 May	Conv	0	Yes	Yes	80	High	ELS	-	High ELS
BsfDry	20 May	Conv	3	No	No	50	Mod	ELS	Yes	Low ELS
AttIrr	17 May	Conv	0	Yes	No	70	High	ELS	-	High ELS
AttDry	17 May	Conv	1	Yes	Yes	70	High	ELS	Yes	Low ELS
RDC	26 May	Strip	3+	No	Yes	50	Mod	LLS	-	Mod LLS
2011										
BsfIrr	25 May	Conv	0	Yes	Yes	85	High	ELS	-	High ELS
BsfDry	25 May	Conv	0	Yes	No	75	High	ELS	Yes	Low ELS
AttIrr	23 May	Conv	1	Yes	Yes	75	High	ELS	-	High ELS
AttDry	23 May	Conv	0	Yes	No	75	High	ELS	Yes	Low ELS
Plains	18 May	Conv	3+	No	Yes	55	Mod	LLS	-	Mod LLS
RDC	20 May	Strip	3+	No	Yes	45	Mod	LLS	-	Mod LLS
2012										
BsfIrr	23 May	Conv	1	Yes	Yes	75	High	ELS	-	High ELS
BsfDry	23 May	Conv	0	Yes	No	75	High	ELS	No	High ELS
MAR	17 May	Conv	2	No	Yes	70	High	LLS	-	High LLS
AttIrr	9 May	Conv	3	No	Yes	60	Mod	ELS	-	Mod ELS
AttDry	9 May	Conv	2	No	No	55	Mod	ELS	No	Mod ELS
RDC	2 May	Strip	3+	No	Yes	50	Mod	LLS	-	Mod LLS

[u] Locations are abbreviated as follows: BsfIrr and BsfDry (irrigated and dryland fields at Black Shank farm, Tifton GA, respectively); AttIrr and AttDry (irrigated and dryland fields in Attapulgus, GA, respectively); RDC (RDC Pivot, Tifton GA); Plains (Plains, GA); MAR (Marianna, FL).

[v] Number of years rotated away from peanut.

[w] This is a more subjective factor and is based on whether a grower recalls if a major leaf spot epidemic had occurred in recent years.

[x] Risk categories are based upon the total points accumulated from Peanut Rx factors (Kemerait et al., 2017a) where high risk = 65 to 100 points, moderate risk = 40 to 60 points and low risk = 10 to 35 points. These scores were calculated based on the points given to the runner cultivar, Georgia-06G, which is a 20. Valencia market types generally have a greater level of susceptibility, but risk points have yet to be assigned.

[y] Each location generally has a history of being predominantly early or late leaf spot; this information was obtained from records kept by research personnel.

[z] Field risk was adjusted based on precipitation anomalies. If the monthly rainfall during July, August or September for a given location was 50% or less of the 30-year average, fields were reduced to low risk. Monthly rainfall records were obtained from the Georgia Automated Environmental Monitoring Network provided by the University of Georgia (www.Georgiaweather.net).

Table 3.2. Prescription and weather-based fungicide programs against peanut leaf spots used in 2011 and 2012.

Program[v]	# Applications[w]	Timing[x]	Fungicide	Formulation	Rate (kg a.i./ha)
AU-Pnut	6 (6.5)[z]	1, 2, 4	Propiconazole + Chlorothalonil	Tilt Bravo™	0.06 0.84
		3, 5	Azoxystrobin	Abound®	0.33
		6, 7	Chlorothalonil	Bravo Weather Stik®	1.26
High	7	1, 2, 4	Propiconazole + Chlorothalonil	Tilt Bravo™	0.06 0.84
		3, 5	Azoxystrobin	Abound®	0.33
		6, 7	Chlorothalonil	Bravo Weather Stik®	1.26
Mod	6	1.5	Propiconazole + Chlorothalonil	Tilt Bravo™	0.09 1.26
		3, 5	Azoxystrobin	Abound®	0.33
		4	Propiconazole + Chlorothalonil	Tilt Bravo™	0.06 0.84
		6.5	Chlorothalonil	Bravo Weather Stik®	1.26
Low	4	2	Propiconazole + Chlorothalonil	Tilt Bravo™	0.06 1.26
		3.5, 5	Azoxystrobin + Chlorothalonil	Abound® Bravo Weather Stik®	0.22 0.84
		6.5	Chlorothalonil	Bravo Weather Stik®	1.26
AU-Pnut	6 (6.5)	1, 2, 7	Chlorothalonil	Echo® 720 AG	1.26
		3, 4, 5, 6	Tebuconazole + Chlorothalonil	Muscle® ADV Echo® 720 AG	0.23 0.84
High	7	1, 2, 7	Chlorothalonil	Echo® 720 AG	1.26
		3, 4, 5, 6	Tebuconazole + Chlorothalonil	Muscle® ADV Echo® 720 AG	0.23 0.84
Mod	6	1.5, 6.5	Chlorothalonil	Echo® 720 AG	1.26
		3, 4, 5	Tebuconazole + Chlorothalonil	Muscle® ADV Echo® 720 AG	0.23 0.84
Low	4	2, 6.5	Chlorothalonil	Echo® 720 AG	1.26
		3.5, 5	Tebuconazole + Chlorothalonil	Muscle® ADV Echo® 720 AG	0.23 1.26
Untreated		-	-	-	-

[v] Prescription fungicide programs based on Peanut Rx are labeled high, mod (moderate) and low. AU-Pnut is the weather-based advisory used in these studies.

[w] Total number of applications made each season for each fungicide program.
[x] Number 1 represents 30 days after planting; subsequent whole numbers stand for 14 day intervals. Applications began at 30, 30, 37, and 44 days after planting for the AU-Pnut, high, moderate and low programs, respectively. Fungicides listed at each timing for AU-Pnut programs were made only if triggered by weather conditions.

Table 3.3. *P*-values from the linear mixed model analysis of variance for all leaf spot related variables, stem rot and yield during 2011 and 2012.

	Predominance[s]	Onset[t]		Rate[u]					
Source of variation	ProLLS	ELS	LLS	ELS	LLS	stAUDPC[v]	FL 1-10[w]	Stem rot[x]	Yield[y]
Risk group (R)[z]	<0.0001	<0.0001	<0.0001	<0.0001	<0.0001	<0.0001	<0.0001	<0.0001	<0.0001
Market type (M)	0.0003	<0.0001	<0.0001	0.2130	<0.0001	<0.0001	<0.0001	<0.0001	<0.0001
R × M	<0.0001	0.7562	<0.0001	0.0009	<0.0001	<0.0001	<0.0001	<0.0001	<0.0001
Fungicide (F)	0.0328	0.0440	0.1917	<0.0001	<0.0001	<0.0001	<0.0001	<0.0001	<0.0001
R × F	0.2957	0.9996	0.9740	0.1287	0.0001	<0.0001	0.0255	0.0178	0.3920
M × F	0.7043	0.9850	0.9502	0.9970	0.3158	0.0611	0.4721	0.0269	0.0964
R × M × F	0.8871	0.9998	0.9995	0.9912	0.6123	0.0001	0.8092	0.0449	0.8016

[s]Proportion of the total leaf spot epidemic attributed to late leaf spot. Calculated by dividing the standardized AUDPC of late leaf spot by the total standardized AUDPC of early and late leaf spot combined.

[t]Number of days after planting to reach 1 % disease incidence for early leaf spot (ELS) and late leaf spot (LLS).

[u]Rate of disease progress estimated with the Gompertz model for both early and late leaf spot incidence over time.

[v]Combined standardized AUDPC of early and late leaf spot.

[w]Florida 1 to 10 visual rating scale, where 1 = no disease and 10 = a dead plant.

[x]Percent number of 30.5-cm sections per linear row with at least one disease locus caused by *Sclerotium rolfsii*.

[y]Pod yield measured in kg/ha and adjusted to 10% moisture.

[z]Refers to the five modified risk groups: ELS High, ELS Mod, ELS Low, LLS High, LLS Mod as defined in Table 3.1.

Table 3.4. Effect of risk group and peanut market type on the predominance, onset and rate of early and late leaf spot in 2011 and 2012.

		Predominance[t]	Disease onset[u]			Rate of disease progress[v]		
Market type	Risk group	ProLLS	ELS	LLS	Diff[w]	ELS	LLS	Diff[x]
Runner	ELS High	0.10 c[y] *[z]	82 c *	121 b *	38	0.046 b	0.024 c *	-0.022
	ELS Mod	0.05 d *	105 b *	129 a *	24	0.061 a *	0.019 c *	-0.042
	ELS Low	0.25 b	89 c *	131 a *	45	0.012 c	0.001 d	-0.011
	LLS High	0.74 a *	108 b	111 c	3	0.065 a	0.078 a *	0.012
	LLS Mod	0.80 a	131 a *	129 a *	-3	0.013 c	0.059 b *	0.046
Valencia	ELS High	0.01 c	70 c	109 a	38	0.042 b	0.015 c	-0.028
	ELS Mod	0.01 c	96 b	110 a	14	0.045 b	0.005 d	-0.041
	ELS Low	0.32 b	78 c	106 a	26	0.012 c	0.002 d	-0.010
	LLS High	0.39 b	101 ab	107 a	6	0.066 a	0.060 a	-0.006
	LLS Mod	0.84 a	112 a	109 a	-3	0.007 c	0.038 b	0.031

[t] Proportion of the total leaf spot epidemic attributed to late leaf spot. Calculated by dividing the standardized AUDPC of late leaf spot by the total standardized AUDPC of early and late leaf spot.

[u] Number of days after planting to reach 1 % disease incidence for early leaf spot (ELS) and late leaf spot (LLS).

[v] Rate (1/day) of disease progress estimated with the Gompertz model for both early and late leaf spot incidence over time.

[w] Difference in days between the onset of late leaf spot relative to that of early leaf spot.

[x] Difference between the rate of late leaf spot by that of early leaf spot.

[y] Means within columns with the same letters are not significantly different based upon Tukey's honestly significant difference test ($\alpha = 0.05$).

[z] Asterisks indicate that the runner cultivar was significantly different from the Valencia cultivars in the corresponding risk group ($\alpha = 0.05$).

Table 3.5. Effect of fungicide program on the development of early and late leaf spot by risk group and across peanut market types in 2011 and 2012.

	Fungicide[t]		Predominance[u]	Disease onset[v]			Rate of disease progress[w]		
Risk group	Product	Program	ProLLS	ELS	LLS	Diff[x]	ELS	LLS	Diff[y]
ELS High	Axy	AuPnut	0.04 a[z]	89 a	118 a	30	0.038 b	0.019 a	-0.018
		High	0.02 a	87 ab	120 a	33	0.037 b	0.016 a	-0.021
		Mod	0.03 a	83 ab	117 a	35	0.042 b	0.020 a	-0.022
		Low	0.01 a	81 ab	119 a	40	0.043 b	0.017 a	-0.026
	Teb	AuPnut	0.02 a	82 ab	117 a	37	0.040 b	0.020 a	-0.020
		High	0.02 a	81 ab	120 a	39	0.038 b	0.018 a	-0.021
		Mod	0.03 a	83 ab	116 a	34	0.049 b	0.022 a	-0.028
		Low	0.01 a	75 b	117 a	44	0.043 b	0.020 a	-0.024
	-	Untreated	0.00 a	80 ab	118 a	39	0.067 a	0.021 a	-0.046
ELS Mod	Axy	AuPnut	0.16 a	76 a	111 a	33	0.041 bc	0.013 a	-0.029
		High	0.12 a	74 ab	112 a	35	0.035 c	0.009 a	-0.026
		Mod	0.10 a	70 ab	110 a	35	0.045 bc	0.012 a	-0.033
		Low	0.08 a	65 ab	109 a	42	0.058 ab	0.012 a	-0.047
	Teb	AuPnut	0.14 a	72 ab	104 a	30	0.057 b	0.016 a	-0.042
		High	0.08 a	69 ab	109 a	34	0.050 bc	0.009 a	-0.041
		Mod	0.08 a	69 ab	109 a	38	0.054 bc	0.011 a	-0.044
		Low	0.07 a	62 ab	109 a	42	0.060 ab	0.011 a	-0.049
	-	Untreated	0.07 a	58 b	109 a	47	0.078 a	0.012 a	-0.066
ELS Low	Axy	AuPnut	0.57 a	100 ab	122 a	23	0.011 a	0.002 a	-0.009
		High	0.56 a	102 ab	120 a	14	0.009 a	0.001 a	-0.008
		Mod	0.40 a	100 ab	117 a	17	0.010 a	0.000 a	-0.010
		Low	0.38 a	96 ab	118 a	32	0.012 a	0.002 a	-0.010
	Teb	AuPnut	0.43 a	105 ab	119 a	21	0.012 a	0.002 a	-0.010
		High	0.49 a	96 ab	121 a	26	0.008 a	0.001 a	-0.007
		Mod	0.42 a	106 a	122 a	20	0.013 a	0.000 a	-0.012
		Low	0.53 a	87 b	121 a	35	0.011 a	0.001 a	-0.009
	-	Untreated	0.37 a	88 b	116 a	30	0.024 a	0.006 a	-0.018
LLS High	Axy	AuPnut	0.69 a	95 a	110 a	14	0.058 b	0.062 a	0.004
		High	0.68 a	93 a	108 a	14	0.055 b	0.063 a	0.008

		Mod	0.71 a	89 a	104 a	15	0.061 b	0.067 a	0.006
		Low	0.59 ab	80 a	108 a	27	0.059 b	0.070 a	0.011
	Teb	AuPnut	0.55 ab	96 a	110 a	13	0.067 ab	0.070 a	0.003
		High	0.63 a	91 a	104 a	12	0.060 b	0.068 a	0.008
		Mod	0.45 ab	89 a	108 a	18	0.069 ab	0.067 a	-0.003
		Low	0.62 a	87 a	104 a	16	0.073 ab	0.072 a	-0.001
	-	Untreated	0.34 b	78 a	102 a	23	0.090 a	0.083 a	-0.007
LLS Mod	Axy	AuPnut	0.84 a	133 a	128 a	-1	0.004 a	0.046 b	0.042
		High	0.74 ab	136 a	126 a	-8	0.007 a	0.041 b	0.034
		Mod	0.81 a	134 a	126 a	-7	0.012 a	0.050 b	0.038
		Low	0.76 ab	134 a	127 a	-3	0.009 a	0.044 b	0.036
	Teb	AuPnut	0.80 ab	136 a	126 a	-5	0.010 a	0.048 b	0.038
		High	0.82 a	139 a	126 a	-11	0.006 a	0.034 b	0.027
		Mod	0.58 b	133 a	126 a	-4	0.012 a	0.043 b	0.031
		Low	0.79 ab	134 a	125 ab	-6	0.014 a	0.048 b	0.034
	-	Untreated	0.89 a	129 a	117 b	-10	0.017 a	0.082 a	0.065

[t] Weather-based (AU-Pnut) and prescription fungicide programs are grouped by azoxystrobin (Axy) and tebuconazole (Teb) based programs.

[u] Proportion of the total leaf spot epidemic attributed to late leaf spot. Calculated by dividing the standardized AUDPC of late leaf spot by the total standardized AUDPC of early and late leaf spot combined.

[v] Number of days after planting to reach 1 % disease incidence for early leaf spot (ELS) and late leaf spot (LLS).

[w] Rate (1/day) of disease progress was estimated with the Gompertz model for both early and late leaf spot incidence over time.

[x] Difference in days between the onset of late leaf spot relative that of early leaf spot.

[y] Difference between the rate of late leaf spot relative to that of early leaf spot.

[z] Means within columns with the same letters are not significantly different based upon Tukey's honestly significant difference test.

Table 3.6. Effect of fungicide program on leaf spot intensity, stem rot incidence and yield for each level of risk and peanut market type during 2011 and 2012.

	Fungicide[u]		Total AUDPC[v]		FL 1-10[w]		Stem rot[x]		Yield[y]	
Risk group	Product	Program	Runner	Valencia	Runner	Valencia	Runner	Valencia	Runner	Valencia
ELS High	Axy	AuPnut	0.47 c[z]	0.42 c	2.6 e	2.0 d	3.7 b	12.1 a	6733 a	2013 a
		High	0.51 c	0.67 cb	2.4 e	2.2 cd	3.4 b	9.4 ab	6097 ab	1759 ab
		Mod	0.78 bc	0.86 cb	3.1 de	2.2 bcd	3.4 b	10.9 ab	6202 ab	1761 ab
		Low	0.92 bc	1.20 b	3.6 cd	3.0 b	4.2 b	12.1 a	5873 ab	1606 ab
	Teb	AuPnut	0.88 bc	0.74 cb	3.1 de	2.4 bcd	4.0 b	10.9 ab	6331 ab	1801 a
		High	0.75 bc	1.01 b	2.7 e	2.6 bc	5.2 b	8.8 ab	6010 ab	1538 ab
		Mod	1.17 ab	1.17 b	3.9 bc	2.8 bc	5.5 b	6.2 b	5790 ab	1493 ab
		Low	1.42 ab	1.27 b	4.6 b	2.8 bc	8.3 ab	13.3 a	5427 b	1744 ab
	-	Untreated	2.22 a	2.90 a	8.3 a	4.8 a	23.5 a	13.9 a	2855 c	1228 b
ELS Mod	Axy	AuPnut	0.60 cd	0.06 cd	3.3 c	1.8 c	3.2 a	9.0 a	5806 a	3947 ab
		High	0.48 d	0.02 d	3.2 c	1.6 c	5.0 a	8.1 a	5597 a	4122 ab
		Mod	0.93 abcd	0.11 bcd	4.3 bc	1.7 c	4.4 a	7.9 a	5000 a	4394 a
		Low	1.16 abcd	0.25 bc	4.5 bc	2.4 bc	2.5 a	8.1 a	5297 a	4065 ab
	Teb	AuPnut	1.63 ab	0.17 bc	5.4 b	2.3 bc	4.4 a	10.2 a	5584 a	4313 a
		High	0.84 bcd	0.26 abc	4.4 bc	2.4 bc	4.0 a	10.5 a	5239 a	3711 ab
		Mod	1.25 abc	0.11 bcd	4.7 bc	2.4 bc	8.8 a	10.3 a	4960 a	3442 ab
		Low	1.38 abc	0.56 ab	4.9 bc	3.1 b	7.9 a	8.9 a	5036 a	4000 ab
	-	Untreated	2.38 a	1.42 a	8.0 a	5.5 a	9.0 a	12.5 a	4555 a	3141 b
ELS Low	Axy	AuPnut	0.01 ab	0.03 a	1.0 b	1.2 b	2.0 a	4.3 a	2046 a	1248 a
		High	0.01 ab	0.01 a	1.0 b	1.1 b	3.2 a	4.4 a	2203 a	1463 a
		Mod	0.01 ab	0.01 a	1.1 b	1.1 b	3.0 a	4.0 a	2285 a	1221 a
		Low	0.01 ab	0.01 a	1.2 b	1.2 b	3.2 a	7.4 a	2322 a	1410 a
	Teb	AuPnut	0.01 b	0.00 a	1.1 b	1.2 b	6.9 a	4.4 a	1874 a	1385 a
		High	0.00 b	0.00 a	1.1 b	1.2 b	4.1 a	6.8 a	2216 a	1317 a
		Mod	0.00 b	0.01 a	1.2 b	1.2 b	4.3 a	6.9 a	2559 a	1264 a
		Low	0.01 ab	0.02 a	1.1 b	1.2 b	2.3 a	5.2 a	2284 a	1350 a
	-	Untreated	0.19 a	0.06 a	2.2 a	1.7 a	5.8 a	6.3 a	1985 a	1348 a
LLS High	Axy	AuPnut	4.03 ab	0.08 c	4.0 bc	1.5 b	2.1 a	3.3 a	6703 a	2501 a

		High	1.91	ab	0.10	bc	2.8	e	1.3	b	1.7	a	11.5	a	5970	a	2870	a
		Mod	4.59	a	0.19	abc	3.8	cde	1.5	b	2.4	a	5.1	a	5661	a	2897	a
		Low	5.22	a	0.43	abc	4.6	bc	1.7	b	3.0	a	3.0	a	6599	a	3046	a
	Teb	AuPnut	4.04	ab	0.14	bc	4.1	bc	1.4	b	3.3	a	3.3	a	6488	a	2737	a
		High	1.14	b	0.42	abc	3.3	de	1.6	b	.	.	4.7	a	6445	a	3236	a
		Mod	3.87	ab	0.57	ab	5.6	b	2.0	b	2.4	a	6.6	a	5929	a	2935	a
		Low	6.33	a	0.49	ab	5.5	b	2.3	ab	5.0	a	3.7	a	6472	a	3076	a
	-	Untreated	5.00	a	1.08	a	7.2	a	3.3	a	5.3	a	3.5	a	5726	a	2816	a
LLS Mod	Axy	AuPnut	0.46	ab	0.01	bc	3.8	cd	1.5	b	7.0	bc	1.4	ab	5833	a	4170	a
		High	0.14	b	0.03	bc	3.5	d	1.5	b	8.4	abc	2.9	ab	5380	ab	3818	a
		Mod	0.46	ab	0.02	bc	4.6	bc	1.6	b	5.2	bc	1.3	b	5222	ab	3823	a
		Low	0.53	ab	0.01	bc	4.2	bcd	1.5	b	4.2	c	2.4	ab	5040	ab	3423	a
	Teb	AuPnut	0.30	b	0.01	bc	3.9	bcd	1.3	b	8.2	abc	1.3	ab	5199	ab	3926	a
		High	0.25	b	0.00	c	3.7	cd	1.4	b	8.0	b	1.7	ab	5694	a	3675	a
		Mod	0.24	b	0.01	bc	4.9	b	1.5	b	10.7	ab	1.9	ab	4932	ab	3826	a
		Low	0.48	ab	0.08	ab	4.7	c	1.7	b	11.3	ab	3.0	ab	5183	ab	3569	a
	-	Untreated	1.72	a	1.04	a	7.0	a	3.1	a	17.9	a	4.4	a	3957	b	3330	a

[u]Weather-based (AU-Pnut) and prescription fungicide programs are grouped by azoxystrobin (Axy) or tebuconazole (Teb) based programs.
[v]The total standardized AUDPC of the combined number of early and late leaf spot lesions.
[w]Florida 1 to 10 rating scale where 1 = no disease and 10 = dead plant.
[x]Percent of 30.5-cm sections per linear row with at least one disease locus.
[y]Pod yield measured in kg/ha and adjusted to 10% moisture.
[z]Means within columns with the same letters are not significantly different based upon Tukey's honestly significant difference test ($\alpha = 0.05$).

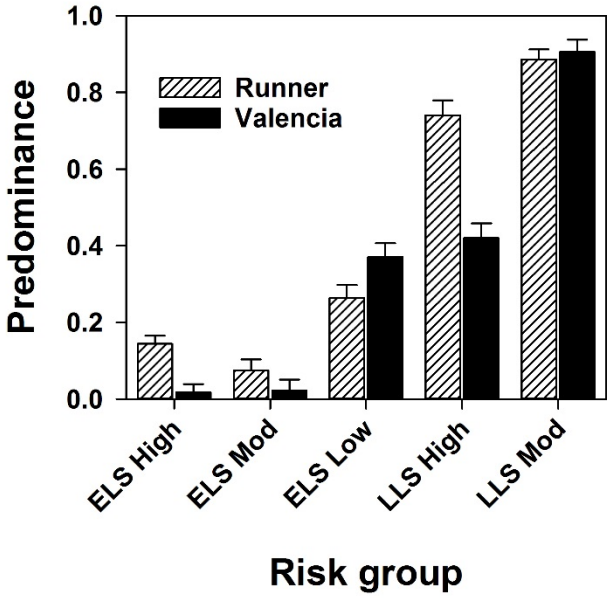


Fig. 3.1. Predominance (proportion of the total leaf spot epidemic attributed to late leaf spot, LLS; calculated by dividing the standardized AUDPC of LLS by the total standardized AUDPC of early (ELS) and LLS combined) on runner (Cultivar Georgia-06G) and Valencia peanut market types (Cultivar New Mexico Valencia C or Georgia Valencia (LLS Mod only)) compared across the five risk groups evaluated in 2011 and 2012. Estimates of the mean (n = 36 to 135) are pooled across fungicide treatments, and error bars indicate the standard error of the mean. Table 3.1 explains the risk groupings.

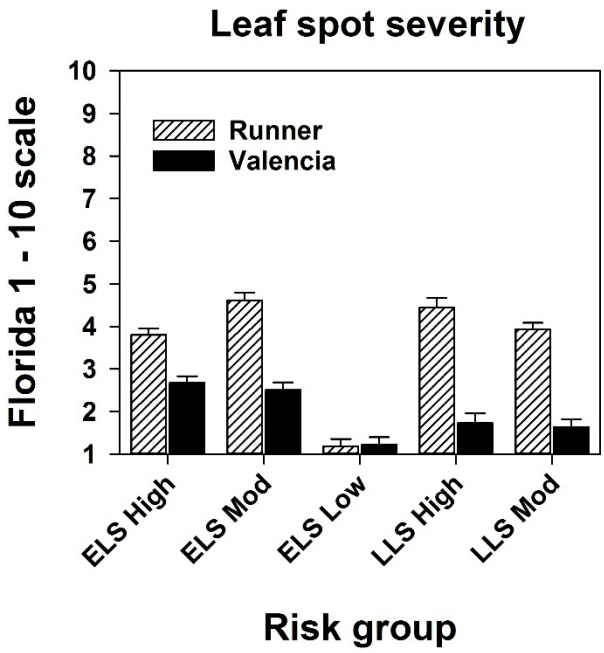


Fig. 3.2. Final leaf spot severity (based on Florida 1-10 scale visual rating where 1 = no disease and 10 = dead plant) on runner (Cultivar Georgia-06G) and Valencia peanut market types (Cultivar New Mexico Valencia C or Georgia Valencia (LLS Mod only)) compared across the five risk groups evaluated in 2011 and 2012. Estimates of the mean (n = 36 to 135) are pooled across fungicide treatments, and error bars indicate the standard error of the mean. Table 3.1 explains the risk groupings.

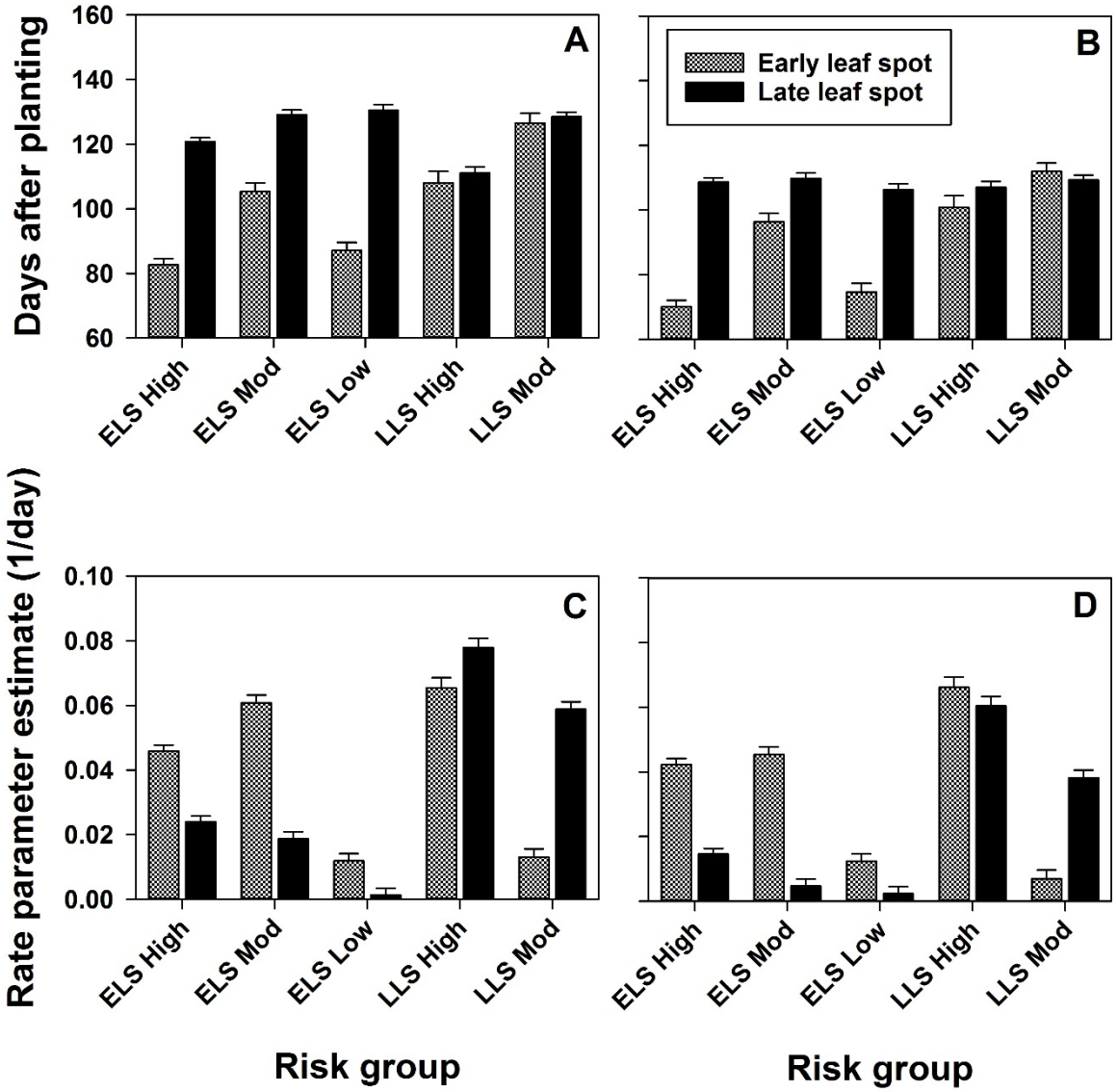


Fig. 3.3. Disease onset (days after planting to reach 1% incidence) of early (ELS) and late leaf spot (LLS) on (**A**) runner (cultivar Georgia-06G) and (**B**) Valencia peanut market types (cultivar New Mexico Valencia C and Georgia Valencia (LLS Mod only)), and rate (estimated rate parameter from the fitted Gompertz model) of ELS and LLS incidence over time on (**C**) runner (cultivar Georgia-06G) and (**D**) Valencia market types (cultivar New Mexico Valencia C and Georgia Valencia (LLS Mod only)) compared across the five risk groups evaluated in 2011 and 2012. Data points are estimates of the mean (n = 36 to 135) pooled across fungicide treatments, and error bars indicate the standard error of the mean. Table 3.1 explains risk groupings.

Supplementary Table S3.1. Prescription fungicide programs used in 2010 field trials at Attapulgus and the Black Shank farm (Tifton, GA).

Program	# Applications[w]	Timing[x]	Fungicide	Formulation	Rate[y] kg a.i./ha
High	7	1, 2, 4	Propiconazole	Tilt Bravo™	0.06
			+ Chlorothalonil		0.84
		3, 5	Azoxystrobin	Abound®	0.33
		6, 7	Chlorothalonil	Bravo Weather Stik®	1.26
Mod	6	1.5	Propiconazole	Tilt Bravo™	0.09
			+ Chlorothalonil		1.26
		3, 5	Azoxystrobin	Abound®	0.33
		4	Propiconazole	Tilt Bravo™	0.06
			+ Chlorothalonil		0.84
		6.5	Chlorothalonil	Bravo Weather Stik®	1.26
Low	4	2	Propiconazole	Tilt Bravo™	0.06
			+ Chlorothalonil		1.26
		3.5, 5	Azoxystrobin	Abound®	0.22
			Chlorothalonil	Bravo Weather Stik®	0.84
		6.5	Chlorothalonil	Bravo Weather Stik®	1.26
High	7	1.5	Pyraclostrobin	Headline 2.09EC	0.16
		2.5, 4, 5, 6	Flutolanil	Artisan	0.42
			+ Propiconazole		0.08
		2.5, 4, 5	Chlorothalonil	Bravo Weather Stik®	0.84
		5	Thiophanate-methyl	Topsin 4.5FL®	0.20
		7	Chlorothalonil	Bravo Weather Stik®	1.26
Mod	5	1.5	Pyraclostrobin	Headline 2.09EC	0.16
		2.5, 4.5, 6	Flutolanil	Artisan	0.44
			+ Propiconazole		0.09
		2.5, 6, 7	Chlorothalonil	Bravo Weather Stik®	0.84
		4.5, 7	Thiophanate-methyl	Topsin 4.5FL®	0.20
Low	4	2	Pyraclostrobin	Headline 2.09EC	0.16
		3.5, 5	Flutolanil	Artisan	0.68
			+ Propiconazole		0.12
		3.5, 5, 6.5	Chlorothalonil	Bravo Weather Stik®	0.84
		6.5	Thiophanate-methyl	Topsin 4.5FL®	0.20

[w] Total number of applications made each season for each fungicide program.

[x] Number 1 represents 30 days after planting; subsequent whole numbers stand for 14 day intervals. Applications began at 30, 30, 37, and 44 days after planting for the high, moderate and low programs. Applications with the same number were tank mixed.

[y] Rate of each fungicide applied is expressed as kg a.i./ha.

Supplementary Table S3.2. Prescription fungicide programs used at the RDC Pivot in Tifton during 2010.

Program	Total[w]	Timing[x]	Fungicide	Formulation	Rate[y] kg ai/ha
High	7	1, 2, 6.5	Chlorothalonil	Bravo Weather Stik®	1.26
		2.5, 3.5, 4.5, 5.5	Prothioconazole	Provost 433 SC	0.08
			+ Tebuconazole		0.17
Mod	5	1.5, 6.5	Chlorothalonil	Bravo Weather Stik®	1.26
		2.5, 4, 5.5	Prothioconazole	Provost 433 SC	0.11
			+ Tebuconazole		0.22
Low		1.5, 6.5	Chlorothalonil	Bravo Weather Stik®	1.26
		3, 5	Prothioconazole	Provost 433 SC	0.11
			+ Tebuconazole		0.22
High	7	1, 2, 2.5, 3.5, 4.5, 5.5	Chlorothalonil	Echo® 720 AG	0.84
		1, 2	Tetraconazole	Eminent	0.06
		2.5, 3.5, 4.5, 5.5	Tebuconazole	Muscle® ADV	0.23
		6.5	Chlorothalonil	Echo® 720 AG	1.26
Mod	5	1.5, 2.5, 3.5, 4.5, 6	Chlorothalonil	Echo® 720 AG	0.84
		1.5, 6	Tetraconazole	Eminent	0.06
		2.5, 3.5, 4.5	Tebuconazole	Muscle® ADV	0.23
Low	4	1.5, 3, 4.5, 6	Chlorothalonil	Echo® 720 AG	1.26
		1.5	Tetraconazole	Eminent	0.06
		3, 4.5, 6	Tebuconazole	Muscle® ADV	0.23
High	7	1.5	Pyraclostrobin	Headline 2.09EC	0.16
		2.5, 3.5, 4.5, 5.5	Flutolanil	Artisan	0.42
			+ Propiconazole		0.08
		2.5, 3.5, 4.5, 5.5	Chlorothalonil	Bravo Weather Stik®	0.84
		5.5	Thiophanate-methyl	Topsin 4.5FL®	0.20
		6.5	Chlorothalonil	Bravo Weather Stik®	1.26
Mod	5	1.5	Pyraclostrobin	Headline 2.09EC	0.16
		2.5, 4, 5.5	Flutolanil	Artisan	0.44
			+ Propiconazole		0.09
		2.5, 4, 5.5, 6.5	Chlorothalonil	Bravo Weather Stik®	0.84
		6.5	Thiophanate-methyl	Topsin 4.5FL®	0.20
Low	4	2	Pyraclostrobin	Headline 2.09EC	0.16
		3, 5	Flutolanil	Artisan	0.68
			+ Propiconazole		0.12
		3, 4.5, 6	Chlorothalonil	Bravo Weather Stik®	0.84
		6	Thiophanate-methyl	Topsin 4.5FL®	0.20

[w]Total number of applications made each season for each fungicide program.

[x]Number 1 represents 30 days after planting; subsequent whole numbers stand for 14 day intervals. Applications began at 30, 30, 37, and 44 days after planting for the high, moderate and low programs. Applications with the same number were tank-mixed.

Supplementary Table S3.3. . Schedule of fungicide applications made at each location 2011 and 2012 based on the AU-Pnuts weather advisory in comparison to high risk prescription program.

Location[w]	Irrigation status[x]	Application schedules (days after planting)[y]				Mean # apps. per year
		2011		2012		
		Calendar[z]	AU-Pnut	Calendar	AU-Pnut	AU-Pnut
Att	Dry	30, 45, 59, 72, 86, 100, 114	30, 45, 64, 93, 107, 120	30, 43, 57, 71, 86, 98, 112	30, 50, 63, 78, 96, 112	6
Att	Irr	30, 45, 59, 72, 86, 100, 114	30, 45, 64, 88, 102, 120	30, 43, 57, 71, 86, 98, 112	30, 50, 63, 78, 96, 112	6
Bsf	Dry	30, 44, 58, 72, 85, 100, 114	30, 44, 58, 72, 100, 118	35, 49, 62, 76, 90, 104, 117	35, 49, 64,76, 90, 104, 117	6.5
Bsf	Irr	30, 44, 58, 72, 85, 100, 114	30, 44, 58, 72 93, 118	35, 49, 62, 76, 90, 104, 117	35, 49, 64,76, 90, 104, 117	6.5
RDC	Irr	28, 42, 56, 69, 84, 98, 112	28, 42, 53, 69, 84, 105, 123	34, 48, 62, 76, 93, 106, 118	34, 56, 70,85, 96, 111, 125	7
Plains	Irr	30, 42, 57, 69, 85, 99, 113	30, 49, 69, 85, 106	-	-	6
MAR	Irr	-	-	27, 42, 57, 69, 85, 97, 112	27, 42, 57, 74, 88, 104	6

[w] Location and environment of field trials are abbreviated as follows: Att = Attapulgus Research and Education Center in Attapulgus, Georgia, Bsf = Black Shank farm in Tifton, Georgia, RDC = RDC Pivot in Tifton, Georgia, Plains = Southwest Georgia Research & Education Center in Plains, Georgia, MAR = Marianna, Florida,

[x] Dry = Dryland, and Irr = Irrigated.

[y] Days after planting date when fungicide applications were made. Planting dates were as follows: AttDry and AttIrr 23 May, 2011 and 9 May 2012; BsfDry and BsfIrr 25 May, 2011 and 23 May, 2012; RDC 20 May, 2011 and 2 May, 2012; Plains 18 May, 2011; Marianna 17 May, 2012.

[z] High-risk prescription fungicide program: starts approximately 30 days after planting with subsequent applications every 14 days.

Supplementary Table S3.4. Effect of fungicide groups and spray programs on the development of early and late leaf spot, stem rot and yield in the peanut cultivar Georgia-06G at four locations grouped into three risk groups in 2010 and one risk group at Plains in 2011.

Year	Risk group	Fungicide[t]		Disease onset[u]		Predominance (ProLLS)[v]	AUDPC[w]	FL 1-10[x]	Stem rot[y]	Yield (kg/ha)
		Product	Program	ELS	LLS					
2010	ELS High	Azy	High	96 a[z]	121 a	0.01 a	0.48 b	1.8 bc	19 b	5653 a
			Mod	89 a	120 a	0.01 a	0.66 b	2.8 bc	24 b	5388 ab
			Low	91 a	119 a	0.02 a	0.80 b	3.6 b	36 ab	4721 bc
		Pyr	High	94 a	118 a	0.04 a	0.47 b	1.5 c	14 b	5761 a
			Mod	90 a	116 a	0.04 a	0.79 b	2.8 bc	21 b	5442 ab
			Low	90 a	116 a	0.01 a	0.94 b	2.5 bc	14 b	5239 ab
		-	Untreated	82 a	117 a	0.04 a	3.68 a	7.9 a	52 a	4309 c
2010	ELS Low	Azy	High	105 ab	127 a	0.04 a	0.11 a	1.1 b	3 a	3668 a
			Mod	119 a	119 a	0.02 a	0.16 a	1.0 b	4 a	3354 a
			Low	116 ab	117 a	0.03 a	0.13 a	1.4 b	3 a	3122 a
		Pyr	High	122 a	105 a	0.03 a	0.13 a	1.1 b	3 a	3292 a
			Mod	96 b	96 a	0.00 a	0.06 a	1.4 b	3 a	3210 a
			Low	121 a	95 a	0.03 a	0.11 a	1.0 b	2 a	3344 a
		-	Untreated	99 b	115 a	0.06 a	0.24 a	3.9 a	4 a	3385 a
2010	LLS Mod	Teb	High	138 a	122 ab	0.98 a	0.30 ab	1.6 bc	9 ab	8452 a
			Mod	111 a	117 b	0.99 a	0.56 ab	2.1 bc	7 ab	8672 a
			Low	120 a	128 ab	0.99 a	0.61 ab	2.4 bc	6 ab	8622 a
		Pyr	High	138 a	131 a	0.96 a	0.28 ab	1.8 bc	2 b	8841 a
			Mod	117 a	128 ab	0.98 a	0.42 ab	1.8 bc	3 ab	8878 a
			Low	117 a	131 a	0.92 a	0.64 ab	2.3 bc	3 b	8959 a
		Pro	High	138 a	131 a	0.99 a	0.29 ab	1.5 c	8 ab	8750 a
			Mod	128 a	128 ab	0.99 a	0.13 b	1.8 bc	3 ab	9113 a
			Low	128 a	124 ab	0.99 a	1.66 a	2.9 b	6 ab	8990 a
		-	Untreated	126 a	124 ab	0.97 a	1.70 a	4.9 a	13 a	7084 b
2011	LLS Mod	Axy	AuPnut	140 b	140 b	0.49 a	0.00 b	1.3 b	11 a	6295 a
			High	140 b	140 b	0.01 a	0.00 b	1.4 b	7 a	5097 a

	Mod	140 b	140 b	0.54 a	0.00 b	1.5 b	6 a	5123 a
	Low	140 b	140 b	0.08 a	0.00 b	1.5 b	3 a	4891 a
Teb	AuPnut	140 b	140 b	0.40 a	0.00 b	1.5 b	9 a	5055 a
	High	140 b	140 b	0.99 a	0.01 ab	1.3 b	12 a	5845 a
	Mod	140 b	140 b	0.07 a	0.00 b	1.7 b	15 a	4965 a
	Low	140 b	140 b	0.83 a	0.00 b	1.5 b	12 a	6007 a
-	Untreated	126 a	126 a	0.80 a	0.59 a	3.7 a	18 a	4343 a

[t] Weather-based (AU-Pnut) and prescription fungicide programs are grouped by azoxystrobin (Axy), pyraclostrobin (Pyr), tebuconazole (Teb) or Prothioconazole (Pro) based programs.

[u] Number of days after planting to reach 1 % disease incidence for early leaf spot (ELS) and late leaf spot (LLS).

[v] Proportion of the total leaf spot epidemic attributed to late leaf spot. Calculated by dividing the standardized AUDPC of late leaf spot by the total standardized AUDPC of early and late leaf spot combined.

[w] The total standardized AUDPC of the combined number of early and late leaf spot lesions.

[x] Florida 1 to 10 rating scale where 1 = no disease and 10 = a dead plant.

[y] Percent of 30.5-cm sections per linear row with at least one disease locus.

[z] Means within columns with the same letters are not significantly different based upon Tukey's honestly significant difference test ($\alpha = 0.05$).

Supplementary Table S3.5. Effect of fungicide program on max number of early and late leaf spot lesions for each level of risk and market type during 2011 and 2012.

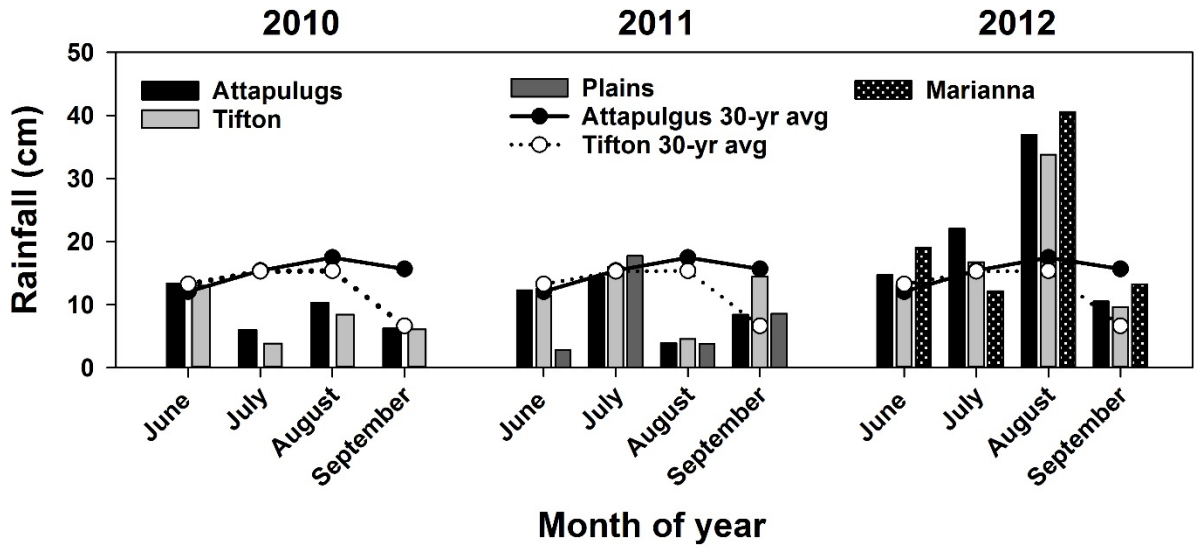
	Fungicide		Maximum number of lesions per leaf[x]			
			Runner		Valencia	
Risk group	Product	Program	ELS[y]	LLS	ELS	LLS
ELS High	Axy	AuPnut	2.90 d[z]	0.87 a	2.47 c	0.11 a
		High	3.38 d	0.84 a	3.40 bc	0.03 a
		Mod	5.33 bcd	1.27 a	4.58 bc	0.07 a
		Low	7.59 abc	0.77 a	5.93 b	0.03 a
	Teb	AuPnut	4.57 cd	1.34 a	4.21 bc	0.08 a
		High	4.46 cd	1.10 a	5.44 b	0.06 a
		Mod	7.00 abc	1.62 a	6.59 b	0.15 a
		Low	9.36 a	1.23 a	6.25 b	0.12 a
	-	UT	9.23 ab	0.33 a	13.21 a	0.35 a
ELS Mod	Axy	AuPnut	4.03 b	0.30 a	0.58 cd	0.01 a
		High	4.06 b	0.09 a	0.24 d	0.02 a
		Mod	7.47 ab	0.22 a	1.20 cd	0.01 a
		Low	9.25 a	0.20 a	1.94 bcd	0.01 a
	Teb	AuPnut	11.27 a	0.86 a	1.49 bcd	0.03 a
		High	7.60 ab	0.17 a	2.63 bc	0.01 a
		Mod	10.59 a	0.30 a	1.35 bcd	0.02 a
		Low	10.55 a	0.30 a	4.64 b	0.01 a
	-	UT	13.12 a	0.30 a	11.51 a	0.05 a
ELS Low	Axy	AuPnut	0.03 b	0.01 a	0.03 a	0.15 a
		High	0.03 b	0.02 a	0.03 a	0.04 a
		Mod	0.05 b	0.03 a	0.02 a	0.01 a
		Low	0.06 b	0.04 a	0.04 a	0.01 a
	Teb	AuPnut	0.09 b	0.01 a	0.03 a	0.01 a
		High	0.05 b	0.01 a	0.02 a	0.01 a
		Mod	0.03 b	0.01 a	0.08 a	0.01 a
		Low	0.04 b	0.01 a	0.13 a	0.04 a
	-	UT	2.11 a	0.13 a	0.66 a	0.11 a
LLS High	Axy	AuPnut	3.90 ab	16.41 ab	0.35 b	0.32 a
		High	1.85 b	7.84 b	0.33 b	0.33 a
		Mod	4.42 ab	17.49 ab	0.49 b	1.01 a
		Low	6.28 ab	17.31 ab	1.64 ab	1.62 a
	Teb	AuPnut	4.78 ab	14.30 ab	0.69 b	0.58 a
		High	1.86 b	7.32 b	1.30 ab	1.27 a
		Mod	6.26 ab	15.23 ab	1.86 ab	0.95 a
		Low	6.58 ab	23.66 a	1.86 ab	1.28 a
	-	UT	8.75 a	11.87 ab	6.14 a	0.70 a
LLS Mod	Axy	AuPnut	0.04 a	3.36 b	0.01 a	0.11 b
		High	0.03 a	1.76 b	0.03 a	0.30 b
		Mod	0.06 a	3.63 b	0.01 a	0.24 b
		Low	0.05 a	3.66 b	0.01 a	0.14 b
	Teb	AuPnut	0.06 a	2.88 b	0.01 a	0.03 b

	High	0.05 a	1.79 b	0.01 a	0.08 b
	Mod	0.04 a	3.63 b	0.03 a	0.08 b
	Low	0.14 a	2.56 b	0.03 a	0.67 b
-	UT	0.22 a	8.91 a	0.08 a	10.23 a

[x] Defined as the greatest number of lesions counted at any rating date throughout the evaluation period, based on the average count of each the four leaflets per leaf

[y] ELS = early leaf spot; LLS = late leaf spot

[z] Means within columns with the same letters are not significantly different based upon Tukey's honestly significant difference test ($\alpha = 0.05$).



Supplementary Figure S3.1. Total monthly rainfall at Attapulgus, Tifton (Black Shank and RDC Pivot) and Plains, GA, or Marianna, FL, during the 2010, 2011 and 2012 growing seasons. Thirty-year averages were calculated only at Attapulgus and Tifton. Monthly rainfall records were obtained from the Georgia Automated Environmental Monitoring Network provided by the University of Georgia (www.Georgiaweather.net).

Chapter 4

EFFECT OF INFESTED PEANUT RESIDUE INOCULUM AND FALL TILLAGE ON EPIDEMCIS OF EARLY AND LATE LEAF SPOT OF PEANUT[1]

[1]Fulmer, A.M., Kemerait, R.C., Brenneman, T.B., Scherm, H., Cantonwine, E.G., Culbreath, A.K., and Stevenson, K.L. 2017. To be submitted to *Plant Disease*.

ABSTRACT

Early (ELS) and late (LLS) leaf spot peanut, caused by the fungi *Cercospora arachidicola* and *Cercosporidium personatum*, respectively, are similar with respect to disease cycle, symptomology and damage to the peanut plant. The epidemiological behavior of these two diseases can differ greatly, but factors responsible for this are unknown. Field trials were conducted in Tifton and Reidsville, Georgia in 2014 and 2015 to determine the effect of initial inoculum and tillage on the predominance and development of each leaf spot disease. Inoculum treatments consisted of peanut residue infested with *C. arachidicla* (*Ca*) alone, *C. personatum* (*Cp*) alone, a 50/50 mix of *Ca* + *Cp* and a no residue control. Plots were subjected to three tillage treatments: fall harrow + spring harrow and rototill, spring harrow and rototill, and strip till (no cover crop). Across trials, LLS predominance (proportion of epidemic attributed to LLS) increased over time, and final values ranged from ~0.2 to ~0.8. Inoculum treatment had a significant effect ($P < 0.05$) on predominance, time of onset, rate of disease progression and defoliation. Within each trial, plots with *Ca* had significantly lower levels of LLS than those treated with *Cp* and vice versa. Plots treated with *Ca* + *Cp* generally had numerically or statistically more ELS than those treated with *Cp* alone or in residue-free plots, and the latter generally had statistically more LLS compared with those with *Ca* alone. Across trials and tillage treatments, the onset of ELS ranged from 58 to 78 days after planting (DAP), whereas the onset of LLS ranged from 89 to 100 DAP, and each disease was first detected in plots with *Ca* and *Cp*, respectively. Regardless of trial or inoculum treatment, the rate of LLS progression was statistically higher than that of ELS, and in all plots, defoliation increased exponentially, resulting in 100% defoliation by ~140 DAP. Fall tillage did not affect predominance or reduce the rate of either disease. Compared with the strip tillage, fall tillage significantly delayed the onset of LLS by 8 days, but the effect on the onset of ELS was less consistent. Results from these trials indicate that the inoculum source is a primary factor governing the prevalence and onset of each disease, and that the development of LLS tends to be more suppressed than the development of ELS when the inoculum source of both diseases is present.

INTRODUCTION

Woodruff (1933) showed that there are two similar, yet distinct leaf spot diseases of peanut (*Arachis hypogeae* L.). These have since been referred to as early leaf spot (ELS) (caused by *Cercospora arachidicola* Hori) and late leaf spot (LLS) (caused *Cercosporidium personatum* (Berk and M. A. Curtis) Deighton)), and are considered to be two of the most damaging foliar diseases of peanut worldwide (Garren and Wilson, 1951). Both are polycyclic diseases with similar symptoms of lesions on leaflets, petioles, stems and pegs (Shokes and Culbreath, 1997). In untreated, high-risk fields, both diseases can cause complete defoliation of the peanut plant resulting in yield losses ranging from 40% to as high as 70 % (Backman and Crawford, 1984; McDonald et al., 1985). Management relies heavily on fungicide inputs, but cultural practices such as rotation, planting date, and tillage are also recommended as part of an integrated management program (Shokes and Culbreath, 1997). From a management standpoint, both leaf spots are generally considered a single disease, and it is generally assumed that most recommended practices are equally effective against ELS and LLS (Garren and Wilson, 1951).

From an epidemiological standpoint, however, the development of these diseases can differ greatly. The most characteristic difference is the time of disease onset. A previous report suggested that when inoculum of both pathogens is present in a field, the onset of ELS typically occurs 30 to 50 days after planting (DAP) and is generally 3 to 4 weeks prior to that of LLS (Smith and Littrell, 1980). However, there are reports of both diseases first appearing at 21 to 35 DAP (McDonald et al., 1985), and as late as 130 DAP (Chapters 3 and 8). LLS is thought to be the more destructive of the two due to a greater amount of sporulation per lesion and thus a higher rate of development toward the end of the season (Backman and Crawford, 1984; Hemingway, 1955; Jenkins, 1938). Although capable of co-existing in the same field and even on the same leaflet, the relative abundance of each disease tends to vary over time both regionally and locally (Jackson, 1981). In the late 1930s, Jenkins (1938) stated that LLS only occurred 2 out of every 5 years in Georgia, and Miller (1953) reported that ELS was the most widespread of the two diseases in the southeastern United States. In Georgia, ELS was the prevalent

disease until 1976 when a shift took place and LLS prevailed until the mid-1990s (Cantonwine et al., 2008; Smith and Littrell, 1980). In recent years, there has been a resurgence of LLS in Georgia (Cantonwine et al., 2008), and now it is possible to have nearby fields that are predominantly one disease or the other (Chapter 3).

The mechanisms governing the differences in the temporal dynamics of ELS and LLS are still not clear. Rotation, variety, fungicide chemistry and/or program and late harvest dates have been speculated as factors that could cause a shift and/or reduction in inoculum potential. As a result, these may affect predominance, onset, rate of epidemic development and final disease intensity (Smith and Littrell, 1980). In a recent study, we found that although both fungicide program and peanut cultivar can have some effect on the predominance and development of each disease, our results strongly suggest that the most extreme variability is largely explained by the quantity and proximity of the initial inoculum (Chapter 3).

Most reports agree that the primary form of initial inoculum for both *C. arachidicola* and *C. personatum* comes from conidia produced on stromata that overwinter in the peanut residue (Hemingway, 1954; Jackson and Bell, 1969; Shanta, 1960) It has been proposed that conidia, mycelial fragments, ascospores and chlamydospores residing in the soil could also serve as potential inoculum sources, but since the time of Jenkins (1938) and Woodroof (1933), the latter two have rarely, if ever, been observed (Shokes and Culbreath, 1997). In the tropics, volunteer peanuts play a major role in continual inoculum perpetuation between seasons (McDonald et al., 1985). In the southeastern U.S., volunteer peanuts likely do not serve as continual inoculum source since most plants die due to freezing weather. However, if not controlled, volunteer peanuts can serve as a bridge for the local inoculum source to perpetuate between rotational crops (A. Fulmer, unpublished data; Shokes and Culbreath, 1997).

The length of time that leaf spot fungi can survive within the peanut residue has proven difficult to fully elucidate. Nuesry (1981) found that sporulation of *C. arachidicola* on stromata occurred after infested leaflets, petioles and stems had been buried in the field at a depth of 10 and 20 cm for 6 months. The same author found that stromata could sporulate after being stored in a cool, closed environment for more than 1.5 years. In a similar study, Wolf (1914) showed that leaves infested with *C. personatum* left

to overwinter out in the field following harvest in October, could still cause infections in May. In contrast, separate studies in India reported that the LLS pathogen within infested leaves and/or stems remained viable for 30 to 60 days (Rao et al., 1993) and at most up to 20 weeks (Shanta, 1960).

It can also be inferred from other published studies that survival of the leaf spot fungi is influenced by cultural practices. In one study, a 1-year rotation away from peanut significantly reduced LLS severity (Kucharek, 1975). Brenneman et al. (1995) found that leaf spot severity decreased when peanuts were rotated to bahiagrass for 0, 1, 2 or 3 years. In Georgia, peanuts are generally rotated with at least 1 year of cotton or corn but a rotation longer than 3 years is rare (S. Monfort, personal communication). Thus, it is possible that some amount of the leaf spot fungi could survive in the soil during the normal rotational period. Taken together, it is probable that inoculum levels of both leaf spot fungi decrease with each year rotated away from peanut, and after 3 years are mostly eliminated.

Tillage practices also have a direct or indirect impact on the level of initial inoculum. In a conventional system, a mold board plow is used to bury any remaining peanut residue to a depth of at least 15 to 30 cm prior to planting peanut (Jackson and Bell, 1969). In a conservation tillage system, a cover crop of wheat or rye is planted in the winter and killed at least 4 weeks prior to planting the peanuts. Immediately prior to planting, a strip-till rig is used to press down the cover crop, and sub soil shanks spaced ~0.91 m apart followed by a combination of fluted coulters and press wheels break up larger clods on the surface to create a narrow seeding bed ~ 30 cm wide (Paulk, personal communication). Interestingly, conservation tillage has been shown to have more of a suppressive effect on ELS epidemics than in a conventional tillage, and this is thought to be due to the barrier created by the cover crop which hinders splash dispersal of the conidia in the soilborne residue from reaching the plant (Cantonwine et al., 2007; Porter and Wright, 1991).

During peanut harvest, growers use a rotating straw spreader attached to the back of the combine to evenly distribute the peanut residue on the soil surface. Thereafter, growers will either bale the peanut residue for hay or allow the residue to remain on the soil surface. If a winter wheat or cover crop is planted, they will begin to harrow in the peanut residue within a few weeks post-harvest. Otherwise,

some growers will begin to harrow in their peanut reside as early as December or January in preparation for planting corn, cotton, etc. in the spring, while others will allow the residue to remain on the surface of the ground throughout the winter season. In a similar pathosystem, De Nazareno et al. (1992) hypothesized that since buried *Cercospora zeae-maydis* in infested corn residue does not survive the overwintering process, tillage of the residue in the fall could substantially reduce the initial inoculum level. Because the survival of the leaf spot pathogens of peanut is evidently longer than that of *C. zeae-maydis*, it is unknown whether there is an effect of this fall tillage on the inoculum level of either leaf spot pathogen.

In order to continue to develop effective integrated management strategies, the factors that drive the differences in the behavior of these two peanut leaf spot diseases needs to be understood. This is particularly relevant in a time when either disease can occur in a given field. However, it is difficult to compare the epidemiology of ELS and LLS when the level of overwintering inoculum of each leaf spot pathogen is unknown or very different. Therefore, the primary objective of this study was to characterize the temporal dynamics of ELS and LLS in field plots with known levels of infested peanut residue harboring each leaf spot pathogen. Secondary objectives were to determine the effect of co-inoculating plots with both leaf spot pathogens was to determine if harrowing infested residue into the soil in the late fall/early winter would have an effect on the development of each leaf spot.

MATERIALS AND METHODS

Field sites and experimental design. Field trials were conducted in 2014 and 2015 at the Coastal Plain Experiment Station in Tifton and the Vidalia Onion and Vegetable Research Center located in Reidsville, Georgia. At the Coastal Plain Experiment Station main campus farm in Tifton, studies were conducted at the east and west side of the FireTower field during 2014 and 2015, respectively. The distance separating these fields was approximately 25 m. An additional trial was also performed at the Coastal Plain Experiment Station Ponder farm during 2015. At Reidsville, the field site selected in 2015 was located approximately 150 m apart from that of 2014. All fields were specifically selected because they had not been planted to peanut for more than 10 years, or had no prior history of peanut.

For both trials at Reidsville and for the FireTower and Ponder farm trials in 2015, plots were laid out in a split-block design with four replications with inoculum source and tillage as factors. The effect of inoculum source alone was evaluated in the 2014 trial at the FireTower field in Tifton as a randomized complete block design with four replications. Inoculum treatments consisted of four combinations of infested peanut residue: *C. arachidicla* (*Ca*) alone, *C. personatum* (*Cp*) alone, a 50/50 mix of *Ca* + *Cp* and a non-treated control. Peanut residue was collected from fields in Tifton, Georgia, with severe epidemics of either (>90%) ELS or LLS, and stored separately. In most cases, the residue came from untreated plots that had nearly 100% severity (a "10" on the Florida 1-10 scale). However, in 2014 at Reidsville, the *Cp* inoculum came from a field with ~ 80% severity, and in 2015, the *Ca* inoculum at Reidsville came from a trial with ~ 70 % severity. Peanut residue including leaflets, petioles and stems, was collected within a few days after harvesting peanuts (late October and November) stored in a large peanut wagon under an open shed for 4 to 6 weeks. In December, plots (1.8 × 7.6 m) were harrowed and rototilled, and dry peanut residue (~4.5 kg) for each treatment was spread across the middle (4.5 m) section of each plot as evenly as possible. For the spring and strip tillage plots, the peanut residue applied to the plots was allowed to overwinter on the surface of the soil. Each plot was bordered by two residue-free buffer rows of peanut on each side, and separated by 3 m between each block.

The experiments included three tillage treatments: fall, spring and strip. For the fall tillage, plots were harrowed (at a depth of ~10.2 to 15.2 cm) three or four times within a day after applying the peanut residue. Plots were harrowed several times each month throughout January and February until the residue was completely incorporated into the soil. For both the spring and fall tillage, plots were harrowed a few weeks prior to planting and rototilled within a day of planting. The spring tillage was meant to serve as a control to confirm that any effects of fall tillage were not simply due to the tillage required to prep the plots prior to planting. For the strip tillage, a KMC (Kelley Manufacturing Co., Tifton, GA) rip/strip rig with two shanks with a dragging depth of 45.7 cm was used to prepare two rows (~ 10 to 15 cm wide) for seeding. The strip tillage did not include a cover crop, and was meant to serve as a positive control since it would leave the greatest amount of residue on the soil surface. To reduce inter-plot contamination,

tillage instruments were lifted from the soil between plots, and any trapped inoculum removed prior to proceeding to the next plot. Approximately 4 weeks prior to planting, plots were treated with an application of glyphosate (Roundup 4 EC, Monsanto, Kansas City, MO) at a rate of 1.2 kg a.i./ha to kill emerged vegetation.

Following the overwintering period, two rows spaced 0.91 m apart were planted to the cultivar Georgia-06G at a rate of 6 seed per 30.5 cm. Plots were planted at Reidsville on 22 May 2014 and 21 May 2015, at the FireTower field on 19 May 2014 and 13 May 2015 and at the Ponder farm on 29 May 2015. At 60 and 90 DAP, flutolanil (Convoy, Nichino America, Wilmington, DE) was applied to all plots at a rate of 0.91 kg/ha to suppress stem rot caused by *Sclerotium rolfsii*. Applications were made with a CO_2-pressurized backpack sprayer, and a boom equipped with four 8002 flat tip nozzles (Teejet Technologies, Springfield, IL) calibrated to deliver 188 liters/ha. Plots were supplemented with irrigation as needed, and all herbicides, insecticides and fertilizers were applied following recommendations of the University of Georgia Cooperative Extension Service (Monfort, 2017).

Environmental data. Environmental conditions within the peanut canopy were measured at Reidsville and the FireTower field in Tifton during 2014 and 2015. Leaf wetness and temperature was measured with leaf wetness and VP-3 sensors (Decagon Devices Inc., Pullman, WA), respectively, placed within the middle part of the peanut canopy. Six sensors of each type were placed arbitrarily throughout the field, and Decagon Em50 data loggers were set to record hourly observations (the average of 60 sensor readings taken each minute within the last hour previously logged). For the leaf wetness sensors, wet and dry events were based on values $\geq$ or $<$ the 450 raw count threshold, respectively. For each location, rainfall and temperature records were obtained from the nearest weather station operated by the Georgia Automated Environmental Monitoring Network (www.Georgiaweather.net). When calibrated in the field, daytime (9:00 a.m. to 9:00 p.m.) temperature readings with the Decagon devices were often found to be 4 to 10 C higher than actual temperatures. Therefore data logged with Decagon devices during daytime hours were excluded from this report. Instead, daytime temperatures reported here are based on the data from the Georgia Automated Environmental Monitoring Network.

Environmental variables were calculated for each month of the main growing season (June, July, August and September). Mean temperature and total leaf wetness hours were calculated for day (9:01 a.m. to 9:00 p.m.) and night (9:01 p.m. to 9:00 a.m.). Rain events was divided into two categories: > 0.25 cm or > 0.63 cm of rainfall. Because LLS is thought to be favored more by cooler weather, two categories of favorable events were calculated: cooler events (18 to 24 °C) and warmer events (20 to 26 °C). A third category (18 to 28 °C) was calculated to show the total range of favorable events at which both leaf spots can still cause infection. For each category, an event was defined as ≥ 12 hours of continuous leaf wetness within each specific temperature range. Because temperatures within a 12 hour period can vary by at least 6 °C, favorable weather events were meant to encompass the relative range of temperatures for cooler events vs. warmer events, and as a result, necessitated some overlap between the two temperature categories.

Disease assessment. Beginning approximately 30 days after planting (DAP), plots were visually monitored for leaf spot symptoms. Upon detecting the first leaf spot symptoms, a more intensive evaluation was initiated and continued approximately on a bi-weekly schedule until harvest. Five main stems per plot were arbitrarily selected for destructive sampling and transported to the lab where the number of present and missing leaflets, and the number of ELS and LLS lesions per leaflet were counted. ELS and LLS were differentiated by symptoms and signs associated with each causal pathogen. In 2014, main stems were sampled at the following days after planting (DAP): FireTower – 42, 56, 70, 89, 99, 113, 126, 143; Reidsville – 32, 42, 60, 74, 89, 103, 116, 132, 144. In 2015, locations were sampled at the following DAP: FireTower – 40, 54, 66, 78, 93, 103, 117, 131, 146; Ponder – 42, 53, 61, 76, 94, 108, 122, 140; Reidsville – 41, 53, 67, 82, 96, 111, 124, 148.

Incidence of each disease for each rating date was calculated by dividing the number of leaflets with at least one lesion by the total number of leaflets counted across all five main stems. Area under the disease progress curve (AUDPC) was calculated for each leaf spot disease based on the number of lesions per leaflet. AUDPC values were standardized (stAUDPC) by dividing AUDPC values by the duration of the epidemic (Campbell and Madden, 1990). Predominance was defined as the proportion of the total leaf

spot epidemic attributed to LLS, and was calculated by dividing the stAUDPC of LLS by the total stAUDPC of ELS and LLS. Time of disease onset was defined as the number of DAP for ELS or LLS to reach 1% incidence. For lesion counts, the maximum rate of increase of lesion numbers per leaflet from one assessment date to the next was calculated for both ELS and LLS (Campbell and Madden, 1990).

Temporal progress of ELS and LLS incidence and of percent defoliation were modeled using linear regression to fit linearized forms of the exponential [log(y)], logistic [ln(y/1-y)] and Gompertz [-ln(-ln y)] models on days after planting. The coefficient of determination (R^2) and mean square error (MSE) from regression of backtransformed predicted on observed values were then compared. Residual plots (observed – predicted values) were also examined for each model. The model with the highest R^2, lowest MSE and the best residual distribution was selected as the best fit for the data. If the model failed to fit the data satisfactorily, parameter estimates were excluded from the analysis. The rate of increase in in incidence and defoliation and time to reach 50 % incidence (TDO50) and time to reach 50 % defoliation, were estimated based on model with the best fit.

Statistical analysis. Predominance, disease onset, rate of disease incidence and maximum rate of lesion increase of both leaf spot pathogens as well as rate of defoliation and time to 50 % defoliation were subjected to linear mixed model analysis of variance with PROC GLIMMIX (SAS 9.4 Institute, Cary, NC). The 2014 trial at the FireTower field in Tifton was analyzed separately, with inoculum as a fixed effect and replication as a random effect. Otherwise, the model was analyzed as a split-strip block with trial, tillage and inoculum considered as fixed effects, and replication × trial, replication × tillage and replication ×inoculum as random effects. The SLICE option was used to explore 2-way interactions when there was no significant 3-way interaction. In all analyses, the Kenward-Roger option was used to adjust the denominator degrees of freedom due to repeated measures, and differences in the least square means were tested by Tukey's multiple comparisons test. When data violated the assumptions of normality, dependent variables were transformed as follows: predominance was arcsin square root transformed, and the square root transformation was used for disease onset and the time to reach 50% defoliation. Back-transformed means of all transformed variables are presented in the results. Depending

on the interaction between main effects, pairwise t-tests were used to directly compare the response of ELS vs. LLS for each level of inoculum level and trial or across each factor. It was assumed that the parameters related to ELS and LLS epidemics were not independent, but rather shared the same variance since they were estimated from the same plant.

RESULTS

Environmental variables. For each month during the growing season, mean day and night temperatures were similar for each year and location (Table 4.1). Generally, June, July and August shared a similar mean temperature, followed by a drop of 1 to 2°C in September. Regardless of the month, however, the mean nightly temperature was in the range of 20 to 24°C – a range generally accepted as highly conducive for either leaf spot (Shokes and Culbreath, 1997). Similarly, average maximum and minimum nightly temperatures were similar for each month across location and years (data not shown). In 2014, the average maximum and minimum nightly temperatures, respectively, for June, July, August and September were 27, 29, 28, 26 and 18, 16, 18, 13°C at the FireTower, and 30, 30, 27, 26 and 18, 18, 16, 14°C at Reidsville. In 2015, the average maximum and minimum nightly temperatures, respectively, for June, July, August and September at were 30, 27, 27, 26 and 19, 18, 16, 12°C at the FireTower, and 30, 29, 26, 26 and 19, 19, 18, 11°C at Reidsville.

Overall, 2014 was a dry year (Table 4.1). Although not always correlated, this is evident from similar patterns in the number of rain events, total rainfall and leaf wetness duration (hours). In 2014, the number of combined rain events and total rainfall was lowest in July and August but increased sharply during September. Conversely, rainfall in 2015 was highest during July and August, but decreased sharply during September. Generally, the total number of leaf wetness hours per month increased as the season progressed regardless of rainfall; this trend was more evident during night hours.

Regardless of year and locations, there were generally fewer cooler events (18 to 24°C) than warmer events (20 to 26°C). A similar trend was that the greatest number of events generally occurred in months that had the highest levels of rainfall (Table 4.1). Overall, the FireTower field had a greater number of warmer events than cooler events in both 2014 and 2015, with the exception of September in

2014. At Reidsville, the numbers of cooler and warmer events were similar; however, in 2015, there were more warmer events than cooler events in almost every summer month (Table 4.1).

Predominance. For all field trials, the proportion of LLS increased over time regardless of tillage or inoculum treatment (Fig. 4.1). Generally, the proportion of LLS lesions reached a peak around 100 to 120 days after planting, and thereafter, began to decrease towards the last rating date (Fig. 4.1). Predominance was significantly affected by trial, inoculum and tillage, but one or more of the treatments were not always constant given that there were significant 2-way interactions between all the factors (Table 4.2). For all trials, plots with *C. personatum*-infested residue had significantly higher proportions of LLS than those with *C. arachidicola*-infested residue, and were always numerically higher than plots with both leaf spot pathogens (Fig. 4.2). In all cases, untreated plots had numerically and often significantly more LLS than plots with both pathogens or with *C. arachidicola* alone (Fig. 4.2). The effect of tillage was inconsistent, but fall and spring tillage tended to have less LLS than plots that were strip tilled (Fig. 4.3). Compared with all other trials, Reidsville had significantly lower and higher proportions of LLS in 2014 and 2015, respectively (data not shown).

Disease onset. The effects of trial, tillage and inoculum for the onset of both ELS and LLS were significant (Table 4.2). For ELS onset there was also a significant trial × tillage interaction (Table 4.2). For all trials, ELS onset was significantly different among plots that received inoculum treatments, and was significantly later in the residue-free control (Table 4.3). The onset of LLS was numerically and significantly earlier in plots with *C. personatum*-infested residue, at the FireTower field in 2014, and for all split-block trials, respectively (Table 4.3). LLS was always last to appear in plots with *C. arachidicola*-infested residue (Table 4.3). In all cases, the onset of ELS was earlier than that of LLS. However, the difference between the onset of the two diseases was reduced in plots with *C. personatum* alone, and in plots where no residue was applied; this difference was significant for all the spilt-block trials, but not at the FireTower field in 2014 (Table 4.3).

Fall tillage significantly delayed the onset of ELS at Reidsville in 2014, in which case onset was generally numerically delayed when compared with spring or strip tillage. Likewise, fall tillage

significantly delayed the onset of LLS as compared with strip tillage control. For both diseases, onset tended to be earlier in plots with the strip tillage, followed by the spring tillage and lastly in the fall tillage (Table 4.4). Regardless of tillage treatment, onset of ELS was significantly earlier at the Ponder farm in 2015, and generally latest at Reidsville in 2014 and 2015. LLS onset was significantly earlier at Ponder in 2015, and significantly latest at Reidsville in 2014 (Table 4.4).

Disease progress. The Gompertz model provided the best fit to the incidence progress curves of ELS and LLS. The rate of progress of both diseases was significantly affected by trial and inoculant treatment, and the rate of progress of ELS was significantly affected by tillage, trial × tillage and trial × inoculum (Table 4.2). There was no consistent effect of inoculum treatment on the rate of either disease (Table 4.5). Regardless of inoculum treatment, the rate of progress of LLS was greater than that of ELS, and the difference was almost always significant (Table 4.5). Although the effect of tillage was inconsistent, the rate of disease progress was almost always greater in the fall and spring tillage and lowest in the strip tillage (data not shown).

The maximum increase in the number of LLS lesions, defined as the greatest increase in the number of lesions between two rating dates, was significantly affected by the inoculum level (Table 4.2); however, the maximum increase in the number of ELS lesions was only affected by trial and tillage for ELS (Table 4.2). For LLS, the maximum increase in lesion number was greatest in plots with *C. personatum*-infested residue and in plots that did not receive any residue (Table 4.7). Similar to the rate of incidence, the difference in the maximum increase in lesion number between the two diseases was numerically and often significantly greater for LLS except in plots where *C. arachidicola* residue was applied (Table 4.7). Tillage had only a minor effect on maximum increase in the number of lesions of either disease, but the effect of trial was highly significant for ELS (Table 4.8). In 2014 the maximum increase of the number of ELS lesions was significantly greater at Reidsville than all other trials, and more than 5 times greater than the trial at Reidsville in 2015 (Table 4.8). Conversely, the trial at Reidsville in 2015 had a numerically or significantly greater maximum increase in lesion numbers for all tillage treatments (Table 4.8).

Main stem defoliation increased over the course of the epidemic for all treatments in all trials conducted in these studies (Fig. 4.4), and the Exponential model provided the best fit to the defoliation progress curves. The effects of trial, tillage, trial × tillage, inoculum and trial × inoculum on the time to 50 % defoliation (T_{50}) and on the rate of defoliation were significant (Table 4.2). In 2014, T_{50} was shorter in plots with *C. arachidicola*-infested residue alone or with both leaf spot pathogens compared with plots with *C. personatum* alone and those where no residue was applied (Table 4.5). In 2015, T_{50} in plots with *C. personatum*-infested residue was less than or similar to that in plots with *C. arachidicola*-infested residue (Table 4.5). T_{50} was longest in the untreated plots, but the difference was not always significant (Table 4.5). In all trials except Ponder, fall tillage significantly increased the T_{50} by 4 to 9 days, whereas the spring and strip tillage treatments were never significantly different (data not shown).

In general, the rate of defoliation was greatest in plots that did not receive any peanut residue (Table 4.5). In 2014, the rate of defoliation was highest in plots with *C. personatum*-infested residue, but was only significantly different from plots with *C. arachidicola*-infested residue at Reidsville (Table 4.5). In 2015, the rate of defoliation was not significantly different for any peanut residue treatments (Table 4.5). The effect of tillage on the rate of defoliation was not consistent for any of the treatments (data not shown). Fall tillage had significantly higher rates of defoliation than spring and strip tillage at Reidsville in 2014 and at FireTower in 2015; otherwise, there was no difference among tillage treatments (data not shown).

DISCUSSION

To the best of our knowledge, this is first study to directly compare the epidemics of ELS and LLS when the relative amount of the initial inoculum source of both pathogens is known. Previous reports have stated that the primary inoculum source for both ELS and LLS is derived from mycelial stromata residing within the peanut residue (Hemingway, 1954; Nuesry, 1981; Shanta, 1960). Several studies have indirectly correlated the amount of residue in the soil with crop rotation (Kucharek, 1975) and provided evidence that the level of inoculum decreases with increasing years out of peanut (Brenneman et al., 1995). Our studies provide additional evidence that the leaf spot fungi overwinter

within the peanut residue for at least one season. Furthermore, by periodically collecting infested residue samples from main stems left near the plots in these studies, we confirmed that both leaf spot fungi produce conidia on old lesions as early as late-April (data not shown).

Unsurprisingly, we found that plots treated with *C. arachidicola*-infested residue had the greatest amount of ELS, and plots treated with *C. personatum*-infested residue had the greatest amount of LLS (Fig. 4.1). Similarly, it was expected that both diseases should occur in the same plot since the inoculum was not 100% pure and given the potential for contamination through tillage. Interestingly, however, plots treated with residue infested with both leaf spot fungi generally had more ELS than LLS when compared to treatments consisting of residue infested with only one pathogen (Fig. 4.2). Possible explanations include differences in the actual amount of inoculum between *C. arachidicola* and *C. personatum*-infested residue, differences in spore production from the residue (including timing and amount) and induced resistance, such that initial infections by the ELS pathogen could have triggered resistance to the late leaf spot pathogen. Another explanation is interspecific competition (Fitt et al., 2006), in which *C. arachidicola* may outcompete *C. personatum* for resources. Niche differences might be explained in several ways. Firstly, *C. arachidicola* may be more biologically active earlier in the season, leading to fewer leaflets to infect once *C. personatum* becomes more active. In fact, we recently conducted planting date trials that show that LLS predominance increases incrementally for April, May and June planting dates (Chapter 5). Secondly, recent field and greenhouse trials strongly suggests that Georgia-06G is more susceptible to *C. arachidicola* than *C. personatum* (Chapters 6 and 7). In the same trials, we found that when both pathogens were co-inoculated on the same leaflet, ELS was more abundant, suggesting that an indirect or direct type of antagonism between the two fungi could hinder the development of LLS.

The onset of both diseases was latest in residue-free plots (Table 4.3), yet both diseases eventually occurred, resulting in ~ 100% defoliation (Fig. 4.4). Interestingly, LLS was often statistically higher in these plots than in the plots treated with *C. arachidicola* alone, and numerically higher than plots treated with both fungi (Fig. 4.2). The fact that both diseases occurred in the residue-free plots can

partially be explained by the limitations in the experimental design. In these studies, the inoculum was applied in the late fall/early winter, and small pieces of residue would have been capable of being transported by wind and rain to residue-free plots. Secondly, during the season, the plots were not surrounded with tall buffer rows, but rather by two rows of peanuts on either side. Furthermore, both leaf spot pathogens are disseminated by wind, rain and insects (Shokes and Culbreath, 1997). Conidia of both fungi have been detected at heights > 2.5 m above the peanut canopy (Mallaiah and Rao, 1980; Smith and Crosby, 1973). Alderman et al. (1989) found a steep dispersal gradient for LLS (within 1 m) for the first few weeks after the introduction of an inoculum source, but after the 5th week of detection, a shallow gradient (within 9 m) was observed. Little is known about the long-distance dispersal of either leaf spot pathogen (Shokes and Culbreath, 1997). However, in one study, we found that the onset of both diseases occurred at ~ 111 days after planting in a peanut field in Tifton with no prior history of the crop. The closest known inoculum sources for *C. arachidicola* and *C. personatum* were located 2.6 and 1.2 km away, respectively, suggesting that conidia of both fungi are readily disseminated (Chapter 3). This corroborates Smith and Littrell (1980) who proposed that the initial inoculum was local or derived from a source within a 1.6-km radius. In terms of differences in the predominance, the LLS pathogen is generally considered to produce more spores per lesion, and to have a greater number of lesions per leaflet (Backman and Crawford, 1984; Hemingway, 1955). Although there is no published data to directly support these generalizations just stated, it may be that the LLS pathogen had the advantage in residue-free plots simply by the sheer quantity of inoculum being produced.

In all plots, the proportion of LLS lesions increased over time, but never reached more than 0.9 (Fig. 4.1). This partially confirms previous reports that have stated that the LLS pathogen often overwhelms the ELS pathogen towards the end of the season (Hemingway, 1955; Smith and Littrell, 1980). Surprisingly, however, we found that there were a number of instances where the proportion of LLS lesions decreased sharply towards the latter part of the epidemic (Fig. 4.1). However, it should be noted that this was not a consistent response, and at the point of decrease there were few remaining leaflets as some treatments were completely defoliated prior to the last rating date (Fig. 4.4). Differences

in microenvironment or leaf age susceptibility are two possible explanations to account for this since the few remaining leaflets were located at the top of the main stem (data not shown). Therefore, these would have been the youngest leaflets and exposed to a different environment than that within the canopy. Also, as previously pointed out, the ELS pathogen may have had an advantage due to differences in host resistance.

Our results show that the while the predominance is significantly affected by inoculum treatment, it also varied by year and location (Fig. 4.1). Many researchers have speculated that this is due to LLS thriving in cool, wet conditions (Sommartya and Beute, 1986). The evidence from the literature is that both leaf spot pathogens require prolonged leaf wetness ≥ 12 h, and temperature ranges from 20 to 24°C for optimal infections to occur (Shew et al., 1988; Wu et al., 1999). However, the literature indicates that there are differences in germination and sporulation between the leaf spot pathogens. The optimum temperatures for conidial germination were reported to be 19 to 25°C for *C. arachidicola* (Alderman and Beute, 1986), but only 16 to 20°C for *C. personatum* (Sommartya and Beute, 1986). In the same studies, germination decreased at increasing temperatures beyond the optimum for both fungal conidia, yet the LLS pathogen still had 60 % germination at 28°C, whereas the ELS pathogen was reduced to 40% germination at 28°C. Alderman and Beute (1987) found that *C. arachidicola* had the highest sporulation at 24 to 28°C, but sporulation was three times less at 16°C. Alderman and Nutter (1994) found that *C. personatum* had the highest sporulation at 20°C, and that sporulation was higher at 15°C than at 25°C.

While the environment may be a factor that could shift the predominance of one leaf spot to another, our data indicate that it would have relatively little influence in irrigated fields in Georgia. For example, in 2015, the onset of LLS was 18 days earlier, and the proportion of the lesions attributed to LLS at the trial at Reidsville was approximately twice as much when compared with 2014 (Fig. 4.2). This corresponds to the differences in source of initial inoculum for both of these trials (see Materials and Methods). Briefly, in 2014, the source of *C. arachidicola* came from a field that was ~ 100% defoliated at the end of the season, whereas the source of the *C. personatum* residue came from a field that was ~ 80% defoliated; in 2015, the opposite situation occurred. In contrast, the differences in the behavior of

ELS and LLS in these trials do not match the environmental conditions that would presumably favor LLS (cooler events). For example, the environment at Reidsville was considerably different in 2014 compared to 2015 (Table 4.1). In 2014, Reidsville had similar favorable events in the 18 to 24°C range as in the 20 to 26°C range, with most of each occurring in September. In 2015, Reidsville had more favorable events in the 20 to 26°C range, with almost twice as many in August. In 2014, the number of LLS lesions should theoretically have escalated in September (~110 to 140 DAP), and in 2015, the number of ELS lesions should have theoretically escalated in August and September (~80 to 140 DAP). However, the opposite situation occurred in both years (Fig. 4.1). Therefore, we conclude that the differences we observed in the behavior of ELS and LLS between trials were mainly due to the difference in the quantity of each leaf spot pathogen in the infested residue.

In all cases, the onset of ELS was earlier than that of LLS (Table 4.2). Yet the gap between the two diseases decreased in plots with *C. personatum*-infested residue as well as in the residue-free plots (Table 4.2). The gap was reduced in plots with *C. personatum*-infested residue because the onset of ELS was later and onset of LLS was earlier. In the residue-free plots, the onset of both leaf spots was later thereby decreasing the gap. A similar trend was observed in a recent study by Fulmer et al. (Chapter 3), where the inoculum level was linked to previous disease history. While these studies document the importance of initial inoculum, it is clear that this factor cannot fully explain the differences in the onset of each disease. Similar to the predominance, we have recently found that other factors such as planting date and varietal resistance also affect the difference in the onset of the two diseases (Chapters 5 and 6).

Inoculum did not have a consistent effect on the rate of disease progress for either leaf spot, but did have a major effect on the maximum lesion increase in lesion number of LLS (Table 4.7). Furthermore, our results confirm previous reports (Backman and Crawford, 1984; Hemingway, 1955) that the rate of disease progress (based on incidence or lesion development) is higher for LLS than ELS (Tables 4.5 and 4.7). However, plots with *C. personatum*-infested residue did not always have a higher rate of defoliation (Table 4.5). One explanation is that although the carrying capacity of LLS is greater, i.e., more lesions per leaflet, the physiological effect on the plant may be similar. The difference in the

carrying capacity is partially explained due to the fact that the average size (diameter) of the ELS lesions on the cultivar Georgia-06G is ~1 mm greater than that of LLS (Chapter 7), and therefore more LLS lesions per leaflet area are possible. Interestingly, in most situations, the plots without residue consistently had the highest rates of defoliation (Table 4.5), regardless of the proportion of LLS (Table 4.5). Similar results were observed in plots with fall tillage (data not shown). Several reasons may explain these results: 1) although disease onset occurred later in these plots (Table 4.3), there would have been more leaflets to defoliate, 2) the aerial inoculum load would have been greater at this point in the season, 3) the environment was generally more conducive for disease development towards the end of the season (more leaf wetness hours, Table 4.1), and 4) there is some evidence to suggest that once the main pod load is set, the peanut plant may become more susceptible to both leaf spot pathogens (Tillman, personal communication). Taken together, these factors would allow rapid defoliation to occur, regardless of the causal leaf spot pathogen.

In studies conducted in Tanzania, Hemingway (1954) reported that there was a reduction in disease severity by plowing and harrowing *C. personatum*-infested peanut residue into the soil, but this was not enough to delay disease onset. However, the details of the experiment are minimal, and it is not clear how long the interval was between crops, for example. Our studies indicate that incorporating the infested residue into the soil did not affect the predominance nor the rate of progress for either disease (Fig. 4.3), but did delay onset of both diseases (Table 4.4). Although the effect on disease onset was more consistent for LLS than ELS, lack of a shift in predominance suggests that the inoculum level of each pathogen can be equally reduced by this practice. Further studies would be needed to determine if the survival of either pathogen is actually affected, or if the soil simply provides a physical barrier to the inoculum. Future research would also need to look at the effect of fall tillage in more realistic field scenarios that include longer rotational intervals as this would presumably provide more time for the residue to break down.

In conclusion, we found that the predominance and onset of each disease in a given year and location is largely explained by inoculum source. However, our results show that inoculum alone does

not fully explain the differences, but there were no obvious differences temperature and leaf wetness patterns to suggest that the environmental conditions were responsible for differences in the behavior of the two diseases. Based on our other recent studies, we hypothesize that the predominance and onset of each disease is an additive effect that depends on other factors such as planting date and cultivar, and that more accurate prediction will require knowledge of the interactions of these factors. Furthermore, by treating plots with peanut residue infested with both leaf spot pathogens, we found that the development of LLS was more negatively affected by the presence of ELS than ELS by the presence of LLS. We hypothesize that there is a degree of competitive exclusion in which the ELS pathogen outcompetes the LLS pathogen early in the season, and that this may be an additional factor that influences the prevalence and the delay in the onset of LLS. Our results also suggest that the quantity of initial inoculum can be reduced by incorporating the residue into the ground in the late fall/early winter. Yet defoliation increased exponentially across all treatments for all the trials conducted in these studies. This latter point emphasizes the impact of the infested peanut residue on the epidemics of these diseases, and we therefore suggest that more research be conducted that is geared towards its destruction. A better understanding of the survival of these fungi in relation to current cultural practices such as tillage and crop rotation may help accomplish this end.

ACKNOWLEDGMENTS

We thank the personnel at the University of Georgia research stations for all of the assistance provided. Particularly, we thank John Paulk, Dewayne Dales, Andy Carter, Cliff Riner, Randy Hill, Denny Thigpin, Lina Flor Young, Jeannette Mixon, Jhen Bennett and Edna Shannon for technical support; and Xuelin Luo for statistical assistance. Weather data was kindly provided by University of Georgia Automated Environmental Monitoring Network. A brief summary of this research has previously been published (Fulmer and Kemerait Jr, 2016)

LITERATURE CITED

Alderman, S., and Beute, M. 1986. Influence of temperature and moisture on germination and germ tube elongation of *Cercospora arachidicola*. Phytopathology 76:715-719.

Alderman, S., and Beute, M. 1987. Influence of temperature, lesion water potential, and cyclic wet-dry periods on sporulation *Cercospora arachidicola* on peanut. Phytopathology 77:960-963.

Alderman, S., and Nutter, F. 1994. Effect of temperature and relative humidity on development of *Cercosporidium personatum* on peanut in Georgia. Plant Dis. 78:690-694.

Alderman, S., Nutter Jr, F., and Labrinos, J. 1989. Spatial and temporal analysis of spread of late leaf spot of peanut. Phytopathology 79:837-844.

Backman, P., and Crawford, M. 1984. Relationship between yield loss and severity of early and late leafspot diseases of peanut. Phytopathology 74:1101-1103.

Brenneman, T., Sumner, D., Baird, R., Burton, G., and Minton, N. 1995. Suppression of foliar and soilborne peanut diseases in bahiagrass rotations. Phytopathology 85:948-952.

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

Cantonwine, E., Culbreath, A., and Stevenson, K. 2007. Characterization of early leaf spot suppression by strip tillage in peanut. Phytopathology 97:187-194.

Cantonwine, E., Culbreath, A., Holbrook, C., and Gorbet, D. 2008. Disease progress of early leaf spot and components of resistance to *Cercospora arachidicola* and *Cercosporidium personatum* in runner-type peanut cultivars. Peanut Sci. 35:1-10.

De Nazareno, N., Lipps, P., and Madden, L. 1992. Survival of Cercospora zeae-maydis in corn residue in Ohio. Plant Dis. 76:560-563.

Fitt, B. D., Huang, Y.-J., van den Bosch, F., and West, J. S. 2006. Coexistence of related pathogen species on arable crops in space and time. Annu. Rev. Phytopathol. 44:163-182.

Fulmer, A., and Kemerait Jr, R. 2016. Effect of inoculum level, planting date and variety on the onset and predominance of early and late leaf spot of peanut. Proc. Amer. Peanut Res. Ed. Soc. 48:94 (Abstr.).

Garren, K., and Wilson, C. 1951. Peanut diseases. Pages 262-324 in: The Peanut, the Unpredictable Legume. Nat. Fertilizer Assoc., Washington, D.C.

Hemingway, J. 1954. Cercospora leafspots of groundnuts in Tanganyika. E. Afr. Agricultural Journal 19:263-271.

Hemingway, J. 1955. The prevalence of two species of Cercospora on groundnuts. Trans. Brit. Mycol. Soc. 38:243-246.

Jackson, C. R., and Bell, D. K. 1969. Diseases of Peanut (Groundnut) Caused by Fungi. Research Bull. 56, College of Agric. Exp. Stations University of Georgia., Athens.

Jackson, L. 1981. Distribution and severity of peanut leafspot in Florida. Phytopathology 71:324-328.

Jenkins, W. A. 1938. Two fungi causing leaf spot of peanut. J. Agr. Res. 56:317-332.

Kucharek, T. 1975. Reduction of Cercospora leafspots of peanut with crop rotation. Plant Disease Reporter 59:822-823.

Mallaiah, K., and Rao, A. 1980. Aerobiology of two species of Cercospora pathogenic to groundnut. Proceedings of the Indian National Science Academy, B 46:215-222.

McDonald, D., Subrahmanyam, P., Gibbons, R., and Smith, D. 1985. Early and Late Leaf Spots of Groundnut. Information Bull. No. 21. ICRISAT, Patancheru.

Miller, L. I. 1953. Studies of the parasitism of *Cercospora arachidicola* Hori and *Cercospora personata* (Berk. & M.A. Curtis). Ph.D. Dissertation. University of Minnesota, Minneapolis.

Monfort, W., ed. 2017. 2017 Peanut Update. Coop. Ext. Ser. College of Agric. Environ. Sci. University of Georgia., Athens.

Nuesry, S. M. 1981. Survival of *Cercospora arachidicola, Cercosporidium personatum,* and primary infection of peanuts in Oklahoma. M.S. thesis. Oklahoma State University, Stillwater.

Porter, D., and Wright, F. 1991. Early leafspot of peanuts: effect of conservational tillage practices on disease development. Peanut Sci. 18:76-79.

Rao, A., McDonald, D., and Reddy, K. 1993. Perpetuation of peanut leaf spots pathogens. Oléagineux (Paris) 48:77-82.

Shanta, P. 1960. Studies on Cercospora leaf spot of Groundnuts (*Arachis hypogaea*). I. Effect of the environmental factors on disease incidence and on survival of the pathogen. J. Madras Univ. 30:167-177.

Shew, B., Beute, M., and Wynne, J. 1988. Effects of temperature and relative humidity on expression of resistance to *Cercosporidium personatum* in peanut. Phytopathology 78:493-498.

Shokes, F., and Culbreath, A. K. 1997. Early and late leaf spots. Pages 17-20 in: Compendium of Peanut Diseases, 2nd Ed. N. Kokalis-Burelle, D. Porter, R. Rodriguez-Kabana, D. Smith and P. Subrahmanyam, eds. American Phytopathological Society Press, St. Paul, MN.

Smith, D., and Crosby, F. 1973. Aerobiology of two peanut leafspot fungi. Phytopathology 63:703-707.

Smith, D. H., and Littrell, R. H. 1980. Management of peanut foliar diseases with fungicides. Plant Dis. 64:356-361.

Sommartya, T., and Beute, M. 1986. Temperature effects on germination and comparative morphology of conidia for Thai and USA isolates of *Cercosporidium personatum*. Peanut Sci. 13:67-70.

Wolf, F. A. 1914. Leaf spot and some fruit rots of peanut. Ala. Agr. Exp. Sta. Bul. 180, Alabama Polytechnic Institute Opelika.

Woodroof, N. C. 1933. Two leaf spots of the peanut (Arachis hypogaea L.). Phytopathology 23:627-640.

Wu, L., Damicone, J., Duthie, J., and Melouk, H. 1999. Effects of temperature and wetness duration on infection of peanut cultivars by *Cercospora arachidicola*. Phytopathology 89:653-659.

Table 4.1. Summary of the relevant environmental conditions recorded during the main part of the growing season for each experimental location in 2014 and 2015.

		Temperature (°C)[u]		Rain events[v]		Rainfall	Leaf wetness hours[w]		Number of favorable events[x]		
Year, location, month		Day	Night	>0.25 cm	>0.63 cm	cm	Day	Night	18-24°C	20-26°C	18-28°C
2014											
Tifton											
	June	28.3 ±3.0[y]	22.7 ±1.8	11	4	7	27 ±5	126 ±19	4 ±1	7 ±2	12 ±6
	July	28.3 ±2.9	22.6 ±2.2	10	3	8	65 ±15	171 ±29	6 ±3	9 ±4	11 ±4
	August	29.3 ±3.2	22.3 ±1.6	10	1	4	82 ±22	213 ±24	13 ±6	20 ±5	25 ±6
	September	25.6 ±3.8	21.1 ±2.3	11	7	15	141 ±93	260 ±26	22 ±8	18 ±6	27 ±11
Reidsville											
	June	28.9 ±3.2	22.5 ±1.9	12	6	10	41 ±6	158 ±6	4 ±1	5 ±2	5 ±1
	July	29.6 ±3.0	23.3 ±1.9	5	2	6	50 ±3	165 ±9	3 ±1	4 ±1	5 ±0
	August	29.9 ±3.5	22.6 ±1.7	7	4	5	75 ±20	205 ±32	5 ±2	6 ±2	5 ±1
	September	25.8 ±3.7	22.5 ±4.3	10	6	21	117 ±16	235 ±27	10 ±2	11 ±3	11 ±1
2015											
Tifton											
	June	28.7 ±3.7	22.8 ±2.0	13	5	9	55 ±9	174 ±45	7 ±1	7 ±1	10 ±2
	July	29.3 ±3.4	23.2 ±1.6	14	10	33	80 ±22	239 ±27	5 ±2	10 ±1	14 ±4
	August	28.6 ±3.1	23.0 ±1.7	9	7	11	110 ±34	245 ±39	6 ±1	14 ±2	17 ±4
	September	25.5 ±3.5	20.7 ±2.6	8	4	5	74 ±23	224 ±44	8 ±3	10 ±3	12 ±4
Reidsville											
	June	29.1 ±3.7	23.4 ±2.1	13	3	5	53 ±9	133 ±16	5 ±0	5 ±2	6 ±1
	July	29.8 ±3.4	23.6 ±1.7	13	6	15	84 ±21	217 ±48	7 ±1	9 ±2	12 ±3
	August	28.1 ±2.9	22.7 ±1.4	10	5	10	153 ±58	279 ±61	9 ±4	17 ±6	21 ±7
	September	25.3 ±3.3	20.8 ±2.7	8	4	8	140 ±67	237 ±78	7 ±4	13 ±7	18 ±8
Ponder[z]											
	June	28.6 ±3.7	23.1 ±2.1	11	5	10	-	-	-	-	-
	July	29.3 ±3.3	24.0 ±1.6	14	7	23	-	-	-	-	-
	August	28.5 ±2.9	23.5 ±1.7	8	5	14	-	-	-	-	-
	September	25.4 ±3.4	21.0 ±2.5	8	5	9	-	-	-	-	-

[u] Day= 9:00 a.m. to 8:59 p.m. Night=9 p.m. to 8:59 a.m. Day temperatures were obtained from the nearest Georgia Automated Environmental Monitoring Network station (www.Georgiaweather.net). Night temperature was measured with Decagon devices placed within the peanut canopy.

[v] Days that received > 0.25 cm or > 0.63 cm of rain. Rainfall records were obtained from the nearest Georgia Automated Environmental Monitoring Network station.

[w] Leaf wetness hours = total number of hours measured as wet according to Decagon leaf wetness sensors placed within the peanut canopy.
[x] A favorable event is defined as a period of continuous leaf wetness was ≥ 12 h and during which temperatures ranged between 18 and 24°C, 20 and 26°C or 18 and 28°C.
[y] Standard deviation based on hourly observations recorded by 6 sensors at each location.
[z] All data for this location was obtained from the Georgia Automated Environmental Monitoring Network.

Table 4.2. P-values from the three-way linear mixed model analysis of variance (ANOVA) for all leaf spot-related variables for split-block trials conducted during 2014 and 2015 and a separate one-way linear mixed model ANOVA for the FireTower field in 2014.

Trial,	Predominance[v]	Disease onset[w]		Rate of incidence[x]		Max increase of lesions[y]		Defoliation[z]	
source of variation	ProLLS	ELS	LLS	ELS	LLS	ELS	LLS	Rate	T_{50}
FireTower (2014)									
Inoculum	0.0269	0.0349	0.0846	0.0038	0.4851	0.2185	0.0172	0.0021	<0.0001
Strip-block trials									
Trial (T)	0.0001	0.0001	0.0002	0.0017	0.0039	<0.0001	0.0582	0.0003	0.0005
Tillage (Till)	0.0290	<0.0001	0.0225	<0.0001	0.1494	0.0408	0.9397	<0.0001	<0.0001
T × Till	0.0034	0.0019	0.5781	0.0073	0.4162	0.1696	0.0038	0.0076	<0.0001
Inoculum (Inoc)	0.0002	<0.0001	0.0014	0.0146	0.0269	0.0862	<0.0001	0.0012	0.0003
T × Inoc	0.0139	0.1469	0.4228	0.0194	0.3574	0.2488	0.1283	0.0166	<0.0001
Till × Inoc	0.0353	0.3793	0.0864	0.7719	0.3073	0.0880	0.7532	0.4549	0.7631
T × Till × Inoc	0.1922	0.4985	0.2838	0.1460	0.9049	0.5309	0.3142	0.2911	0.3908

[v] Predominance (proportion of the total leaf spot epidemic attributed to late leaf spot; calculated by dividing the standardized AUDPC of late leaf spot by the total standardized AUDPC of early and late leaf spot).

[w] Number of days after planting to reach > 1 % disease incidence for early leaf spot (ELS) or late leaf spot (LLS).

[x] Rate parameters for both diseases were estimated for fitted Gompertz models of incidence over time.

[y] Maximum rate of increase of lesion numbers per leaflet from one assessment date to the next.

[z] Rate and T_{50} (days to reach 50% defoliation) parameters were estimated from fitted exponential models of % defoliation over time.

Table 4.3. Effect of inoculum treatment on the onset of early and late leaf spot.

	Disease onset[w]		
Trial, inoculum	ELS	LLS	Diff[x]
FireTower (2014)			
Cercospora arachidicola (*C.a.*)	59 b	99 a	39 **[z]
Cercosporidium personatum (*C.p.*)	67 b	80 a	6 ns
C.a. + *C.p.*	56 b	94 a	38 **
None	89 a	96 a	8 ns
Split block trials			
C.a.	58 b	100 a	38 ***
C.p.	61 b	89 b	25 ***
C.a. + *C.p.*	59 b	96 a	31 ***
None	78 a	99 a	16 ***

[w] Number of days after planting to reach > 1 % disease incidence for early leaf spot (ELS) and late leaf spot (LLS).

[x] Difference between the onset of late leaf spot to that of early leaf spot.

[z] Asterisks indicate that differences were significant for each inoculum treatment based upon pairwise t-tests at $\alpha = 0.05$.

Table 4.4. Effect of trial and tillage type on the onset of early and late leaf spot in 2014 and 2015.

	Onset early leaf spot[w]				Onset late leaf spot[w]			
	Tillage treatment				Tillage treatment			
Year, trial	Fall	Spring	Strip	Mean	Fall	Spring	Strip	Mean
2014, Reidsville	90 a[x]	77 b	64 c	77[y]	113	112	108	111 A[z]
2015, Reidsville	78 a	79 a	70 a	76	98	91	91	93 B
2015, FireTower	64 a	51 b	55 ab	57	98	92	88	93 B
2015, Ponder	50 a	46 a	51 a	49	92	89	81	87 C
Average	70	62	60	64	100 a	96 ab	92 b	96

[w] Number of days after planting to reach >1 % disease incidence.

[x] For tillage treatments, means within rows with the same lower case letters are not significantly different based upon Tukey's honestly significant difference test ($\alpha = 0.05$).

[y] Due to a significant trial × tillage interaction for early leaf spot onset, we only show the means of the trial effect. Results are presented in the text.

[z] For trial effect, means within the same column with the same upper case letters are not significantly different.

Table 4.5. Effect of inoculum treatment on the rate of progress of early and late leaf spot incidence, and parameters for the progress of defoliation for each trial conducted in 2014 and 2015.

	Rate of disease incidence[w]			Defoliation[x]	
Year, trial, inoculum[v]	ELS	LLS	Diff	Rate	T_{50}
2014					
FireTower					
C.a.	0.036 b[y]	0.049 a	0.013 n.s[z]	0.012 b	89 c
C.p.	0.037 b	0.055 a	0.018 *	0.017 ab	104 ab
C.a. + *C.p.*	0.032 b	0.053 a	0.021 n.s.	0.015 b	98 b
None	0.052 a	0.057 a	0.005 n.s.	0.022 a	113 a
Reidsville					
C.a.	0.052 a	0.069 b	0.017 *	0.020 c	109 c
C.p.	0.058 a	0.094 ab	0.036 **	0.027 ab	118 b
C.a. + *C.p.*	0.066 a	0.089 ab	0.023 **	0.022 bc	113 bc
None	0.059 a	0.098 a	0.039 **	0.027 a	126 a
2015					
Reidsville					
C.a.	0.038 a	0.070 a	0.032 **	0.017 b	109 ab
C.p.	0.029 c	0.059 a	0.03 **	0.018 b	107 b
C.a. + *C.p.*	0.030 bc	0.055 a	0.025 **	0.018 b	106 b
None	0.036 ab	0.072 a	0.036 ***	0.022 a	114 a
FireTower					
C.a.	0.037 a	0.051 a	0.014 **	0.016 b	102 b
C.p.	0.034 a	0.035 b	0.001 n.s.	0.016 b	101 b
C.a. + *C.p.*	0.033 a	0.043 ab	0.01 n.s.	0.016 b	104 b
None	0.039 a	0.048 ab	0.009 **	0.022 a	113 a
Ponder					
C.a.	0.042 a	0.069 a	0.027 ***	0.024 a	95 a
C.p.	0.041 a	0.068 a	0.027 ***	0.025 a	97 a
C.a. + *C.p.*	0.042 a	0.069 a	0.027 **	0.024 a	95 a
None	0.048 a	0.081 a	0.033 **	0.028 a	99 a

[v] Peanut residue infested with *Cercospora arachidicola* (*C.a.*) or *Cercosporidium personatum* (*C.p.*) or *C.a.* + *C.p.*

[w] Rate parameters for both diseases were estimated for fitted Gompertz models of incidence over time. Diff = difference between the disease progess rate (1/day) of early (ELS) and late (LLS) leaf spot.

[x] Rate of defoliation (1/day) and T_{50} (days to reach 50 % defoliation) were estimated from fitted exponential models of defoliation over time.

[y] Means within columns with the same letters are not significantly different based upon Tukey's honestly significant difference test ($\alpha = 0.05$).

[z] Asterisks indicate that differences were significant for each inoculum treatment based upon pairwise t-tests at $\alpha = 0.05$; n.s. = not significant.

Table 4.7. Effect of inoculum level on the maximum increase in numbers of early and late leaf spot lesions per leaflet in 2014 and 2015.

	Maximum lesion increase[x]		
Trial, inoculum	ELS	LLS	Diff
FireTower (2014)			
C. arachidicola	0.85 a[y]	1.07 b	0.22 ns[z]
C. personatum	0.54 a	2.61 a	2.07 *
C.a. + C.p.	0.72 a	1.46 ab	0.74 ns
None	0.43 a	2.15 ab	1.72 ns
All other trials			
C. arachidicola	0.20 a	0.18 c	-0.02 ***
C. personatum	0.17 a	0.33 a	0.16 ***
C.a. + C.p.	0.19 a	0.23 c	0.04 ***
None	0.15 a	0.27 a	0.12 ***

[x] Maximum increase in number of lesions per leaflet that occurred between two rating dates. Diff = difference between the maximum increase in number of early (ELS) and late (LLS) leaf spot lesions.
[y] Means within columns with the same letters are not significantly different based upon Tukey's honestly significant difference test (α = 0.05).
[z] Asterisks indicate that differences were significant for each inoculum treatment based upon pairwise t-tests at α = 0.05; n.s. = not significant.

Table 4.8. Effect of trial and tillage type on the maximum increase in numbers of early and late leaf spot lesions per leaflet in 2014 and 2015.

	Max. increase of ELS lesions[w]				Max. increase of LLS lesions[w]			
	Tillage treatment				Tillage treatment			
Year, trial	Fall	Spring	Strip	Mean	Fall	Spring	Strip	Mean
2014, Reidsville	0.32	0.44	0.32	0.36 A[x]	0.23 AB	0.20 B	0.18 A	0.20
2015, Reidsville	0.06	0.09	0.06	0.07 C	0.37 A	0.42 A	0.27 A	0.35
2015, FireTower	0.16	0.17	0.19	0.17 B	0.27 AB	0.27 AB	0.32 A	0.29
2015, Ponder	0.18	0.21	0.15	0.18 B	0.14 B	0.17 B	0.24 A	0.18
Mean	0.17 a[y]	0.21 a	0.17 a	0.20	0.25 [z]	0.26	0.25	0.26

[w] Maximum increase in number of lesions per leaflet that occurred between two rating dates.
[x] For trial effect, means within the same column with the same upper case letters are not significantly different (α = 0.05).
[y] For tillage treatments, means across rows with the same lower case letters are not significantly different based upon Tukey's honestly significant difference test (α = 0.05).
[z] Due to a significant trial × tillage interaction for late leaf spot, we only show the means of the tillage effect. Results are presented in the text.

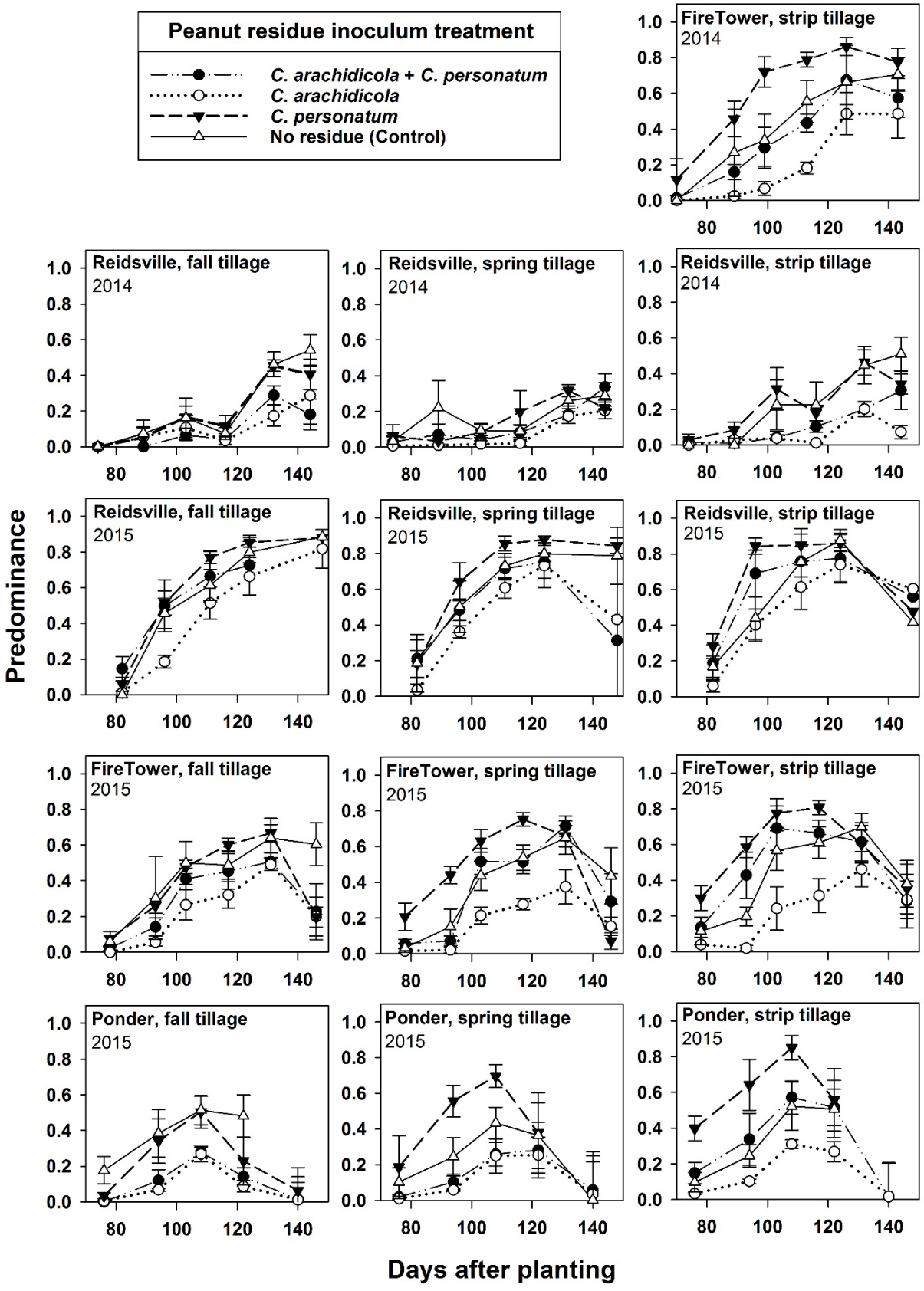

Peanut residue inoculum treatment
C. arachidicola + C. personatum
C. arachidicola
C. personatum
No residue (Control)
FireTower, strip tillage
2014
Reidsville, fall tillage
2014
Reidsville, spring tillage
2014
Reidsville, strip tillage
2014
Reidsville, fall tillage
2015
Reidsville, spring tillage
2015
Reidsville, strip tillage
2015
FireTower, fall tillage
2015
FireTower, spring tillage
2015
FireTower, strip tillage
2015
Ponder, fall tillage
2015
Ponder, spring tillage
2015
Ponder, strip tillage
2015
Predominance
Days after planting

Fig. 4.1. Estimates of the mean late leaf spot predominance (proportion of the total leaf spot epidemic attributed to late leaf spot; calculated by dividing the number late leaf spot lesions by the total number of early and late leaf spot lesions at each rating date) over time for each tillage and inoculum treatment in the trials conducted in 2014 and 2015. Error bars indicate the standard error of the mean based on four replications.

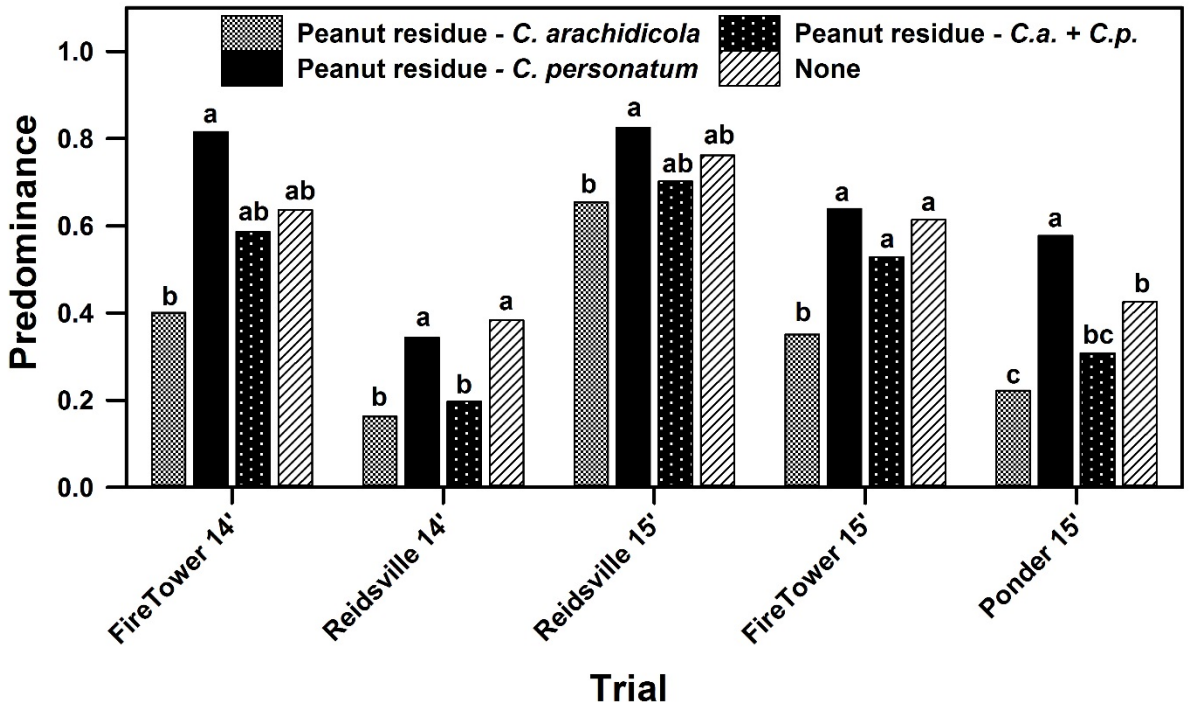


Fig. 4.2. Effect of inoculum on predominance (proportion of the total leaf spot epidemic attributed to late leaf spot; calculated by dividing the standardized AUDPC of late leaf spot by the total standardized AUDPC of early and late leaf spot combined). The inoculum treatments consisted of peanut residue infested with *Cercospora arachidicola*, *Cercosporidium personatum*, a 50/50 mixture of *C. arachidicola* + *C. personatum* and an untreated control. Estimates of the mean were pooled across tillage treatments via the SLICE statement in SAS 9.4 for all trials except the FireTower field in 2014 which did not have the tillage factor included. Grouped bars for each trial with the same letters are not significantly different based upon Tukey's honestly significant difference test ($\alpha = 0.05$).

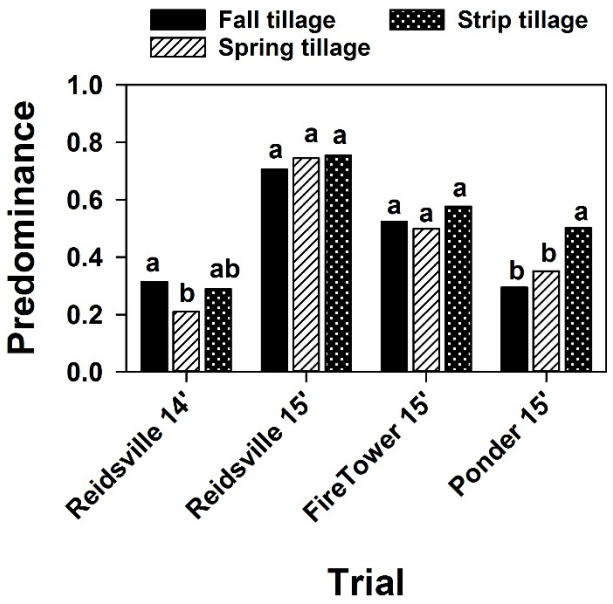


Fig. 4.3. Effect of tillage treatment on predominance (proportion of the total leaf spot epidemic attributed to late leaf spot; calculated by dividing the standardized AUDPC of late leaf spot by the total standardized AUDPC of early and late leaf spot combined). Estimates of the mean were pooled across inoculum treatments via the SLICE statement in SAS 9.4. Grouped bars for each trial with the same letters are not significantly different based upon Tukey's honestly significant difference test ($\alpha = 0.05$).

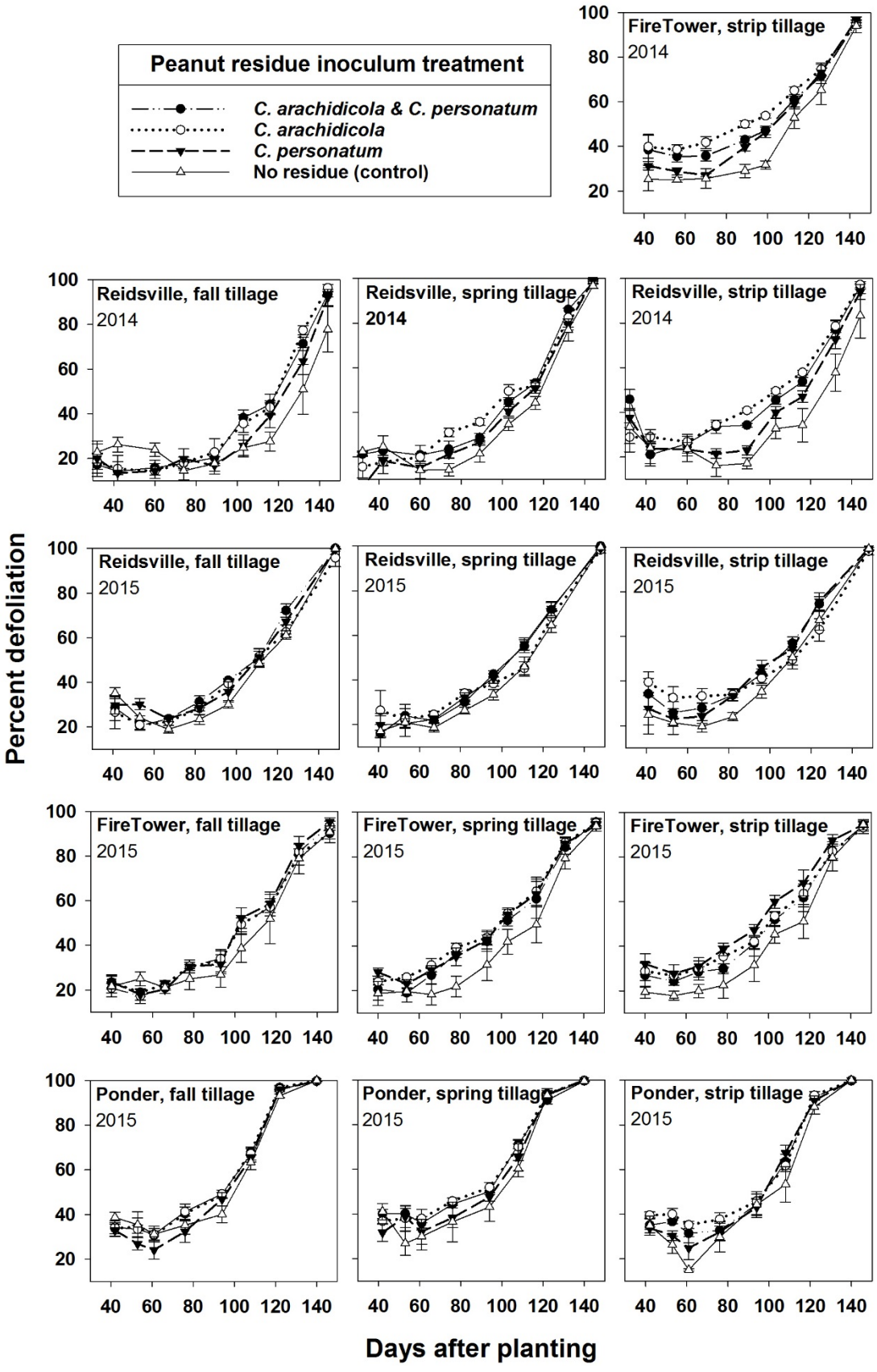

Peanut residue inoculum treatment
C. arachidicola & C. personatum
C. arachidicola
C. personatum
No residue (control)
FireTower, strip tillage
2014
Reidsville, fall tillage
2014
Reidsville, spring tillage
2014
Reidsville, strip tillage
2014
Reidsville, fall tillage
2015
Reidsville, spring tillage
2015
Reidsville, strip tillage
2015
FireTower, fall tillage
2015
FireTower, spring tillage
2015
FireTower, strip tillage
2015
Ponder, fall tillage
2015
Ponder, spring tillage
2015
Ponder, strip tillage
2015
Percent defoliation
Days after planting

Fig. 4.4. Effect of different inoculum treatments on the defoliation of peanut over the course of the epidemic in plots with different tillage treatments in the 2 years that these trials were conducted. The inoculum treatments consisted of peanut residue infested with *Cercospora arachidicola*, *Cercosporidium personatum*, a 50/50 mixture of *C. arachidicola* + *C. personatum* and an untreated control. Data points signify mean defoliation, and error bars indicate the standard error of the mean based on four replications.

CHAPTER 5

INTERACTIVE EFFECTS OF INOCULUM LEVEL AND PLANTING DATE ON THE TEMPORAL DYNAMICS OF EARLY AND LATE LEAF SPOT OF PEANUT[1]

[1]Fulmer, A.M., Kemerait, R.C., Brenneman, T.B., Scherm, H., Cantonwine, E.G., Culbreath, A.K., and Stevenson, K.L. 2017. To be submitted to *Plant Disease*.

ABSTRACT

The temporal dynamics of early (ELS) and late (LLS) leaf spot of peanut, caused by *Cercospora arachidicola* (*Ca*) and *Cercosporidium personatum* (*Cp*), respectively, often differ between fields and seasons. To better understand differences in their behavior, field trials were conducted at multiple locations in Georgia and Florida in 2014 and 2015 to determine the effect of planting date and inoculum level/location on the predominance and development of each disease. The peanut cultivar Georgia-06G was planted in late April, mid-May and early June. Plots were amended with infested peanut residue (*Ca* alone, *Cp* alone, a 50/50 mix of *Ca* + *Cp* and a non-treated control), or had a history of ELS or LLS. Across trials, LLS predominance (proportion of the total leaf spot epidemic attributed to LLS) ranged from 0.009 to 0.95, and tended to increase with later planting dates in fields or plots with more *Ca* inoculum, but not in fields with a history of LLS or in plots treated with *Cp* alone. The onset (days after planting (DAP) to reach 1% incidence) of both leaf spots, and their difference, generally decreased with later planting dates. Across trials, ELS onset ranged from ~ day of year (DOY) 160 to DOY 260, and LLS onset ranged from ~ DOY 200 to DOY 260. Planting date and inoculum treatment significantly affected the time to reach 50% incidence (T50%) for both diseases. T50% ELS ranged from 82 to 134 DAP and 92 to 140 DAP, and LLS ranged from 103 to 140 and 96 to 139 DAP in 2014 and 2015, respectively. In 2014, ELS generally reached T50% prior to LLS, but the difference was least for the June planting date. In 2015, LLS reached T50% prior to ELS in all but one location. In all trials, defoliation at 130 DAP was significantly lower for peanuts planted in April compared to those planted in June.

INTRODUCTION

Two of the most damaging foliar diseases of peanut are caused by the fungi *Cercospora arachidicola* (causing early leaf spot) and *Cercosporidium personatum* (causing late leaf spot) (Shokes and Culbreath, 1997). Both are globally distributed, polycyclic diseases that can coexist in the same field and even on the same leaflet. Together or separately, they can cause complete defoliation of the peanut plant, resulting in yield losses > 50% in unsprayed, high-risk fields (Backman and Crawford, 1984; Shokes and Culbreath, 1997). In Georgia, management relies heavily on fungicide inputs, but integrated management strategies that combine varietal resistance, rotation, knowledge of previous disease history, tillage, irrigation and planting date have recently been very successful in reducing the overall severity of leaf spot epidemic with fewer fungicide applications (Cantonwine et al., 2006; Woodward et al., 2010).

Because of similarities in appearance, disease cycle and potential yield loss, both leaf spots are generally managed as a single disease (Garren and Wilson, 1951). However, the relative abundance of each disease varies in space and time. In Georgia, late leaf spot (LLS) was predominant from 1976 to the early 1990s; otherwise early leaf spot (ELS) has generally been the most prevalent (Cantonwine et al., 2008; Smith and Littrell, 1980). In recent years, the prevalence of LLS has increased and LLS predominance over ELS varies for each field (Chapter 3). Furthermore, these diseases generally differ in relation to onset and rate of progress. Depending on predisposing field risk, the onset of both diseases can range from 30 to 140 days after planting (DAP) (Chapter 8), but ELS generally appears 2 to 4 weeks prior to LLS (Shokes and Culbreath, 1997; Smith and Littrell, 1980). Nevertheless, LLS is thought to be more destructive of the two due to a greater rate of increase (Backman and Crawford, 1984; Hemingway, 1955; Jenkins, 1938).

It is still not clear what factors drive the variations in predominance or the differences in disease onset. Smith and Littrell (1980) speculated that rotation, variety, fungicide chemistry and/or program and late harvest dates could be factors that affect the differences in the behavior of ELS and LLS. Others have proposed that prolonged cool, wet weather favors the development of LLS relative over that of ELS (Sommartya and Beute, 1986). In a recent study, we characterized the effect of initial inoculum on the

behavior of both leaf spot diseases (Chapter 4). Our results showed that the predominance and onset of each disease was largely explained by the initial inoculum source, but not to any obvious environmental patterns (Chapter 4). However, an inoculum source consisting of a 50/50 mix of peanut residue infested with each leaf spot pathogen did not significantly accelerate the onset of LLS. Furthermore, these plots tended to have more ELS than residue-free plots where the onset of both diseases was delayed and greater levels of LLS were observed (Chapter 4). While the inoculum source was clearly a major factor for predominance and onset in these studies, it did not fully explain the variability observed (Chapter 4).

Planting date may be a factor that could influence the differences in the dynamics of epidemics of these two diseases. As part of the management principle of avoidance, adjusting the planting date may allow the host to evade contact with the pathogen. This is an important cultural practice recommended in Georgia as part of an integrated disease management program to manage *Tomato spotted wilt virus* (TSWV) (Culbreath et al., 2010) and leaf spot (Kemerait et al., 2017). In Georgia, peanuts are planted from early April to mid-June, thus providing a large window of opportunity to avoid certain diseases. Early plantings are exposed to cooler temperatures early in the season while late planted peanuts are exposed to cooler temperatures late in the season. Manipulation of planting date could therefore lead to a differential effect for more than one disease.

The effect of planting date on leaf spot has been reported in several studies. Hemingway (1954) evaluated the effect of five different planning dates ranging from mid-January to mid-March on the development of late leaf spot in Tanzania and found that time to defoliation increased linearly with each consecutive planting date. In Senegal, Chevaugeon (1952) reported a similar trend for LLS across five planting dates ranging from 15 June to 25 July. In contrast, Farrell et al. (1967) reported that the development of ELS in Malawi decreased linearly when planted during the first weeks of December, January and February. In the southeastern United States, where both leaf spots are present, Shokes et al. (1982) reported a linear increase of LLS severity in peanuts planted in late April, early May and mid-May. In a more recent study in Georgia, Jordan and Culbreath (2016) evaluated six planting dates from early April to late May and found that LLS increased with each of the consecutive later planting dates.

Together, these data demonstrate the effect of planting date on the overall leaf spot epidemic. However, the comparative effect on the temporal development of each leaf spot is still unknown.

Detailed knowledge of disease dynamics is requisite for recommending the most effective integrated disease management programs (Fry, 1982). Given the increased prevalence of LLS in Georgia in recent years, a more detailed understanding of the mechanisms involved in the differing behavior of the two diseases comprising the peanut leaf spot complex is essential. Therefore, the primary objective of this study was to test for interactive effects of inoculum level and planting date on the temporal dynamics of both ELS and LLS. A secondary objective was to assess the overall effect of planting date on the general leaf spot epidemic under a wide range of locations and environments.

MATERIALS AND METHODS

Field sites and experimental design. Split-block trials were conducted at the Vidalia Onion and Vegetable Research Center located in Reidsville, Georgia, in 2014 and 2015. Factors included planting date and inoculum source with four replications. Treatments included three target planting dates: late April, mid-May and early June, but in some cases actual dates did not match target dates due to weather interference (see Table 5.1 for specific dates). Inoculum treatments consisted of four combinations of infested peanut residue: *C. arachidicla* (*Ca*) alone, *C. personatum* (*Cp*) alone, a 50/50 mix of *Ca* + *Cp* and a non-treated control. Peanut residue was collected from fields in Tifton, Georgia, with severe epidemics of either (>90%) ELS or LLS, and stored separately. In most cases, the residue came from untreated plots that had nearly 100% severity (a "10" on the Florida 1-10 scale). However, in 2014 at Reidsville, the *Cp* inoculum came from a field with ~ 80% severity, and in 2015, the *Ca* inoculum at Reidsville came from a trial with ~ 70 % severity. Peanut residue including leaflets, petioles and stems, was collected within a few days after harvesting peanuts (late October and November) stored in a large peanut wagon under an open shed for 4 to 6 weeks. In December, plots were harrowed and rototilled, and dry peanut residue (~4.5 kg) for each treatment was spread across the middle (4.5 m) section of each plots (1.8 × 7.6 m) as evenly as possible. Each plot was bordered by 1.8 m residue free buffer, and was separated by a 3-m alley between each block. Fields used in these studies had been out of peanut for

more than 10 years, or had no prior history of peanut production. For each planting season, the peanut residue was collected in the fall, and allowed to overwinter in the field. Approximately 4 weeks prior to planting, plots were treated with an application of glyphosate (Roundup 4 EC, Monsanto, Kansas City, MO) at 1.2 kg a.i./ha in order to kill emerged vegetation. Just prior to planting, a KMC (Kelley Manufacturing Co., Tifton, GA) rip/strip implement with two shanks with dragging depth of 45.7 cm. was used to prepare two rows (~ 10 to 15 cm wide) for seeding.

Additional experiments with the same target planting dates were conducted in 2014 and 2015 at the Coastal Plain Experiment Station in Tifton, Georgia, the Attapulgus Research and Education Center in Attapulgus, Georgia, and the North Florida Research and Education Center in Marianna, Florida. An additional trial was also conducted at the Plant Science Research and Education Unit in Citra, Florida during 2014. The purpose of these studies was to provide additional locations with different inoculum levels of *Ca* and *Cp* based upon knowledge of previous field history. All trials were laid out in a randomized complete block design with four replications, and included a non-planted border bed (1.8 m) between plots and 3-m alley between blocks.

For the trials in Tifton, the same field was used in both years. In 2014, plots with no prior history of peanut were amended with a 50/50 mix of *Ca* + *Cp* using the same quantity of residue as that of Reidsville. In 2015, peanuts were planted back to the same plots. Approximately 4 weeks prior to planting, plots were treated with an application of glyphosate (Roundup 4 EC, Monsanto, Kansas City, MO) at 1.2 kg a.i./ha in order to kill emerged vegetation. Just prior to planting, a KMC (Kelley Manufacturing Co., Tifton, GA) rip/strip implement with two shanks with dragging depth of 45.7 cm. was used to prepare two rows (~ 10 to 15 cm wide) for seeding in plots measuring 1.8 m x 7.6 m.

For the trials at Attapulgus, the same field was used in both years. In 2013 the field had been planted to peanut and had a severe ELS (>90% of total lesions were ELS) epidemic in all plots. Field plots were prepared with conventional tillage using a moldboard plow. In Marianna, trials were conducted in different sections of the research farm in 2014 and 2015. Each year, peanut followed a previous cropping sequence of corn, cotton, and peanut, and each site had a previous history of LLS

(>90% of total lesions were LLS). In Citra, the field had been planted the previous 2 years with sesame and cotton, and continuously cultivated peanuts with a history of ELS were located in nearby fields (0.5 km). At all locations, blocks were separated by a 3-m alley and plots were 1.8 m wide by 4.6 m long at Marianna and 7.6 m long at the other locations.

In all trials, two-row plots spaced 0.91 m apart were planted to the cultivar Georgia-06G at a rate of four to six seed per 30.5 cm (see Table 5.1 for planting dates). At 60 and 90 days after planting (DAP), flutolanil (Convoy, Nichino America) was applied to all plots at a rate of 0.91 kg/ha to suppress stem rot caused by *Sclerotium rolfsii*. Applications were made with a CO_2-pressurized backpack sprayer, and a boom equipped with four 8002 flat tip nozzles (Teejet Technologies, Springfield, IL) calibrated to deliver 188 liters/ha. Plots were supplemented with irrigation as needed and all herbicides, insecticides and fertilizers were applied following recommendations of the University of Georgia Cooperative Extension Service (Monfort, 2017).

Disease assessment. Beginning approximately 30 days after planting (DAP), plots were visually monitored for leaf spot symptoms. Upon detecting the first leaf spot symptoms, a more intensive evaluation was initiated and continued approximately on a bi-weekly schedule until harvest. Five main stems per plot were arbitrarily selected for destructive sampling and transported to the lab where the number of present and missing leaflets, and the number of ELS and LLS lesions per leaflet were counted. ELS and LLS were differentiated by symptoms and signs associated with each causal pathogen.

Incidence of each disease for each rating date was calculated by dividing the number of leaflets with at least one lesion by the total number of leaflets counted across all five main stems. Area under the disease progress curve (AUDPC) was calculated for each leaf spot disease based on the number of lesions per leaflet. AUDPC values were standardized (stAUDPC) by dividing AUDPC values by the duration of the epidemic (Campbell and Madden, 1990). Predominance was defined as the proportion of the total leaf spot epidemic attributed to LLS, and was calculated by dividing the stAUDPC of LLS by the total stAUDPC of ELS and LLS. Time of disease onset was defined as the number of DAP for ELS or LLS to

reach 1% incidence. For lesion counts, the maximum number of ELS and LLS lesions per leaflet was the highest value that occurred during the season.

Temporal progress of ELS and LLS incidence and of percent defoliation were modeled using linear regression to fit linearized forms of the exponential [log(y)], logistic [ln(y/1-y)] and Gompertz [-ln(-ln y)] models on days after planting. The coefficient of determination (R^2) and mean square error (MSE) from regression of backtransformed predicted on observed values were then compared. Residual plots (observed – predicted values) were also examined for each model. The model with the highest R^2, lowest MSE and the best residual distribution was selected as the best fit for the data. If the model failed to fit the data satisfactorily, parameter estimates were excluded from the analysis. The rate of increase in in incidence and defoliation and time to reach 50 % incidence (T50%) were estimated based on the model with the best fit. Due to differences in rating dates and some treatments with 100% defoliation values prior to 140 DAP, defoliation at 130 DAP was estimated based on the model with the best fit, and used as a measure of severity.

Statistical analysis. Predominance, disease onset, rate of disease incidence, T50%, maximum number of early and late leaf spot lesions and defoliation at 130 DAP were subjected to linear mixed model analysis of variance with PROC GLIMMIX (SAS 9.4 Institute, Cary, NC). Due to differences in planting dates between years, all locations were analyzed separately by year. At Reidsville, the model was analyzed as a strip block design with planting date and inoculum considered as fixed effects, and replication, replication × planting date and replication × inoculum as random effects. The SLICE option was used to explore two-way interactions when necessary. For all other locations, the planting date was considered a fixed effect and replication was considered a random effect. In all analyses, the Kenward-Roger option was used to adjust the denominator degrees of freedom due to repeated measures, and differences in the least-square means were tested by Tukey's multiple comparisons test. When data violated the assumptions of normality, dependent variables were transformed as follows: predominance was arcsin square root transformed, and the square root transformation was used for disease onset, maximum lesion number and T50%. Back-transformed means of all transformed variables are presented

in the results. To explore the relationship between planting date (day of year) and onset (day of year and day after planting) of ELS and LLS, Pearson correlation coefficients were calculated using PROC CORR in SAS.

RESULTS

Environmental conditions. Overall, 2014 was a drier year than 2015, but variations were observed among locations (Table 5.1). Generally, precipitation patterns were more similar for northern locations (Tifton and Reidsville), or the more southwestern locations (Attapulgus and Marianna) (Table 5.1). In 2014, May and September generally had the greatest precipitation, and, with the exception of Citra, August was the driest month; Attapulgus, Marianna and Citra also had high levels of rainfall in July (Table 5.1). In 2015, July and August were extremely wet at Tifton and Reidsville, and May and September were the driest; however, Attapulgus and Marianna had more rainfall than the other locations in June, July and September (Table 5.1). Mean air temperatures were similar across years and locations (Table 5.1). The lowest temperatures were observed in May and September, and there was little difference in June, July and August (Table 5.1).

Predominance. In 2014 in Reidsville, planting date, inoculum and planting date × inoculum had highly significant effects on predominance, but in 2015, only inoculum was significant (Table 5.3). Based on comparisons of planning dates within inoculum treatments in 2014, only plots with *C. personatum*-infested residue were not significantly affected by planting date (Fig. 5.1). Planting date also had a significant effect in Tifton in 2014 and in Attapulgus in 2015 (Table 5.3). In cases where the planting date was significant, LLS predominance increased with later planting dates (Fig. 5.1).

Similar to other recent trials (Chapter 4), plots with *C. personatum*-infested residue had significantly higher proportions of LLS than those with *C. arachidicola*-infested residue, and numerically more LLS than plots inoculated with residue infested with both pathogens. Untreated plots had numerically and often significantly more LLS than plots treated with residue infested with both pathogens or with *C. arachidicola* alone (data not shown). However, the effect of inoculum level became less evident with later planting dates (Fig. 5.1). In 2014, comparison of inoculum treatment within planting

date showed that there was no significant difference among inoculum treatments for the planting date in June, but that there were significant differences among inoculum treatments in April and May (data not shown). Similarly, predominance was greatly affected by location (Fig. 5.1). Regardless of year, Attapulgus was almost exclusively ELS, Marianna exclusively LLS, and Tifton had similar levels of both; Citra was predominantly ELS in 2014 (Fig. 5.1). In all of these situations, the final predominance at each location reflected the historical leaf spot prevalence (ELS, LLS or both diseases) at each location.

Disease onset. With the exception of Marianna, the onset of ELS was earlier than that of LLS across planting dates, inoculum levels and locations (Fig. 5.2). Furthermore, the time to the onset of both diseases tended to decrease with later planting dates, but this trend differed based upon inoculum treatment and location with differing disease histories. Overall, time of onset of LLS decreased the most between the April and June planting dates (Fig. 5.2), and the onset of LLS was more consistently affected by planting date than the onset of ELS regardless of inoculum treatment and location (Table 5.3). In Reidsville, the effect of planting date on the onset of both diseases was significant in both years although there was a significant planting date × inoculum interaction for LLS in 2014 (Table 5.3). ELS was only significantly affected by planting date in Citra, but in all other locations, LLS was always significantly affected by planting date (Table 5.3). Generally, in plots planted in April, disease onset was significantly later than those planted in June, but neither planting date was significantly different from disease onset in plots planted in May (data not shown).

The effect of planting date on the onset of both diseases was also dependent upon inoculum treatment and locations with differing disease histories (Table 5.3). Onset of ELS was not significantly affected planting date at Attapulgus (a high -risk ELS situation), and at Marianna (presumably due to low inoculum levels of *C. arachidicola*). Generally, onset of LLS was significantly earlier in plots treated with *C. personatum*-infested residue (Fig. 5.2) and at locations with a prior history of LLS (e.g., Marianna). As a result, these treatments resulted in little or no difference between the onset of the two diseases (data not shown). Likewise, the difference between the onset of the two diseases was smallest in plots where no residue was applied and in the June planting dates (across inoculum treatments) (Fig. 5.2).

Across years, locations and inoculum levels, onset of both diseases based on day of year (DOY) and DAP was significantly correlated with planting date (DOY) (Fig. 5.3). For the onset of both diseases, planting date was positively with DOY and negative correlated with DAP. However, the highest correlation coefficient for ELS onset occurred with DOY (0.45), whereas onset based on DAP provided the best correlation coefficient (-0.54) for LLS (Fig. 5.3). Although more variable, similar correlations were observed when analyzed by year, location and inoculum level (data not shown).

Disease progress. The Gompertz model provided the best fit to the incidence progress curves of ELS and LLS. The rate of disease progress for both diseases was not consistently affected by planting date, inoculum level or location (Table 5.3). However, the rate of disease progress of both diseases tended to increase with later planting dates, and was statistically higher in plots planted in June than April in 2014 in Reidsville (data not shown). When analyzed across all trials, the rates of progress of ELS and LLS were positively correlated with planting date. Correlation coefficients were 0.217 and 0.292 and associated *P*-values were 0.0048 and 0.0002, respectively.

Across years, time to reach 50 % incidence (T50%) was significantly affected by planting date and inoculum level in Reidsville, and by planting date at almost all other locations (Table 5.3). For both years, ELS T50% in April plantings was significantly longer compared to the June plantings, but T50% for April, May and June plantings were almost all statistically different for LLS (Table 5.4). Similar to onset, inoculum treatment had a major effect on the T50% of both diseases (Table 5.3). In Reidsville, ELS epidemics in plots treated with residue infested with *C. arachidicola* and *C.a. + C.p.* reached T50% ELS before those in plots treated with residue infested with *C. personatum* or with no residue; conversely, LLS epidemics in plots treated with residue infested with *C. personatum* or with no residue reached T50% LLS before those in plots treated with residue infested with *C. arachidicola* and *C.a. + C.p.* (data not shown).

The difference in the T50% of ELS and LLS was similar to onset in 2014, but not for 2015 (Table 5.4). In 2014, T50% ELS was always less than T50% LLS, but the differences were smaller (or reversed) in those plots planted in June. In 2015, in all locations except Attapulgus T50% LLS was prior to T50%

ELS, regardless of planting date, and the difference was generally significant (Table 5.4). The effect of planting date was similar for all inoculum treatments, but plots treated with *C. personatum*-infested residue were the least affected (Table 5.4).

The maximum number of ELS and LLS lesions per leaflet was not consistently affected by planting date or inoculum level (Table 5.3). Planting date did have a highly significant effect on maximum LLS lesions in 2014 (Table 5.3), and lesion counts were significantly higher lesion counts in later plantings than in April plantings (Table 5.5). Inoculum treatment also had a significant effect in 2014 at Reidsville (Table 5.3). Plots treated with residue infested with *C. arachidicola* or *C.a.* + *C.p.* had statistically greater ELS lesions counts per leaflet compared with those inoculated with *C. personatum* or with no residue. The reverse was observed for LLS (data not shown). Similarly, there were significant differences between maximum ELS and LLS lesion counts at each location, with the highest counts consistent with previous disease history of each location (Table 5.5). Fields with a history of LLS had higher LLS lesion counts and vice versa (Table 5.5).

The effect of planting date on main stem defoliation at 130 DAP was highly significant across all locations and years in which these studies were conducted (Table 5.3). Regardless of inoculum level, year or location, defoliation increased with later planting dates, and % defoliation in April plantings were statistically lower than June plantings in all cases (Fig. 5.4). In 2014 and 2015 in Reidsville, there was a significant planting date × inoculum interaction (Table 5.3), and comparisons of inoculum treatment within planting date revealed that in plots planted in April or May, defoliation was greater in plots with peanut residue compared with the untreated check, but there was no difference in defoliation among inoculum treatments in plots planted in June (data not shown). Defoliation differed between locations and between years within location, but there was no consistent pattern in fields with differing disease histories (Fig. 5.4).

DISCUSSION

In these studies, planting date influenced the comparative development of ELS and LLS, but the magnitude of the effect greatly depended on the quantity and or proximity of the initial inoculum source of each leaf spot pathogen. Furthermore, while both diseases were affected by planting date, the results from these studies suggest that planting date has a greater impact on the onset, rate of progress and prevalence of LLS than ELS. To the best of our knowledge this has not previously been specifically linked with planting date. However, although we report differences in the predominance and development of each disease, these studies confirm previous reports that LLS-incited defoliation is reduced by planting peanuts earlier in the season, (Jordan and Culbreath, 2016; Shokes et al., 1982), and also demonstrate that earlier planting dates can be an effective management tactic for reducing ELS-incited defoliation.

We found that LLS tended to be more predominant in later plantings, but differences were observed between inoculum treatments and locations. When the primary local inoculum in the field was almost exclusively comprised of the LLS pathogen or treated with peanut residue infested with *C. personatum*, there is little effect of planting date on predominance, but more of an effect is seen in plots treated with peanut residue infested with *C. arachidicola* or *C.a. + C.p.* or in locations with a history of ELS. For example, in Marianna, no shift in the prevalence of ELS to LLS was observed across planting dates, but in Attapulgus LLS numerically or statistically increased in plots planted in June in both years (Fig. 5.1). However, when both leaf spot pathogens were present in the field in roughly equal quantities, the effect of planting date became more obvious. This was the case in 2014 in Reidsville and Tifton, but the same response was not seen in these locations in 2015 (Fig. 5.1). We believe that this may be explained by a greater amount of *C. personatum* inoculum in the latter year. The peanut residue infested with *C. arachidicola* used in the experiment at Reidsville in 2015 was obtained from a moderately infected field of ELS, whereas the residue infested with *C. personatum* was obtained from a field with complete losses to LLS (see Material and Methods). Likewise, the inoculum source at Tifton in 2014 was comprised of an approximately equal mixture of peanut residue infested with each pathogen, but by the

end of the season LLS was more prevalent. Thus, when these same plots were replanted to peanut in 2015, we presume that there was a greater amount of *C. personatum* inoculum.

There are many examples in the literature of the manipulation of planting date as a cultural practice to reduce or avoid disease intensity in many different pathosystems, e.g., (Culbreath et al., 2010; Mani et al., 2016; Marburger et al., 2016), yet there are few studies that actually investigate the differential response of planting date on the coexisting diseases. However, a few reports do link planting date to shifts in prevalence or differential response to some aspect of development. Eyal (1999) reported that planting date was one of several factors led to the shift from *Stagonospora nodorum* to *Septoria tritici* in the UK. In studies conducted on the eyespot of wheat pathogens, *Oculimacula yallundae* and *O. acuformis,* Wan et al. (2005) reported a reduction in the incidence of *O. yallundae* affected by planting date but not for *O. acuformis.* In the larger biological context, planting date has also been shown to affect the species composition of weeds (Milberg et al., 2001) and insects (Shrestha and Parajulee, 2010). When taken together with the data we present in this paper, we provide data and biological precedence in support for the role of planting date as a contributing factor to the relative abundance of ELS and LLS in a given field or season.

The onset of both diseases was generally earlier with later planting dates (Fig. 5.2). In most cases, ELS appeared before LLS, but the difference in onset between the two diseases tended to decrease with later planting dates and in plots that were inoculated with *C. personatum*; the difference also decreased at Marianna, where LLS was predominant (Fig. 5.2). This difference in the onset of the two diseases attributed to planting date was likely partially explained by less variability in the onset of ELS as opposed to that of LLS which tended to decline more sharply in June plantings (Fig. 5.2). This was particularly evident in plots inoculated with both leaf spot pathogens, and at Attapulgus (predominantly ELS with no rotation). Previous research has not specifically focused on the onset of either disease in relation to planting date, but the general assumption has been that LLS appears 3 to 4 weeks after the onset of ELS, assuming the inoculum of both pathogens is present (Smith and Littrell, 1980). While our results demonstrate that the lag between the onset of these diseases is indeed dependent on the inoculum

level of each pathogen, it is a less accurate predictor without consideration of planting date. Nevertheless, the variability we observed in disease onset not correlated with inoculum source and planting date suggests that there are additional contributing factors that need to be determined before we can fully understand the differences in the onset of early and late leaf spot.

Results from these studies illustrate that the rate of progress of both leaf spot diseases was weakly correlated with planting date, yet the response was not statistically consistent (Table 5.3). Both diseases tended to have a slightly higher rate of incidence in the June planting dates. However, the principal difference we observed was the time to reach 50 % incidence (T50%), which illustrates the faster rate of progress of LLS compared with that of ELS, particularly at later planting dates. In spite of a delay in the onset of LLS, the T50% of LLS was much closer to or earlier than ELS (Table 5.4). In 2014 in Reidsville, the difference in T50% between ELS and LLS disappeared by the last planting date (Table 5.4). And in 2015, LLS reached 50% incidence sooner than ELS across inoculum treatments, regardless of planting date. These results support the traditional view that LLS increases at a faster rate than ELS (Backman and Crawford, 1984; Hemingway, 1955), but also suggests that the differential rate of disease development depends on the planting date.

Taken together, these studies indicate that LLS is more active on the runner variety 'Georgia-06G' when peanuts are planted later in the season; conversely, the development of ELS tends to remain relatively stable across planting dates. One way to better understand this is by considering the onset of each disease in relation to day of year (DOY). Accordingly, when assessed across years, locations and planting dates, ELS onset ranged from ~ DOY 160 to 260, (Fig. 5.3) corresponding to early June to mid-September. LLS onset ranged from ~ DOY 200 to 260 (mid-July to mid-September), with the majority occurring from 200 to 240 DOY (mid-July to the end of August) (Fig. 5.3). These results suggest that LLS onset is more of a function of the time of year rather than by limitations of initial inoculum source.

Previous reports have suggested that the LLS is more prevalent toward the season due to cool, wet conditions (Sommartya and Beute, 1986) but our results indicate that the time frame in question differs based upon planting date, i.e., the end-of-season prevalence in April-planted peanuts is different

from that of June-planted peanuts. Furthermore, in terms of disease onset, the majority of LLS actually occurred in late July to the end of August, during the hottest part of the season (Table 5.2). Although these fields were irrigated, rainfall patterns varied greatly from mid-July to late-August, ranging from the some of the driest periods of the season in 2014 to some of the wettest periods of the season in 2015 (Table 5.2). However, because the majority of disease progress of LLS tends to occur in late August and September, regardless of planting date, and given that germination and sporulation of the LLS pathogen is highest at cooler temperatures (16 to 20 °C) (Alderman and Nutter, 1994; Sommartya and Beute, 1986), we cannot exclude the cooler environment as a contributing factor to an increase in LLS abundance later in August and September, particularly when the inoculum source of both leaf spot pathogens is present (Table 5.2).

The tendency of LLS to occur toward the end of July through late August is likely caused by a number of other interacting factors in addition to planting date and inoculum source, such as differences in environment, fungal biology, and host resistance that create a unique ecological niche (Fitt et al., 2006). With respect to ELS and LLS, cooler temperatures in May would theoretically favor the sporulation of *C. personatum* (Sommartya and Beute, 1986), but on a cultivar with more resistance to LLS (e.g., Georgia-06G, the cultivar used in these studies) (Chapters 6 and 7), then ELS would have the advantage of early onset and the initiation of secondary inoculum. The LLS pathogen would then be at a further disadvantage due to warmer temperatures in June and July (Table 5.2) and possibly interspecific competition. However, after the onset of LLS, secondary inoculum of *C. personatum* would begin to increase toward early September when temperatures have begun to cool down. Due to a greater sporulation potential compared with *C. arachidicola* (Shokes and Culbreath, 1997), the LLS pathogen would then have a greater advantage to colonize any remaining plant material. However, this hypothesis has yet to be tested or demonstrated.

As pointed out by (Chevaugeon, 1952), an inherent limitation of planting date studies in small plot designs is that the close proximity of earlier planted peanuts may have led to artificially high inoculum levels of each leaf spot pathogen for the May and June-planted peanuts. Peanuts planted in

June would have been subjected to the large quantity of secondary inoculum generated by the earlier planted peanuts. Therefore the results presented here may not completely reflect the effect of planting date in a normal field situation since there are generally larger spatial divisions between fields. However, because conidia of both leaf spot pathogens can be aerially dispersed (Shokes and Culbreath, 1997), and distance between peanut fields is often less than 1 km distance, we cannot preclude a greater aerial inoculum load at the macroscale having a similar effect in actual grower situations.

Regardless of the differences observed in the behavior of ELS and LLS, our results confirm the efficacy of planting date as a tactic to reduce the severity of the overall leaf spot epidemic. In all trials conducted, the defoliation at 130 DAP was significantly lower for peanuts planted in April than in later plantings (Fig. 5.4), and this confirms several previous reports (Chevaugeon, 1952; Hemingway, 1954; Shokes et al., 1982). However, from a forecasting standpoint, it is clear that the onset of each disease cannot be predicted based on planting date alone.

In conclusion, in this study we compared the epidemiology of ELS and LLS, and found that, in later planting dates, the onset of both diseases generally occurs earlier (in DAP), and the rate of progress and defoliation increases. More importantly, however, we found that in situations where the inoculum source of *C. personatum* is less prevalent than that of *C. arachidicola*, later planting dates can result in a greater abundance of LLS. Additionally, later planted peanuts resulted in less of a difference in onset of these two diseases, but the smallest differences in onset were observed in fields with a history of LLS or in plots treated with *C. personatum*-infested residue. While there are almost certainly other factors that influence the development of these two diseases, the knowledge gained from the study of the interaction between the inoculum source and the planting date for each leaf spot is an additional step towards a more complete understanding of the mechanisms responsible for differences in the epidemiology of these two diseases. Such information is critical for recommending the most accurate and precise forecasting systems.

ACKNOWLEDGMENTS

We thank the personnel at the University of Georgia and University of Florida research stations for all of the assistance provided. Particularly, we thank Billy Mills, Lee Hitson, John Paulk, Andy Carter, Mark Gomillion, Jim Boyer, Cliff Riner, Randy Hill, Denny Thigpin, Lina Flor Young, Jeannette Mixon, Jhen Bennett and Edna Shannon for technical support; and Xuelin Luo for statistical assistance. Weather data was provided by the University of Georgia Automated Environmental Monitoring Network. A brief summary of this research has previously been published (Fulmer and Kemerait Jr, 2016).

LITERATURE CITED

Alderman, S., and Nutter, F. 1994. Effect of temperature and relative humidity on development of *Cercosporidium personatum* on peanut in Georgia. Plant Dis. 78:690-694.

Backman, P., and Crawford, M. 1984. Relationship between yield loss and severity of early and late leafspot diseases of peanut. Phytopathology 74:1101-1103.

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

Cantonwine, E., Culbreath, A., Holbrook, C., and Gorbet, D. 2008. Disease progress of early leaf spot and components of resistance to *Cercospora arachidicola* and *Cercosporidium personatum* in runner-type peanut cultivars. Peanut Sci. 35:1-10.

Cantonwine, E. G., Culbreath, A. K., Stevenson, K. L., Kemerait, R. C., Brenneman, T. B., Smith, N. B., and Mullinix, B. G. 2006. Integrated disease management of leaf spot and spotted wilt of peanut. Plant Dis. 90:493-500.

Chevaugeon, J. 1952. Recherches sur la Cercosporiose de l'arachide en Moyenne Casamance. Ann. Inst. Nat. Rech. Agron. Ser. C 3:489-510.

Culbreath, A. K., Tillman, B. L., Tubbs, R. S., Beasley, J. P., Kemerait, R. C., and Brenneman, T. B. 2010. Interactive effects of planting date and cultivar on tomato spotted wilt of peanut. Plant Dis. 94:898-904.

Eyal, Z. 1999. The *Septoria tritici* and *Stagonospora nodorum* blotch diseases of wheat. Eur. J. Plant Pathol. 105:629-641.

Farrell, J., Bailey, B., and Mills, W. 1967. The effects of time of planting, spacing and fungicide on Cercospora leaf spots of groundnuts in Malawi. Rhod. Zambia Malawi J. Agric. Res 5:241-247.

Fitt, B. D., Huang, Y.-J., van den Bosch, F., and West, J. S. 2006. Coexistence of related pathogen species on arable crops in space and time. Annu. Rev. Phytopathol. 44:163-182.

Fry, W. E. 1982. Principles of Plant Disease Management. Academic Press, Orlando.

Fulmer, A., and Kemerait Jr, R. 2016. Effect of inoculum level, planting date and variety on the onset and predominance of early and late leaf spot of peanut. Proc. Amer. Peanut Res. Ed. Soc. 48:94 (Abstr.).

Garren, K., and Wilson, C. 1951. Peanut diseases. Pages 262-324 in: The Peanut, the Unpredictable Legume. Nat. Fertilizer Assoc., Washington, D.C.

Hemingway, J. 1954. Cercospora leafspots of groundnuts in Tanganyika. E. Afr. Agricultural Journal 19:263-271.

Hemingway, J. 1955. The prevalence of two species of Cercospora on groundnuts. Trans. Brit. Mycol. Soc. 38:243-246.

Jenkins, W. A. 1938. Two fungi causing leaf spot of peanut. J. Agr. Res. 56:317-332.

Jordan, B., and Culbreath, A. 2016. Effect of planting date and peanut cultivar on leaf spot epidemics and pod yield with implications for organic production. (Abstr.). Phytopathology 106:S2.6. http://dx.doi.org/10.1094/PHYTO-106-4-S2.6.

Kemerait, R. C., Jr., Brenneman, T. B., and Culbreath, A. K. 2017. Peanut disease update. Pages 23-62 in: 2017 peanut update. W. Monfort, ed. Coop. Ext. Ser. College of Agric. Environ. Sci. University of Georgia., Athens.

Mani, K. K., Hollier, C. A., and Groth, D. E. 2016. Effect of planting date, fungicide timing and cultivar susceptibility on severity of narrow brown leaf spot and yield of rice. Crop Protect. 90:186-190.

Marburger, D. A., Smith, D. L., and Conley, S. P. 2016. Revisiting planting date and cultivar effects on soybean sudden death syndrome development and yield loss. Plant Dis. 100:2152-2157.

Milberg, P., Hallgren, E., and Palmer, M. 2001. Timing of disturbance and vegetation development: how sowing date affects the weed flora in spring-sown crops. Journal of Vegetation Science 12:93-98.

Monfort, W., ed. 2017. 2017 Peanut Update. Coop. Ext. Ser. College of Agric. Environ. Sci. University of Georgia., Athens.

Shokes, F., and Culbreath, A. K. 1997. Early and late leaf spots. Pages 17-20 in: Compendium of Peanut Diseases, 2nd Ed. N. Kokalis-Burelle, D. Porter, R. Rodriguez-Kabana, D. Smith and P. Subrahmanyam, eds. American Phytopathological Society Press, St. Paul, MN.

Shokes, F., Gorbet, D., and Sanden, G. 1982. Effect of planting date and date of spray initiation on control of peanut leaf spots in Florida. Plant Dis. 66:574-575.

Shrestha, R., and Parajulee, M. 2010. Effect of tillage and planting date on seasonal abundance and diversity of predacious ground beetles in cotton. Journal of Insect Science 10:1-17.

Smith, D. H., and Littrell, R. H. 1980. Management of peanut foliar diseases with fungicides. Plant Dis. 64:356-361.

Sommartya, T., and Beute, M. 1986. Temperature effects on germination and comparative morphology of conidia for Thai and USA isolates of *Cercosporidium personatum*. Peanut Sci. 13:67-70.

Wan, A., Bock, C., Fitt, B. D., Harvey, J., and Jenkyn, J. 2005. Development of *Oculimacula yallundae* and *O. acuformis* (eyespot) on leaf sheaths of winter wheat in the UK in relation to thermal time. Plant Pathol. 54:144-155.

Woodward, J., Brenneman, T., Kemerait, R., Culbreath, A., and Smith, N. 2010. Management of peanut diseases with reduced input fungicide programs in fields with varying levels of disease risk. Crop Protect. 29:222-229.

Table 5.1. Planting dates used in the field experiments conducted at locations in Georgia and Florida in 2014 and 2015.

	Day and month planted			Day of year planted		
Year, location	1	2	3	1	2	3
2014						
Reidsville	28 April	22 May	5 June	118	142	156
Tifton	24 April	14 May	6 June	114	134	157
Attapulgus	28 April	19 May	6 June	118	139	157
Marianna	28 April	22 May	1 June	118	142	152
Citra	23 April	14 May	4 June	113	134	155
2015						
Reidsville	5 May	21 May	4 June	125	141	155
Tifton	24 April	13 May	5 June	114	133	156
Attapulgus	27 April	21 May	4 June	117	141	155
Marianna	5 May	14 May	2 June	125	134	153

Table 5.2. Accumulated monthly rainfall (mm) and average, minimum and maximum monthly air temperature (°C) from 1 May to 30 September for each location in 2014 and 2015.

Year, location[y]	Month	2014 Rainfall	2014 Temperature (°C)			2015 Rainfall	2015 Temperature (°C)		
		mm	Mean	Minimum	Maximum	mm	Mean	Minimum	Maximum
Reidsville[z]									
	May	103	23.3	9.6	33.6	30	23.7	9.4	33.3
	June	97	26.1	19.0	34.6	54	26.4	19.9	36.8
	July	60	27.0	18.9	35.5	147	27.2	19.8	35.9
	August	46	27.0	17.8	36.3	102	25.8	18.4	34.7
	September	207	23.9	13.8	33.4	84	23.5	12.8	33.0
Attapulgus									
	May	73	22.3	9.8	33.3	40	23.4	9.2	32.9
	June	52	25.7	18.1	34.6	98	25.9	18.4	35.6
	July	115	25.8	16.7	34.5	91	27.2	20.4	35.6
	August	15	26.7	17.6	37.3	63	26.4	15.9	34.4
	September	194	23.9	16.3	35.4	213	23.6	11.9	32.7
Marianna									
	May	102	23.2	9.6	36.4	90	24.5	9.7	35.5
	June	59	26.7	19.5	36.8	102	27.0	19.8	37.4
	July	144	26.9	17.2	37.0	123	28.1	19.9	38.1
	August	65	27.4	18.7	37.7	70	27.4	15.4	37.5
	September	153	24.8	14.3	36.6	105	24.7	10.9	36.7
Tifton									
	May	205	22.7	9.6	32.0	23	23.9	9.9	32.1
	June	73	25.8	19.1	33.6	87	26.2	19.1	36.1
	July	76	25.9	17.9	34.2	326	26.8	20.2	34.6
	August	38	26.5	19.1	36.0	110	26.2	16.5	34.7
	September	151	23.7	14.2	34.2	45	23.4	12.9	33.7
Citra									
	May	95	23.7	-	35.4	-	-	-	-
	June	103	26.0	-	36.3	-	-	-	-
	July	163	26.6	-	36.5	-	-	-	-
	August	117	27.3	-	37.1	-	-	-	-
	September	117	25.2	-	35.3	-	-	-	-

[y] Weather data were recorded at onsite weather stations, obtained from the Georgia Automated Environmental Monitoring Network (www.Georgiaweather.net), or the Florida Automated Weather Network (http://fawn.ifas.ufl.edu).
[z] At Reidsville, the nearest weather station was located 17.5 km away .

Table 5.3. *P*-values from the linear mixed model analysis of variance for all leaf spot variables for split-block trials conducted during 2014 and 2015 with a separate one-way ANOVA for additional planting date studies at locations with different disease histories.

Location, year	Predominance[t]	Disease onset[u]		Rate of progress[v]	
source of variation	ProLLS	ELS	LLS	ELS	LLS
Reidsville					
2014					
Planting Date (P)	0.0012	0.0382	0.0002	0.0523	0.0018
Inoculum (I)	0.0001	<0.0001	0.0020	0.7569	0.1518
P x I	<0.0001	0.2270	0.0336	0.5978	0.1169
2015					
Planting Date (P)	0.9073	0.0008	<0.0001	0.1324	0.0614
Inoculum (I)	0.0204	<0.0001	0.0970	0.6049	0.0185
P x I	0.5849	0.3357	0.8862	0.4218	0.4003
Additional studies					
2014					
Attapulgus (P)	0.5572	0.2469	0.0043	0.0643	-[z]
Marianna (P)	0.5602	0.1918	<0.0001	-	0.2085
Tifton (P)	0.0038	0.1942	0.0037	0.8420	0.4538
Citra (P)	0.8051	0.0040	<0.0001	0.0012	0.1272
2015					
Attapulgus (P)	0.0050	0.4362	0.0029	0.0402	-
Marianna (P)	0.2857	0.5892	0.0064	-	0.4084
Tifton (P)	0.9235	0.0857	0.0006	0.3177	0.9675

Location, year	Defoliation[w]	Days to 50% incidence[x]		Max lesion number[y]	
source of variation	130 DAP	ELS	LLS	ELS	LLS
Reidsville					
2014					
Planting Date (P)	0.0024	0.0006	<0.0001	0.2214	<0.0001
Inoculum (I)	<0.0001	0.0086	0.0219	0.0002	0.0008
P x I	0.0003	0.9850	0.3076	0.1450	0.0060
2015					
Planting Date (P)	<0.0001	0.0015	<0.0001	0.0893	0.3455
Inoculum (I)	0.0002	0.0614	0.0196	0.1216	0.0133
P x I	0.0034	0.3126	0.2572	0.5662	0.2163
Additional studies					
2014					
Attapulgus (P)	<0.0001	0.0001	-	0.4673	0.5193
Marianna (P)	0.0004	-	0.1765	0.0868	0.8398
Tifton (P)	<0.0001	0.0205	<0.0001	0.0674	0.0028
Citra (P)	<0.0001	0.0004	-	0.5059	0.7491
2015					
Attapulgus (P)	<0.0001	<0.0001	-	0.4535	0.0826
Marianna (P)	0.0324	-	0.0250	0.2286	0.1238
Tifton (P)	0.0002	0.0555	0.0020	0.8892	0.3313

[t] Predominance (proportion of the total leaf spot epidemic attributed to late leaf spot; calculated by dividing the standardized AUDPC of late leaf spot by the total standardized AUDPC of early and late leaf spot combined).
[u] Number of days after planting to reach > 1 % disease incidence for early leaf spot (ELS) and late leaf spot (LLS).
[v] Rate (1/day) parameters for both diseases were estimated from fitted Gompertz models of incidence over time.
[w] Percent defoliation at 130 DAP. Estimates based on the exponential model of defoliation over time.
[x] Number of days after planting to reach 50% disease incidence. Estimates based on fitted Gompertz models of incidence over time.
[y] Maximum number of lesions per leaflet observed during the season.
[z] Incidence levels were too low to model the data.

Table 5.4. Effect of planting date on the days required to reach 50 % incidence of early and late leaf spot in relation to infested peanut residue or locations with different disease histories in 2014 and 2015.

Location, inoculum[t]	Planting date	Days to reach 50% incidence[u] 2014 ELS	2014 LLS	2014 Diff[v]	2015 ELS	2015 LLS	2015 Diff
Reidsville							
C. arachidicola	April	122 a[w]	140 a	-18 *	133 a	125 a	12 *
	May	108 b	127 b	-21 *	120 b	115 b	5 ns
	June	103 b	109 c	-7 ns	113 b	101 c	12 ns
C. personatum	April	130 a	131 a	-1 ns	138 a	123 a	12 ns
	May	116 b	121 b	-5 ns	132 a	108 b	24 *
	June	109 b	106 c	3 ns	116 b	98 c	18 ns
C.a. + C.p.	April	125 a	139 a	-17 ns	135 a	123 a	13 *
	May	108 b	126 b	-20 *	129 a	115 b	13 *
	June	106 b	109 c	-4 ns	110 b	101 c	10 **
None	April	133 a	138 a	-5 ns	140 a	124 a	16 **
	May	121 ab	120 b	1 ns	136 a	112 b	24 **
	June	115 b	108 c	7 *	122 b	101 c	21 *
Other locations[x]							
Attapulgus	April	106 a	-[y]	-	111 a	139 a	-26 *
	May	90 b	-	-	98 b	128 b	-30 *
	June	82 c	-	-	92 c	111 c	-19 *
Marianna	April	-	126 a	-	-	114 a	-
	May	-	114 a	-	-	102 ab	-
	June	-	105 a	-	-	96 b	-
Tifton	April	134 a	140 a	-8 ns	137 a	122 a	14 ns
	May	112 ab	119 b	-5 ns	119 a	112 b	3 ns
	June	103 b	103 c	-5 ns	115 a	98 c	18 ns
Citra	April	118 a	-[z]	-	-	-	-
	May	107 b	-	-	-	-	-
	June	103 b	-	-	-	-	-

[t] Inoculum treatments consisted of peanut residue infested with *Cercospora arachidicola* (*C.a.*), *Cercosporidium personatum* (*C.p.*), a 50/50 mixture of *C.a.*+ *C.p.* and a residue free control (None).

[u] Number of days after planting to reach 50% disease incidence. Estimates based on fitted Gompertz models of incidence over time. ELS = early leaf spot; LLS=late leaf spot.

[v] Number of days after planting to reach 50% ELS subtracted from days to reach 50% LLS incidence. Asterisks indicate that differences were significant for each inoculum treatment based on pairwise t-tests at $\alpha = 0.05$ (*) or $\alpha = 0.01$ (**); n.s. = not significant.

[w] For each inoculum treatment or location, means within the same column with the same lower case letters are not significantly different based upon Tukey's honestly significant difference test ($\alpha = 0.05$).

[x] Locations with differing inoculum levels were selected based upon previous historical predominance or amending plots with infested peanut residue. Attapulgus, historically early leaf spot; Marianna,

historically late leaf spot; Tifton, a 50/50 mixture of *C.a.*+ *C.p.* in 2014, and replanted to same plots in 2015; Citra, long rotation, but history of early leaf spot in nearby (0.5 km) fields.

[y] Incidence levels were not high enough to model the data.

[z] No trial conducted at Citra in 2015.

Table 5.5. Effect of planting date on the maximum number of early and late leaf spot lesions per leaflet in relation to infested peanut residue or locations with different disease histories in 2014 and 2015.

		Maximum number of lesions per leaflet[w]			
		2014		2015	
Location, inoculum[v]	Planting date	ELS	LLS	ELS	LLS
Reidsville					
C. arachidicola	April	3.9 b[x]	0.2 b	1.7 b	8.2 a
	May	8.5 a	5.9 a	2.3 ab	7.8 a
	June	6.0 ab	10.0 a	3.8 a	10.3 a
C. personatum	April	2.8 a	6.1 b	1.1 a	13.8 a
	May	3.5 a	11.2 a	1.4 a	15.4 a
	June	3.2 a	12.7 a	1.8 a	14.8 a
C.a. + C.p.	April	3.7 a	1.1 c	1.4 b	11.4 a
	May	7.3 a	5.8 b	1.5 ab	7.8 a
	June	4.0 a	13.2 a	3.3 a	9.2 a
None	April	2.8 a	2.4 b	1.1 a	10.0 a
	May	3.0 a	9.1 a	1.0 a	12.3 a
	June	3.6 a	10.4 a	1.9 a	16.1 a
Other locations[y]					
Attapulgus	April	12.2 a	0.3 a	9.5 a	1.0 a
	May	10.5 a	0.3 a	11.6 a	4.0 a
	June	11.4 a	0.2 a	9.6 a	2.6 a
Marianna	April	1.0 a	14.8 a	0.6 a	9.2 a
	May	1.7 a	16.9 a	1.2 a	11.8 a
	June	2.1 a	18.0 a	1.2 a	25.1 a
Tifton	April	2.6 a	2.2 b	1.5 a	10.2 a
	May	3.3 a	8.1 ab	1.5 a	9.1 a
	June	3.8 a	10.2 a	1.2 a	7.4 a
Citra	April	6.9 a	1.3 a	-[z]	-
	May	8.8 a	1.6 a	-	-
	June	7.9 a	0.9 a	-	-

[v] Inoculum treatments consisted of peanut residue infested with *Cercospora arachidicola* (*C.a.*), *Cercosporidium personatum* (*C.p.*), a 50/50 mixture of *C.a.*+ *C.p.* and a residue free control (None).

[w] Maximum number of lesions per leaflet observed during the season. ELS = early leaf spot; LLS=late leaf spot.

[x] For each treatment or location, means within the same column with the same lower case letters are not significantly different based upon Tukey's honestly significant difference test ($\alpha = 0.05$).

[y] Locations with differing inoculum levels were selected based upon previous disease history or amending plots with infested peanut residue. Attapulgus, historically early leaf spot; Marianna, historically late leaf spot; Tifton, a 50/50 mixture of *C.a.*+ *C.p.* in 2014, and replanted to same plots in 2015; Citra, long rotation, but history of early leaf spot in nearby (0.5 km) fields.

[z] No trial conducted at Citra in 2015.

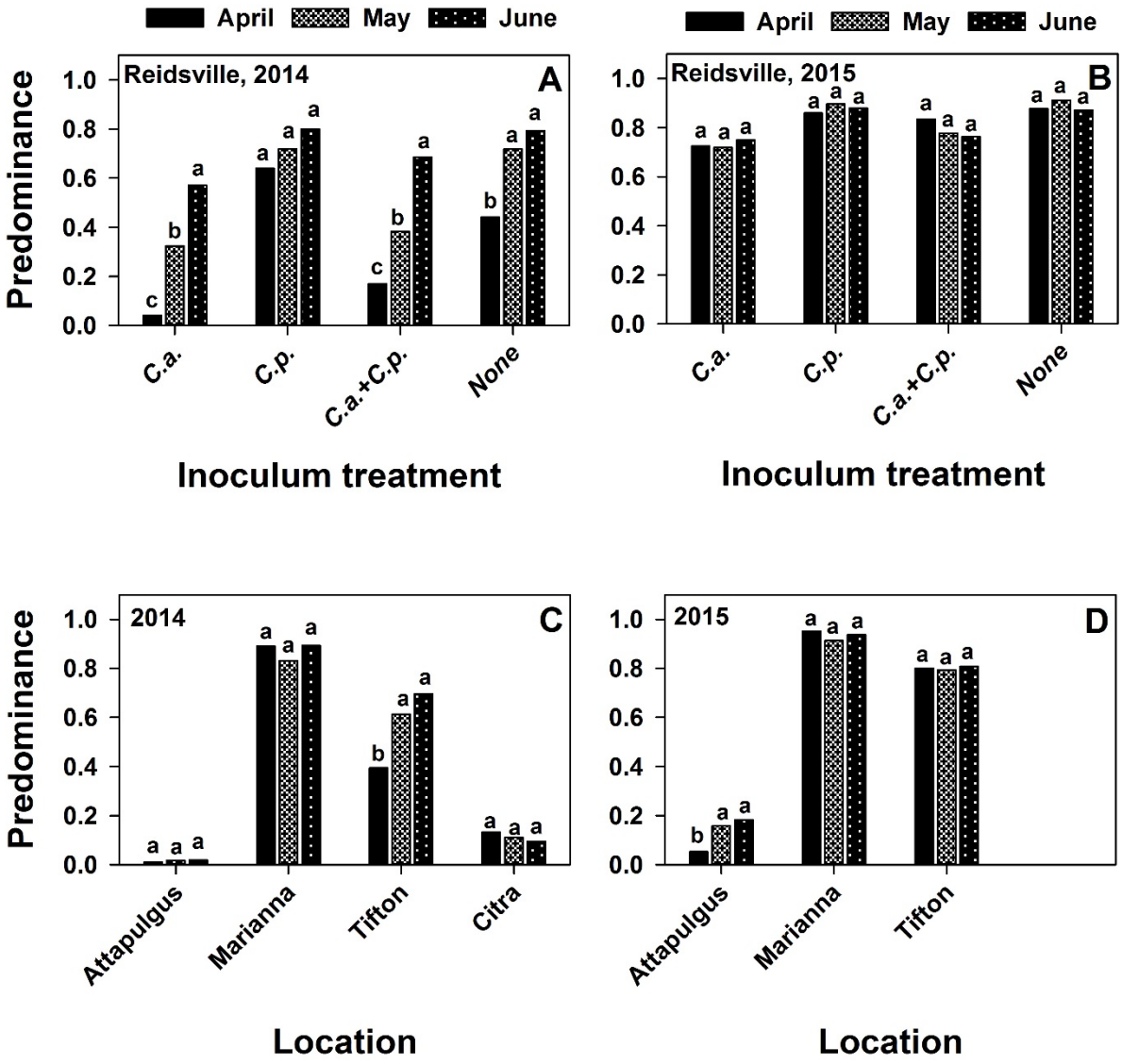


Fig. 5.1. Effect of planting date on predominance (proportion of the total leaf spot epidemic attributed to late leaf spot; calculated by dividing the standardized AUDPC of late leaf spot by the total standardized AUDPC of early and late leaf spot combined) in relation to inoculum level of each leaf spot pathogen or previous disease history for each location. **A** and **B**, The inoculum treatments consisted of peanut residue infested with *Cercospora arachidicola* (*C.a.*), *Cercosporidium personatum* (*C.p.*), a 50/50 mixture of *C.a.*+ *C.p.* and a residue-free control (none). **C and D**, Locations with differing inoculum levels (inferred based on previous disease history or direct application of infested peanut residue). Attapulgus, historically early leaf spot; Marianna, historically late leaf spot; Tifton, a 50/50 mixture of *C.a.*+ *C.p.* in 2014, and replanted to same plots in 2015; Citra, 2 year rotation out of peanut, but history of early leaf spot in nearby (0.5 km) fields. Grouped bars for each inoculum treatment or location with the same letters are not significantly different based upon Tukey's honestly significant difference test ($\alpha = 0.05$).

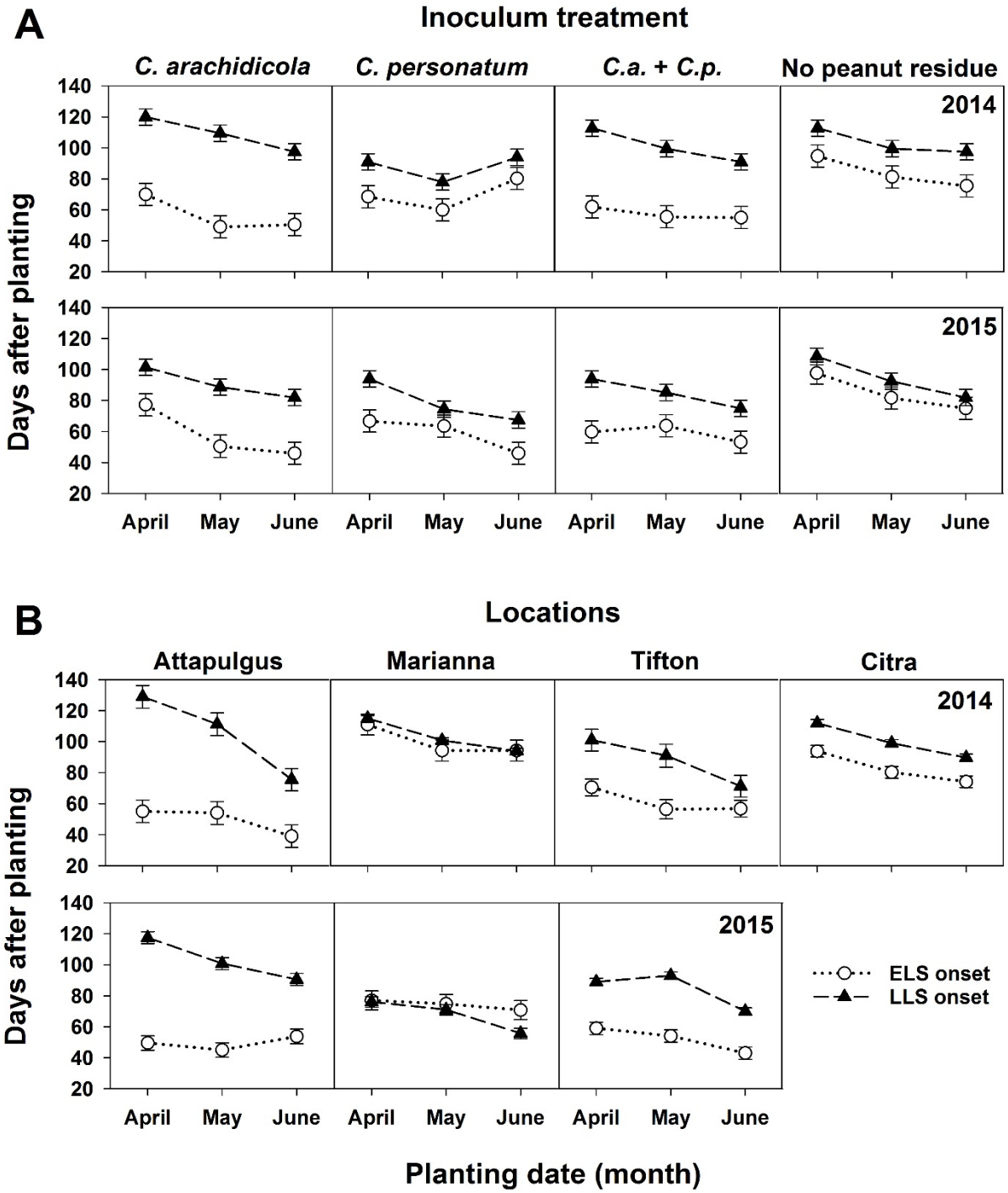


Fig. 5.2. Effect of planting date on the onset (days after planting to reach 1 % disease incidence) of early leaf spot (ELS) and late leaf spot (LLS) in relation to inoculum level of each leaf spot pathogen or previous disease history for each location. **A**, Trials conducted at Reidsville had the following inoculum treatments: peanut residue infested with *Cercospora arachidicola* (*C.a.*), *Cercosporidium personatum* (*C.p.*), a 50/50 mixture of *C.a.*+ *C.p.* and a residue free control (none). **B**, Locations with differing inoculum levels (inferred based on previous disease history or direct application of infested peanut residue). Attapulgus, historically early leaf spot; Marianna, historically late leaf spot; Tifton, a 50/50 mixture of *C.a.*+ *C.p.* in 2014, and replanted to same plots in 2015; Citra, 2-year rotation out of peanut,

but history of early leaf spot in nearby (0.5 km) fields. Data points for each inoculum treatment or location represent the mean value of each level and error bars represent the standard error of the mean.

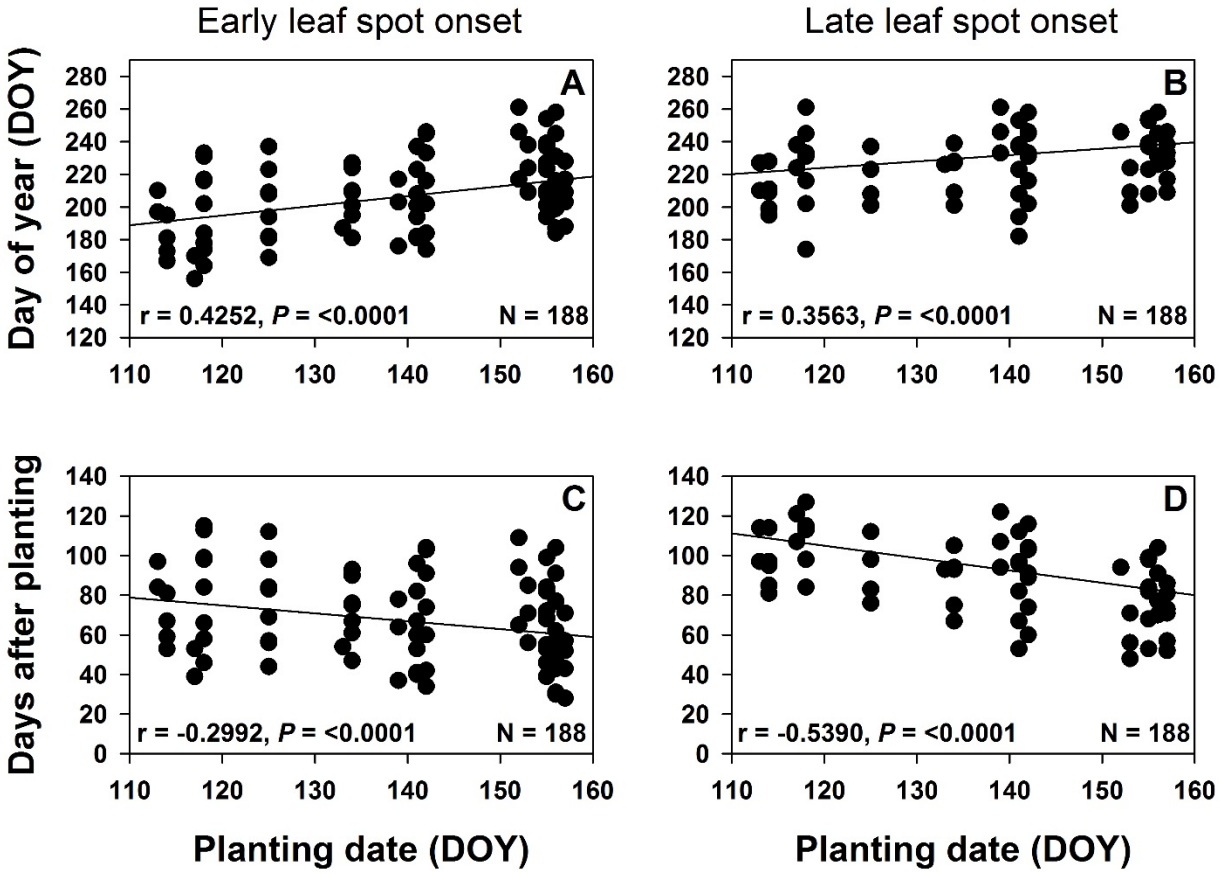


Fig. 5.3. Correlation of planting date (day of year (DOY) planted) with the onset of early (ELS) and late (LLS) leaf spot based on the DOY (**A** and **B**) or days after planting (**C** and **D**). Data were combined across years, locations and inoculum treatments, and the resulting Pearson correlation coefficients, *r*, and associated *P*-values are provided.

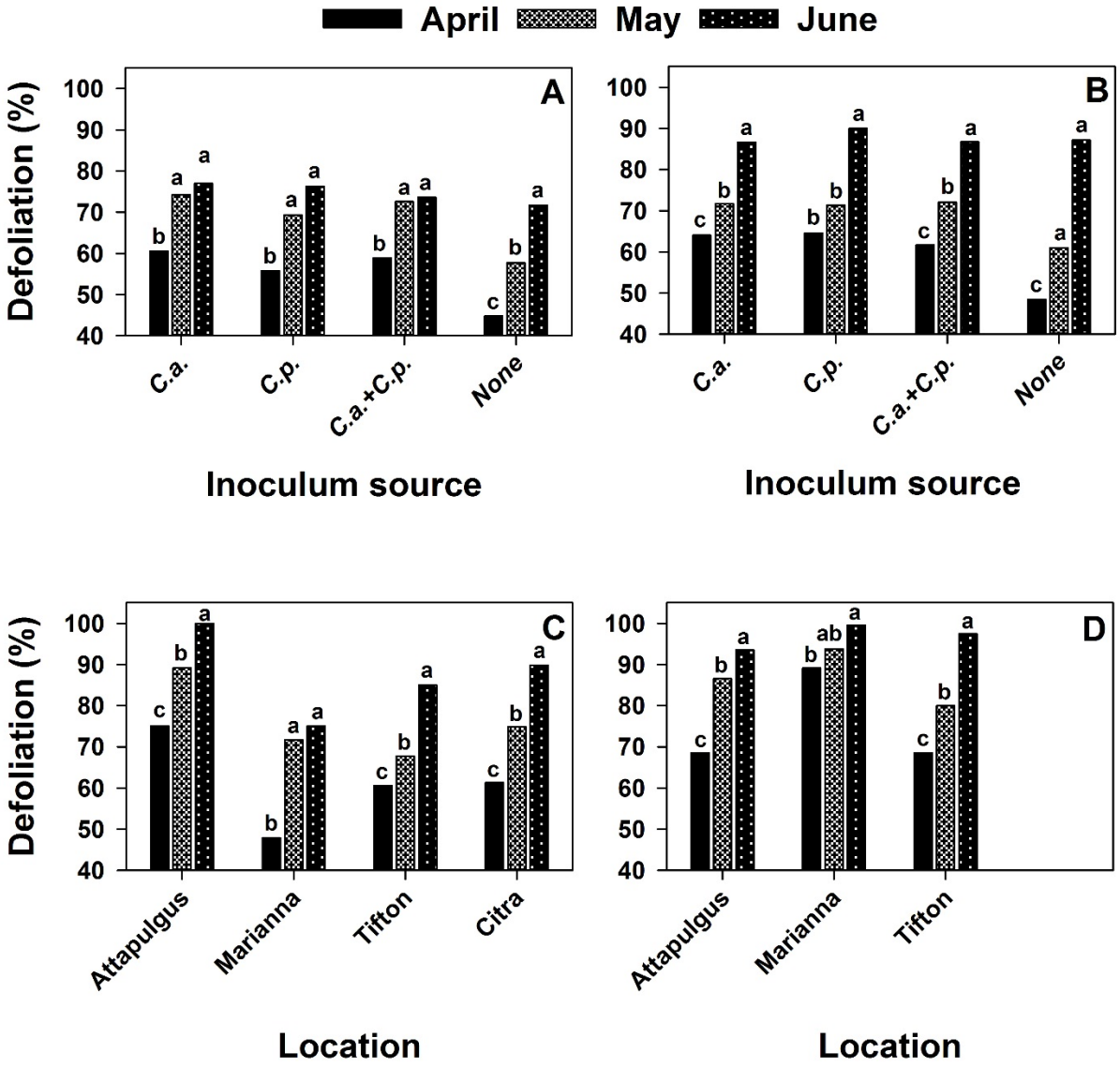


Fig. 5.4. Effect of planting date on the defoliation of peanut at 130 days after planting as predicted with the exponential model. **A** and **B**, The inoculum treatments consisted of peanut residue infested with *Cercospora arachidicola* (*C.a.*), *Cercosporidium personatum* (*C.p.*), a 50/50 mixture of *C.a.*+ *C.p.* and a residue free control (none) at Reidsville in 2014 (A) and 2015 (B). **C and D**, Locations with differing disease histories or direct application of infested peanut residue in 2014 (C) and 2015 (D). Attapulgus, historically early leaf spot; Marianna, historically late leaf spot; Tifton, a 50/50 mixture of *C.a.*+ *C.p.* in 2014, and replanted to same plots in 2015; Citra, 2 year rotation out of peanut, but history of early leaf spot in nearby (0.5 km) fields. Grouped bars for each inoculum treatment or location with the same letters are not significantly different based upon Tukey's honestly significant difference test ($\alpha = 0.05$).

CHAPTER 6

EFFECT OF CULTIVAR ON THE DEVELOPMENT OF EARLY AND LATE LEAF SPOT IN FIELDS WITH DIFFERING PLANTING DATES AND INOCULUM LEVELS[1]

[1]Fulmer, A.M., Kemerait, R.C., Brenneman, T.B., Scherm, H., Cantonwine, E.G., Culbreath, A.K., and Stevenson, K.L. 2017. To be submitted to *Plant Disease*.

ABSTRACT

Field trials were conducted in Tifton, Georgia and Marianna, Florida, in 2014, 2015 and 2016, to determine the role of host resistance on the differences in the prevalence, onset and severity of early (ELS, *Cercospora arachidicola*) and late (LLS, *Cercosporidium personatum*) leaf spot. The peanut cultivars Florunner, Georgia Green and Georgia-06G were screened against a wide range of known resistant or susceptible standards; planting dates ranged from mid-May to late-August; fields were either amended with peanut residue infested with *C. arachidicola* (*Ca*) alone, *C. personatum* (*Cp*) alone, *Ca* + *Cp* or a non-inoculated control, or had a previous history of ELS or LLS. Epidemics were differentiated by sampling five main stems on a bi-weekly basis and counting the number of lesions of each leaf spot. Onset and standardized AUDPC (stAUDPC) of lesions per leaflet of both diseases was earliest and highest, respectively, in fields where the inoculum source of each pathogen was present and for later planting dates. Among trials, onset of ELS ranged from 26 to 63 days after planting (DAP), but generally did not statistically differ among cultivars. LLS onset ranged from 27 to 88 DAP, and in most trials planted in May and June, was significantly earlier on Florunner and cultivars known to be more susceptible to LLS, compared with Georgia Green and Georgia-06G. However, LLS onset did not differ among cultivars when planted in late July and August in fields with a prior history of LLS. ELS stAUDPC was similar for Florunner, Georgia Green and Georgia-06G, and was almost always significantly higher on New Mexico Valencia C. LLS stAUDPC was always numerically and often statistically higher on Florunner and on cultivars known to be more susceptible to LLS. Georgia Green and Georgia-06G stAUDPC values were similar to the LLS-resistant cultivars, C99R and Southern Runner. Percent defoliation on the main stem was similar among cultivars, and generally lowest on New Mexico Valencia C. Results from this study indicate that differential susceptibility of peanut cultivars to ELS and LLS can partially explain differences in the prevalence and onset of ELS and LLS, but this is dependent on planting date and inoculum level.

INTRODUCTION

Early (ELS) and late (LLS) leaf spot of peanut, caused by *Cercospora arachidicola* and *Cercosporidium personatum*, respectively, are two of the most important foliar diseases of peanut (Shokes and Culbreath, 1997). Both are globally distributed, polycyclic diseases that can coexist in the same field and even on the same leaflet. Together or separately, they can cause complete defoliation of the peanut plant, resulting in yield losses > 50% in unsprayed, high-risk fields (Backman and Crawford, 1984; Shokes and Culbreath, 1997). In Georgia, management relies heavily on fungicide inputs, but more recently, a risk index, Peanut Rx, has been developed that allows growers to utilize a prescription fungicide program appropriate for high, moderate or low-risk fields (Woodward et al., 2008). Risk factors include cultivar, planting date, rotation, field history, and irrigation vs dryland (Kemerait et al., 2017).

Because of similarities in appearance, disease cycle and potential impact on yield, both leaf spots are generally managed as a single disease (Garren and Wilson, 1951). However, the development and relative abundance of each disease often varies across fields, regions and years. Depending on predisposing field risk, the onset of both diseases can range from 30 to 140 DAP, but ELS generally appears 2 to 4 weeks prior to LLS (McDonald et al., 1985; Smith and Littrell, 1980). LLS is thought to be more destructive of the two due to a higher rate of disease progress (Backman and Crawford, 1984; Hemingway, 1955; Jenkins, 1938). In Georgia, LLS was predominant from 1976 to the early 1990s; otherwise ELS has generally been the most prevalent (Cantonwine et al., 2008; Smith and Littrell, 1980). In recent years, the prevalence of LLS has increased again (Cantonwine et al., 2008) and predominance can vary between fields located 2 km apart (Chapter 3).

The factors that drive the variations in predominance and the differences in disease onset between the two leaf spots remain unclear. In recent studies, we characterized the effect of initial inoculum source and planting date on the epidemiological behavior of both leaf spot diseases (Chapters 4 and 5). Our results showed that the predominance and onset of ELS and LLS were associated with amendments of infested peanut residue of *C. arachidicola* alone or *C. personatum* alone, respectively. However, a 50/50

mix of peanut residue infested with each pathogen did not significantly increase the onset of LLS and tended to have more ELS than residue-free plots where the onset of both diseases was delayed and greater levels of LLS were observed (Chapter 4). Furthermore, results from previous studies also demonstrated that the difference in the onset of the two diseases tended to decrease with later planting dates, and that LLS was generally more prevalent in later planted peanuts (Chapter 5). However, neither of these factors fully explained the differences observed in the onset and prevalence of each disease.

Smith and Littrell (1980) suggested that the shift from ELS to LLS in Georgia during 1976 could have been linked to the wide-spread introduction of a new cultivar (Florunner) and that differences in host resistance could influence the differential development of ELS and LLS. This hypothesis is reasonable because studies indicate that genes for partial components of resistance to *C. arachidicola* and *C. personatum* are inherited independently (Abdou et al., 1974; Cantonwine et al., 2008; Walls et al., 1985). Yet, with most research emphasis directed toward managing the predominant leaf spot in a given region, few studies have directly compared the relative susceptibility of these cultivars to each of the two leaf spot diseases, or shown how such differences would affect the temporal dynamics of each leaf spot disease (Cantonwine et al., 2008). However, there is circumstantial evidence to suggest that a single cultivar can indeed have differential or equal levels of resistance/susceptibility to ELS and LLS. For example, in one study, the cultivar NC 3303 was shown to have high levels of resistance to ELS (Ricker et al., 1985), but in another study, was shown to be highly susceptible to LLS (Walls et al., 1985). In contrast, the cultivar Florigiant was found to be highly susceptible to both leaf spot diseases (Ricker et al., 1985; Walls et al., 1985).

Since the 1970s, there have been three predominant peanut cultivars grown in southeastern United States: Florunner (1970-1996), Georgia Green (1996-2008), and Georgia-06G (2006-present) (J. Beasley, personal communication). Florunner was considered highly susceptible while the latter two cultivars are considered only moderately susceptible (A. Culbreath, personal communication). However, it is unknown if the relative susceptibility of these cultivars differs between leaf spot diseases and there is little evidence in the literature to support a consistent inference. Monasterios de La Torre (1980) screened

multiple cultivars, including Florunner, for resistance to ELS and LLS, and his results suggested that Florunner seemed equally susceptible to both pathogens. Backman and Crawford (1984) attributed similar yield losses to both leaf spot diseases. In contrast, while not the main part of their study, Branch and Culbreath (1995) observed that Florunner appeared to have more resistance to ELS than LLS. Anderson et al. (1993) also reported greater LLS on Florunner, but suggested that the observation was more a function of a negative interaction between leaf spot pathogens rather than a difference in host susceptibility. Georgia Green was reported to have more resistance to both leaf spot diseases compared to Florunner (Cantonwine et al., 2008). Georgia Green was derived from a cross with Sunbelt Runner and Southern Runner, with the latter cultivar known to have a moderate level of resistance to LLS (Branch, 1996; Gorbet et al., 1987). Although not specifically compared in the same study, results from detached leaf assays indicated that Georgia Green could have slightly higher levels of susceptibility to ELS than LLS (Cantonwine et al., 2008). In that study, infection frequency was high for both diseases, but it appeared that the LLS pathogen had much lower levels of sporulation on Georgia Green compared with the ELS pathogen (Cantonwine et al., 2008). Georgia-06G is thought to have similar levels of resistance as Georgia Green, and was derived from a cross between Georgia Green and C99R, a cultivar with resistance to LLS (Branch, 2007; Gorbet and Shokes, 2006).

Detailed knowledge of disease dynamics is a prerequisite for recommending the most effective integrated disease management programs (Fry, 1982). Given the increased prevalence of LLS in Georgia, a more detailed understanding of the mechanisms involved in the differing epidemic development of the two leaf spot diseases is essential. For risk indices such as Peanut Rx, cultivar susceptibility is a major factor in determining risk in a given field (Kemerait et al., 2017). Differences in the relative susceptibility of each cultivar to ELS and/or LLS could enhance our ability to predict the risk in fields that have historically been predominantly ELS or LLS. Therefore, the objective of this study was to determine the effect of important historic and current cultivars on the differential development of ELS and LLS in fields with differing inoculum levels and planting dates.

MATERIALS AND METHODS

2014 Field sites and experimental design. Three preliminary trials were conducted in three separate fields, two of which were located at the Coastal Plain Experiment Station (RDC Pivot and the FireTower field) located in Tifton, Georgia, and one located at the North Florida Research and Education Center in Marianna, Florida. The experiments were laid out in a randomized complete block design with two replications. Treatments consisted of six cultivars (see Table 6.1 for details) and were planted at Tifton on 29 August and at Marianna on 21 August at a rate of four to six seeds per 30.5 cm. Plots consisted of a single row that was 3-m long with 0.91 m between rows, and the block was separated by 2.4 or 3-m alleys. Each field was surrounded by peanuts (within 10 m of plots) which were heavily infected with both diseases (FireTower field in Tifton) or with only LLS (RDC Pivot and Marianna).

2015 Field sites and experimental design. Two trials were conducted in fields located at the University of Georgia Coastal Plain Experiment Station's Black Shank farm and the RDC research farm located in Tifton, Georgia, and one trial at the University of Florida North Florida Research and Education Center in Marianna, Florida. The trial at the RDC farm was set up as a split block design with three replications with inoculum source and cultivar as treatment factors. Inoculum treatments consisted of four combinations of peanut residue infested with *C. arachidicla* (*Ca*) alone, *C. personatum* (*Cp*) alone, a 50/50 mix of *Ca + Cp*, and a non-treated control. Peanut residue was collected from fields in Tifton, Georgia, with severe epidemics of either (>90%) ELS or LLS, and stored separately. Peanut residue, including leaflets, petioles and stems, was collected within a few days after harvesting peanuts (late November) and stored in a large peanut wagon under an open shed for 4 to 6 weeks. In December, dry peanut residue (approximately4.5 kg) for each treatment was spread across the middle (4.5-m) section of each plot (1.8 × 7.6 m) as evenly as possible. Each plot was bordered by a 1.8-m residue-free buffer zone, and was separated by a 3-m alley between each block. The field used in this study had not been planted to peanut for more than 10 years. Six cultivars (see Table 6.1) were planted on 14 May at a rate of 4 seed per 30.5 cm. Plots consisted of a single row 3-m long with 0.91 cm between rows, and blocks were separated by 3-m alleys.

The trial at the Black Shank farm was set up as a split plot design with four replications. Inoculum was the main plot and cultivar was the sub-plot. The field had previously been consecutively cropped to peanut for 3 years and had a history of severe ELS epidemics. Inoculum consisted of plots inoculated with dry peanut residue infested with *C. personatum* (approximately4.5 kg) or non-inoculated plots. Residue was spread across each plot as evenly as possible and incorporated into the soil with a rototiller 1 day prior to planting. Peanut residue was collected from fields in Tifton, Georgia, with a heavy leaf spot epidemic (9 on the Florida 1 to 10 scale) in 2014, and stored in a peanut wagon under an open shed during winter and spring. Seventeen cultivars (see Table 6.1) were planted on 24 June at a rate of six seed per 30.5 cm. Plots consisted of a single row, 3-m long, with 0.91 cm between rows, and blocks were separated by 2.4-m alleys.

The field at Marianna had been out of peanut production for the previous 2 years, but was located in close proximity of a large block of peanuts (within 50 m of the plots) that had been planted a few months earlier and was heavily infected with LLS. Seventeen cultivars (see Table 6.1) were planted on 28 July at a rate of four seed per 30.5 cm. Plots consisted of a single row, 3-m long, with 0.91 cm between rows, and blocks were separated by 2.4-m alleys.

2016 Field sites and experimental design. Two trials were conducted in fields located at the University of Georgia Coastal Plain Experiment Station's research farms in Tift Co., Georgia. One field was located at the Ponder farm and the other field was at the RDC (FireTower) field. Each field had previously been planted to peanut in which severe epidemics of both ELS and LLS occurred in 2015. A split-plot design with four replications was used for each location with planting date as the main plot and cultivar as the subplots. The two target planting dates were mid-May and early June and varieties included important past and present runner and Valencia peanut cultivars (see Table 6.1 for specific planting dates and cultivars). Plots were planted at the Ponder farm on 12 May and 10 June, and at the FireTower field on 14 May and 10 June at a rate of 6 seed per 30.5 cm. Plots consisted of a single row that was 3 m long with 1.8 m between rows. The single treatment row was planted between two border

rows of the cultivar Georgia-06G spaced 0.91 m apart and planted on the same day. Blocks were separated by 3-m alleys at the Ponder farm and 9.1-m alleys at the FireTower field.

Disease assessment. Beginning approximately 20 days after planting (DAP), plots were visually inspected for initial leaf spot symptoms. Upon first detection of leaf spot symptoms, a more intensive evaluation was initiated and continued approximately every other week until the end of the peanut season. Five main stems per plot were arbitrarily selected for destructive sampling. For the trial at the Tifton RDC farm in 2015, a different sampling method was used and consisted of arbitrarily collecting 10 leaves from the interior of the peanut canopy of each plot. All samples were returned to the lab where the number of present and missing leaflets, and the number of ELS and LLS lesions per leaflet were counted. ELS and LLS were differentiated by symptoms and signs associated with each causal pathogen.

In 2014, the two trials in Tifton were sampled at 28, 49, 62 and 77 DAP, and the trial in Marianna was sampled at 28, 48, 64 and 85 DAP. In 2015, the trial in the Tifton RDC farm was sampled at 43, 54, 66, 77 and 92 DAP; the trial in the Tifton Black Shank farm was sampled at 44, 56, 71 and 85 DAP; the trial in Marianna was sampled at 29, 44, 58 and 73 DAP. Because of the extreme delay in planting dates for trials in 2014 and 2015, sampling was terminated prior to 100 DAP due the change in environmental conditions that occurred in the late fall, or due to complete defoliation prior to 100 DAP (e.g., Black Shank farm trial in 2015). In 2016, the trial at the RDC farm in Tifton was sampled at 42, 56, 70, 84, 98, 110, 125 and 137 DAP for the May planting date, and at 29, 43, 57, 71, 83, 110 and 125 DAP for the June planting date. The trial at the Ponder farm was sampled at 43, 58, 69, 86, 104, 120 and 134 for the May planting date, and at 29, 40, 57, 75, 91, 105, 118 and 127 DAP for the June planting date.

Based on the data collected, incidence of ELS and LLS for each rating date was calculated by dividing the number of leaflets with at least one lesion by the total number of leaflets collected for plot. Area under the disease progress curves (AUDPC) were calculated for each leaf spot based on the number of lesions per leaflet. AUDPC values were standardized (stAUDPC) by dividing AUDPC values by the number of days between the first and last rating date (Campbell and Madden, 1990). Predominance was estimated as the proportion of the total leaf spot epidemic attributed to LLS, and was calculated by

dividing the standardized AUDPC of LLS by the total standardized AUDPC of ELS and LLS. Time of disease onset was defined as the number of DAP to reach 1% incidence of ELS or LLS. For lesion counts, the maximum number of ELS and LLS lesions per leaflet was calculated as the highest value that occurred during the season. Percent defoliation was calculated by dividing the total number of leaflets counted by the total number of possible leaflet per main stem. Area under the defoliation curves were calculated and standardized (stAUDefol) by dividing AUDPC values by the number of days between the first and last rating date (Campbell and Madden, 1990).

Statistical analysis. Predominance, disease onset, stAUDPC of each leaf spot, maximum number of early and late leaf spot lesions, final defoliation and stAUDefol were subjected to linear mixed model analysis of variance with PROC GLIMMIX (SAS 9.4 Institute, Cary, NC). Due to differences in the cultivars or experimental design, each year was analyzed separately. In 2014, cultivar was considered a fixed effect and replication, location and replication × location as random effects. In 2015, the trial at the Tifton RDC farm was analyzed as a split block design with inoculum treatment and cultivar as fixed effects and replication, replication × inoculum treatment and replication × cultivar as random effects. The trial at the Tifton Black Shank farm was analyzed as a split plot design, with inoculum treatment and cultivar as fixed effects and replication and replication × inoculum treatment as random effects. In the same year, the trial in Marianna was analyzed as a randomized complete block design with cultivar as a fixed effect and replication as a random effect. In 2016, the model was analyzed as a split plot design, with planting date and cultivar as fixed effects, and replication, replication × location and replication × planting date as random effects. The SLICE option was used to explore two-way interactions when necessary. In all analyses, the Kenward-Roger option was used to adjust the denominator degrees of freedom, and differences in the least square means were tested by Tukey's multiple comparisons test. When data violated the assumptions of normality, transformations were used. Predominance and final defoliation were arcsin square root transformed, the square root transformation was used for disease onset and maximum lesion number per leaflet, and the natural log transformation was used for stAUDPC based on lesions per leaflet. Back-transformed means of all transformed variables are presented in the results.

RESULTS

Weather conditions. More rainfall was recorded during the first 30 DAP compared to the middle or latter part of the growing season across years and locations, but temperatures were more variable among trials due to a broad range of planting dates (Table 6.2). In 2014, the trials at Tifton and Marianna were planted late and both locations received above-average rainfall in September; average temperatures were similar for both locations (Table 6.2). In 2015, plots planted in Tifton in May received approximately a third less rainfall in the first 30 DAP than those planted at the Black Shank farm in June (Table 6.2). The trial at Marianna received less rainfall during the first 30 DAP, and mean temperatures were approximately 1°C higher compared with the trials at Tifton (Table 6.2). For the 2016 trials in Tifton, the overall conditions at the RDC farm and the Ponder farm were very similar for each month. The lowest amount of rainfall occurred in May and July, and mean temperatures were lowest in May and September (Table 6.2).

Predominance. Overall, there were no cultivar × inoculum treatment and cultivar × planting date interactions (Table 6.3), and the prevalent disease across cultivars for each year and location reflected the previous history of each location or inoculum treatment (Tables 6.4 and 6.5), and. Across trials, cultivar tended to affect LLS predominance if there was a prior history of both leaf spot diseases. With the exception of Marianna in 2015, Georgia Valencia, Carver and Florunner had numerically and often statistically greater LLS predominance compared with Georgia Green, Georgia-06G and C99R (Table 6.4). In 2015 and 2016, New Mexico Valencia C had the lowest LLS predominance, and Southern Runner was similar to Georgia Green, Georgia-06G and C99R. Where included, NC 3303 was similar to Georgia Valencia (Table 6.4). A wider range of cultivars included in the study at the Tifton Black Shank farm and Marianna in 2015 reflected previously known levels of susceptibility and/or partial resistance to ELS and LLS (Table 6.1 and Supplementary Tables 6.1 and 6.2). Addition of peanut residue infested with *C. personatum* did not significantly affect the predominance at the Black Shank farm in Tifton (Table 6.5). At the RDC farm, LLS predominance was numerically or significantly lower in plots treated with *Ca*-infested residue or with no added residue compared with plots treated with peanut residue

infested with *Cp* alone or *Ca* + *Cp* (Table 6.5). Planting date did not affect predominance in the trials at Tifton in 2016 (Table 6.5).

Disease onset. Across trials, there was less variability among cultivars in the onset of ELS compared with that of LLS (Table 6.6). Apart from 2014, there were no significant differences in the onset of ELS among cultivars (Table 6.6). The onset of LLS was generally numerically or statistically earlier on Georgia Valencia, Carver and Florunner, and tended to be significantly later on Georgia Green, Georgia-06G, C99R and Southern Runner (Table 6.6). Planting date had a significant effect on the onset of both diseases, but only LLS was affected by inoculum treatment (Table 6.3). Onset of LLS was numerically or statistically earlier in plots where *Cp* was present (Table 6.5). In 2016, onset of both diseases was significantly earlier in plots planted in June compared to plots planted in May (Table 6.5). Generally, LLS onset was the same time or earlier than ELS onset in trials planted in late June, July and August (Table 6.5).

Leaf spot severity. Cultivar had a highly significant effect on the stAUDPC of LLS and in most cases for ELS (Table 6.3). Across trials, New Mexico Valencia C and Georgia Valencia tended to have the highest levels of ELS, and there were generally no differences between Florunner, Georgia Green and Georgia-06G (Figs. 6.1 and 6.2). Georgia Valencia consistently had the highest LLS stAUDPC values, and values for Carver and Florunner were always numerically, and often statistically higher than Georgia Green, Georgia-06G, C99R and Southern Runner. For the trial Black Shank farm in Tifton and the trial in Marianna in 2015, Georgia-12Y and Bailey consistently had the lowest levels of LLS, but Georgia-12Y had above average levels of ELS (Fig. 6.2). NC 3303 had the lowest levels of ELS, but was among the cultivars with the highest LLS severity (Fig. 6.2). For both trials in Tifton in 2015, inoculum treatment had a significant effect on the stAUDPC of LLS, but not for ELS, and there were no significant inoculum × cultivar interactions (Table 6.3). Plots that received *Cp* or *Ca* + *Cp* had the highest levels of LLS (Table 6.5). In 2016, planting date had a significant effect on the stAUDPC of ELS but not for LLS (Table 6.3). ELS and LLS stAUDPC values were significantly and numerically higher, respectively, in plots planted in May compared with those planted in June (Table 6.5). Across trials, maximum number

of lesions per leaflet for ELS and LLS values generally showed similar trends as stAUDPC values (Table 6.7).

Defoliation. Across trials, cultivar almost always had a highly significant effect on final defoliation and stAUDefol values (Table 6.3). New Mexico Valencia C had the lowest defoliation values across trials (Table 6.8). Otherwise, there was rarely a statistically significant difference among cultivars (Table 6.8). Inoculum treatment had a significant effect on the stAUDefol at the Black Shank farm in Tifton in 2015 and planting date had a significant effect on final defoliation and stAUDefol in 2016 (Table 6.3). stAUDefol was significantly higher in plots treated with peanut residue infested with *Cp* compared with non-treated plots (Table 6.5). In 2016, final defoliation and stAUDefol was significantly greater in plots planted in June than in those planted in May (Table 6.5).

DISCUSSION

The results of these studies clearly illustrate that differences in the epidemiological behavior of ELS and LLS depend on multiple factors, including cultivar, inoculum treatment and planting date. Cultivar had the greatest effect on the onset and severity of LLS, but generally did not affect the onset of ELS. When planted in May and June, cultivar, planting date and inoculum treatment tended to have additive effects on the onset and severity of LLS, and on the severity but not onset of ELS. However, these factors did not always have an additive affect. In peanuts planted in late July or August in fields with a previous history of LLS, the effect of cultivar did not affect the onset of LLS, but did affect the severity of LLS.

These studies confirm previous reports that all peanut cultivars are susceptible to infection by both leaf spot pathogens (Abdou et al., 1974), but some cultivars are partially resistant to one or both leaf spot diseases (Ricker et al., 1985; Walls et al., 1985). Similar results have been reported in other pathosystems that feature the coexistence of closely related species. For example, in the Sigatoka complex of banana, *Mycosphaerella fijiensis*, the cause of black sigatoka, is highly virulent on all of the cultivars that are resistant to *Mycosphaerella musicola*, the cause of yellow sigatoka (Ploetz et al., 2003). Georgia Valencia appears to be an example of a cultivar that is highly-susceptible to both ELS and LLS

(Figs. 6.1 and 6.2), similar to what was observed on the older cultivar, Florigiant (Ricker et al., 1985; Walls et al., 1985). NC 3303 and NC 12C were previously shown to have resistance to ELS but highly susceptible to LLS (Ricker et al., 1985; Walls et al., 1985) and our results from two field trials in 2015 corroborate these findings (Fig. 6.2). Southern Runner and C99R consistently had among the lowest levels of LLS ratings (Figs. 6.1 and 6.2) and confirms previous reports of LLS resistance for these cultivars (Gorbet et al., 1987; Gorbet and Shokes, 2006). New Mexico Valencia C consistently had the highest ELS severity (Figs. 6.1 and 6.2) and generally the lowest LLS predominance, suggesting that it is more susceptible to ELS than LLS, which is similar to results obtained in our previous studies conducted in 2011 and 2012 (Chapter 3). Based on these studies, Georgia-12Y, a relatively new cultivar recently released from the UGA breeding program (Branch, 2013), is more susceptible to ELS than LLS (Fig. 6.2).

Smith and Littrell (1980) suggested that the shift from ELS to LLS was, in part, due to the wide-spread introduction of the Florunner cultivar. We found that ELS onset and intensity was similar on Florunner, Georgia Green and Georgia-06, but that LLS onset was earlier and stAUDPC and lesions per leaflet were higher in Florunner compared with the latter two cultivars (Figs. 6.1 and 6.2; Tables 6.5 and 6.6). Our results suggest that while Florunner is susceptible to ELS, its extreme susceptibility to LLS could have indeed been a factor in shifting the predominance from ELS to LLS back in 1976 (Smith and Littrell, 1980). Similarly, the lower levels of LLS observed on Georgia Green suggest that the wide-spread adaptation of this cultivar in the southeastern United States could have played a role in the shift from LLS to ELS in the early to late 1990s (Cantonwine et al., 2008). The lower levels of LLS on Georgia Green and Georgia-06G were similar to that of Southern Runner and C99R (Figs. 6.1 and 6.2). This is not surprising since Southern Runner and C99R were each a parent line of Georgia Green and Georgia-06G, respectively (Branch, 1996, 2007; Branch and Culbreath, 1995). While LLS severity did not significantly differ between Georgia-06G and Georgia Green, severity values were numerically higher for Georgia-06G in most studies (Figs. 6.1 and 6.2). Taken together, this suggests that, while the recent resurgence of LLS may be partially linked to the introduction of Georgia-06G, it is likely due to additional factors as well.

It should be noted that our hypothesis of the effect of cultivar on the shift in predominance is based on the assumption that the population of each leaf spot pathogen has remained relatively stable over time with respect to virulence. While we cannot be absolutely certain that this is the case, there are several factors in favor of stability. First, the sexual stage of each fungus has not been observed since the time of Jenkins (1938), and most researchers agree that it rarely occurs in nature (Shokes and Culbreath, 1997). Second, the additional cultivars used in these studies with known levels of resistance to LLS (Southern Runner and C99R), and cultivars with known levels of resistance to LLS, and also with resistance to ELS (NC 3303 and NC 12C) had the lowest levels of LLS and ELS, respectively. The data supporting the resistance levels of these cultivars was generated over 30 years ago, and we report very similar results from multiple locations and years in the present study.

Our results show that the onset of ELS was not affected by the cultivars evaluated in these studies (Table 6.5). However, the onset of LLS was generally highly affected by cultivar (Table 6.5). Exceptions occurred in trials planted in late July and August (Table 6.5). In these trials, the aerial inoculum level was most likely extremely high due to heavy LLS epidemics in surrounding fields at the time of planting, coupled with the fact that the environmental conditions were ideal for infection (Table 6.2) (Alderman and Nutter, 1994). We have previously documented that the onset of both ELS and LLS occurs earlier with later planting dates (Chapter 5), but this is the first time we have documented onset of LLS prior to 60 DAP. The fact that the onset of LLS was later in trials planted in May and June can be partly, but not fully, explained by cultivar (Table 6.5). Another explanation may be competition between the ELS and LLS pathogen (Anderson et al., 1993). In these situations, the ELS pathogen was present in all plots at an early stage (Tables 6.5 and 6.8), and usually preceded the onset of LLS by 20 to 30 days. A degree of competitive exclusion (Fitt et al., 2006) could have occurred in which the early leaf spot pathogen out-competed the LLS pathogen for resources, or had an antagonistic effect on the LLS pathogen. This is similar to what we have reported elsewhere (Chapters 3, 4 and 5).

We did not observe major differences in final defoliation nor in the stAUDPC defoliation values for the majority of cultivars monitored in these studies (Table 6.7). This suggests that although more

susceptible cultivars have a greater carrying capacity (lesions per leaflet) of ELS and/or LLS, defoliation progresses at a similar rate in all cultivars. Components of resistance to ELS and LLS have previously been linked to fewer lesions per leaf as well a delay in the time to defoliation (Nevill, 1981; Ricker et al., 1985), but this did not appear to be the case in these studies. However, it should be noted that the disease assessment dates in the studies conducted in 2014 and 2015 were terminated between 70 and 100 DAP (mainly due to delayed planting dates). This may have influenced results for defoliation related values, as well as predominance and severity. However, similar results were seen in the 2016 trials, in which disease was assessed over the entire growing season. This suggests that the overall results would likely have been similar.

In conclusion, these studies illustrate that peanut cultivar can result in the differential development of ELS and LLS in field studies. This is likely due to cultivars with differing levels of resistance to ELS and LLS. The main difference we observed was the influence of cultivar on the prevalence and onset of LLS. The onset of LLS appeared to be much more dependent on peanut cultivar than ELS, but was also dependent on the time of planting and the level of initial inoculum of each pathogen present in the field. Because the prevalence of each leaf spot can be influenced by cultivar, we support the hypothesis that Smith and Littrell (1980) proposed that cultivar could be an important factor in the regional shift from one disease to another. This information is important for breeders, particularly in the context of integrating a cultivar into an integrated management system. As pointed out by Walls et al. (1985), the release of a cultivar with a high level of resistance to only one leaf spot pathogen may be less effective if its introduction leads to a shift to the more aggressive pathogen. Furthermore, for risk indices, such as Peanut Rx, it is critical to know if the risk associated with a given cultivar is applicable to fields with one or both leaf spots. These studies also reinforce the importance of the local inoculum source on the prevalence of each leaf spot. We suggest that it may be possible to use the knowledge of previous field history to select a cultivar with more resistance to the predominant leaf spot.

ACKNOWLEDGEMENTS

We than personnel at the University of Georgia and University of Florida research stations for all of the assistance provided. Particularly, we thank John Paulk, Dewayne Dales, Dan Everts, Andy Carter, Mark Gomillion, Lina Flor Young, Jeannette Mixon, Jhen Bennett and Edna Shannon for technical support; and Xuelin Luo for statistical assistance. Weather data was obtained from the University of Georgia Automated Environmental Monitoring Network.

LITERATURE CITED

Abdou, Y. A.-M., Gregory, W. C., and Cooper, W. E. 1974. Sources and nature of resistance to *Cercospora arachidicola* Hori and *Cercosporidium personatum* (Beck & Curtis) Deighton in *Arachis* species. Peanut Sci. 1:6-11.

Alderman, S., and Nutter, F. 1994. Effect of temperature and relative humidity on development of *Cercosporidium personatum* on peanut in Georgia. Plant Dis. 78:690-694.

Anderson, W., Holbrook, C., and Brenneman, T. 1993. Resistance to *Cercosporidium personatum* within peanut germplasm. Peanut Sci. 20:53-57.

Backman, P., and Crawford, M. 1984. Relationship between yield loss and severity of early and late leafspot diseases of peanut. Phytopathology 74:1101-1103.

Branch, W. 1996. Registration of ‘Georgia Green’ peanut. Crop Sci. 36:806-806.

Branch, W. 2007. Registration of ‘Georgia-06G’ peanut. J Plant Regist 1:120-120.

Branch, W. 2013. Registration of ‘Georgia-12Y’peanut. Journal of Plant Registrations 7:151-153.

Branch, W., and Culbreath, A. 1995. Combination of early maturity and leaf spot tolerance within an advanced Georgia peanut breeding line. Peanut Sci. 22:106-108.

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

Cantonwine, E., Culbreath, A., Holbrook, C., and Gorbet, D. 2008. Disease progress of early leaf spot and components of resistance to *Cercospora arachidicola* and *Cercosporidium personatum* in runner-type peanut cultivars. Peanut Sci. 35:1-10.

Fitt, B. D., Huang, Y.-J., van den Bosch, F., and West, J. S. 2006. Coexistence of related pathogen species on arable crops in space and time. Annu. Rev. Phytopathol. 44:163-182.

Fry, W. E. 1982. Principles of Plant Disease Management. Academic Press, Orlando.

Garren, K., and Wilson, C. 1951. Peanut diseases. Pages 262-324 in: The Peanut, the Unpredictable Legume. Nat. Fertilizer Assoc., Washington, D.C.

Gorbet, D., and Shokes, F. 2006. Registration of 'C99R' peanut. Crop Sci. 46:2713-2714.

Gorbet, D., Norden, A., Shokes, F., Knauft, D., Porter, K., Worrall, W., Gardenhire, J., Gilmore, E., McDaniel, M., and Tuleen, N. 1987. Registration of 'Southern Runner' peanut. Crop Sci. 27:817-817.

Hemingway, J. 1955. The prevalence of two species of Cercospora on groundnuts. Trans. Brit. Mycol. Soc. 38:243-246.

Jenkins, W. A. 1938. Two fungi causing leaf spot of peanut. J. Agr. Res. 56:317-332.

Kemerait, R. C., Jr., Brenneman, T. B., and Culbreath, A. K. 2017. Peanut disease update. Pages 23-62 in: 2017 peanut update. W. Monfort, ed. Coop. Ext. Ser. College of Agric. Environ. Sci. University of Georgia., Athens.

McDonald, D., Subrahmanyam, P., Gibbons, R., and Smith, D. 1985. Early and Late Leaf Spots of Groundnut. Information Bull. No. 21. ICRISAT, Patancheru.

Nevill, D. 1981. Components of resistance to *Cercospora arachidicola* and *Cercosporidium personatum* in groundnuts. Ann. Appl. Biol. 99:77-86.

Ploetz, R., Thomas, J., and Slabaugh, W. 2003. Diseases of banana and plantain. Pages 73-134 in: Diseases of Tropical Fruit Crops. R. Ploetz, ed. CABI Publishing, Wallingford, UK.

Ricker, M., Beute, M., and Campbell, C. 1985. Components of resistance in peanut to *Cercospora arachidicola*. Plant Dis. 69:1059-1064.

Shokes, F., and Culbreath, A. K. 1997. Early and late leaf spots. Pages 17-20 in: Compendium of Peanut Diseases, 2nd Ed. N. Kokalis-Burelle, D. Porter, R. Rodriguez-Kabana, D. Smith and P. Subrahmanyam, eds. American Phytopathological Society Press, St. Paul, MN.

Smith, D. H., and Littrell, R. H. 1980. Management of peanut foliar diseases with fungicides. Plant Dis. 64:356-361.

Walls, S. B., Wynne, J., and Beute, M. 1985. Resistance to late leafspot peanut of progenies selected for resistance to early leafspot. Peanut Sci. 12:17-22.

Woodward, J. E., Brenneman, T. B., Kemerait, R. C., Smith, N. B., Culbreath, A. K., and Stevenson, K. L. 2008. Use of resistant cultivars and reduced fungicide programs to manage peanut diseases in irrigated and nonirrigated fields. Plant Dis. 92:896-902.

Table 6.1. Peanut cultivars used in field trials conducted at Marianna, Florida, and Tifton, Georgia, in 2014, 2015 and 2016.

	Year planted			Description		
Cultivar	2014	2015	2016	Susceptibility[w]	Partial resistance[y]	Growth habit
Georgia Valencia	+[z]	+	+	High	Unknown	Erect
Carver	+	+	+	High	Unknown	Prostrate
Florunner	+	+	+	High	Unknown	Prostrate
Georgia Green	+	+	+	Moderate	Unknown	Prostrate
Georgia-06G	+	+	+	Moderate	Unknown	Prostrate
C99R	+	+	+	Moderate	*C.p.*	Prostrate
Southern Runner		+	+	Low	*C.p.*	Prostrate
New Mexico Valencia C		+	+	High	Unknown	Erect
New Mexico Valencia A		+		High	Unknown	Erect
NC 3303		+		Moderate	*C.a.*	Erect
NC 12C		+		Moderate	*C.a.*	Erect
Perry		+		Moderate	*C.a.*	Erect
Flavor Runner 458		+		High	Unknown	Prostrate
FloRun 107		+		High	Unknown	Prostrate
Georgia-12Y		+		Moderate	Unknown	Prostrate
Tifguard		+		Moderate	Unknown	Prostrate
Bailey		+		Low	*C.a.* + *C.p.*	Prostrate

[w] Susceptibility is generalized for both leaf spots, and is based on personal communications with breeders, and published reports of field evaluations where *Cercospora arachidicola* or *Cercosporidium personatum* or both leaf spot pathogens were present in the field.

[x] Differences in the susceptibility to each pathogen is unknown.

[y] Known to have resistance to *C. arachidicola* (*C.a.*) and/or *C. personatum* (*C.p*).

[z] (+) sign signifies that the cultivar was included in the field trials for a given year.

Table 6.2. Accumulated monthly rainfall (mm) and mean, minimum and maximum monthly air temperature (°C) during the months in which studies took place from 2014 to 2016.

		Tifton (RDC)[y]					Marianna[y]				
		Rainfall		Temperature (°C)			Rainfall		Temperature (°C)		
Year	Month	mm	Days[z]	Mean	Min	Max	mm	Days	Mean	Min	Max
2014											
	September	15.1	19	23.8	20.1	29.6	15.3	10	24.8	14.3	36.6
	October	5.6	5	19.5	13.1	26.6	5.2	3	19.9	5.3	33.5
2015											
	June	8.6	15	26.2	21.4	32.0	10.2	7	27.0	19.8	37.4
	July	32.5	13	26.8	22.0	33.3	12.3	9	28.1	19.9	38.1
	August	11.0	10	26.2	21.9	32.4	7.0	6	27.4	15.4	37.5
	September	4.5	9	23.5	19.2	29.0	10.5	6	24.7	10.9	36.7
	October	5.0	10	19.3	14.0	25.6	2.5	3	20.5	5.5	33.3
		Tifton (RDC farm)					Tift Co. (Ponder farm)				
		Rainfall		Temperature (°C)			Rainfall		Temperature (°C)		
Year	Month	mm	Days	Mean	Min	Max	mm	Days	Mean	Min	Max
2016											
	May	3.7	8	22.4	16.3	28.7	6.6	6	22.7	16.5	29.2
	June	10.0	10	26.3	20.9	32.3	12.2	11	26.5	21.2	32.7
	July	8.6	9	27.5	22.2	34.0	3.9	7	27.8	22.5	34.6
	August	16.0	19	26.5	22.2	32.7	9.4	16	26.8	22.5	33.2
	September	15.6	7	24.7	19.7	31.1	14.8	7	24.9	19.9	31.5
30 yr. avg.											
	May	6.7	-	23.9	17.5	30.4	10.2	-	24.0	17.6	30.4
	June	13.3	-	27.0	21.1	33.0	14.6	-	27.1	21.4	32.8
	July	15.3	-	27.7	22.1	33.4	17.1	-	28.2	22.9	33.5
	August	15.4	-	27.8	22.0	33.7	15.5	-	28.6	23.3	33.9
	September	6.7	-	25.2	19.2	31.2	12.1	-	25.5	20.3	30.7

[y] Weather data were obtained from the Georgia Automated Environmental Monitoring Network (www.Georgiaweather.net), or the Florida Automated Weather Network (http://fawn.ifas.ufl.edu).
[z] Number of days with > 0.254 cm of rain.

Table 6.3. *P*-values from the linear mixed model analysis of variance for all peanut leaf spot related variables for trials conducted from 2014 to 2016 in Tifton, Georgia and Marianna, Florida.

Trial,	Predominance[v]	Onset[w]		Max lesions per leaflet[x]		st AUDPC lesions per leaf leaflet[y]		Defoliation[z]	
source of variance	ProLLS	ELS	LLS	ELS	LLS	ELS	LLS	Final	stAUDefol
2014 trials									
Cultivar	0.0526	0.0078	0.0071	0.0675	0.0002	0.0008	<0.0001	0.0051	0.0005
2015 Marianna									
Cultivar	0.0297	0.7315	0.2632	0.5415	<0.0001	0.5382	0.0107	0.0024	0.0020
2015 Tifton (Bsf)									
Inoculum (I)	0.0613	0.3499	0.0618	0.1938	0.0395	0.0766	0.0444	0.1765	0.0316
Cultivar	<0.0001	0.0286	<0.0001	<0.0001	<0.0001	<0.0001	<.0001	<0.0001	<0.0001
I x Cultivar	0.2458	0.9042	0.8302	0.7961	0.4793	0.9936	0.4868	0.5922	0.2263
2015 Tifton (RDC)									
Inoculum (I)	0.0031	0.2431	0.0004	<0.0001	<0.0001	0.5410	0.0044	-	-
Cultivar	<0.0001	0.1104	0.0064	<0.0001	<0.0001	<0.0001	<0.0001	-	-
I x Cultivar	0.2799	0.8424	0.1963	0.5212	0.6352	0.5838	0.2100	-	-
2016 trials									
Planting date (P)	0.2662	0.0002	<0.0001	0.6454	<0.0001	0.0154	0.5018	<0.0001	<0.0001
Cultivar	<0.0001	0.1261	<0.0001	<0.0001	<0.0001	<0.0001	<0.0001	0.2628	<0.0001
P x Cultivar	0.2706	0.5079	0.1051	0.5212	0.6352	0.7464	0.5350	0.2628	0.2141

[v] Predominance (proportion of the total leaf spot epidemic attributed to late leaf spot; calculated by dividing the standardized AUDPC of late leaf spot by the total standardized AUDPC of early and late leaf spot).

[w] Number of days after planting to reach > 1 % disease incidence for early leaf spot (ELS) and late leaf spot (LLS).

[x] Maximum number of lesions per leaflet; calculated by dividing the total number of lesions by total leaflets per main stem.

[y] stAUDPC = Standardized area under the disease progress curve of the number of per leaflet.

[z] Defoliation at the last rating date; stAUDefol = Standardized area under the defoliation progress curve.

Table 6.4. Effect of peanut cultivar on predominance in field trials conducted in Tifton, Georgia and Marianna, Florida, from 2014 to 2016.

	Predominance[s]				
Cultivar	2014[t]	2015 Tifton (RDC)[u]	2015 Marianna[v]	2015 Tifton (Bsf)[w]	2016[x]
Georgia Valencia	0.78 a[y]	0.54 a	0.99 a	0.86 ab	0.78 a
Carver	0.88 a	0.54 a	0.99 ab	0.82 abc	0.79 a
Florunner	0.92 a	0.36 a	0.99 a	0.79 abc	0.71 a
Georgia Green	0.72 a	0.17 b	0.97 ab	0.64 abcd	0.54 b
Georgia-06G	0.77 a	0.16 b	0.99 ab	0.62 bced	0.57 b
C99R	0.76 a	0.15 b	0.98 ab	0.69 abcd	0.57 b
Southern Runner	-[z]	-	0.97 abc	0.66 abcd	0.53 b
NM Valencia C	-	-	0.99 ab	0.48 de	0.40 c
NC 3303	-	-	0.99 a	0.89 a	-

[s] Predominance (proportion of the total leaf spot epidemic attributed to late leaf spot; calculated by dividing the standardized AUDPC of late leaf spot by the total standardized AUDPC of early and late leaf spot).

[t] Results from three locations (two in Tifton, one in Marianna) that all had a previous history of late leaf spot and were planted in late August.

[u] Results are averaged across four inoculum treatments. Inoculum treatments consisted of peanut residue infested with *Cercospora arachidicola*, *Cercosporidium personatum*, *Ca + Cp*, and a residue-free control. Plots were planted on 14 May.

[v] Plots were planted on 28 July in a field with a previous history of late leaf spot.

[w] Plots were planted on 24 June in a field with a previous history of early leaf spot. Results are averaged across plots inoculated with peanut residue infested with *C. personatum* and non-inoculated plots.

[x] Results are from two separate field trials in Tifton and are averaged across plots planted on 14 May and 10 June; both fields were peanut behind peanut with severe early and late leaf spot epidemics in the previous year.

[y] Means within columns with the same letters are not significantly different based upon Tukey's honestly significant difference test ($\alpha = 0.05$).

[z] Cultivars not planted in these trials.

Table 6.5. Effect of inoculum treatment and planting date on the development of early and late leaf spot at the Black Shank farm in field trials conducted in Tifton, Georgia, 2015 and 2016, respectively.

		Predominance[u]	Onset[v]		Max lesions per leaflet[w]		stAUDPC [x] (lesions per leaf leaflet)		Defoliation[y]	
Trial	Factor	ProLLS	ELS	LLS	ELS	LLS	ELS	LLS	Final	stAUDefol
2015 Tifton	Inoculum									
(Bsf)	*Ca*	0.58 a[z]	41 a	48 a	2.4 a	8.3 b	0.7 a	1.4 b	56.5 a	24.6 b
	Ca + Cp	0.79 a	38 a	37 a	1.6 a	13.1 a	0.5 a	3.0 a	60.4 a	28.2 a
2015 Tifton	Inoculum									
(RDC)	*Ca*	0.02 b	45 a	82 a	3.4 a	0.2 c	0.72 a	0.03 b	-	-
	Cp	0.68 a	46 a	55 b	1.1 ab	8.2 a	0.36 a	1.19 a	-	-
	Ca + Cp	0.40 a	48 a	62 b	1.2 ab	4.6 ab	0.41 a	0.33 a	-	-
	None	0.33 ab	63 a	67 ab	0.6 b	1.2 bc	0.34 a	0.16 ab	-	-
2016 Tift Co.	Planting date									
	14 May	0.62 a	48 a	88 a	2.7 a	7.7 a	0.8 a	1.2 a	86.5 b	34.0 b
	10 June	0.58 a	38 b	72 b	2.9 a	5.8 b	0.6 b	0.9 a	97.8 a	41.2 a

[u] Predominance (proportion of the total leaf spot epidemic attributed to late leaf spot; calculated by dividing the standardized AUDPC of late leaf spot by the total standardized AUDPC of early and late leaf spot combined).

[v] Number of days after planting to reach > 1 % disease incidence for early leaf spot (ELS) and late leaf spot (LLS).

[w] Maximum number of lesions per leaflet; calculated by dividing the total number of lesions by total leaflets per main stem.

[x] stAUDPC = Standardized area under the disease progress curve of the number of per leaflet.

[y] Defoliation at the last rating date; stAUDefol = Standardized area under the defoliation progress curve.

[z] Means within columns with the same letters are not significantly different based upon Tukey's honestly significant difference test ($\alpha = 0.05$).

Table 6.6. Effect of cultivar on the onset of early and late leaf spot in field trials conducted in Tifton, Georgia and Marianna, Florida, from 2014 to 2016.

	2014[s]		2015 Tifton (RDC)[t]		2015 Marianna[u]		2015 Tifton (Bsf)[v]		2016[w]	
Cultivar	ELS	LLS	ELS	LLS	ELS	LLS	ELS	LLS	ELS	LLS
Georgia Valencia	[x]26 b[y]	39 ab	46 a	55 b	48 a	29 a	36 a	34 c	42 a	63 d
Carver	43 ab	27 b	49 a	62 b	47 a	29 a	42 a	40 bc	43 a	77 bc
Florunner	43 ab	28 b	51 a	60 b	58 a	29 a	42 a	41 bc	44 a	75 c
Georgia Green	41 ab	43 ab	53 a	67 ab	48 a	39 a	44 a	50 ab	45 a	88 a
Georgia-06G	32 ab	37 ab	53 a	70 ab	58 a	34 a	42 a	48 abc	45 a	88 a
C99R	62 a	47 a	50 a	83 a	48 a	39 a	44 a	46 abc	45 a	84 ab
Southern Runner	- [z]	-	-	-	49 a	29 a	40 a	45 abc	43 a	88 a
NM Valencia C	-	-	-	-	53 a	34 a	32 a	38 bc	41 a	77 bc
NC 3303	-	-	-	-	63 a	39 a	38 a	40 bc	-	-

[s] Results from three locations (two in Tifton, one in Marianna) that all had a previous history of late leaf spot and planted in late August.

[t] Results are averaged across four inoculum treatments. Inoculum treatments consisted of peanut residue infested with *Cercospora arachidicola*, *Cercosporidium personatum*, *Ca + Cp*, and a residue-free control. Plots were planted on 14 May.

[u] Plots were planted on 28 July in a field with a previous history of late leaf spot.

[v] Plots were planted on 24 June in a field with a previous history of early leaf spot. Results are averaged across plots inoculated with peanut residue infested with *C. personatum* and non-inoculated plots.

[w] Results are from two separate field trials in Tifton, Georgia and are averaged across plots planted on 14 May and 10 June; both fields were peanut behind peanut with severe early and late leaf spot epidemics in the previous year.

[x] Number of days after planting to reach > 1 % disease incidence for early leaf spot (ELS) and late leaf spot (LLS).

[y] Means within columns with the same letters are not significantly different based upon Tukey's honestly significant difference test ($\alpha = 0.05$).

[z] Cultivars not planted in these trials.

Table 6.7. Effect of cultivar on the maximum number of early and late leaf spot lesions per leaflet in field trials conducted in Tifton, Georgia and Marianna, Florida, from 2014 to 2016.

	2014[t]		2015 Tifton (RDC)[u]		2015 Marianna[v]		2015 Tifton (Bsf)[w]		2016[x]	
Cultivar	ELS	LLS	ELS	LLS	ELS	LLS	ELS	LLS	ELS	LLS
Georgia Valencia	1.2 a[y]	7.9 ab	3.1 a	14.1 a	0.1 a	16.6 a	2.0 bcd	27.0 a	2.8 b	11.4 a
Carver	0.5 a	9.5 a	0.8 c	5.0 b	0.1 a	6.9 bcde	1.5 cd	15.4 bc	2.4 b	11.2 a
Florunner	0.5 a	9.6 a	2.0 ab	3.7 b	0.1 a	10.5 ab	1.8 cd	13.9 bcd	2.8 b	8.9 a
Georgia Green	0.9 a	3.0 c	1.2 bc	0.4 c	0.1 a	5.2 cde	2.1 bcd	7.2 efg	2.8 b	5.1 b
Georgia-06G	0.9 a	4.0 bc	1.0 bc	0.5 c	0.1 a	7.3 bcde	2.3 bcd	7.0 efg	2.9 b	5.6 b
C99R	0.6 a	4.2 bc	0.8 c	0.3 c	0.1 a	6.0 bcde	1.9 cd	9.1 cdefg	2.4 b	4.7 b
Southern Runner	-[z]	-	-	-	0.2 a	4.6 cde	1.7 cd	6.6 efg	1.9 b	3.9 b
NM Valencia C	-	-	-	-	0.2 a	10.7 ab	4.6 a	9.4 cdef	5.2 a	5.0 b
NC 3303	-	-	-	-	0.1 a	10.5 ab	0.9 d	17.6 ab	-	-

[t] Results from three locations (two in Tifton, one in Marianna) that all had a previous history of late leaf spot and were planted in late August.

[u] Results are averaged across four inoculum treatments. Inoculum treatments consisted of peanut residue infested with *Cercospora arachidicola*, *Cercosporidium personatum*, *Ca + Cp*, and a residue-free control. Plots were planted on 14 May.

[v] Plots were planted on 28 July in a field with a previous history of late leaf spot.

[w] Plots were planted on 24 June in a field with a previous history of early leaf spot. Results are averaged across plots inoculated with peanut residue infested with *C. personatum* and non-inoculated plots.

[x] Results are from two separate field trials in Tifton and are averaged across plots planted on 14 May and 10 June; both fields were peanut behind peanut with severe early and late leaf spot epidemics in the previous year.

[y] Means within columns with the same letters are not significantly different based upon Tukey's honestly significant difference test ($\alpha = 0.05$).

[z] Cultivars not planted in these trials.

Table 6.8. Effect of cultivar on the final defoliation and the standardized AUDPC of defoliation in field trials conducted in Tifton, Georgia and Marianna, Florida, from 2014 to 2016.

	2014[u]		2015 Marianna[v]		2015 Tifton[w]		2016[x]	
Cultivar	Final	stAUDefol	Final	stAUDefol	Final	stAUDefol	Final	stAUDefol
Georgia Valencia	46 b[y]	23.0 b	49 a	20.6 a	66.4 abc	22.9 c	93.6 a	37.0 b
Carver	68 a	43.1 a	55 a	27.9 a	58.6 abc	29.2 ab	91.7 a	37.2 b
Florunner	61 a	37.5 a	59 a	30.7 a	61.0 abc	27.7 abc	93.0 a	37.7 b
Georgia Green	60 a	39.0 a	58 a	29.6 a	61.4 abc	28.3 ab	90.4 a	38.6 ab
Georgia-06G	61 a	38.3 a	47 ab	21.6 a	61.9 abc	30.3 ab	92.2 a	40.3 a
C99R	59 ab	37.5 a	62 a	29.6 a	59.5 abc	29.0 ab	91.4 a	37.7 b
Southern Runner	- [z]	-	49 ab	27.6 a	63.8 abc	28.5 ab	91.1 a	38.9 ab
New Mexico Valencia C	-	-	23 bc	9.5 bc	42.5 e	15.4 d	92.3 a	32.5 c
NC 3303	-	-	44 ab	19.8 ab	60.3 abc	28.0 abc	-	-

[u] Results from three locations (two in Tifton, one in Marianna) that all had a previous history of late leaf spot and planted in late August.

[v] Plots were planted on 28 July in a field with a previous history of late leaf spot.

[w] Plots were planted on 24 June in a field with a previous history of early leaf spot. Results are averaged across plots inoculated with peanut residue infested with *C. personatum* and non-inoculated plots.

[x] Results are from two separate field trials in Tifton, Georgia and are averaged across plots planted on 14 May and 10 June; both fields were peanut behind peanut with severe early and late leaf spot epidemics in the previous year.

[y] Means within columns with the same letters are not significantly different based upon Tukey's honestly significant difference test ($\alpha = 0.05$).

[z] Cultivars not planted in these trials.

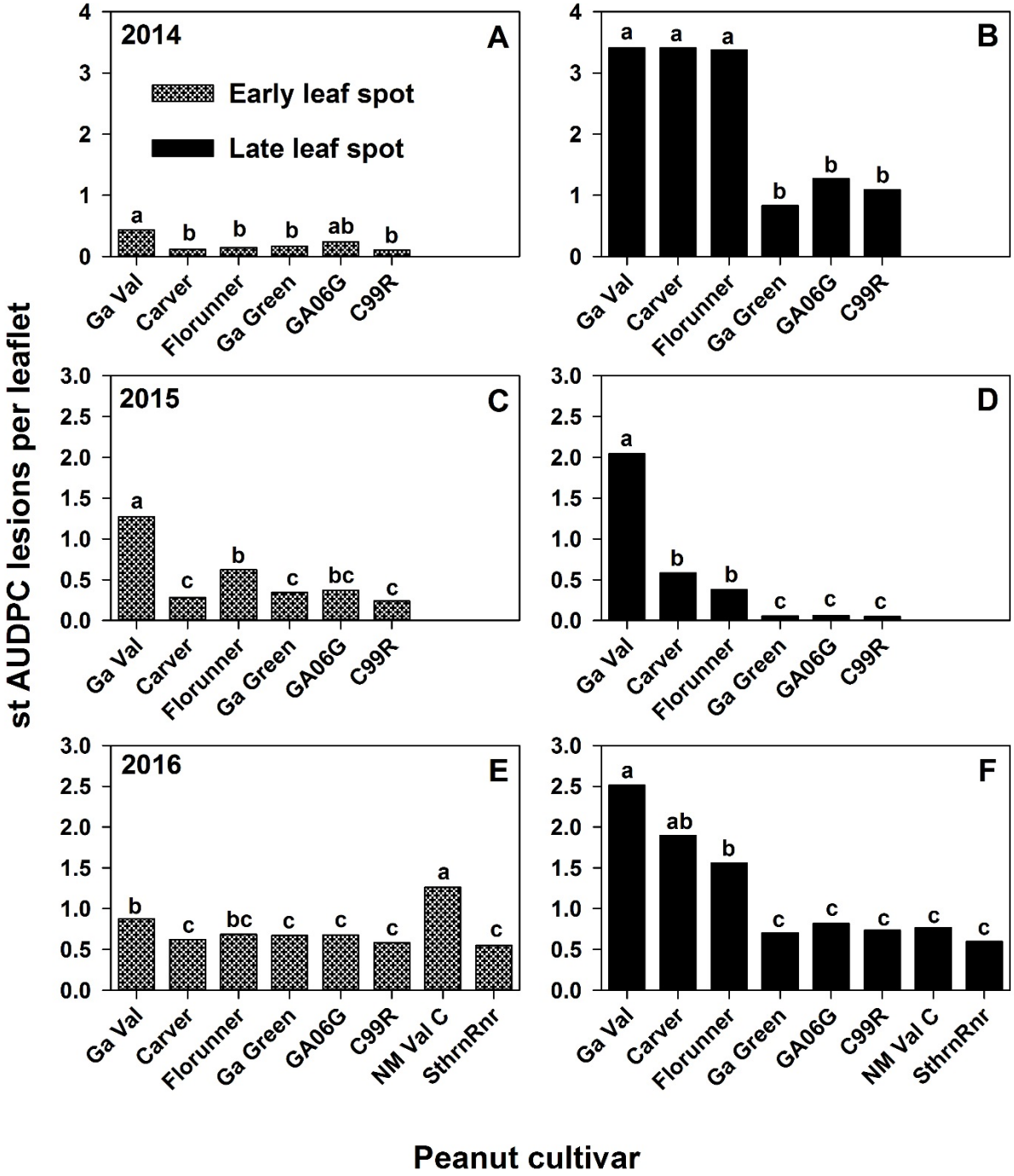


Fig. 6.1. Effect of peanut cultivar on the standardized AUDPC (area under the disease progress curve) of early or late leaf spot lesions per leaflet. **A** and **B.** Results from three field trials conducted in 2014 at Tifton and Marianna in fields with a prior history of late leaf spot. Two fields at Tifton were planted on 29 August and in Marianna on 21 August and monitored until 77 and 85 days after planting, respectively. **C** and **D**. Results are from a single field trial conducted in Tifton, Georgia, in 2015, and are averaged across four inoculum treatments. **E** and **F**. Results are averaged across two planting dates (12 May and 10 June) in two separate, non-rotated fields in Tifton; both fields had a previous history of severe early and late leaf spot epidemics. Bars with the same letters indicate means are not significantly different based upon Tukey's honestly significant difference test ($\alpha = 0.05$).

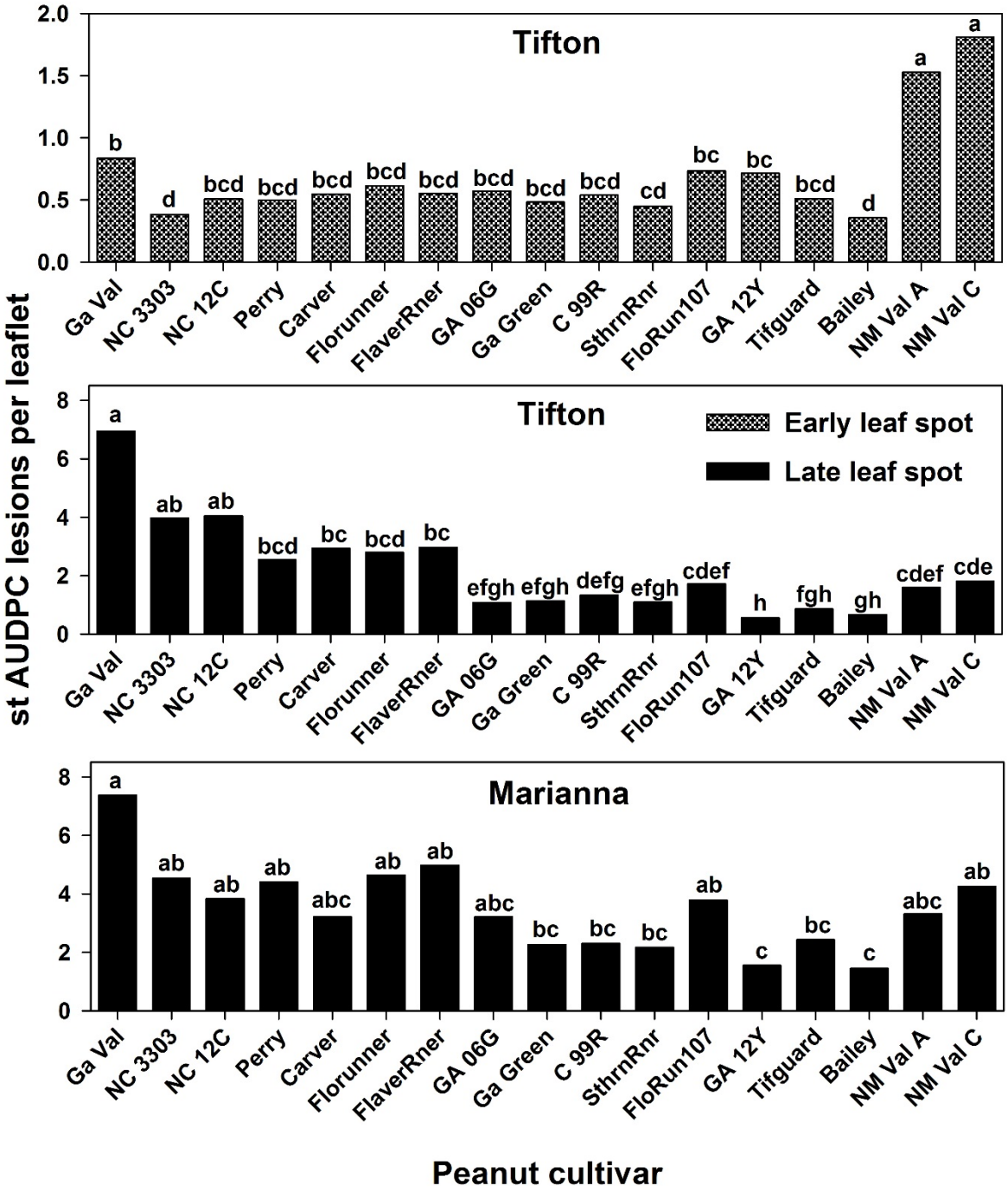


Fig. 6.2. Effect of cultivar on the standardized AUDPC (area under the disease progress curve) of early or late leaf spot lesions per leaflet in fields with a prior history of early leaf spot (Tifton) or late leaf spot (Marianna) during 2015. Results at Tifton are averaged across plots with or without the addition of peanut residue infested with *Cercosporidium personatum*. Plots were planted on 24 June and 28 July and evaluated and rated until 85 and 73 DAP at Tifton and Marianna, respectively. Grouped bars with the same letters indicate means are not significantly different based upon Tukey's honestly significant difference test ($\alpha = 0.05$).

Supplementary Table 6.1. Effect of all cultivars on the development of early and late leaf spot at the Black Shank farm in Tifton, Georgia, 2015.

	Predominance[u]	Onset[v]		Max lesions per leaflet[w]		Defoliation[x]	
Cultivar	ProLLS	ELS	LLS	ELS	LLS	Final	stAUDefol
Georgia Valencia	0.86 ab[y]	36 a	34 c	2.0 bcd	27.0 a	66.4 abc	22.9 c
NC 3303	0.89 a	38 a	40 bc	0.9 d	17.6 ab	60.3 abc	28.0 abc
NC 12C	0.85 ab	38 a	34 c	1.5 cd	17.8 ab	73.0 a	33.9 abc
Perry	0.80 abc	38 a	40 bc	1.3 cd	12.8 bcde	67.2 ab	30.5 ab
Carver	0.82 abc	42 a	40 bc	1.5 cd	15.4 bc	58.6 abc	29.2 ab
Florunner	0.79 abc	42 a	41 bc	1.8 cd	13.9 bcd	61.0 abc	27.7 abc
Flavor Runner	0.81 abc	42 a	38 bc	1.5 cd	14.8 bcd	61.5 abc	31.7 a
Georgia Green	0.64 abcd	44 a	50 ab	2.1 bcd	7.2 efg	61.4 abc	28.3 ab
Georgia-06G	0.62 bced	42 a	48 abc	2.3 bcd	7.0 efg	61.9 abc	30.3 ab
C99R	0.69 abcd	44 a	46 abc	1.9 cd	9.1 cdefg	59.5 abc	29.0 ab
Southern Runner	0.66 abcd	40 a	45 abc	1.7 cd	6.6 efg	63.8 abc	28.5 ab
FloRun 107	0.66 abcd	38 a	46 abc	2.3 bcd	10.4 bcde	54.7 bcde	25.5 bc
Georgia-12Y	0.42 e	41 a	46 abc	2.9 abc	3.7 g	59.4 abc	28.8 ab
Tifguard	0.59 cde	44 a	61 a	1.7 cd	6.5 efg	51.6 edc	25.4 bc
Bailey	0.62 bced	44 a	46 abc	1.2 cd	4.4 fg	55.6 bcd	28.1 abc
NM Valencia A	0.49 de	36 a	38 bc	4.1 ab	8.0 defg	43.8 ed	14.6 d
NM Valencia C	0.48 de	32 a	38 bc	4.6 a	9.4 cdef	42.5 e	15.4 d
ANOVA[z]	*P* > *F*	*P* > *F*	*P* > *F*	*P* > *F*	*P* > *F*	*P* > *F*	*P* > *F*
Inoculum (I)	0.0613	0.3499	0.0618	0.1938	0.0395	0.1765	0.0316
Cultivar (C)	<0.0001	0.0286	<0.0001	<0.0001	<0.0001	<0.0001	<0.0001
I x G	0.2458	0.9042	0.8302	0.7961	0.4793	0.5922	0.2263

[u] Predominance (proportion of the total leaf spot epidemic attributed to late leaf spot; calculated by dividing the standardized AUDPC of late leaf spot by the total standardized AUDPC of early and late leaf spot combined).

[v] Number of days after planting to reach > 1 % disease incidence for early leaf spot (ELS) and late leaf spot (LLS).

[w] Maximum number of lesions per leaflet; calculated by dividing the total number of lesions by total leaflets per main stem.

[x] Defoliation at the last rating date; stAUDefol = Standardized area under the defoliation progress curve.

[y] Means within columns with different letters are significantly different based upon Tukey's honestly significant difference test ($\alpha = 0.05$).

[z] *P*-values from the linear mixed model analysis of variance.

Supplementary Table 6.2. Effect of all cultivars on the development of early and late leaf spot in a field with a history of late leaf spot in Marianna, Florida, 2015.

	Predominance[u]	Onset[v]		Max lesions per leaflet[w]		Defoliation[x]	
Cultivar	ProLLS	ELS	LLS	ELS	LLS	Final	stAUDefol
Georgia Valencia	0.99 a[y]	48 a	29 a	0.1 a	16.6 a	49 a	20.6 a
NC 3303	0.99 a	63 a	39 a	0.1 a	10.5 ab	44 ab	19.8 ab
NC 12C	0.99 a	53 a	34 a	0.1 a	7.4 bcd	59 a	30.8 a
Perry	0.99 a	44 a	29 a	0.1 a	9.0 bcd	68 a	29.9 a
Carver	0.99 ab	47 a	29 a	0.1 a	6.9 bcde	55 a	27.9 a
Florunner	0.99 a	58 a	29 a	0.1 a	10.5 ab	59 a	30.7 a
Flavor Runner	0.98 ab	53 a	29 a	0.3 a	10.7 ab	61 a	32.4 a
Georgia Green	0.97 ab	48 a	39 a	0.1 a	5.2 cde	58 a	29.6 a
Georgia-06G	0.99 ab	58 a	34 a	0.1 a	7.3 bcde	47 ab	21.6 a
C99R	0.98 ab	48 a	39 a	0.1 a	6.0 bcde	62 a	29.6 a
Southern Runner	0.97 abc	49 a	29 a	0.2 a	4.6 cde	49 ab	27.6 a
FloRun 107	0.99 ab	53 a	39 a	0.1 a	9.1 bc	52 a	23.4 a
Georgia-12Y	0.93 c	43 a	29 a	0.4 a	3.9 de	59 a	27.5 a
Tifguard	0.95 bc	43 a	39 a	0.3 a	5.2 cde	45 ab	26.1 a
Bailey	0.97 ab	53 a	39 a	0.1 a	3.1 e	57 a	29.9 a
NM Valencia A	0.98 ab	48 a	44 a	0.1 a	8.6 bc	19 c	6.8 c
NM Valencia C	0.99 ab	53 a	34 a	0.2 a	10.7 ab	23 bc	9.5 bc
ANOVA[z]	*P* > *F*	*P* > *F*	*P* > *F*	*P* > *F*	*P* > *F*	*P* > *F*	*P* > *F*
Cultivar	<0.0001	0.6669	0.0588	0.1131	<0.0001	<0.0001	<0.0001

[u] Predominance (proportion of the total leaf spot epidemic attributed to late leaf spot; calculated by dividing the standardized AUDPC of late leaf spot by the total standardized AUDPC of early and late leaf spot combined).

[v] Number of days after planting to reach > 1 % disease incidence for early leaf spot (ELS) and late leaf spot (LLS).

[w] Maximum number of lesions per leaflet; calculated by dividing the total number of lesions by total leaflets per main stem.

[x] Defoliation at the last rating date; stAUDefol = Standardized area under the defoliation progress curve.

[y] Means within columns with different letters are significantly different based upon Tukey's honestly significant difference test ($\alpha = 0.05$).

[z] *P*-values from the linear mixed model analysis of variance.

CHAPTER 7

COMPONENTS OF RESISTANCE TO AND ASSOCIATION BETWEEN EARLY AND LATE LEAF SPOT ON THREE WIDELY PLANTED PEANUT CULTIVARS IN THE SOUTHEASTERN UNITED STATES[1]

[1]Fulmer, A.M., Kemerait, R.C., Brenneman, T.B., Scherm, H., Cantonwine, E.G., Culbreath, A.K., and Stevenson, K.L. 2017. To be submitted to *Plant Disease*.

ABSTRACT

Cercospora arachidicola (*Ca*) and *Cercosporidium personatum* (*Cp*), the pathogens that cause early (ELS) and late (LLS) leaf spot, respectively, were inoculated separately or together in two greenhouse studies on the historically dominant peanut cultivars Florunner, Georgia Green and Georgia-06G, and on one susceptible cultivar, Georgia Valencia. Except for time to defoliation, there were no statistically significant interactions among cultivar and inoculum. For both pathogens, incubation period and percent incubation at 17 days after inoculation (PI) did not differ among cultivars, but incubation period was 2 days shorter and PI was 30% higher for *Ca* compared with *Cp*. Infection frequencies of *Ca* and *Cp* were highest on Georgia Valencia, but did not significantly differ among the other cultivars. Lesion diameter for both pathogens was significantly smaller on Georgia Green and Georgia-06G compared to the other cultivars. Sporulation of *Ca* did not differ between cultivars, but *Cp* sporulation was significantly higher on Georgia Valencia and Florunner than on Georgia Green and Georgia-06G. Time to defoliation did not differ among cultivars inoculated with *Ca* or *Ca + Cp*, but defoliation was numerically or significantly later on all cultivars inoculated with *Cp*. Final defoliation values were lowest for leaflets inoculated with *Cp* and, averaged across inoculum treatments, lowest on Georgia Valencia. Co-inoculations of *Ca + Cp* resulted in a 33% reduction in the number of ELS lesions and a 73% reduction in the number of LLS when compared to *Ca* alone and *Cp* alone, respectively. Incidence, incubation period and lesion diameter of LLS were also significantly lower when inoculated with *Ca + Cp* compared to single inoculations, but the same was not observed for ELS. Results from this study indicate that components of resistance to ELS are similar among cultivars tested, but that Florunner is more susceptible to LLS than Georgia Green and Georgia-06G. Co-inoculations indicate that there is a negative association between ELS and LLS, but that LLS is more suppressed in the presence of ELS. Taken together, these results suggest that differences in host resistance between the two diseases and a negative association between ELS and LLS may contribute to shifts in prevalence and differences in the epidemiological behavior of ELS and LLS observed in the field.

INTRODUCTION

Early (ELS) and late (LLS) leaf spot are two of the most important foliar diseases of peanut worldwide, and are caused by *Cercospora arachidicola* Hori (*Ca*) and *Cercosporidium personatum* (Berk & M.A. Curtis) Deighton (*Cp*) (Shokes and Culbreath, 1997). Although similar in appearance, disease cycle and damage to the crop, the comparative behavior of each disease often differs. In Georgia, LLS was predominant from 1976 to the early 1990s; otherwise ELS has generally been the most prevalent (Cantonwine et al., 2008; Smith and Littrell, 1980). In recent years, the prevalence of LLS has increased (Cantonwine et al., 2008), and predominance can differ between nearby fields (Chapter 3). Depending on the inoculum level in the field, the onset of both diseases can range from 30 to 140 days after planting (DAP) (Chapters 3, 4, 5 and 6), but ELS generally appears 2 to 4 weeks prior to LLS (Shokes and Culbreath, 1997; Smith and Littrell, 1980). Nevertheless, LLS is thought to be the more destructive of the two due to a greater rate of disease increase (Backman and Crawford, 1984; Hemingway, 1955; Jenkins, 1938).

Smith and Littrell (1980) suggested that the shift from ELS to LLS in Georgia and Florida during 1976 could have been linked to the widespread introduction of a new cultivar (Florunner). Because resistance to ELS and LLS is partial and thought to be inherited independently for each leaf spot pathogen (Abdou et al., 1974; Cantonwine et al., 2008), it is possible to have cultivars with differing or similar levels of resistance to each disease. For example, in one study, the cultivar NC 3303 was shown to have high levels of resistance to ELS (Ricker et al., 1985), but in another study, it was shown to be highly susceptible to LLS (Walls et al., 1985). In contrast, the cultivar Florigiant was found to be highly susceptible to both leaf spot diseases (Ricker et al., 1985; Walls et al., 1985). For both diseases, partial resistance of peanut cultivars is comprised of one or more components that are related to rate-reducing resistance (Abdou et al., 1974; Cantonwine et al., 2008; Nevill, 1981; Walls et al., 1985). These include delayed incubation and/or latent periods, and reduced infection frequency, lesion size, sporulation and defoliation. These components are often correlated (Chiteka et al., 1988a; Walls et al., 1985), but some components appear to vary between cultivars and environments (Shew et al., 1988). However, a longer

latent period and reduced sporulation appear to be the most consistent measures of resistance to both leaf spot pathogens (Anderson et al., 1993; Aquino et al., 1995; Cantonwine et al., 2008; Chiteka et al., 1988b; Nevill, 1981; Ricker et al., 1985; Shew et al., 1988; Walls et al., 1985).

Three main cultivars have dominated the peanut acreage in the southeastern United States over the past 50 years: Florunner (1970-1996), Georgia Green (1996-2008), and Georgia06-G (2006-present) (Beasley, personal communication). Interestingly, the shifts in prevalence from ELS to LLS, LLS to ELS and, more recently, ELS to LLS are somewhat correlated with the introduction of Florunner, Georgia Green and Georgia-06G, respectively. In recent field studies, we demonstrated that a number of cultivars, including Florunner, appeared much more susceptible to LLS than the more recent cultivars, Georgia Green and Georgia-06G, but all cultivars appeared equally susceptible to ELS (Chapter 6). Alhough not statistically different, results from the same studies showed that Georgia-06G tended to have more LLS than Georgia Green (Chapter 6). While these data suggest that these cultivars could have differential levels of resistance to each disease, the actual relative resistance for each cultivar to ELS compared to that of LLS is less clear.

Monasterios de La Torre (1980) screened multiple cultivars, including Florunner, for resistance to ELS and LLS, and his results suggested that Florunner seemed equally susceptible to both diseases. In a different study, Branch and Culbreath (1995) observed that Florunner appeared to have more resistance to ELS than LLS. Georgia Green was reported to have more resistance to both leaf spot diseases compared with Florunner (Cantonwine et al., 2008). Georgia Green was derived from a cross between Sunbelt Runner and Southern Runner, with the latter cultivar having a moderate level of resistance to LLS (Branch, 1996; Gorbet et al., 1987). Though not specifically compared in the same study, results from detached leaf assays indicated that Georgia Green could have slightly higher levels of susceptibility to ELS than to LLS (Cantonwine et al., 2008). In that study, the infection frequency was high for both diseases, but it appeared that the LLS pathogen had much lower levels of sporulation on Georgia Green compared with ELS (Cantonwine et al., 2008). Georgia-06G is thought to have similar levels of

resistance as Georgia Green, and was derived from a cross between Georgia Green and C99R, a cultivar with resistance to LLS (Branch, 2007; Gorbet and Shokes, 2006).

In ecological theory, there are three basic interactions between pathogens occupying the same resource: negative, neutral or synergistic effects on neighboring species (Le May et al., 2009). Negative interactions are a result of interspecific competition between pathogens when resources are in short supply, and according to the competitive exclusion principle, species sharing the same niche will eventually drive competitors to extinction or exclusion from a given area (Fitt et al., 2006). Anderson et al. (1993) reported higher levels of LLS on Florunner compared with ELS, but suggested that this was due to a negative interaction between leaf spot pathogens rather than a difference in host susceptibility. Monasterios de La Torre (1980) also reported the possibility of a negative interaction between *Ca* and *Cp* when co-inoculations were compared with inoculations of each pathogen alone on multiple cultivars in a greenhouse study. For the cultivar Florunner, he reported that when both pathogens were inoculated together at a combined concentration of 20,000 spores/ml, the number of ELS lesions was reduced by 36% compared to inoculations with *Ca* alone at a concentration of 20,000 spores/ml. In contrast, the number of LLS lesions was reduced by 92% compared with inoculations of *Cp* alone at a concentration of 20,000 spores/ml (Monasterios de La Torre, 1980). The author attributed this reduction to three possibilities: a negative interaction between species, differences in spore concentrations, or different levels of susceptibility in Florunner (Monasterios de La Torre, 1980).

Integrated disease management programs are dependent on knowing the onset of the disease in question and the expected level of severity in the absence of intervention (Fry, 1982). Given the increased prevalence of LLS in Georgia, a more detailed understanding of the mechanisms involved in the differing epidemiological behavior of the two leaf spot diseases is essential. Knowledge of differential levels of resistance of peanut cultivars to ELS and LLS could foreseeably aid our ability to predict the initiation and/or development of a given leaf spot epidemic. Additionally, a more detailed understanding of the interaction between the leaf spot pathogens could help to elucidate the role of resistance versus that of competition for resources. Therefore, the objectives of this study were to 1) compare the components

of resistance to ELS and LLS in the cultivars Georgia Valencia, Florunner, Georgia Green and Georgia-06G, and 2) determine whether co-inoculations of *Ca* and *Cp* would result in a negative, positive or neutral interaction between ELS and LLS for each cultivar.

MATERIALS AND METHODS

Experimental design and treatments. Two greenhouse trials were conducted during the summer and fall of 2016 at the University of Georgia's South Milledge Greenhouse Complex located in Athens, Georgia. Both trials were conducted in separate greenhouse sections. Each trial was laid out in split plot design with four replications with inoculum as the main plots and cultivar as the subplots. This experimental design was selected in order to decrease the potential for contamination between inoculum treatments. There were four cultivars: Georgia Valencia, Florunner, Georgia Green and Georgia-06G. Seed of the first three cultivars was obtained from breeder stock and the latter was obtained from Georgia Foundation Seed. Each greenhouse section was equipped with four benches ~ 1.5 m wide × 9.1 m long with ~ 0.91 m between each bench. Each replication was confined to a single bench and within each inoculum treatment, 4 pots (each containing 3 plants of each cultivar) were placed ~ 0.35 m apart within a ~1.5 × 2 m section on the bench according to each sub-plot randomization. For each cultivar and each replication, three seeds pre-inoculated with *Rhizobium* spp. (Peanut Power, E-Commerce Biologics LLC, Sioux Falls, SD) were planted at a depth of 1.3 cm in a 7.5 liter plastic pot. Each pot was filled with a Fafard RSi #3 grower mix composed of the following ingredients: 35-45% Canadian sphagnum peat moss, aged pine bark, perlite, vermiculite, dolomitic lime and wetting agent (Sun Gro Horticulture, Agawam, MA). The first trial was planted on 27 June and the second trial on 31 August.

Inoculum treatments consisted of 30,000 conidia/ml of *C. arachidicola* (*Ca*) alone, *C. personatum* (*Cp*) alone, a 30,000/30,000 conidia/ml mix of *Ca* + *Cp* and a non-inoculated control (distilled water + 0.005% Tween 20). Field inoculum was used in both greenhouse studies. Leaves were collected from fields that were predominantly early or late leaf spot and allowed to dry at room temperature for 1 to 2 weeks. Sporulating lesions of each leaf spot pathogen were excised and stored in separate vials at approximately 4.0°C until needed. For the first trial, inoculum was collected in June and

July of 2016. For the second trial, inoculum was collected in July and August of 2016. Conidial suspensions were prepared by first vortexing approximately 50 sporulating lesions in 30 ml of distilled water for 5 min, then filtering contents through sterilized cheese cloth. The process was repeated until sufficient conidia were obtained. Final concentrations were obtained by diluting with distilled water + 0.005% Tween 20, and concentrations were confirmed twice with a hemacytometer.

Peanut plants were inoculated at 30 days after planting, in the late afternoon on the same day as the inoculum was prepared. For each pot, five newly expanded leaves located on the 3rd to 4th node of the terminal end of the main or lateral stem were tagged for inoculation. At least one leaf from each of the three plants in each pot was selected for inoculation. Each pot was carried outside of the greenhouse chamber, and the adaxial side of each leaflet of every tagged leaf was held upright, pulled away from the plant, and sprayed with each inoculum treatment or the water + Tween 20 control for 1 s with a Spra-Tool spray gun (Aervoe Industries, Inc., Gardnerville, NV) at a distance of ~ 3 cm. Plants were returned to the greenhouse bench and allowed to dry for 2 h. Afterwards, each plant was misted until leaves were visibly wet, and then bagged with a clear, 189.2-liter plastic bag (The Home Depot Inc.), and tied around the top (0.6 m) of a bamboo stake located in the center of each pot. After 14 h (late morning), plants were uncovered and allowed to dry during daylight hours, and the same leaf wetting/bagging process that evening. The wet/dry incubation cycle continued for the following 2 days, for a total of three nights with a 14-h leaf wetness duration. Plants remained uncovered for the remainder of the study, and received water on a daily basis. Fertility and insecticide/miticide (for spider mites, whiteflies and thrips) applications were made at regular intervals. Temperature and relative humidity within the greenhouse were measured with VP-3 sensors (Decagon Devices Inc., Pulman, WA). Four sensors were placed arbitrarily throughout the greenhouse section, and Decagon Em50 data loggers were set to record hourly observations.

Disease assessment. Disease was assessed beginning 13 and 9 days after inoculation (DAI) in the summer and fall trials, respectively. For each replication, three tagged leaves were evaluated for the presence or absence of each leaflet; the number of ELS and LLS lesions (≥ 1 mm) were counted for each

leaflet, and lesions were examined with a 15x hand lens for sporulation. The same assessments were made every other day until 34 DAI and then once a week until the trial was terminated. The summer trial was terminated at 64 DAI and the fall trial was terminated at 72 DAI. For each tagged leaf, a digital caliper (Fisher Scientific, Hampton, NH) was used to measure length and width (mm) of each leaflet. The lesion diameter (mm) was recorded at 23 DAI from 12 mature lesions arbitrarily chosen from the three tagged leaves. From these data, incidence (proportion of inoculated leaflets with at least one lesion) was first calculated on the entire range of inoculated leaflets. Because some leaflets failed to develop symptoms, only leaflets infected with one or more lesions were used to calculate the following variables: incubation period (time to first symptomatic lesion), percent incubation period (number of lesions at 17 DAI/maximum number of lesions per leaflet counted prior to leaflet defoliation), lesions per leaflet (final number of lesions per leaflet), infection frequency (i.e., lesion density - commonly referred to as infection frequency in the literature (Cantonwine et al., 2008), and is defined as the final number of lesions counted per leaflet area (cm^2)), lesion diameter, time to leaflet defoliation, defoliation at 30 DAI (percent missing leaflets), final defoliation (percent number of missing leaflets at the last rating) and percent leaf area necrosis = [(final lesion number prior to defoliation * 3.14(lesion diameter/2)^2)/leaf area]. Because lesions failed to sporulate in greenhouse conditions, the latent period was not determined in these studies. Presumably this was due to the low humidity in the greenhouse (Supplementary Fig. 7.1).

At 30 DAI, two tagged leaves from each treatment were excised from the plant and placed upright in styrofoam hydroponic seed trays. Detached leaves were then incubated in a dew formation chamber (Percival Scientific, Inc., Perry, IA) at 24°C with 12-h diurnal wet/dry, light/dark regime for a total of 72 h. Afterwards, adaxial and abaxial surfaces of 12 mature lesions per treatment were observed under a dissecting microscope (40x), and percent sporulation was rated using a 1 to 5 scale where 1 = few stromata with little or no sporulation, 2 = stromata with slight sporulation, 3 = stromata over most of lesion, moderate sporulation, 4 = stromata on entire lesion, moderate to profuse sporulation, 5 = dense production of stromata with heavy sporulation (Chiteka et al., 1988b).

Statistical analysis. Incubation period, percent incubation period at 17 DAI, lesions per leaflet, infection frequency, lesion diameter, time to leaflet defoliation, defoliation at 30 DAI, final defoliation and percent leaf area necrosis were subjected to linear mixed model analysis of variance with PROC GLIMMIX (SAS 9.4 Institute, Cary, NC). The model was analyzed as a split plot design with inoculum and cultivar considered as fixed effects. Replication, trial, replication × trial and replication × inoculum were considered random effects. The SLICE option was used to explore two-way interactions when necessary. In all analyses the Kenward-Roger option was used to adjust the denominator degrees of freedom due to repeated measures, and differences in the least square means were tested by Tukey's multiple comparisons test. When data violated the assumptions of normality, transformations were used. Proportion of lesions that were LLS (ProLLS), incidence and percent defoliation were arcsin-square root transformed. The square root transformation was used for incubation period, percent incubation, infection frequency and time to defoliation. The natural log transformation was used for stAUDPC of defoliation. Back-transformed means of all transformed variables are presented in the results. Two sample t-tests were used to directly compare the components of resistance to ELS vs. LLS for single inoculations of *Ca* and *Cp* across cultivars and experiments. To explore the relationship between components of resistance for each disease, Spearman's rank correlation coefficients were calculated using PROC CORR in SAS.

Supplemental material. In both trials, additional inoculations were made on 90-day-old plants. The experimental design and inoculation procedures were exactly the same on the 90-day-old plants as for the 30-day old plants except that plants were grown 11 liter pots (26.4 cm depth and 24.1 cm height). Due to extreme thrips and spider mite damage in the first trial, and poor infection efficiency in the second trial, these data are presented in supplementary section in addition to the results presented in the main body for plants inoculated at 30 DAI.

RESULTS

Environmental conditions between the two greenhouse trials were more similar with respect to temperature than to relative humidity. The summer trial averaged approximately 2 °C warmer and 20% higher relative humidity compared with the fall trial (Supplementary Fig. 7.1). However, the minimum

(night) temperatures in both the summer (22 - 24°C) and the fall (22°C) were highly conducive for germination and infection of both pathogens (Alderman and Beute, 1986; Alderman and Nutter, 1994; Shew et al., 1988; Sommartya and Beute, 1986; Wu et al., 1999).

With the exception of time to defoliation, there were no interactions between cultivar and inoculum for all the variables measured (Table 7.1). In both trials, conidia used for inoculations had a high percent germination (>90%) for both leaf spot pathogens (data not shown). Incidence of infection was generally high for both *C. arachidicola* and *C. personatum*, but was significantly and numerically lowest on Georgia Green (Table 7.2). No lesions were observed on plants sprayed with the water + Tween 20 (data not shown). The incubation period and percent incubation of ELS and LLS did not differ between cultivars (Table 7.1 and 7.2). Cultivar had a significant effect on the lesion diameter of both diseases (Table 7.1). For both ELS and LLS, lesion diameter was significantly greater on Georgia Valencia and Florunner (Fig. 7.1 A). Cultivar did not affect the sporulation of ELS lesions but had a highly significant effect on the sporulation of LLS lesions (Table 7.1). Sporulation of *Cp* was significantly lower on Georgia Green and Georgia-06G compared with Georgia Valencia and Florunner (Fig. 7.1 B).

The number of lesions per leaflet and the number of lesions per leaflet area (infection frequency) were highly correlated at ($r > 0.95$) for both ELS and LLS (data not shown). Therefore, we only report the number of lesions per leaf area here. Cultivar had a highly significant effect on the infection frequency of ELS and LLS (Table 7.1). Infection frequencies of ELS and LLS were significantly higher on Georgia Valencia than Georgia Green and Georgia-06G (Fig. 7.2 A). Florunner tended to have numerically higher ELS and LLS than Georgia Green and Georgia-06G, but did not differ significantly (Fig. 7.2 A). The proportion of ELS to LLS lesions did not differ among cultivars (Table 7.3). The effect of cultivar and inoculum on the number of lesions per leaflet reported here on plants inoculated at 30 DAI was similar for plants that were inoculated at 90 DAI (Supplementary Fig. 7.3. and supplementary Table 7.1). There was a significant interaction between cultivar and inoculum for time to defoliation, but not for defoliation at 30 DAI or final defoliation (Table 7.1). For time to defoliation, there were no significant

differences between cultivars inoculated with *Ca* alone or *Ca* + *Cp*, but for leaflets inoculated with *Cp* alone, defoliation for Georgia Valencia was significantly delayed compared with Florunner and Georgia-06G (Fig. 7.3). Defoliation at 30 DAI was significantly higher on Georgia-06G than the other cultivars, and the amount of defoliation was significantly or numerically lowest on Georgia Valencia (Table 7.2). Similar trends were observed for final defoliation but only Georgia Valencia was statistically different from the other cultivars (Table 7.2).

There was a significant effect of inoculum on the incubation period of LLS, but not for ELS (Tables 7.1 and 7.3). Co-inoculations with *Ca* + *Cp* resulted in significantly smaller LLS lesions compared to *Cp* alone (Fig. 7.4). Inoculum did not affect sporulation of ELS or LLS lesions (Table 7.1, Fig. 7.4). Inoculum had a highly significant effect on the infection frequency and the percent necrotic leaf area of ELS and LLS (Table 7.1). For both diseases, co-inoculations with *Ca* + *Cp* resulted in significantly lower infection frequencies and percent necrosis (Fig. 7.4). Co-inoculations resulted in a 33% reduction in the number of ELS lesions and a 73% reduction in the number of LLS when compared with *Ca* alone and *Cp* alone, respectively (data not shown).

Across cultivars, the combined number of lesions per leaflet was not statistically different for *Ca* alone (10.7), *Cp* alone (9.1) or *Ca* + *Cp* (8.7) ($P = 0.0987$). Overall, leaflets inoculated with *Ca* alone or *Ca* + *Cp* abscised earlier than leaflets inoculated with *Cp* alone (Fig. 7.3 and Table 7.3). Time to defoliation was only reported in one trial due to a large number of leaflets inoculated with *Cp* alone that did not defoliate in the second trial. Across cultivars, defoliation was significantly highest on leaflets inoculated with *Ca* alone, and was significantly greater on plants co-inoculated with *Ca* + *Cp* than those inoculated with *Cp* alone (Table 7.2). The effect of inoculum reported here on plants inoculated at 30 DAI was similar for plants inoculated at 90 DAI (Supplementary Figs. 7.2 and 7.3 and Supplementary Table 7.1).

For direct comparisons of components of resistance for *Ca* alone vs. *Cp* alone across cultivars, there was no difference in the incidence, infection frequency or sporulation rating of ELS to LLS (Table 7.4). However, for all other variables, ELS was significantly different from LLS (Table 7.4). For ELS,

the incubation period was approximately 2 days shorter, and had 33 and 20 % more lesions per leaf area (cm) at 13 and 17 DAI, respectively, compared with LLS (Table 7.4). For LLS, time to defoliation approximately 14 days longer, and defoliation at 30 DAI was ~ 57% less compared with ELS (Table 7.4). The average diameter of ELS lesions was approximately 1 mm larger than that of LLS lesions (Table 7.4).

Correlations between components tended to differ for leaflets inoculated with *Ca* alone and *Cp* alone (Tables 7.5 and 7.6). Variables related to defoliation were often positively correlated with the infection frequency and sporulation on plants inoculated with *Ca* alone, but not on those inoculated with *Cp* alone (Tables 7.5 and 7.6). On plants inoculated with *Cp* alone, the incubation period was significantly and positively correlated with incidence, lesion size and the number of lesions per leaflet, but not on plants inoculated with *Ca* alone (Tables 7.5 and 7.6). For both pathogens, incidence was correlated with lesions per leaflet (Tables 7.5 and 7.6). Sporulation was correlated with five out of eight *Ca* components and only two out of eight *Cp* components (Tables 7.5 and 7.6). For both pathogens, infection frequency and percent necrotic leaf area were positively correlated with sporulation, and for *Cp* alone, these two components were also positively correlated with lesion size (Tables 7.5 and 7.6).

DISCUSSION

In these studies, we compared the components of resistance to ELS and LLS on a susceptible peanut cultivar Georgia Valencia and three of the most widely planted cultivars in the southeastern United States over the last 50 years: Florunner, Georgia Green and Georgia-06G. While many studies have previously evaluated components of resistance to one or both pathogens on one or more of these cultivars, this is the first study to directly compare all cultivars in the same study with both leaf spot pathogens. These studies demonstrated that Georgia Valencia is highly susceptible to both ELS and LLS and that Florunner, Georgia Green and Georgia-06G have similar levels of resistance to ELS. However, these data indicate that Florunner is more susceptible to LLS than Georgia Green or Georgia-06G. Co-inoculations of *Ca* + *Cp* demonstrated that each leaf spot disease was negatively affected by the presence of the other

regardless of cultivar. Furthermore, our results strongly suggest that the ELS pathogen had a more of negative effect on the development of the LLS pathogen than vice versa.

Unlike most previous studies, this study enabled us to directly compare the components of resistance to each leaf spot pathogen. However, because most of the related literature is based on the evaluation of a single disease, it is necessary to interpret our results accordingly. Overall, results for incidence showed that we were successful in our inoculation methods (Table 7.2). However, as there were some leaflets that failed to develop lesions, we chose to remove these from our further analyses in order to better compare the actual severity of each leaflet. The incubation period for *Ca* or *Cp* did not differ among cultivars, but was more delayed than expected (Shokes and Culbreath, 1997). Previous reports indicate that the incubation period of *Cp* is 9-10 days on Florunner (Aquino et al., 1995; Chiteka et al., 1988b), but in our studies Florunner averaged 15 days for *Cp* (Table 7.2). Some components of resistance, including incubation period, infection frequency and latent period, are known to vary under different environmental conditions (Shew et al., 1988). Germination of *Cp* is greatest at 20°C and 24°C for *Ca* (Alderman and Beute, 1986; Sommartya and Beute, 1986) and thus could . Percent incubation was greater for *Ca* than *Cp* (Table 7.5), and did not differ among cultivars (Table 7.2). The average temperatures in over the course of our studies were 26 - 28°C and were similar to greenhouse trials conducted by (Chiteka et al., 1988b); however, minimum temperatures of 22-24°C reported in our studies vs. a minimum of 19°C in the study by Chiteka et al. (1988b). We report a percent incubation of 91.5 and 59.3% for *Ca* and *Cp* on Georgia Green, which differed from a previous report of 46.2 and 43.5%, respectively (Cantonwine et al., 2008). Environmental conditions in the study by Cantonwine et al. (2008) had > 90 % relative humidity for the duration of the experiment, whereas the average relative humidity in our studies ranged from ~ 70-80% (Supplementary Fig. 7.1).

Our method of evaluating sporulation proved to be very effective, particularly in providing a more controlled and conducive environment (100% relative humidity for 72 h at 24 °C). Our results confirm that sporulation as assessed on a 1 to 5 scale is an important resistance component (Aquino et al.,

1995; Chiteka et al., 1988b) for separating cultivars (Fig. 7.1). Similar to our studies, previous reports have documented heavy sporulation of *Cp* on Florunner (4 to 5 on sporulation scale) (Aquino et al., 1995; Chiteka et al., 1988b). Our report of lower *Cp* sporulation on Georgia Green corroborates the study by Cantonwine et al. (2008) who found that Georgia Green had the lowest levels of *Cp* sporulation among the three cultivars tested. We found that the number of lesions per leaflet was highly correlated ($r > 0.95$) with infection frequency (number of lesions per leaf area). Our results indicate that infection frequency may be an effective component for separating cultivars, but only if the difference between host resistance levels is extreme (Fig. 7.2). Several studies have suggested that infection frequency/lesions per leaflet is not a good measure of resistance due to inconsistent results (Ricker et al., 1985; Subrahmanyam et al., 1982; Waliyar et al., 1995), but others have reported that is an effective component (Cantonwine et al., 2008). In our studies, infection frequency was numerically, but not statistically higher on Florunner compared with Georgia Green and Georgia-06G. However, in recent field studies we reported that Florunner consistently had the highest levels of LLS compared to the latter two cultivars (Chapter 6). Others have also reported extreme levels of susceptibility of Florunner to LLS (Anderson et al., 1993; Branch and Culbreath, 1995; Chiteka et al., 1988b; Culbreath et al., 1991). Our results suggest that Florunner is highly susceptible to both pathogens, but is likely more prone to severe LLS epidemics due to its larger lesions and greater sporulation of *Cp* (Fig. 7.1). Our results suggest that the other cultivars in these studies have similar levels of resistance to ELS and LLS in regards to infection frequencies. Although not specifically compared in the same study, Cantonwine et al. (2008) reported that the infection frequency of ELS and LLS was similar on Georgia Green. Therefore, the lower levels of LLS we have observed in field trials on Georgia Green and Georgia-06G, is likely primarily due to a rate-reducing response caused by limited sporulation of *Cp* (Fig. 7.1).

Measures of defoliation tended to be greatest on Georgia-06G (Table 7.2), but depended on the inoculum treatment. Time to defoliation was only reported in one trial due to a large number of leaflets inoculated with *Cp* alone that did not defoliate in the second trial. However, it was clear from both trials that all cultivars were more prone to pre-mature defoliation when inoculated with *Ca* alone or with *Ca* +

Cp (Fig. 7.3 and Table 7.3). Ricker et al. (1985) found that time to leaflet defoliation was generally correlated with shorter latent period and higher sporulation of *Ca*, but not for lesions per leaflet. In contrast, Nevill (1981) reported that cultivars susceptible to *Cp* had more lesions per leaflet, higher sporulation and leaflets were quicker to defoliate. Our studies suggest that time to leaflet defoliation is not a consistent measure of resistance to *Cp* in that Georgia Valencia had the highest lesions per leaflet, heavy sporulation, but the lowest values for all measures of defoliation (Table 7.2, Fig. 7.3). Furthermore, it is interesting to note that Georgia Green and Georgia-06G both had low infection frequencies and sporulation, but the highest defoliation values (Table 7.2, Fig. 7.3). This suggests that a delay in time to defoliation could actually be considered a measure of susceptibility rather than a component of resistance.

The trial was originally designed to test the differences in plant age (90-day-old plants vs 30 day old plants) as there is field evidence to suggest that many cultivars appear to become more susceptible to LLS with increasing age (Tillman, Culbreath, personal communication). The hypothesis is that increased energy allocated to pod production after the flowering stage reduces the plant's capacity to utilize defense mechanisms. In the field, we observed severe epidemics of LLS on Georgia Green and Georgia-06G after, but not before to 100 DAP. The results of our greenhouse trials on the 90-day-old plants were much more inconsistent, but overall, did not appear to have any differences in susceptibility compared with the 30-day-old plants (Supplementary Figs. 7.2 and 7.3 and Supplementary Table 7.1). However, several factors may explain this. First, we were no successful with 90-day-old plants in the greenhouse because they were much more subject to stress-related factors including, fertility, mites and insects. Secondly, pod development on the plants was limited (data not shown), which may have been due to insufficient container size, and therefore we were not able to test the hypothesis that more pod production increases susceptibility. Furthermore, our results could have been related to leaf age. Older leaves on 90-day-old plants were often necrotic, or had already abscised, so we inoculated the 3rd or 4th newest leaves on the main or lateral stems. Others have found that increasing plant age and increasing leaf age on certain peanut cultivars resulted in higher levels of resistance to *Puccinia arachidis* (Power, 2014; Wang and

Lin, 2000). Additional research is needed to understand how plant age and leaf age may affect leaf spot epidemics.

One of the most interesting aspects of this study was the co-inoculations with *Ca* and *Cp*. Our data indicates that there is a negative interaction between *Ca* and *Cp* in which development of both pathogens is reduced in the presence of the other, but the former clearly dominated the latter. It should be noted that in our studies, the inoculum density for *Ca* + *Cp* was twice as high as in the individual inoculations. Therefore, it is possible that intra-specific competition and inter-specific competition could have reduced the development of both leaf spots.

For ELS, co-inoculations resulted in a reduction in the infection frequency and percent necrotic area (Fig. 7.4). The same was observed for LLS, but also included a reduction in incidence, incubation period and lesion diameter (Fig. 7.4 and Table 7.3). There was a reduction of 43 and 74% in the infection frequency of the ELS and LLS pathogen, respectively, as compared to *Ca* alone or *Cp* alone. Monasterios de La Torre (1980) reported a similar reduction of 36% and 92% on Florunner when co-inoculations of *Ca* + *Cp* were compared to inoculations of each pathogen alone. The author attributed this reduction to three possibilities: a negative interaction between species, differences in spore concentrations, or different levels of susceptibility in Florunner (Monasterios de La Torre, 1980). Co-occurrence has also been documented to inhibit the development of ascochyta blight complex on pea. Le May et al. (2009) showed that simultaneous inoculations of *Mycosphaerella pinodes* and *Phoma medicaginis* var. *pinodella* resulted in reduced lesion size and fewer pycnidia for each disease compared to when each pathogen was inoculated alone. The authors proposed that this was due to direct competition for space, antibiosis or induced plant resistance (Le May et al., 2009). There is evidence that induced plant resistance may affect the disease dynamics of the coexisting *Leptosphaeria* species causing phoma stem canker of oilseed rape (Fitt et al., 2006). Several studies indicated that the development of *L. maculan*s was suppressed when *L. biglobosa* was inoculated prior to *L. maculans* (Liu et al., 2006; Mahuku et al., 1996). Therefore, it is possible that prior infections of *Ca* or *Cp* could suppress the development of LLS or ELS, respectively.

However, it was clear from our studies that when both pathogens were co-inoculated on the same leaflet at the same point in time, the amount of LLS was much more reduced than ELS. Taken together, these data strongly suggest that *Ca* has an indirect or direct antagonistic effect on *Cp* when co-inoculated with both pathogens, particularly due to having occurred across all cultivars. However, future research is needed to address differences in inoculum densities as well as inoculations at differing time points in order to determine whether prior infection by one pathogen could induce plant resistance to the other pathogen.

A negative association may partially explain the differences between onset of ELS and LLS. In our recent field studies, we found that onset of LLS was earliest on the cultivars Georgia Valencia and Florunner in fields with a history of both leaf spot pathogens, but that the onset of ELS was 20 to 40 days earlier than LLS across cultivars (Chapter 6). In contrast, the onset of LLS was earlier or similar to that of ELS when planted in late July and August in fields with a history of LLS (Chapter 6). This suggests that early infections by *Ca* could inhibit the infection or development of *Cp*. Time to defoliation could be another indirect mechanism of competition, and thus a reason that inhibits early detection of LLS. In these studies, we found that leaflets were first to abscise if inoculated with *Ca* alone or with *Ca* + *Cp* (Fig. 7.3 and Table 7.3). Early defoliation due to *Ca* could thus inhibit the initial development of *Cp* by eliminating possible infection courts. While cultivar, inoculum and planting date have previously been shown to affect the differences in the prevalence and onset of these two diseases, they do not fully explain all observed differences in epidemic development (Chapters 4, 5 and 6). We hypothesize that two additional factors include a negative association between the two leaf spot pathogens on the same leaflet, and an indirect effect associated with early defoliation caused by *Ca* infection.

In conclusion, these studies corroborate previous reports that peanut cultivars are often capable of being infected with both leaf spot pathogens (Abdou et al., 1974), but that some cultivars, such as NC 3303, have differing levels of partial resistance to one or both leaf spot pathogens (Ricker et al., 1985; Walls et al., 1985). Smith and Littrell (1980) hypothesized that widespread introduction of Florunner could have helped to shift the predominance from ELS to LLS in the late 1970s. We found that

sporulation of *Cp* was greatest on Florunner, but was limited on Georgia Green. It is therefore possible that widespread adoption of these cultivars in the southeastern United States could have played a role in the variable prevalence of these two diseases. Because components of resistance to *Cp* did not differ between Georgia-06G and Georgia Green, it is likely that the recent resurgence of LLS is due to other factors. Our data also suggests that there is a negative association between *Ca* and *Cp* when co-inoculated on the same leaflet. While the logical inference could partially explain differences in the prevalence or onset of these diseases in the field, we suggest that more research is needed on the competition between the two pathogens and the role of induced plant resistance in order to better understand the differences in the epidemiological behavior of ELS and LLS.

ACKNOWLEDGMENTS

We thank Matt Seader, manager of the University of Georgia Plant Pathology greenhouse operations, for his valuable assistance in managing of the plants used in these trials, and Xuelin Luo for statistical assistance.

LITERATURE CITED

Abdou, Y. A.-M., Gregory, W. C., and Cooper, W. E. 1974. Sources and nature of resistance to *Cercospora arachidicola* Hori and *Cercosporidium personatum* (Beck & Curtis) Deighton in *Arachis* species. Peanut Sci. 1:6-11.

Alderman, S., and Beute, M. 1986. Influence of temperature and moisture on germination and germ tube elongation of *Cercospora arachidicola*. Phytopathology 76:715-719.

Alderman, S., and Nutter, F. 1994. Effect of temperature and relative humidity on development of *Cercosporidium personatum* on peanut in Georgia. Plant Dis. 78:690-694.

Anderson, W., Holbrook, C., and Brenneman, T. 1993. Resistance to *Cercosporidium personatum* within peanut germplasm. Peanut Sci. 20:53-57.

Aquino, V., Shokes, F., Gorbet, D., and Nutter, F. 1995. Late leaf spot progression on peanut as affected by components of partial resistance. Plant Dis. 79:74-78.

Backman, P., and Crawford, M. 1984. Relationship between yield loss and severity of early and late leafspot diseases of peanut. Phytopathology 74:1101-1103.

Branch, W. 1996. Registration of ‘Georgia Green’ peanut. Crop Sci. 36:806-806.

Branch, W. 2007. Registration of ‘Georgia-06G’ peanut. J Plant Regist 1:120-120.

Branch, W., and Culbreath, A. 1995. Combination of early maturity and leaf spot tolerance within an advanced Georgia peanut breeding line. Peanut Sci. 22:106-108.

Cantonwine, E., Culbreath, A., Holbrook, C., and Gorbet, D. 2008. Disease progress of early leaf spot and components of resistance to *Cercospora arachidicola* and *Cercosporidium personatum* in runner-type peanut cultivars. Peanut Sci. 35:1-10.

Chiteka, Z., Gorbet, D., Knauft, D., Shokes, F., and Kucharek, T. 1988a. Components of resistance to late leafspot in peanut. ii. Correlations among components and their significance in breeding for resistance. Peanut Sci. 15:76-81.

Chiteka, Z., Gorbet, D., Shokes, F., Kucharek, T., and Knauft, D. 1988b. Components of resistance to late leafspot in peanut. I. Levels and variability-implications for selection. Peanut Sci. 15:25-30.

Culbreath, A., Brenneman, T., and Shokes, F. 1991. Quantitative comparison of stem lesions caused by *Cercosporidium personatum* in Florunner and Southern Runner peanut cultivars. Peanut Sci. 18:116-121.

Fitt, B. D., Huang, Y.-J., van den Bosch, F., and West, J. S. 2006. Coexistence of related pathogen species on arable crops in space and time. Annu. Rev. Phytopathol. 44:163-182.

Fry, W. E. 1982. Principles of Plant Disease Management. Academic Press, Orlando.

Gorbet, D., and Shokes, F. 2006. Registration of ‘C99R’ peanut. Crop Sci. 46:2713-2714.

Gorbet, D., Norden, A., Shokes, F., Knauft, D., Porter, K., Worrall, W., Gardenhire, J., Gilmore, E., McDaniel, M., and Tuleen, N. 1987. Registration of 'Southern Runner' peanut. Crop Sci. 27:817-817.

Hemingway, J. 1955. The prevalence of two species of Cercospora on groundnuts. Trans. Brit. Mycol. Soc. 38:243-246.

Jenkins, W. A. 1938. Two fungi causing leaf spot of peanut. J. Agr. Res. 56:317-332.

Le May, C., Potage, G., Andrivon, D., Tivoli, B., and Outreman, Y. 2009. Plant disease complex: antagonism and synergism between pathogens of the Ascochyta blight complex on pea. J. Phytopathol. 157:715-721.

Liu, S., Liu, Z., Fitt, B. D., Evans, N., Foster, S., Huang, Y., Latunde-Dada, A., and Lucas, J. 2006. Resistance to *Leptosphaeria maculans* (phoma stem canker) in *Brassica napus* (oilseed rape) induced by *L. biglobosa* and chemical defence activators in field and controlled environments. Plant Pathol. 55:401-412.

Mahuku, G., Hall, R., and Goodwin, P. 1996. Co-infection and induction of systemic acquired resistance by weakly and highly virulent isolates of *Leptosphaeria maculans* in oilseed rape. Physiol. Mol. Plant Pathol. 49:61-72.

Monasterios de La Torre, T. 1980. Genetic resistance to Cercospora leafspot diseases in peanut (*Arachis hypogaea* L.). Ph.D. Dissertation. University of Florida, Gainesville.

Nevill, D. 1981. Components of resistance to *Cercospora arachidicola* and *Cercosporidium personatum* in groundnuts. Ann. Appl. Biol. 99:77-86.

Power, I. 2014. Characterizing peanut rust resistance: determining its mechanisms, and the genetics of the peanut host and *Puccinia arachidis*. Ph.D. Dissertation. University of Georgia, Athens.

Ricker, M., Beute, M., and Campbell, C. 1985. Components of resistance in peanut to *Cercospora arachidicola*. Plant Dis. 69:1059-1064.

Shew, B., Beute, M., and Wynne, J. 1988. Effects of temperature and relative humidity on expression of resistance to *Cercosporidium personatum* in peanut. Phytopathology 78:493-498.

Shokes, F., and Culbreath, A. K. 1997. Early and late leaf spots. Pages 17-20 in: Compendium of Peanut Diseases, 2nd Ed. N. Kokalis-Burelle, D. Porter, R. Rodriguez-Kabana, D. Smith and P. Subrahmanyam, eds. American Phytopathological Society Press, St. Paul, MN.

Smith, D. H., and Littrell, R. H. 1980. Management of peanut foliar diseases with fungicides. Plant Dis. 64:356-361.

Sommartya, T., and Beute, M. 1986. Temperature effects on germination and comparative morphology of conidia for Thai and USA isolates of *Cercosporidium personatum*. Peanut Sci. 13:67-70.

Subrahmanyam, P., McDonald, D., Gibbons, R., Nigam, S., and Nevill, D. 1982. Resistance to rust and late leafspot diseases in Some genotypes of *Arachis hypogaea* Peanut Sci. 9:6-10.

Waliyar, F., Shew, B., Sidahmed, R., and Beute, M. 1995. Effects of host resistance on germination of *Cercospora arachidicola* on peanut leaf durfaces. Peanut Sci. 22:154-157.

Walls, S. B., Wynne, J., and Beute, M. 1985. Resistance to late leafspot peanut of progenies selected for resistance to early leafspot. Peanut Sci. 12:17-22.

Wang, Z., and Lin, K. 2000. Modeling leaf age-related susceptibility and rust eruption dynamics in peanut. Peanut Sci. 27:7-10.

Wu, L., Damicone, J., Duthie, J., and Melouk, H. 1999. Effects of temperature and wetness duration on infection of peanut cultivars by *Cercospora arachidicola*. Phytopathology 89:653-659.

Table 7.1. *P*-values from the linear mixed model analysis of variance for components of resistance to early and late leaf spot on four peanut cultivars in greenhouse trials conducted in the summer and fall of 2016.

Source of variation[q]	Incidence[r]		Incubation period[s]		Percent incubation[t] 17 DAI		Infection frequency[u] 17 DAI		Percent defoliation[v]	
	ELS	LLS	ELS	LLS	ELS	LLS	ELS	LLS	30 DAI	Final
Cultivar (V)	0.0075	0.1689	0.0639	0.6071	0.4154	0.6816	0.0092	0.0437	<0.0001	0.0068
Inoculum (I)	0.2554	<0.0001	0.0874	0.0004	0.0770	0.0883	<0.0001	<0.0001	<0.0001	<0.0001
V*I	0.6641	0.5680	0.5415	0.5942	0.3612	0.5545	0.9897	0.8328	0.8662	0.1565

Source of variation	Infection frequency[w] Final		Percent leaf necrosis[x]		Lesion diameter (mm)		Sporulation[y]		
	ELS	LLS	ELS	LLS	ELS	LLS	ELS	LLS	Time to defoliation[z]
Cultivar (V)	<0.0001	0.0002	<0.0001	0.0007	<0.0001	<0.0001	0.3145	<0.0001	0.0920
Inoculum (I)	<0.0001	<0.0001	0.0026	<0.0001	0.9456	<0.0001	0.0312	0.2678	<0.0001
V*I	0.9340	0.8303	0.8403	0.5291	0.3914	0.6399	0.4766	0.0850	0.0009

[q] Cultivars included Georgia Valencia, Florunner, Georgia Green and Georgia-06G. Inoculum treatments consisted of *Cercospora arachidicola* (*Ca*) alone, *Cercosporidium personatum* (*Cp*) alone and a 100/100 mixture of *Ca* + *Cp* inoculated on 30-day-old peanut plants.
[r] Incidence = number of leaflets with one or more lesions out of the 12 inoculated leaflets per replication.
[s] Incubation period = time to appearance of first lesion.
[t] Percent incubation at 17 DAI = number of lesions at 17 days after inoculation (DAI) divided by the number of lesions prior to defoliation.
[u] Number of lesions per leaf area (cm^2) at 17 DAI.
[v] Percent defoliation at 30 DAI and at the last rating date (64 and 72 DAI for summer and fall, respectively).
[w] Final number of lesions per leaf area (cm^2) prior to defoliation.
[x] Percent leaf area necrosis = [(final lesion number prior to defoliation * 3.14(lesion diameter/2)^2)/leaf area].
[y] Adaxial (ELS) or abaxial (LLS) side of each lesion scored as 1 = no sporulation and 5 = profuse sporulation over the entire lesion.
[z] Days to reach leaflet defoliation.

Table 7.2. Development of early and late leaf spot on four peanut cultivars pooled over inoculation treatments in greenhouse trials conducted in the summer and fall of 2016.

	Incidence[v]		Incubation period[w]		Percent incubation[x]		Defoliation[y]	
Cultivar	ELS	LLS	ELS	LLS	ELS	LLS	30 DAI	Final
Georgia Valencia	99.2 a[z]	93.0 a	12 a	15 a	88.7 a	66.5 a	22.2 c	65.1 b
Florunner	96.5 a	85.6 a	14 a	15 a	83.3 a	71.0 a	38.6 bc	82.2 a
Georgia Green	74.3 b	78.7 a	14 a	16 a	91.5 a	59.3 a	50.4 ab	85.4 a
Georgia-06G	96.3 a	81.7 a	12 a	16 a	89.2 a	65.0 a	73.3 a	90.9 a

[v] Incidence = number of leaflets with one or more lesions out of the 12 inoculated leaflets per rep.
[w] Incubation period = time to appearance of first lesion.
[x] Percent incubation at 17 DAI = number of lesions at 17 days after inoculation (DAI) divided by the number of lesions prior to defoliation.
[y] Percent defoliation at 30 DAI and at the last rating date (64 and 72 DAI for summer and fall, respectively).
[z] Means within columns with the same letters are not significantly different based upon Tukey's honestly significant difference test $\alpha = 0.05$).

Table 7.3. Development of early and late leaf spot in relation to separate or co-inoculations of *Cercospora arachidicola* and *Cercosporidium personatum* in greenhouse trials conducted in the summer and fall of 2016.

	Incidence[v]		Incubation period[w]		Percent incubation[x]		Defoliation[y]	
Inoculum	ELS	LLS	ELS	LLS	ELS	LLS	30 DAI	Final
C.a.	95.4 a[z]	-	13 a	-	91.0 a	-	72.9 a	93.9 a
C.a. + *C.p.*	92.2 a	72.2 b	13 a	17 b	85.3 a	59.9 a	15.7 c	64.9 c
C.p.	-	94.6 a	-	14 a	-	71.1 a	51.9 b	81.8 b

[v] Incidence = number of leaflets with one or more lesions out of the 12 inoculated leaflets per rep.
[w] Incubation period = time to appearance of first lesion.
[x] Percent incubation at 17 DAI = number of lesions at 17 days after inoculation (DAI) divided by the number of lesions prior to defoliation.
[y] Percent defoliation at 30 DAI and at the last rating date (64 DAI and 72 DAI for summer and fall, respectively).
[z] Means were pooled across four cultivars inoculated at 30 days after planting. Means within columns with the same letters are not significantly different based upon Tukey's honestly significant difference test ($\alpha = 0.05$).

Table 7.4. Two sample t-tests showing the differences between the components of resistance to ELS and LLS when analyzed across all peanut cultivars and trials.

Variable	N	Diff (ELS - LLS)[v]	t-value	d.f.	Prob > t
Incidence	32	1.97 [w]	0.64	62	0.5262
Incubation period	32	-1.63 [x]	-3.1	62	0.0030
Percent incubation - 13 DAI	32	0.33 [x]	9.35	62	<0.0001
Percent incubation - 17 DAI	32	0.20 [x]	7.11	62	<0.0001
Infection frequency - final	32	0.074 [x]	1.11	61	0.2718
Percent necrotic leaf area	31	1.48 [w]	4.24	61	<0.0001
Time to defoliation	16	-13.7 [x]	-5.52	30	<0.0001
Defoliation at 30 DAI	32	57.2 [w]	5.04	62	<0.0001
Final defoliation	28	14.1 [y]	3.45	62	0.0010
Lesion diameter (mm)	32	1.08 [z]	9.39	62	<0.0001
Sporulation score	31	0.19 [z]	1.26	59	0.2111

[v] Mean difference between early and late leaf spot.
[w] Based on back-transformations of arcsin-square root transformed data.
[x] Based on back-transformations of square root transformed data.
[y] Based on back-transformations of the log transformed data.
[z] Based on untransformed data.

Table 7.5. Spearman's correlation coefficients and associated *P*-values of components of resistance to early leaf spot across the four peanut cultivars inoculated on 30-day-old plants in two separate greenhouse experiments conducted in the summer and fall of 2016.

Variable[p]	IncPeriod[r]	PerInc17[s]	Pdefol30[t]	Pdefol[u]	T Defol[v]	InfFreq[w]	% Necrosis[x]	Lesion (mm)[y]	Sporulation[z]
Incidence[q]	-0.3247	-0.0338	0.5621	0.6569	-0.2869	0.6010	0.6723	0.2787	0.6980
	0.0698	0.8539	0.0008	<.0001	0.2812	0.0003	<0.0001	0.1224	<0.0001
IncPeriod[r]		0.1811	0.0526	-0.0393	0.33491	-0.2266	-0.2768	-0.2279	-0.1954
		0.3212	0.7747	0.8305	0.2048	0.2122	0.1250	0.2096	0.3007
PerInc17[s]			0.1509	-0.0600	-0.8352	0.2309	0.2514	0.2307	0.2029
			0.4097	0.7441	<0.0001	0.2035	0.165	0.2038	0.2822
Pdefol30[t]				0.8493	-0.7360	0.6480	0.4364	-0.2492	0.5485
				<0.0001	0.0012	<.0001	0.0125	0.1689	0.0017
Pdefol[u]						0.6683	0.5642	-0.0949	0.4500
						<.0001	0.0008	0.6053	0.0126
T Defol[v]						-0.4676	-0.2764	-0.0676	-0.5040
						0.0678	0.2999	0.8034	0.0554
InfFreq[w]							0.8772	0.2540	0.5648
							<0.0001	0.1606	0.0011
% Necrosis[x]								0.6120	0.4755
								0.0002	0.0079
Lesion (mm)[y]									0.1911
									0.3117

[p] Number of observations = 16 for TimeToDefol. For all other variables, N = 32.
[q] Incidence = number of leaflets with one or more lesions out of the 12 inoculated leaflets per rep.
[r] Incubation period = time to appearance of first lesion.
[s] Percent incubation at 17 DAI = number of lesions at 17 days after inoculation (DAI) divided by the number of lesions prior to defoliation.

[t] Pdefol30 = percent defoliation at 30 days after inoculation (DAI).
[u] Percent defoliation at 30 DAI and at the last rating date (64 DAI and 72 DAI for summer and fall, respectively).
[v] TimeToDefol = Days to reach leaflet defoliation.
[w] InfFreq = Final number of lesions per leaf area (cm^2) prior to defoliation.
[x] Percent leaf area necrosis = [(final lesion number prior to defoliation * 3.14(lesion diameter/2)2)/leaf area].
[y] Lesion (mm) = lesion diameter.
[z] Sporulation = adaxial rating where 1 = no sporulation and 5 = profuse sporulation over the entire lesion.

Table 7.6. Spearman's correlation coefficients and associated *P*-values of components of resistance to late leaf spot across the four peanut cultivars inoculated on 30-day-old plants in two separate greenhouse experiments conducted in the summer and fall of 2016.

Variable[p]	IncPeriod[r]	PerInc17[s]	Pdefol30[t]	Pdefol[u]	T Defol[v]	InfFreq[w]	% Necrosis[x]	Lesion (mm)[y]	Sporulation[z]
Incidence[q]	-0.4738	-0.3275	0.3778	0.5865	-0.1400	0.4033	0.4922	0.4761	0.3563
	0.0062	0.0673	0.0330	0.0004	0.6050	0.0245	0.0049	0.0059	0.0491
IncPeriod[r]		0.1620	-0.3538	-0.4692	0.5177	-0.8496	-0.7229	-0.2766	-0.1904
		0.3756	0.0470	0.0068	0.0400	<.0001	<.0001	0.1254	0.3050
PerInc17[s]			0.3662	-0.0050	-0.7912	-0.0573	-0.1496	-0.1256	-0.2337
			0.0393	0.9782	0.0003	0.7596	0.4218	0.4933	0.2058
Pdefol30[t]				0.7302	-0.9184	0.2774	0.1750	0.0908	-0.2278
				<.0001	<.0001	0.1308	0.3465	0.6211	0.2179
Pdefol[u]					-0.7283	0.4254	0.3542	0.3079	0.0128
					0.0014	0.0170	0.0506	0.0864	0.9456
T Defol[v]						-0.2588	-0.0118	0.1353	0.1929
						0.3331	0.9655	0.6174	0.4910
InfFreq[w]							0.8669	0.4335	0.3642
							<.0001	0.0148	0.0478
% Necrosis[x]								0.7264	0.5836
								<.0001	0.0007
Lesion (mm)[y]									0.6650
									<.0001

[p] Number of observations = 16 for TimeToDefol. For all other variables, N = 32.
[q] Incidence = number of leaflets with one or more lesions out of the 12 inoculated leaflets per rep.
[r] Incubation period = time to appearance of first lesion.
[s] Percent incubation at 17 DAI = number of lesions at 17 days after inoculation (DAI) divided by the number of lesions prior to defoliation.

[t] Pdefol30 = percent defoliation at 30 days after inoculation (DAI).
[u] Percent defoliation at 30 DAI and at the last rating date (64 DAI and 72 DAI for summer and fall, respectively).
[v] TimeToDefol = Days to reach leaflet defoliation.
[w] InfFreq = Final number of lesions per leaf area (cm^2) prior to defoliation.
[x] Percent leaf area necrosis = [(final lesion number prior to defoliation * 3.14(lesion diameter/2)2)/leaf area].
[y] Lesion (mm) = lesion diameter.
[z] Sporulation = abaxial rating where 1 = no sporulation and 5 = profuse sporulation over the entire lesi

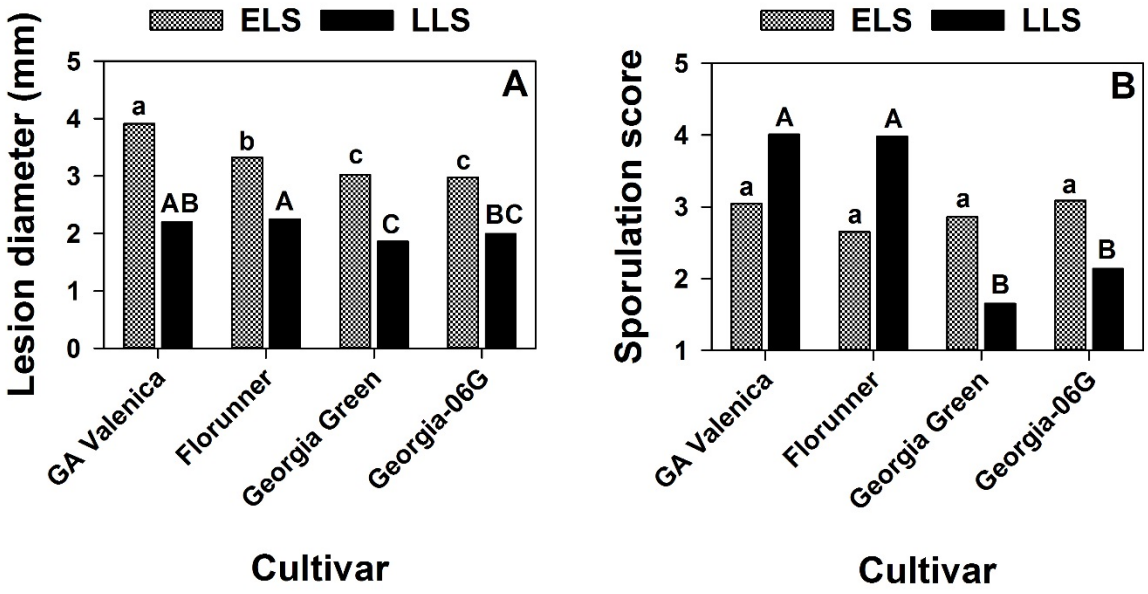


Fig. 7.1. Effect of peanut cultivar on (**A**) lesion diameter and (**B**) sporulation of early (ELS) and late (LLS) leaf spot lesions. Sporulation was rated using a 1 to 5 scale where 1 = few stromata with little or no sporulation, 2 = stromata with slight sporulation, 3 = stromata over most of lesion, moderate sporulation, 4 = stromata on entire lesion, moderate to profuse sporulation, 5 = dense production of stromata with heavy sporulation. Results are pooled across inoculum treatments. Bars with the same lower- or uppercase letters signify that cultivars are not significantly different based upon Tukey's honestly significant difference test ($\alpha = 0.05$).

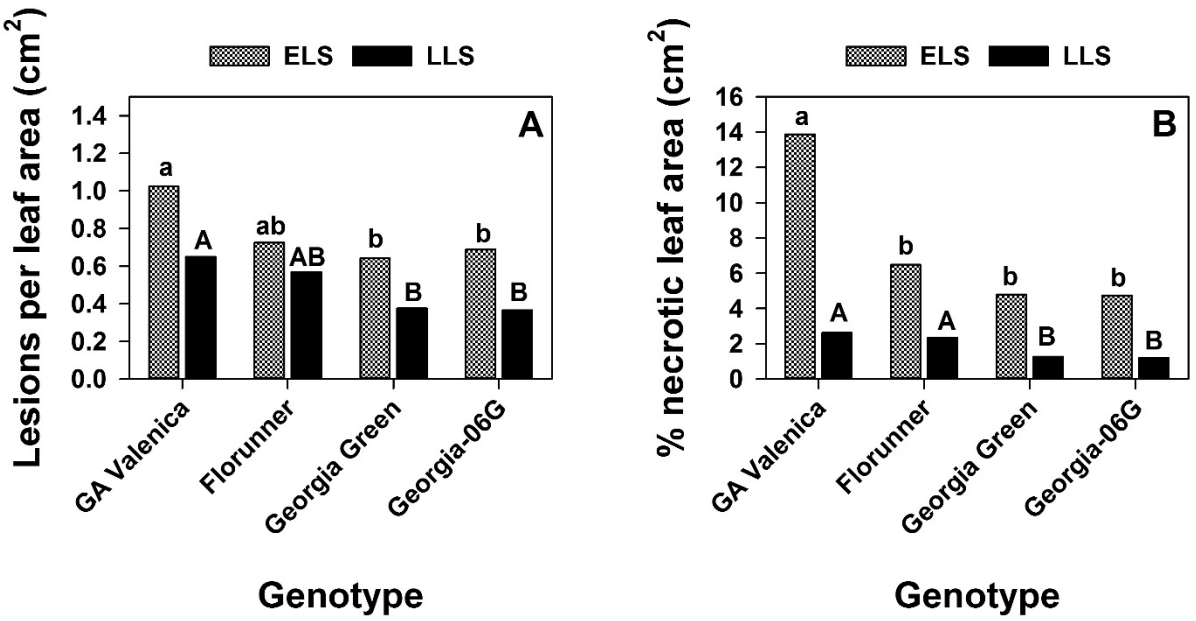


Fig. 7.2. Effect of peanut cultivar on the final number of lesions per leaflet (infection frequency) (**A**) and percent necrotic leaf area (**B**) of early (ELS) and late (LLS) leaf spot. Percent necrotic leaf area was estimates as [(final lesion number prior to defoliation * 3.14(lesion diameter/2)^2)/leaf area]. Results are pooled across inoculum treatments. Bars with the same lower- or uppercase letters signify that cultivars are not significantly different based upon Tukey's honestly significant difference test ($\alpha = 0.05$).

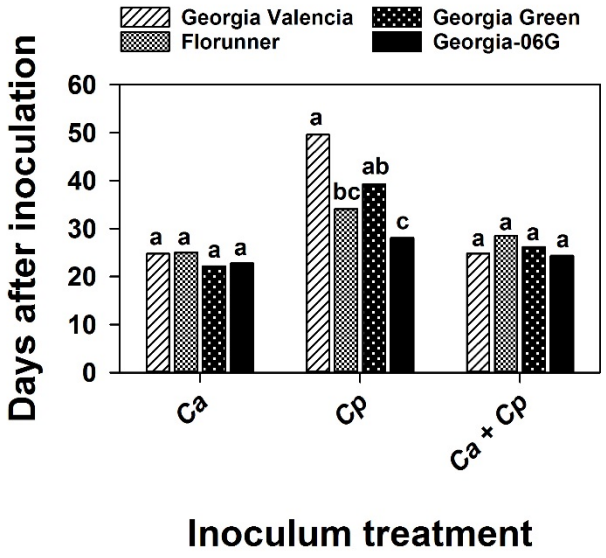


Fig. 7.3. Effect of inoculum treatment and peanut cultivar on the time to leaflet defoliation (measured as days after inoculation). The inoculum treatments consisted of *Cercospora arachidicola* (*Ca*) alone, *Cercosporidium personatum* (*Cp*) alone and a 100/100 mixture of *Ca + Cp*. Due to limited defoliation in the second trial, data reported here is only for the first trial. Grouped bars for each inoculum treatment with the same letters are not significantly different based upon Tukey's honestly significant difference test ($\alpha = 0.05$).

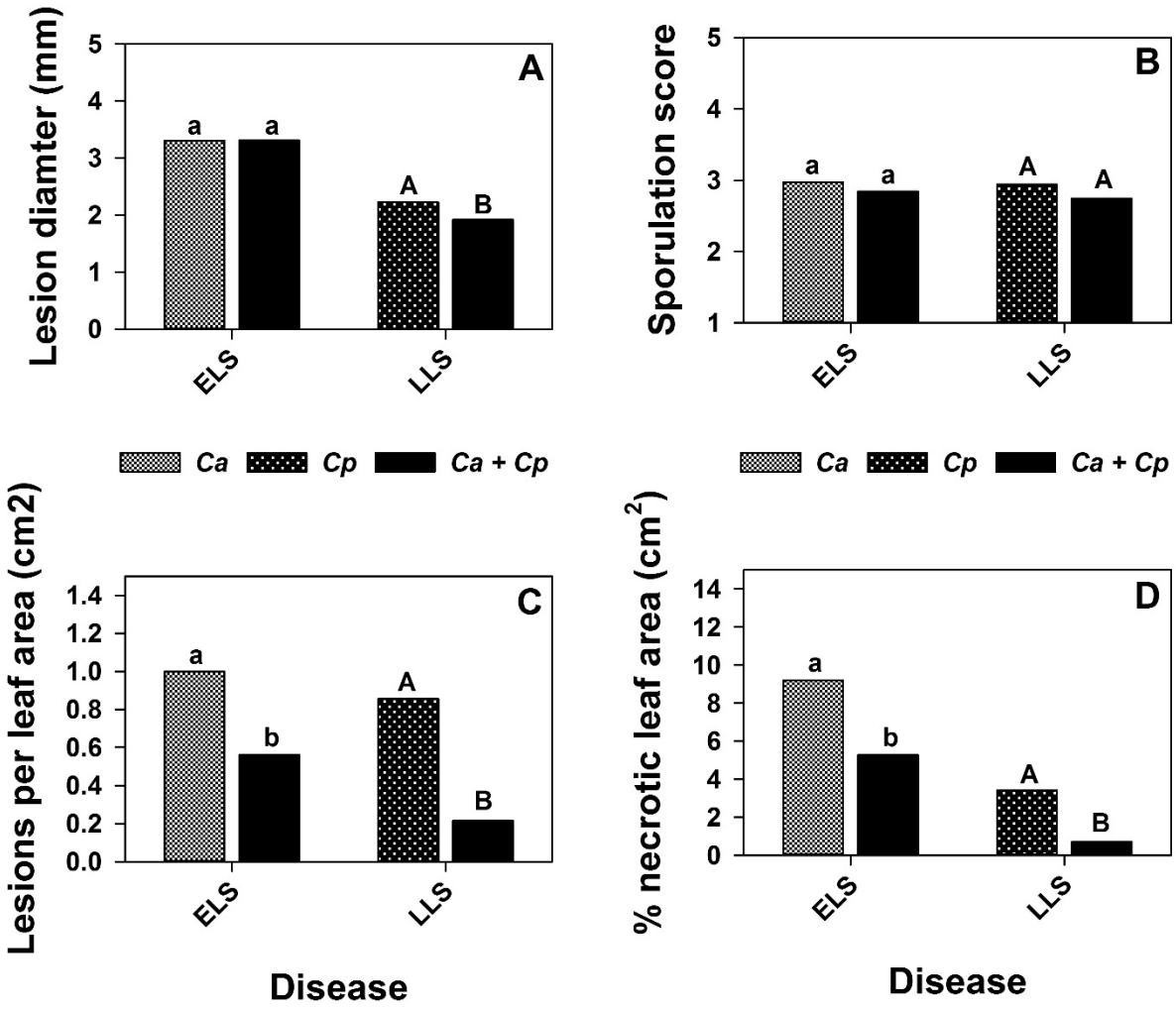
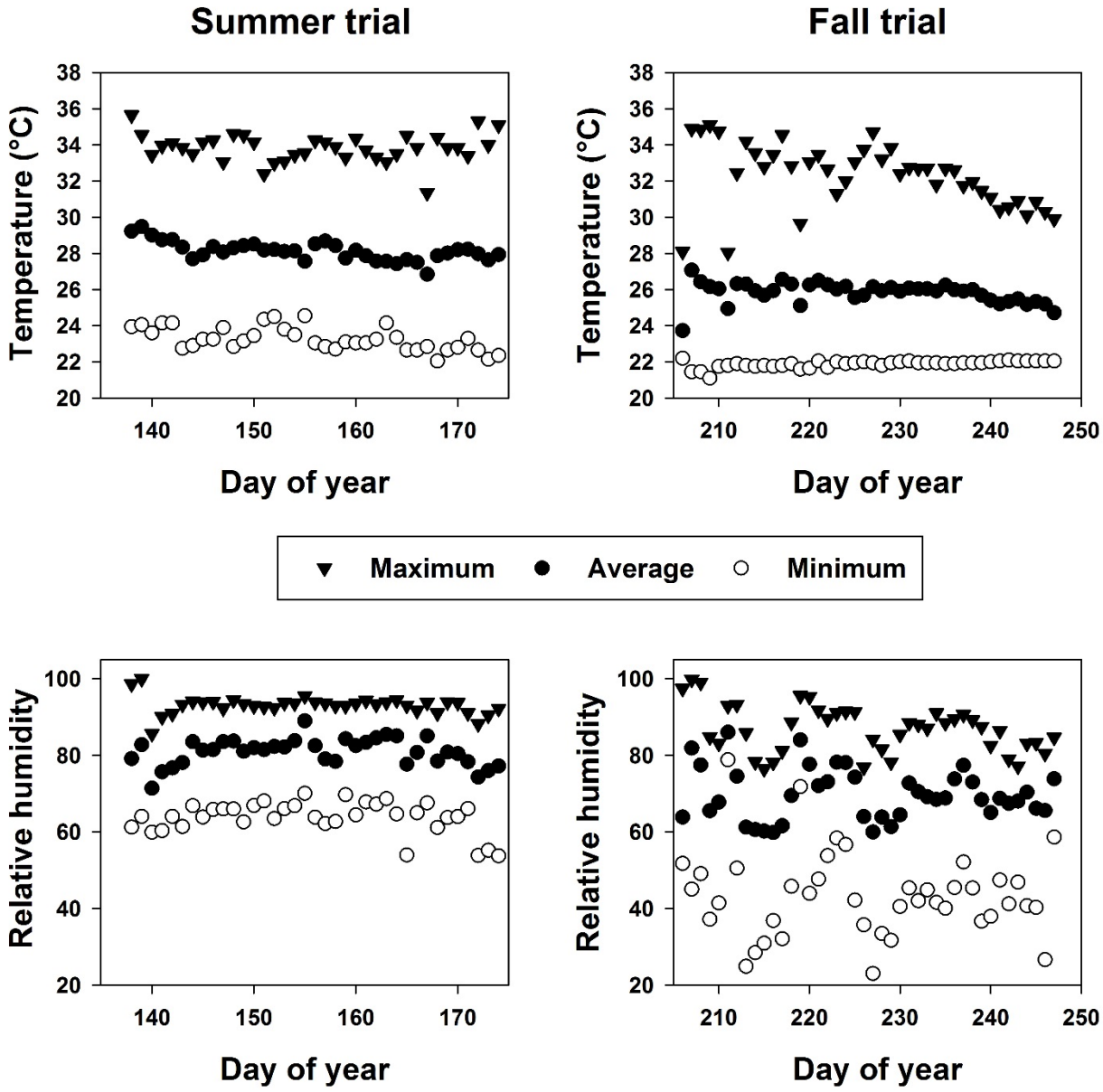


Fig. 7.4. Effect of inoculum treatment on the lesion diameter (**A**), sporulation (**B**), the final number of lesions per leaflet (infection frequency) (**C**), and percent necrotic leaf area (**D**) of early (ELS) and late (LLS) leaf spot. The inoculum treatments consisted of *Cercospora arachidicola* (*Ca*) alone, *Cercosporidium personatum* (*Cp*) alone and a 100/100 mixture of *Ca* + *Cp.* Grouped bars for each disease with the same letters are not significantly different based upon Tukey's honestly significant difference test ($\alpha = 0.05$).

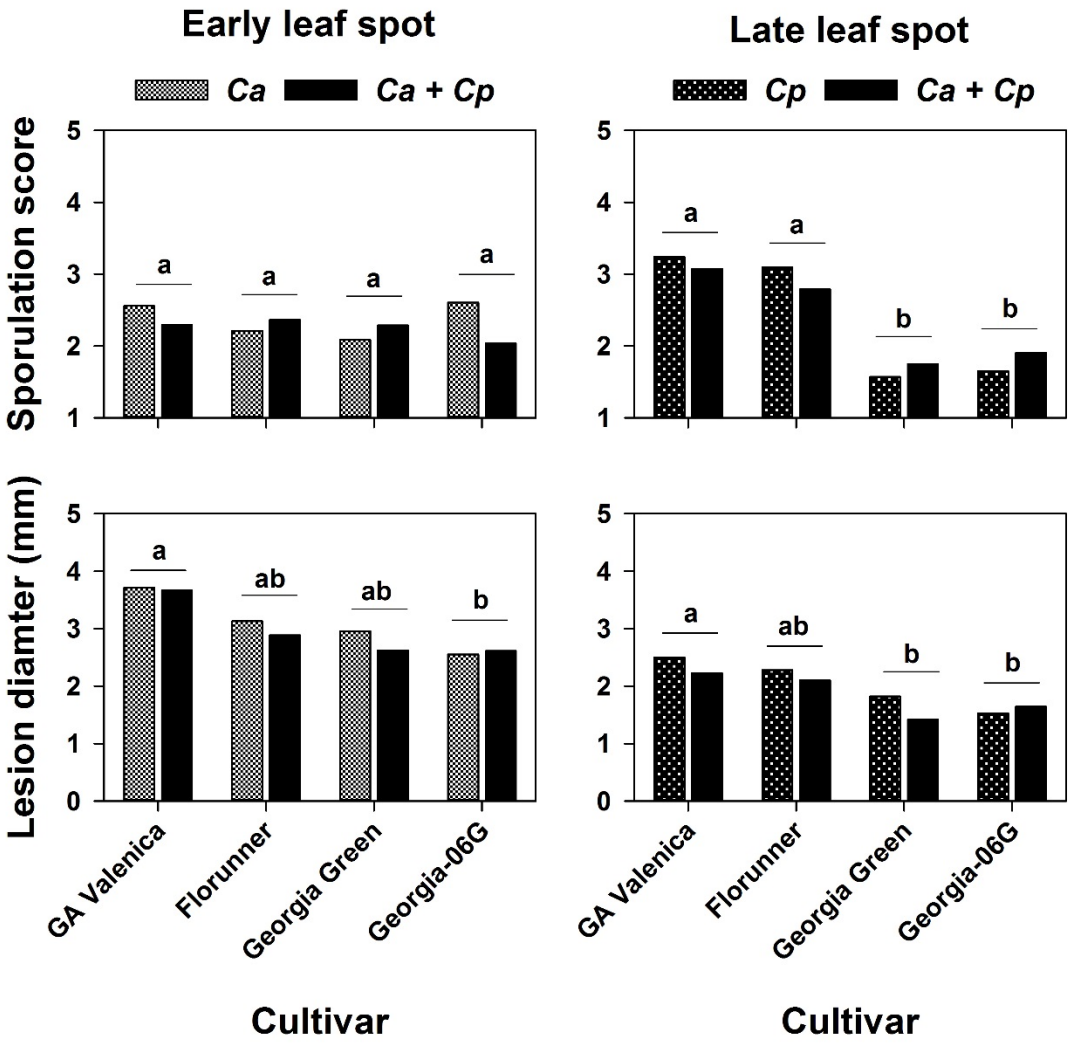
Supplementary Table 7.1. Development of early and late leaf spot on four cultivars in response to separate or co-inoculations of *C. arachidicola* and *C. personatum* made at 90 days after planting in a greenhouse trial conducted in the summer of 2016.

Cultivar, inoculum, ANOVA *P* values	Total lesions per leaflet	Predominance ProLLS	Time to defoliation	Defoliation at 30 DAI	Final defoliation	stAUDPC defoliation
Cultivar						
Georgia Valencia	15.6 a	0.27 a	40.9 a	19.4 a	88.4 a	35.7 a
Florunner	5.4 b	0.32 a	39.5 a	9.9 a	49.2 ab	27.0 a
Georgia Green	3.2 b	0.23 a	39.2 a	8.9 a	44.9 b	17.1 a
Georgia-06G	3.8 b	0.28 a	40.0 a	9.6 a	61.6 ab	28.1 a
Inoculum						
C.a.	7.0 a	0.00 c	35.9 b	35.9 a	85.3 a	45.8 a
C.p.	6.9 ab	1.00 a	42.6 a	3.2 b	43.0 b	17.8 b
C.a. + *C.p.*	4.9 b	0.32 b	41.5 ab	4.9 b	55.0 ab	21.8 b
ANOVA						
Cultivar	0.0003	0.2778	0.9406	0.7025	0.0362	0.1204
Inoculum	0.0335	<0.0001	0.0234	0.0030	0.0153	0.0047
Cultivar x Inoculum	0.0806	0.2174	0.5415	0.7678	0.7278	0.3024

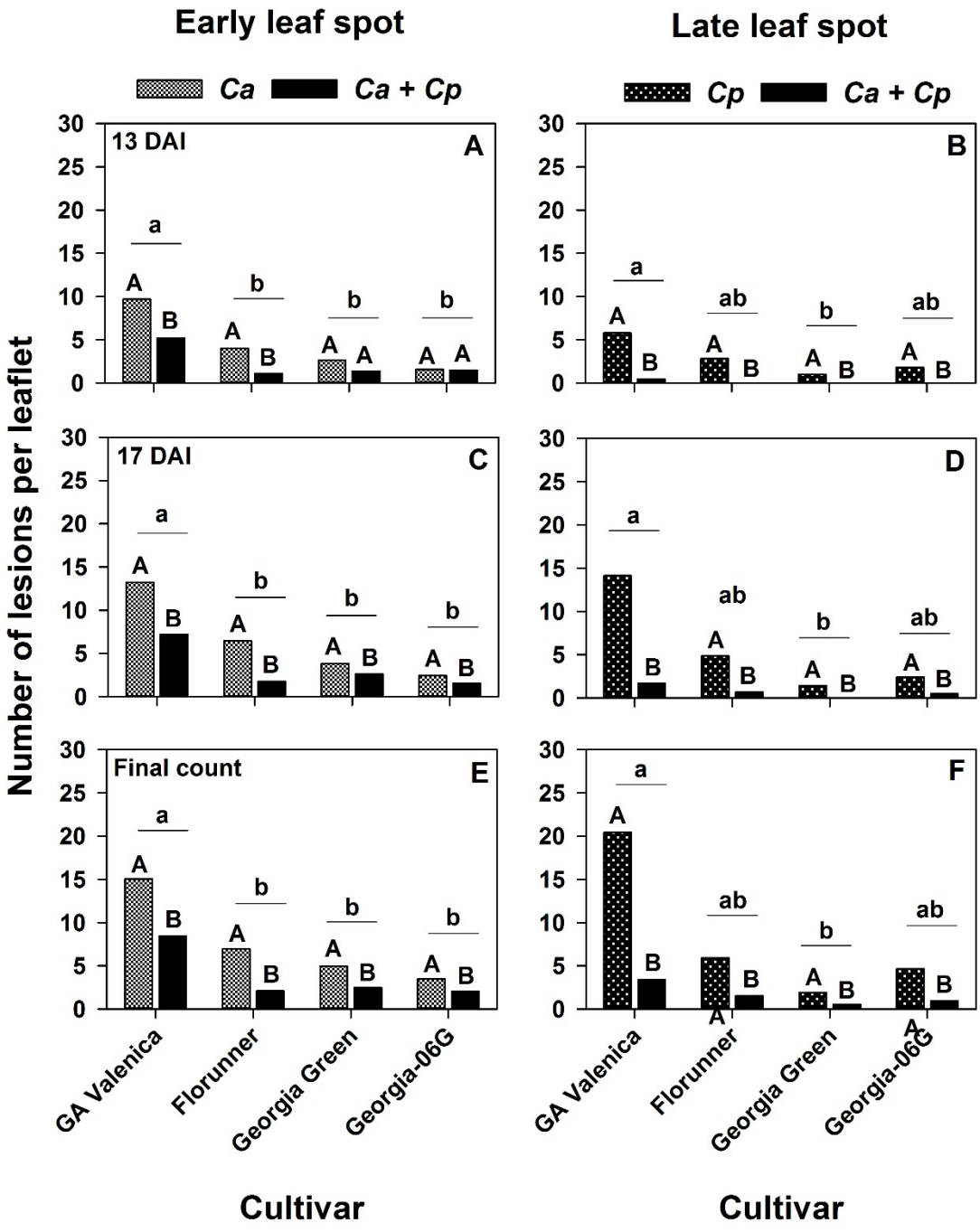
[w] Total number of early plus late leaf spot lesions per leaflet prior to leaflet defoliation.
[t] Predominance (proportion of lesion attributed to late leaf spot; calculated by dividing the final number of late leaf spot lesions by the total early and late leaf spot lesions).
[r] Days to reach leaflet defoliation.
[z] Percent defoliation at 30 days after inoculation (DAI).
[z] Final defoliation made at 64 DAI.
[s] stAUDPC defoliation = standardized area under the defoliation progress curve.



Supplementary Fig. 7.1. Average, minimum and maximum temperature (°C) and relative humidity in greenhouse chambers for the first 40 days after inoculation in trials conducted in the summer and fall of 2016.



Supplementary Fig. 7.2. Effect of inoculum treatment and peanut cultivar on the sporulation and lesion size of early (ELS) and late (LLS) leaf spot lesions. Plants were inoculated at 90 days after planting with *Cercospora arachidicola* (*Ca*), *Cercosporidium personatum* (*Cp*), and a 100/100 mixture of *Ca* + *Cp*. Grouped bars with the same letters signify that cultivars (pooled across inoculum treatments) are not significantly different based upon Tukey's honestly significant difference test ($\alpha = 0.05$). There was no statistical difference between inoculum treatments at ($\alpha = 0.05$).



Supplementary Fig. 7.3. Effect of inoculum and cultivar on the number of early and late leaf spot lesions per leaflet at 13 days after inoculation (DAI) (**A** and **B**), 17 DAI (**C** and **D**) and the final counts prior to defoliation (**E** and **F**). Plants were inoculated at 90 days after planting with *Cercospora arachidicola* (*Ca*), *Cercosporidium personatum* (*Cp*), and a 100/100 mixture of *C.a.* + *C.p.* Grouped bars for each cultivar with the same capital letters indicate that inoculum treatments were not significantly different based upon Tukey's honestly significant difference test ($\alpha = 0.05$). Lowercase letters above each cultivar denote Tukey's mean separations between cultivars ($\alpha = 0.05$). No significant ($P < 0.05$) interaction between inoculum and cultivar was observed in these analyses.

CHAPTER 8

EVALUATION OF PEANUT RX RISK FACTORS AND HISTORICAL DISEASE PREVALENCE TO PREDICT EPIDEMICS OF EARLY AND LATE LEAF SPOT OF PEANUT[1]

[1]Fulmer, A.M., Kemerait, R.C., Brenneman, T.B., Scherm, H., Cantonwine, E.G., Culbreath, A.K., Stevenson, K.L. and Mehra, L. 2017. To be submitted to *Plant Disease*.

ABSTRACT

Previous research has demonstrated the efficacy of prescription fungicide programs, based upon Peanut Rx, to reduce combined effects of early (ELS) and late leaf spot (LLS), caused by *Cercospora arachidicola* and *Cercosporidium personatum*, respectively, but the potential of Peanut Rx to predict each disease has never been formally evaluated. From 2010 to 2016, non-fungicide peanut plots in Georgia and Florida were sampled to monitor the development of ELS and LLS. This resulted in 168 cases (unique combinations of Peanut Rx risk factors) with associated total leaf spot risk points ranging from 40 to 100. Percent defoliation ranged from 13.9 to 100%, and increased linearly with increasing total risk points (R^2 = 0.37; *P* = <0.0001). Leaf spot onset [time in days after planting (DAP) when either leaf spot reached one percent lesion incidence], ELS onset, and LLS onset ranged from 29 to 140, 29 to 142 and 50 to 143 DAP, and decreased linearly with increasing risk points (R^2 0.69, *P* = <0.0001; R^2 0.62, *P* = <0.0001; R^2 0.42, *P* = <0.0001; respectively). stAUDPC of ELS increased linearly with increasing risk points (R^2 0.33, *P* = <0.0001), but not for LLS. Results from multiple regression confirmed that all Peanut Rx risk factors/predictor variables contribute to the variability of at least one measurement of development of combined or separate epidemics of ELS and LLS; however, not all factors affected ELS and LLS equally. Historical leaf spot prevalence, a new potential pre-plant risk factor, significantly contributed to the multiple regression model for predicting the onset and proportion of the epidemic attributed to LLS. Results presented here demonstrate that Peanut Rx is a very effective tool for predicting leaf spot onset regardless of which leaf spot is predominant, but also suggest that associated risk is not the same for each disease. These data will be useful for refining thresholds for differentiating high, moderate and low risk fields, and reevaluating the timing fungicide applications in reduced input programs with respect to disease onset.

INTRODUCTION

Prophylactic application of fungicide is a primary tactic for managing early (ELS) and late (LLS) leaf spot of peanut, caused by *Cercospora arachidicola* Hori, and *Cercosporidium personatum* (Berk & M. A. Curtis) Deighton, respectively (Shokes and Culbreath, 1997). Historically, fungicides have been applied on a calendar-based schedule, beginning just before or at the expected time of disease onset (30 days after planting [DAP]), and subsequently sprayed every 14 days until 2 to three weeks prior to harvest (~140 DAP) (Culbreath et al., 2006). These fungicide applications represent one of the highest input costs for peanut growers in Georgia, where the annual average damage plus cost of control from 2012 to 2014 was estimated at $37.9 million (Kemerait, 2012, 2013, 2014). As a result, a number of strategies have been investigated to reduce the number of applications. Weather-based advisories have been the major focus over the last 50 years and have been particularly successfully in Virginia (Phipps et al., 1997). The AU-Pnut weather advisory (Jacobi et al., 1995) is recommended for growers in Georgia (Kemerait et al., 2017), but has not been widely adopted by growers, likely because they do not consider the long-term gain to be worth the opportunity cost, and therefore prefer the convenience of the calendar-based system (Woodward et al., 2008).

A more recent alternative for reducing fungicide inputs is to utilize prescription programs based on disease risk as predicted with Peanut Rx (Woodward et al., 2014). Peanut Rx is an index that allows growers to estimate the overall risk to the most important foliar and soilborne peanut diseases in a given field prior to planting (Kemerait et al., 2017). For leaf spot, pre-plant risk factors include variety, planting date, rotation, field history, and irrigation (Kemerait et al., 2017). Risk points are assigned to each factor and after calculating the total points, fields are assigned a high, moderate or low level of risk. Prescription fungicide programs feature later initial applications and greater spray intervals with decreasing risk values. High-risk fields are treated according to the standard 14-day calendar based program (seven applications initiated at 30 DAP), whereas moderate- and low-risk fields are initiated at 37 and 44 DAP, and sprayed at 14/21 and 21 day intervals, for a total of five and four applications, respectively (Kemerait et al., 2017). While the general efficacy of prescription fungicide programs has

been previously established in fields with varying risk levels (Woodward et al., 2014; Woodward et al., 2010), the actual predictive potential of Peanut Rx has not been formally assessed. Furthermore, it is not clear how Peanut Rx would predict the development of ELS and LLS if the epidemics were differentiated. This uncertainty is based on the knowledge that while these diseases are similar in many respects, there are several key differences in their epidemic development that may reduce the predictability of one or both diseases.

The most characteristic difference in the epidemiological behavior of ELS and LLS is the time of disease onset. A previous report suggested that, when inoculum of both pathogens is present in a field, the onset of ELS typically occurs 30 to 50 days DAP and is generally 2 to 4 weeks prior to that of LLS (Shokes and Culbreath, 1997; Smith and Littrell, 1980). However, there are reports of both diseases appearing at 21 to 35 DAP in the tropics (McDonald et al., 1985), and more recently we have demonstrated that both diseases can occur as late as 140 DAP in Georgia (Chapter 3). LLS is also thought to be more destructive of the two due to a greater amount of sporulation per lesion and thus a higher rate of development toward the end of the season (Backman and Crawford, 1984; Hemingway, 1955; Jenkins, 1938).

Predicting the epidemic development of these diseases may be further challenged given that there are generally differences in the prevalence of ELS and LLS in a given field. Although capable of co-existing in the same field and even on the same leaflet, the relative abundance of each disease tends to vary over time both regionally (Miller, 1953) and locally (Jackson, 1981). In the late 1930s, Jenkins stated that LLS only occurred 2 out of every 5 years in Georgia, and Miller (1953) reported that ELS was the most widespread of the two diseases in the southeastern U.S. In Georgia, ELS was the most prevalent disease until a shift took place in 1976, and LLS prevailed until the mid-1990s (Cantonwine et al., 2008; Smith and Littrell, 1980). In recent years there has been a resurgence of LLS in Georgia, and it is possible to have nearby fields that are predominantly ELS or LLS (Chapter 3).

In recent studies, we demonstrated that although fungicide program, peanut cultivar and planting date can each contribute varying levels to the predominance and development of each disease, our results

strongly suggest that the amount of initial inoculum is the most important factor (Chapters 3, 4, 5 and 6). In Peanut Rx, crop rotation history is an indirect estimate of the initial inoculum, but considered alone, as it currently stands, it does not provide any indication of whether the inoculum pertains to that of the ELS or LLS pathogen. We have consistently observed that prevalence of ELS or LLS in particular field is strongly correlated with the history of these diseases in the same field. So that ELS is more prevalent in fields with a history of ELS and same for LLS (Chapters 3, 5 and 6). We hypothesize that knowledge of the historical prevalence of each disease in a given field is a good indicator of the relative initial inoculum level of each pathogen for that field, and, as a result, a good predictor of which disease will predominate. This corresponds to Smith and Littrell (1980) who surmised that the initial inoculum came from within the field or within a 1.6 km radius, and that the onset of each disease was dependent on quantity of initial inoculum in the field. Therefore, we hypothesize that historical leaf spot prevalence can be used as a pre-plant risk factor for predicting the prevalence, onset and final severity of ELS and LLS.

Given the lack of data on the actual predictive nature of Peanut Rx, and the fact that the leaf spot complex is comprised of two distinct diseases with differing epidemiological behaviors, there is a very practical need to evaluate the utility of Peanut Rx as a forecasting system. The objectives of this study were to 1) determine the relationship between Peanut Rx risk points and response variables related to key components of the general leaf spot epidemic as well as those related to each leaf spot alone, 2) to evaluate the current weighting system used to assign risk points for leaf spot by using multiple regression to determine the contribution of each risk factor, and 3) to determine if historical/previous leaf spot prevalence could be incorporated into Peanut Rx as a risk factor to predict the prevalence, onset and severity of each leaf spot disease.

MATERIALS AND METHODS

Field sites and data source. Data were obtained from untreated peanut plots from a wide range of studies with differing treatment protocols and in multiple locations during the growing seasons of years 2010 to 2016. From 2010 to 2013, data were obtained from fungicide trials conducted in Georgia and Florida that were planted to peanut cultivar Georgia-06G or Valencia peanut market types. From 2014 to

2016, the data came from several fungicide efficacy trials and epidemiological studies that included multiple cultivars and planting dates. All data included in these studies were from plots not treated with fungicide, were based on naturally occurring inoculum levels, but all herbicides, insecticides and fertilizers were applied following recommendations of the University of Georgia Cooperative Extension Service (Monfort, 2017). Experimental designs included randomized complete block, split-plot, and factorial with three to five replications. All plots consisted of two single rows spaced 91-cm apart and planted at 19.2 seeds/m. Plot length ranged from 4.6 to 12.2 m.

The majority of the field sites were located on the University of Georgia Coastal Plain Experiment Station in Tifton, GA. These fields included the RDC Pivot, Black Shank farm, Lang farm, Rigdon farm, Jones farm, ABAC farm, fire tower field, fishpond field, and the Ponder farm. Additional locations in Georgia included the Attapulgus Research and Education Center (Attapulgus, GA), the Southwest Georgia Research and Education Center (Plains, GA) the Southeast Georgia Research and Education Center (Midville, GA), C.M. Stripling Irrigation Research Park (Camilla, GA), and the Sunbelt Ag Expo (Moultrie, GA). In Florida, field sites included the North Florida Research and Education Center (Quincy and Marianna, FL), and Plant Science Research and Education Unit (Citra, FL). The studies conducted for each year and location are listed in Tables 8.1 and 8.2.

Risk factors and associated risk points. For each year and location, risk points were assigned to each unique combination of the following Peanut Rx factors: cultivar, planting date, rotation, tillage, irrigation and field (disease) history. Points were assigned based on the most recent edition of Peanut Rx (Kemerait et al., 2017). The complete data set that includes each unique combination of Peanut Rx factors for each year and location is listed in Supplementary Table 8.1. The majority of fields were planted to Georgia-06G (Table 8.3), irrigated, conventionally tilled and had a history of previous leaf spot epidemics (Table 8.3). Based on the total risk points, each case was grouped into high-, moderate- or low-risk categories according to the current Peanut Rx edition as follows: high risk, 65 to 100 points; moderate risk, 40 to 60 points; low risk, 10 to 35 points (Kemerait et al., 2017).

Variety. Sixteen varieties were included, but the majority of the studies were planted to Georgia-06G (see Table 8.3 for frequency and risk points assigned to each variety). This resulted in four categories of risk points: 15 (2 cases), 20 (118 cases), 25 (2 cases) and 30 (29 cases). The cultivars not listed on the current or past Peanut Rx sheets were assigned risk points based on the combined knowledge/experience of the authors. These included the following varieties: New Mexico Valencia C (30 points), Florunner (30 points), Southern runner (15 points) and C99R (15 points).

Planting date. The frequency of planting dates for each risk level are listed in Table 8.3. The majority of cases were in the 11 to 25 May category. Risk points for each planting date range were assigned as follows: prior to 1 May (0 points), 1 to 10 May (5 points), 11 to 25 May (10 points), and after 25 May (15 points).

Rotation. The frequency of each rotation category is listed in Table 8.3. Overall, the majority of cases were fairly uniformly distributed among the categories, but the fewest cases were recorded for a 2-year rotation. Risk points for each planting date range were assigned as follows: 0 years out of peanut (25 points), 1 year out of peanut (15 points), 2 years out of peanut (10 points), 3 or more years out of peanut (5 points).

Tillage. The frequency of each tillage type is listed in Table 8.3. Conventional tillage involved disking and deep turning with a moldboard plow. Reduced tillage involved planting a winter cover crop of wheat (*Triticum aestivum*) each year, followed by an application of glyphosate (Roundup 4 EC, Monsanto, Kansas City, MO) at 1.2 kg a.i./ha approximately 2 weeks prior to planting. Plots were prepared for planting using a strip-till implement as described by (Monfort et al., 2004).

Irrigation. The frequency of irrigated and dryland fields used in these studies is listed in Table 8.3. Fields that were irrigated according to moisture recommendations from the University of Georgia Cooperative Extension Service were assigned a risk value of 10 points and non-irrigated fields were assigned a risk value of 0 points.

Field history. This is a more subjective factor and is based on whether a field was known to have had a prior outbreak of a leaf spot epidemic during the previous peanut cropping season. For these

studies, 'Yes' was assigned to fields that had previously been used for fungicide studies that included untreated plots and were assigned a risk value of 10 points. 'No' was assigned to fields with a rotation > 3 years, or for fields where diseases were heavily managed with fungicides in the previous seasons and were assigned a risk value of 0 points.

Previous leaf spot history. Based on the historical prevalence of each leaf spot, fields were assigned one of the following four categories: ELS, LLS, both or none. ELS and LLS signifies that fields had previously been documented to have a history of being predominantly ELS or LLS, respectively. 'Both' signifies that both diseases were known to heavily contribute to the overall leaf spot epidemic in the previous peanut cropping season. 'None' signifies that there was no record or knowledge of which disease was prevalent, and was generally associated with fields that had been out of peanut for > five years. Because previous leaf spot history is not currently listed as risk factor in Peanut Rx, no risk points were associated with this variable. Rather these data were assembled for multiple regression studies to determine if historical prevalence could help explain the variability in the development of early and late leaf spot.

Disease assessment. Beginning approximately 30 days after planting (DAP), plots were visually monitored for initial leaf spot symptoms. Upon first detection of leaf spot symptoms, a more intensive evaluation was initiated and continued approximately on a bi-weekly schedule until the end of the peanut season (~140 DAP). Five main stems per plot were arbitrarily selected for destructive sampling and returned to the lab where the number of present and missing leaflets, and the number of ELS and LLS lesions per leaflet were counted. ELS and LLS were differentiated by symptoms and signs associated with each causal pathogen.

Based on the data collected, incidence of each disease for each assessment date was calculated by dividing the number of leaflets with at least one lesion by the total number of leaflets collected from each plot. Area under the disease progress curves (AUDPC) were calculated for each leaf spot disease based on the number of lesions per leaflet. AUDPC values were standardized (stAUDPC) by dividing AUDPC values by the duration of the epidemic (Campbell and Madden, 1990). Predominance was estimated as

the proportion of the total leaf spot epidemic attributed to LLS, and was calculated by dividing the standardized AUDPC of LLS by the total standardized AUDPC of ELS and LLS combined.

Time of disease onset was defined as the number of DAP for ELS or LLS to reach 1% incidence. Our decision to use 1% incidence as the onset threshold was based on several reasons. First, fungicide timings based upon Peanut Rx are designed to be prophylactic, and therefore a one-percent threshold is the most realistic value for applied purposes. Secondly, because ELS and LLS were not evenly distributed in each trial, there were a number of occasions in which neither leaf spot reached a level greater than 1% incidence. For the same purpose, onset was not based on values calculated from modeling the disease progress curves. Thirdly, 1% incidence, rather than the first lesion detected, would theoretically help ensure that the epidemic was actually commencing rather than detecting a false positive (e.g., a random lesion).

Statistical analyses. To test the overall predictive potential of the current Peanut Rx model, simple linear regression, PROC REG in SAS (version 9.4, SAS Institute, Cary, NC), was used to assess the relationship between Peanut Rx risk points and percent main stem defoliation, total AUDPC (combined stAUDPC and values of the number of ELS and LLS lesions per leaf), leaf spot onset (time in DAP when either leaf spot reached 1% lesion incidence). The same regression approach was used to determine the relationship between Peanut Rx risk points and the separate epidemics of ELS and LLS, and included the following response variables: ELS onset, LLS onset, ELS stAUDPC, LLS stAUDPC and predominance.

To determine the predictive potential of each Peanut Rx factor, and to determine if historical prevalence could be used as a predictive variable, multiple linear regression was performed using PROC REG in SAS. Predictive variables in the model included: variety risk, planting date (day of year), rotation (0, 1, 2, or ≥ 3 years out of peanut), tillage, field history, irrigation, and previous leaf spot history (ELS, LLS, Both or None). Dummy variables were generated for each of the predictive variables based on the categories previously listed for each Peanut Rx factor. The only exception was variety, in which case the actual risk points were used in the multiple regression model. This was due to there being a large number

of cultivars that shared the same risk points as well as due to many of the cultivars were included in only a few studies (see Table 8.3). Response variables included percent main stem defoliation, total AUDPC (combined stAUDPC values of the number of ELS and LLS lesions per leaf), leaf spot onset (time in DAP when either leaf spot reached one percent lesion incidence), ELS onset, LLS onset, ELS stAUDPC, LLS stAUDPC and predominance.

RESULTS

After averaging across replicates, a total of 168 cases (unique combinations of levels of Peanut Rx factors) were obtained from field trials conducted in Georgia and Florida from 2010 to 2016. Based on the current Peanut Rx thresholds, this resulted in 129 high risk cases, 39 moderate risk cases and no low risk cases. Although, it should be noted that several cases were near the low-risk threshold ($\leq$ 35 points). The distribution of final risk points for each case is displayed in Fig. 8.1, showing that there was a good representation of risk points. Summary statistics of each response variable are displayed in Table 8.4, and generally document a wide range of values across the years in which these data were obtained. Percent defoliation on the main stem ranged from 13.9 to 100%, and total stAUDPC ranged from 0 to 23.7 (Table 8.4). Leaf spot onset (time in DAP when either leaf spot reached one percent lesion incidence) ranged from 29 to 140 DAP; however, this value reflected the onset of ELS 90% of the time (data not shown). On average, ELS onset was ~20 days prior to that of LLS, and the earliest observation of ELS and LLS was at 29 and 50 DAP, respectively (Table 8.4). The average and maximum values of ELS stAUDPC were lower than those of LLS stAUDPC, and both diseases had cases with very low severity (values close to 0) (Table 8.4). The predominance of LLS varied significantly between fields, with the maximum proportion of LLS at almost one and the lowest at almost zero (Table 8.4).

Analyses based on risk points. The results from simple linear regression analysis demonstrated that there was a significant relationship between Peanut Rx risk points and all response variables except for LLS stAUDPC (Table 8.5). Among the significant models, the coefficients of determination (R^2) ranged from 0.18 to 0.69 (Table 8.5). Overall, risk points more strongly associated with leaf spot onset ($R^2 = 0.69$), and were a better for ELS onset ($R^2 = 0.62$) than for LLS onset ($R^2 = 0.42$) (Table 8.5). For

all onset variables, there was a negative relationship with risk points (Figs. 8.1 and 8.2; Table 8.6). Percent defoliation had a moderate, positive linear relationship with risk points, but was not as strong for total stAUDPC of lesions per leaf (Fig. 8.1 and Table 8.6). There was a moderate positive linear relationship for ELS stAUDPC ($R^2 = 0.33$), but not for LLS stAUDPC ($R^2 = 0.02$) (Table 8.6 and Fig. 8.2). Predominance had a weak, negative linear relationship with risk points (Table 8.6 and Fig. 8.4), and had a low R^2 value (Table 8.5).

Analyses based on multiple regression. The results from the multiple regression analysis generally resulted in similar differences between response variables compared with those obtained with simple linear regression with total risk points. For example, the multiple regression model provided the best fit for leaf spot onset ($R^2 = 0.72$), and ELS onset had a better fit ($R^2 = 0.71$) than LLS onset ($R^2 = 0.50$) (Table 8.7). Percent defoliation had a better fit to the multiple regression model ($R^2 = 0.56$) than the total stAUDPC of lesions per leaf (0.24), and when both diseases were analyzed separately, ELS stAUDPC had a better fit ($R^2 = 0.43$) than LLS stAUDPC ($R^2 = 0.21$) (Table 8.7).

Rotation was consistently the most important predictor variable, and significantly contributed to the model of seven of the eight response variables (Table 8.8). Variety and irrigation were significant in six of the eight models; field history and historical prevalence contributed to five of the eight models (Table 8.8). Planting date was significant for four of the response variables, tillage was significant for three of the response variables (Table 8.8).

Percent defoliation was significantly affected by all predictor variables except historical prevalence (Table 8.8). For total stAUDPC, the multiple regression analysis indicated that tillage, field history, irrigation and historical prevalence were the most important predictor variables (Table 8.8). For leaf spot onset, and for ELS onset, all predictor variables significantly contributed to the model except for tillage and historical prevalence (Table 8.8). Similar results were obtained for LLS onset, except that historical prevalence had a highly significant effect (Table 8.8). For ELS stAUDPC, planting date, tillage, field history did not contribute to the model, and the same was observed for LLS stAUDPC except that irrigation also failed to contribute to the model (Table 8.8).

The results indicate historical prevalence had a consistent significant effect on response variables related to the behavior of LLS onset, LLS stAUDPC and predominance (ProLLS) (Table 8.8). Onset of LLS was slightly earlier in fields that had a prior history of LLS or both leaf spots, but was generally less variable than ELS (Fig. 8.5). The onset of ELS was earliest in fields with a prior history of ELS or both diseases (Fig. 8.5), however, this did not significantly contribute to the variability of ELS onset in the multiple regression analysis (Table 8.8). Predominance (ProLLS) was primarily associated with the historical prevalence of each leaf spot (Fig. 8.6) and rotation (Table 8.8). As such, ELS was predominant in fields with a prior history of ELS and LLS was predominant in fields with a prior history of LLS; fields with a prior history of 'Both' or 'None' tended to have an equal level of both diseases (Fig. 8.6).

DISCUSSION

Based on 168 unique combinations of levels of Peanut Rx factors gathered from untreated plots over the course of 7 years, the results presented here clearly demonstrate that associated risk points are capable of predicting key components of the overall leaf spot epidemic. Prior to this report, the predictive potential of Peanut Rx has not been assessed on a large scale. These results corroborate previous studies that have documented that prescription fungicide programs based upon Peanut Rx allow growers to make fewer fungicide applications in fields with varying levels of risk without compromising yield (Woodward et al., 2014; Woodward et al., 2010). Furthermore, by differentiating the two leaf spot diseases, we found that ELS onset and stAUDPC had a stronger linear relationship with risk points compared with LLS. However, in relation to predicting the onset of the overall leaf spot epidemic, this was inconsequential. Our overall results indicate that leaf spot onset is the best predicted response variable, and this holds true regardless of which leaf spot is predominant. This is an important discovery given the differences in prevalence of each disease that can occur in a given field. To the best of our knowledge, this is the first formal analysis to identify the most important components of the leaf spot epidemic associated with risk points.

Given the strong negative linear relationship between risk points and leaf spot onset, these data suggest that the current threshold for risk categories may result in an over-estimation of risk. Based on

the current threshold levels, all of the fields were categorized as high-risk, or moderate-risk fields. There were no low-risk cases, and yet the onset of disease was >90 DAP in fields with the lowest risk points. This discovery calls into question the recommended timing of the first fungicide application in the current Peanut Rx-based prescription fungicide programs. Currently, initial applications for high, moderate and low risk prescription fungicide programs are recommended at 30, 37 and 44 DAP, respectively (Kemerait et al., 2017), yet actual disease onset much more delayed in many moderate risk fields. It should be noted that, in the present study, ELS and LLS lesions were differentiated based on signs and symptoms which required detection of a sporulating lesion, and thus does not consider the time required for infection prior to the development of symptoms and signs. The incubation and latent period for both ELS and LLS is ~ 7 to 14 and ~14 to 21 days, respectively, depending on genotype and environment (Cantonwine et al., 2008; Shew et al., 1988). Thus, the infection process for detection of either disease at 100 DAP would have commenced by ~75 to 80 DAP, respectively. Therefore, even when considering the time required for the infection process, these results suggest that current fungicide timings for Peanut Rx-based prescription fungicide programs could be adjusted to better match expected disease onset. To emphasize the point, we recently showed that in fields where the onset of disease is > 100 DAP, four fungicide applications with the first fungicide application delayed up to 65 and 86 DAP still provided excellent leaf spot control (Chapter 11). Therefore, it may be that the data obtained from these studies can be used to reevaluate the thresholds for low-, moderate- and high-risk groups.

Percent defoliation had a stronger relationship with risk points than stAUDPC, but neither were as strong as leaf spot onset. Several reasons may explain these results. The final assessment of defoliation was made between ~ 135 and 145 DAP, and therefore was not uniform across all cases. More importantly, final percent defoliation at this time does not differentiate between fields that were completely defoliated by 120 vs. 140 DAP, which was the case in a number of high risk scenarios (data not shown).

Results of the multiple regression analyses confirmed that all Peanut Rx risk factors/predictor variables contribute to the variability of at least one component of the combined and separate epidemic of

ELS and LLS. However, not all factors affected ELS and LLS equally. We previously documented that inoculum level, planting date and cultivar affect both diseases, but the magnitude of the effect often differs between the two diseases (Chapters 4, 5 and 6). This will be further elaborated on in the following paragraphs that will focus on the individual contribution of each risk factor.

The primary cultivar used in these studies was Georgia-06G, but the multiple regression approach still indicated that variety was a significant predictor variable. We previously documented that the onset of LLS is more delayed and the LLS stAUDPC is lower on Georgia-06G compared with more susceptible varieties such as Florunner and Georgia Valencia (Chapters 6 and 7). This may partially explain why the slope of the line for LLS onset was less than that of ELS. In fact, in a separate simple linear regression analysis with risk points that excluded all other varieties other than Georgia-06G, we found that the R^2 value for LLS was considerably reduced ($R^2 = 0.26$, $P = <0.0001$) compared to when all other varieties were included in the analysis ($R^2 = 0.42$, $P = <0.0001$) while the R^2 value for ELS onset for Georgia-0G alone ($R^2 = 0.64$, $P = 0.0001$) was similar to the result for all cultivars ($R^2 = 0.62$, $P = <0.0001$) (data not shown). Similar results were obtained when ELS and LLS stAUDPC values were regressed on risk points associated with Georgia-06G alone (data not shown). These data corroborate previous reports that indicate that there may be differential levels of resistance to each leaf spot pathogen for a given genotype (Abdou et al., 1974; Walls et al., 1985). Therefore, these data suggest that knowledge of disease prevalence in a given field could be another important source of risk.

Planting date had a highly significant effect on defoliation and leaf spot onset, but not for stAUDPC values. A number of previous studies documented that the onset of both leaf spots generally decreases with later planting dates (Jordan and Culbreath, 2016; Shokes et al., 1982), but we have recently reported that magnitude of the response tends to be greater for LLS (Chapter 5). However, stAUDPC values were not affected by planting date in the studies in present study. This was likely due to the effect of planting date being overshadowed by the differing levels of prevalence between fields. We recently reported a similar effect in fields treated with differing levels of peanut residue (Chapter 4 and 5).

Rotation had the most consistent effect on leaf spot development across response variables, which corroborates results of previous studies. In a previous study, a 1-year rotation away from peanut significantly reduced LLS severity (Kucharek, 1975). Brenneman et al. (1995) found that leaf spot severity decreased when peanuts were rotated to bahiagrass for 0, 1, 2 or 3 years. Currently, apart from variety, the Peanut Rx weighting system places the greatest weight on rotation (Kemerait et al., 2017), and the results from these studies validate this emphasis. Interestingly, we found that LLS tended to be most prevalent in a majority of the fields with the longest rotations away from peanut, i.e., fields with the lowest risk points, as indicated by the heavy LLS predominance associated with low risk points (Fig. 8.4). This could be due to differences in the survival or dispersal of the two pathogens. However, as discussed in Chapter 4, further research is needed to better understand the differences in survival or dispersal of the two pathogens.

The effect of reduced or strip tillage to delay the epidemics of ELS has previously been well documented (Cantonwine et al., 2007; Monfort et al., 2004). However, in our study, there was no effect of tillage on the onset of either disease. It should be noted that in this data set, the majority (~95%) of the fields were conventionally tilled; among the cases with reduced tillage, fields were rotated for 3 or more years, and predominantly LLS. Therefore, this data set did not offer a good opportunity to evaluate the effect of tillage on ELS or LLS epidemics. However, the results do suggest that the effect of reduced or strip tillage may become negligible in a well rotated field, or if LLS is predominant. Cantonwine et al. (2007) suggested that because ELS was the predominant disease in their studies, further work would be needed to determine the effect of tillage on LLS development. We suggest that this statement still applies, and that further work is needed to determine the interactive effects of rotation and tillage for each leaf spot.

It is well known that leaf spot epidemics are dependent upon moisture, and this has been the rationale behind the intense research aimed at developing weather-based advisories for peanut growers (Jacobi et al., 1995; Jensen and Boyle, 1965; Jensen and Boyle, 1966; Phipps et al., 1997). Peanut Rx is unique in that there is no major weather component included other than irrigation, which we found to be a

highly significant pre-plant risk factor in these studies. Therefore, it has been recognized from the beginning that Peanut Rx gives up some predictive potential at the expense of providing a less time demanding tool for growers. However, our results indicate that Peanut Rx risk points provide an excellent prediction of disease onset regardless of variations in the weather patterns across years. It should be noted that most (88%) of the fields in these studies were irrigated, which likely provided a more of uniformly moist environment. Therefore, Peanut Rx may be more accurate under irrigated conditions. However, it is noteworthy that, at worst, prediction of leaf spot onset based on Peanut Rx in a dryland situation would result in an over prediction of risk, in which case, growers would follow a fungicide application schedule as they would in the absence of Peanut Rx.

The results presented here indicate that field history does contribute to the variability in the onset of both diseases, and therefore suggests that it is a useful tool for helping growers estimate disease risk. Field history is one of the most challenging risk factors to quantify because of the subjective nature of the characterization, and as a result, each grower may interpret this differently. The purpose of including this factor is to help provide an indirect estimate of the inoculum potential of each field (along with rotation), by assessing whether a disease outbreak had occurred in the field in recent history. Presumably, a well-managed field would result in very little to no inoculum carryover. In these studies, we attempted to standardize the qualification by assigning 'Yes' to fields that had previously been used for fungicide studies that included untreated plots; 'No' was assigned to fields with a rotation > 3 years, or for fields where diseases were heavily managed with fungicides in the previous seasons. However, by doing so, this may have been testing more of a reflection of the effect of rotation than of field history. While more research on the interaction of these factors may be warranted, the conclusion based on our combined experience, is that field history is worthwhile to include in the Peanut Rx risk index as an additional safety factor.

Smith and Littrell (1980) previously suggested that a number of factors may influence the shift from ELS to LLS including initial inoculum source, variety, and fungicide, among others. We recently reported that while planting date, variety and fungicide program can all affect the predominance of each

leaf spot disease, initial inoculum source appears to have the most significant impact (Chapters 3, 4, 5 and 6). The current study clearly documents that knowledge of previous disease history is an excellent indicator of future predominance in a given field. More importantly, this knowledge can be used to more accurately predict the onset of LLS. To the best of our knowledge, this is a novel discovery that has not been previously documented that helps explain the differences in the onset and predominance of each disease. From a practical standpoint, we suggest that it may be possible to use the knowledge of previous field history as part of an integrated management system by selecting a cultivar with more resistance to the predominant leaf spot (Chapter 6), or by adjusting fungicide timing to better align with expected disease development (Chapter 11).

In conclusion, the results presented here demonstrate that Peanut Rx is a very effective tool for predicting leaf spot onset - regardless of which leaf spot is predominant. These data support previous studies that documented the individual contribution of each risk factor, and confirm that a high level of predictive accuracy can be obtained without knowledge of in-season weather conditions. However, it is clear that the current weighting system is a better predictor of ELS epidemics compared with LLS epidemics, although this was inconsequential in terms of predicting overall leaf spot onset (time in DAP when either leaf spot reached 1% lesion incidence). Furthermore, because of a linear relationship between disease onset and total leaf spot risk points, and resulting unequal distribution of risk groups, these data suggest that the current threshold for differentiating high, moderate and low risk fields may need to be modified. We also report, for the first time, that the knowledge of previous leaf spot prevalence is an excellent predictor of which disease will be predominant, and we hypothesize that this knowledge may be useful for refining our ability to manage each disease with reduced fungicide input programs.

ACKNOWLEDGEMENTS

We thank personnel at the University of Georgia and University of Florida research stations for all of the assistance provided. Particularly, we thank Stan Jones, Billy Mills, Lee Hitson, John Paulk, Andy Carter, Dewayne Dales, Mike Heath, Eric Smith, Hunt Sanders, Mark Gomillion, Jim Boyer,

Maynard Douglas, Dan Everts, Cliff Riner, Randy Hill, Denny Thigpin, Anthony Black, Calvin Perry, Bobby Washington, Caleb Clements, Jarod Walls, Lina Flor Young, Jeannette Mixon, Jhen Bennett and Edna Shannon for technical support.

LITERATURE CITED

Abdou, Y. A.-M., Gregory, W. C., and Cooper, W. E. 1974. Sources and nature of resistance to *Cercospora arachidicola* Hori and *Cercosporidium personatum* (Beck & Curtis) Deighton in *Arachis* species. Peanut Sci. 1:6-11.

Backman, P., and Crawford, M. 1984. Relationship between yield loss and severity of early and late leafspot diseases of peanut. Phytopathology 74:1101-1103.

Brenneman, T., Sumner, D., Baird, R., Burton, G., and Minton, N. 1995. Suppression of foliar and soilborne peanut diseases in bahiagrass rotations. Phytopathology 85:948-952.

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

Cantonwine, E., Culbreath, A., and Stevenson, K. 2007. Characterization of early leaf spot suppression by strip tillage in peanut. Phytopathology 97:187-194.

Cantonwine, E., Culbreath, A., Holbrook, C., and Gorbet, D. 2008. Disease progress of early leaf spot and components of resistance to *Cercospora arachidicola* and *Cercosporidium personatum* in runner-type peanut cultivars. Peanut Sci. 35:1-10.

Culbreath, A., Kemerait Jr, R., and Brenneman, T. 2006. Management of early leaf spot of peanut as affected by fungicide and date of spray program initiation. Plant Health Progress doi:10.1094/PHP-2006-0214-01-RS.

Hemingway, J. 1955. The prevalence of two species of Cercospora on groundnuts. Trans. Brit. Mycol. Soc. 38:243-246.

Jackson, L. 1981. Distribution and severity of peanut leafspot in Florida. Phytopathology 71:324-328.

Jacobi, J., Backman, P., Davis, D., and Brannen, P. 1995. AU-Pnuts advisory I: Development of a rule-based system for scheduling peanut leaf spot fungicide applications. Plant Dis. 79:666-671.

Jenkins, W. A. 1938. Two fungi causing leaf spot of peanut. J. Agr. Res. 56:317-332.

Jensen, R., and Boyle, L. 1965. The effect of temperature, relative humidity and precipitation on peanut leafspot. Plant Dis. Rep 49:975-978.

Jensen, R. E., and Boyle, L. W. 1966. A technique for forecasting leafspot on peanuts. Plant Disease Reporter 50:810-814.

Jordan, B., and Culbreath, A. 2016. Effect of planting date and peanut cultivar on leaf spot epidemics and pod yield with implications for organic production. (Abstr.). Phytopathology 106:S2.6. http://dx.doi.org/10.1094/PHYTO-106-4-S2.6.

Kemerait, R. C., Jr. 2012. Georgia Plant Disease Loss Estimates. Coop. Ext. Ser. Bull. 102-5, University of Georgia, Athens.

Kemerait, R. C., Jr. 2013. Georgia Plant Disease Loss Estimates. Coop. Ext. Ser. Bull. 102-6, University of Georgia, Athens.

Kemerait, R. C., Jr. 2014. Georgia Plant Disease Loss Estimates. Coop. Ext. Ser. Bull. 102-7, University of Georgia, Athens.

Kemerait, R. C., Jr., Brenneman, T. B., and Culbreath, A. K. 2017. Peanut disease update. Pages 23-62 in: 2017 peanut update. W. Monfort, ed. Coop. Ext. Ser. College of Agric. Environ. Sci. University of Georgia., Athens.

Kucharek, T. 1975. Reduction of Cercospora leafspots of peanut with crop rotation. Plant Disease Reporter 59:822-823.

McDonald, D., Subrahmanyam, P., Gibbons, R., and Smith, D. 1985. Early and Late Leaf Spots of Groundnut. Information Bull. No. 21. ICRISAT, Patancheru.

Miller, L. I. 1953. Studies of the parasitism of *Cercospora arachidicola* Hori and *Cercospora personata* (Berk. & M.A. Curtis). Ph.D. Dissertation. University of Minnesota, Minneapolis.

Monfort, W., Culbreath, A., Stevenson, K., Brenneman, T., Gorbet, D., and Phatak, S. 2004. Effects of reduced tillage, resistant cultivars, and reduced fungicide inputs on progress of early leaf spot of peanut *(Arachis hypogaea*). Plant Dis. 88:858-864.

Monfort, W., ed. 2017. 2017 Peanut Update. Coop. Ext. Ser. College of Agric. Environ. Sci. University of Georgia., Athens.

Phipps, P. M., Deck, S. H., and Walker, D. R. 1997. Weather-based crop and disease advisories for peanuts in Virginia. Plant Dis. 81:236-244.

Shew, B., Beute, M., and Wynne, J. 1988. Effects of temperature and relative humidity on expression of resistance to *Cercosporidium personatum* in peanut. Phytopathology 78:493-498.

Shokes, F., and Culbreath, A. K. 1997. Early and late leaf spots. Pages 17-20 in: Compendium of Peanut Diseases, 2nd Ed. N. Kokalis-Burelle, D. Porter, R. Rodriguez-Kabana, D. Smith and P. Subrahmanyam, eds. American Phytopathological Society Press, St. Paul, MN.

Shokes, F., Gorbet, D., and Sanden, G. 1982. Effect of planting date and date of spray initiation on control of peanut leaf spots in Florida. Plant Dis. 66:574-575.

Smith, D. H., and Littrell, R. H. 1980. Management of peanut foliar diseases with fungicides. Plant Dis. 64:356-361.

Walls, S. B., Wynne, J., and Beute, M. 1985. Resistance to late leafspot peanut of progenies selected for resistance to early leafspot. Peanut Sci. 12:17-22.

Woodward, J., Brenneman, T., Kemerait, R., Culbreath, A., and Smith, N. 2010. Management of peanut diseases with reduced input fungicide programs in fields with varying levels of disease risk. Crop Protect. 29:222-229.

Woodward, J., Brenneman, T., Kemerait Jr, R., Culbreath, A., and Smith, N. 2014. On-farm evaluations of reduced input fungicide programs in peanut fields with low, moderate, or high levels of disease risk. Peanut Sci. 41:50-57.

Woodward, J. E., Brenneman, T. B., Kemerait, R. C., Smith, N. B., Culbreath, A. K., and Stevenson, K. L. 2008. Use of resistant cultivars and reduced fungicide programs to manage peanut diseases in irrigated and nonirrigated fields. Plant Dis. 92:896-902.

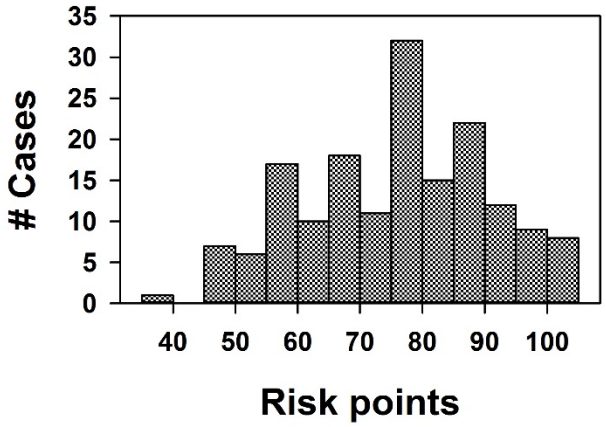


Fig. 8.1. Frequency distribution of total risk points calculated for each of the 168 unique combinations of Peanut Rx factors obtained from multiple field trials conducted in Georgia and Florida during 2010 to 2016.

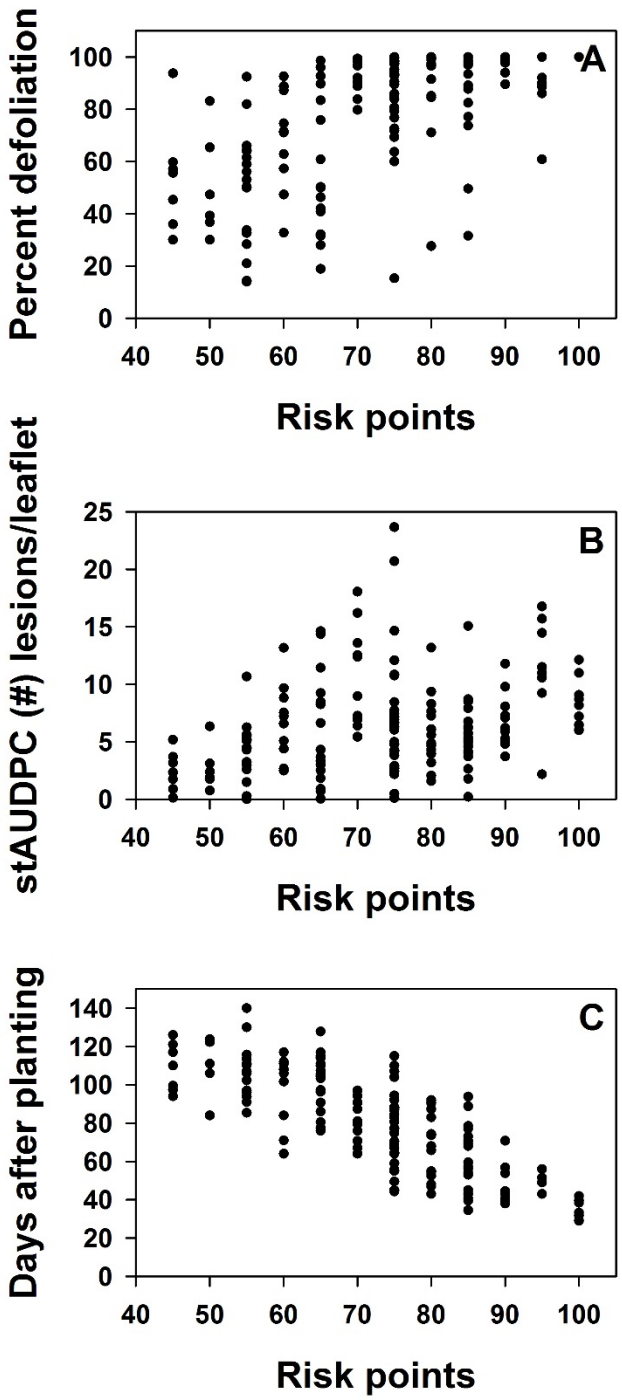


Fig. 8.2. Relationship of **A** final main stem defoliation, **B** stAUDPC of total lesions counted and **C** leaf spot onset (time in DAP when either leaf spot reached 1% lesion incidence) with total risk points calculated for 168 disease cases (unique combinations of Peanut Rx factors) collected from untreated plots in Georgia and Florida during 2010 to 2016.

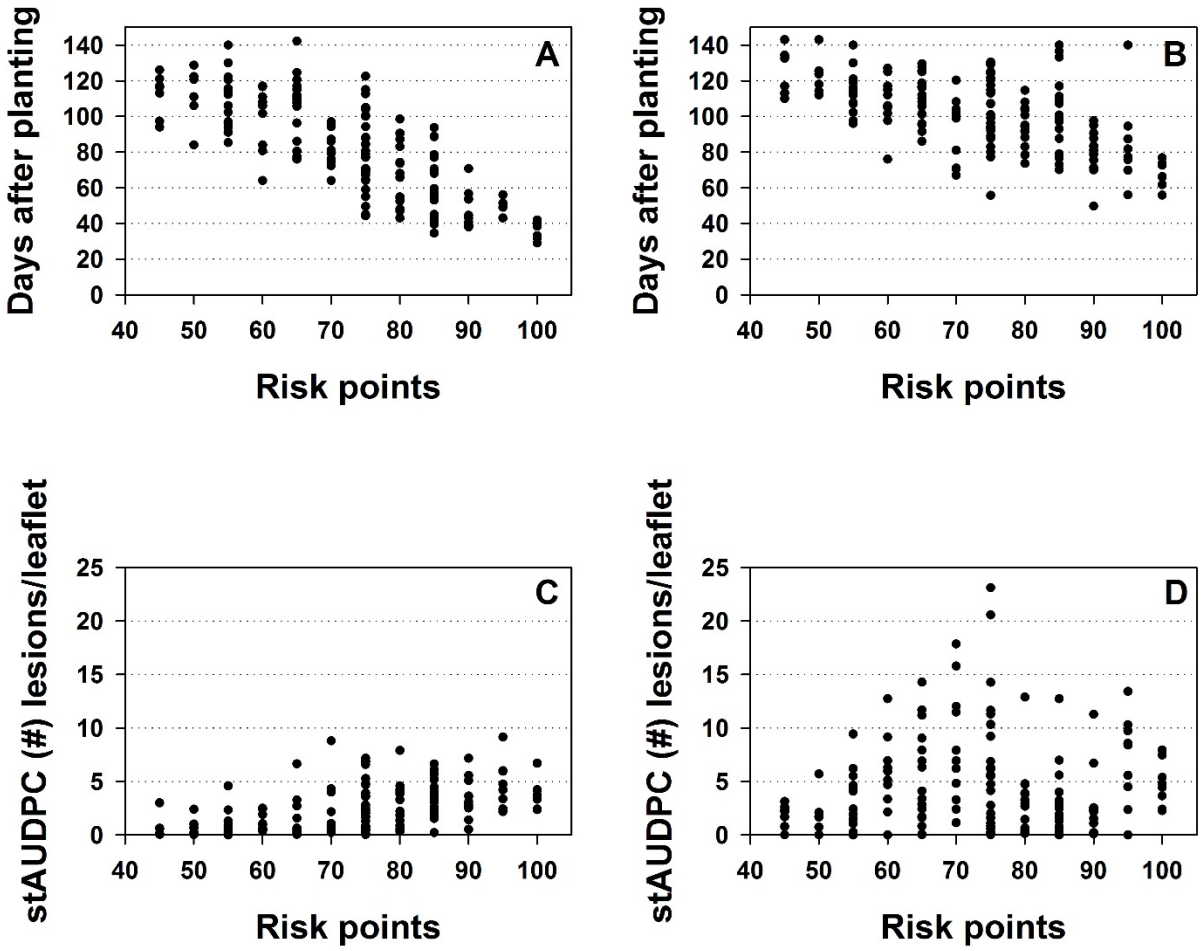


Fig. 8.3. Scatter plots showing the relationship of risk points calculated for 168 disease cases (unique combinations of Peanut Rx factors) collected from untreated plots in Georgia and Florida during 2010 to 2016 with **A** early (ELS) and **B** late (LLS) leaf spot onset (time in DAP to reach 1% lesion incidence) and, **C** ELS and **D** LLS stAUDPC (standardized area under the disease progress curves based on the number of lesions per leaflet).

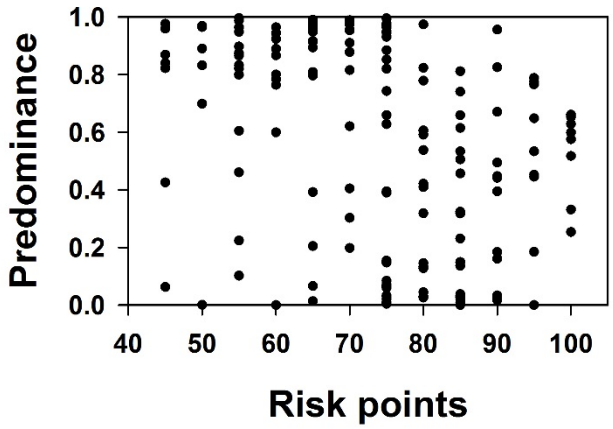


Fig. 8.4. Relationship of predominance (proportion of the stAUDPC (based on the number of lesions per leaflet) attributed to late leaf spot) with Peanut Rx risk points calculated for 168 disease cases (unique combinations of Peanut Rx factors) collected from untreated plots in Georgia and Florida during 2010 to 2016.

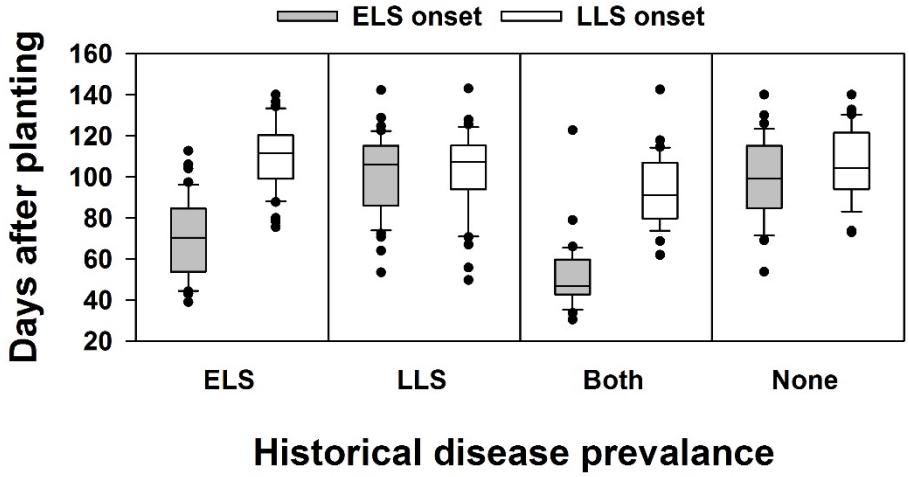


Fig. 8.5. Distribution of early (ELS) and late (LLS) leaf spot onset in relation to the disease history (historical prevalence of each case (unique combination of Peanut Rx risk factors). ELS and LLS signifies that fields had previously been documented to have a history of being predominantly ELS or LLS, respectively. Both signifies that both diseases were known to contribute heavily to the epidemic in the previous peanut cropping season. None signifies that there was no record or knowledge of which disease was prevalent, which was generally associated with fields that had been out of peanut for > 5 years. For ELS, n = 49; LLS, n = 46; Both, n = 39; None, n = 34. Data were collected from untreated plots in Georgia and Florida from 2010 to 2016.

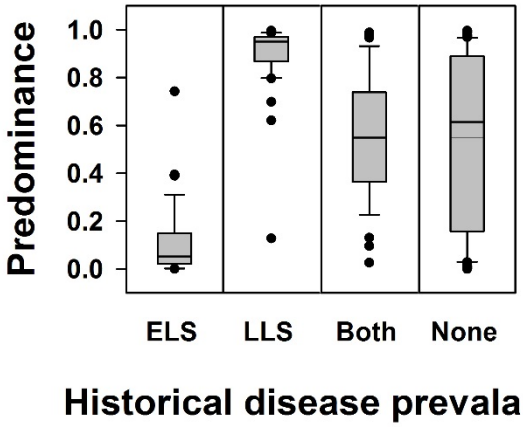


Fig. 8.6. Distribution of predominance (proportion of the stAUDPC (based on the number of lesions per leaflet) attributed to late leaf spot) in relation to the disease history (historical prevalence of each leaf spot). ELS and LLS signifies that fields had previously been documented to have a history of being predominantly ELS or LLS, respectively. Both signifies that both diseases were known to contribute heavily to the epidemic in the previous peanut cropping season. None signifies that there was no record or knowledge of which disease was prevalent, and was generally associated with fields that had been out of peanut for > 5 years. For ELS, n = 49; LLS, n = 46; Both, n = 39; None, n = 34. Data were collected from untreated plots in Georgia and Florida from 2010 to 2016.

Supplementary Table 8.1. Risk factors and associated risk points calculated for each unique combination of Peanut Rx risk factors from untreated plots in Georgia and Florida from 2010 to 2016.

Year	Location	Study	Variety	Tillage	Planting Date	Rotation	Irrigation	Risk Points	Dis History
2010	Attapulgus, GA	Dry	GA06G	Conv	5/17/2010	0	No	75	ELS
2010	Attapulgus, GA	Irr	GA06G	Conv	5/17/2010	1	Yes	75	ELS
2010	Blackshank Farm, Tifton, GA	Irr	GA06G	Conv	5/20/2010	0	Yes	85	ELS
2010	Blackshank Farm, Tifton, GA	Dry	GA06G	Conv	5/20/2010	3	No	55	ELS
2010	RDC, Tifton, GA	Strip	GA06G	Strip	5/26/2010	5	Yes	60	LLS
2011	Attapulgus, GA	Dry	GA06G	Conv	5/23/2011	0	No	75	ELS
2011	Attapulgus, GA	Irr	GA06G	Conv	5/23/2011	1	Yes	75	ELS
2011	Attapulgus, GA	Dry	NMValA	Conv	5/23/2011	0	No	85	ELS
2011	Attapulgus, GA	Irr	NMValA	Conv	5/23/2011	1	Yes	85	ELS
2011	Blackshank Farm, Tifton, GA	Dry	GA06G	Conv	5/25/2011	0	No	75	ELS
2011	Blackshank Farm, Tifton, GA	Irr	GA06G	Conv	5/25/2011	0	Yes	85	ELS
2011	Blackshank Farm, Tifton, GA	Dry	NMValA	Conv	5/25/2011	0	No	85	ELS
2011	Blackshank Farm, Tifton, GA	Irr	NMValA	Conv	5/25/2011	0	Yes	95	ELS
2011	Plains, GA	Irr	GA06G	Conv	5/18/2011	3	Yes	65	None
2011	RDC, Tifton, GA	Strip	GA06G	Strip	5/20/2010	5	Yes	55	LLS
2011	RDC, Tifton, GA	Strip	GaVal	Strip	5/20/2010	5	Yes	65	LLS
2012	Attapulgus, GA	Dry	GA06G	Conv	5/9/2012	2	No	55	ELS
2012	Attapulgus, GA	Irr	GA06G	Conv	5/9/2012	3	Yes	60	ELS
2012	Attapulgus, GA	Dry	NMValA	Conv	5/9/2012	2	No	65	ELS
2012	Attapulgus, GA	Irr	NMValA	Conv	5/9/2012	3	Yes	70	ELS
2012	Blackshank Farm, Tifton, GA	Dry	GA06G	Conv	5/23/2012	0	No	75	ELS
2012	Blackshank Farm, Tifton, GA	Irr	GA06G	Conv	5/23/2012	1	Yes	75	ELS
2012	Blackshank Farm, Tifton, GA	Dry	NMValA	Conv	5/23/2012	0	No	85	ELS
2012	Blackshank Farm, Tifton, GA	Irr	NMValA	Conv	5/23/2012	1	Yes	85	ELS
2012	Marianna, FL	Fung	GA06G	Conv	5/18/2011	2	Yes	70	LLS
2012	Marianna, FL	Fung	NMValA	Conv	5/18/2011	2	Yes	80	LLS
2012	RDC, Tifton, GA	Strip	GA06G	Conv	5/2/2012	3	Yes	60	LLS
2012	RDC, Tifton, GA	Strip	GaVal	Strip	5/2/2012	3	Yes	60	LLS

2013	Attapulgus, GA	Irr	GA06G	Conv	5/22/2013	1	Yes	75	ELS
2013	Attapulgus, GA	Culbreat	GA06G	Conv	5/29/2013	1	Yes	80	ELS
2013	Blackshank Farm, Tifton, GA	Dry	GA06G	Conv	5/29/2013	0	No	80	ELS
2013	Blackshank Farm, Tifton, GA	Irr	GA06G	Conv	6/21/2013	1	Yes	80	ELS
2013	FishPond, Tifton, GA	Dry	GA06G	Conv	5/9/2013	5	No	50	None
2013	Marianna, FL	Fung	GA06G	Conv	6/12/2013	2	Yes	75	LLS
2013	RDC, Tifton, GA	Conv	GA06G	Conv	5/16/2013	3	Yes	65	LLS
2013	RDC, Tifton, GA	Strip	GA06G	Strip	5/16/2013	3	Yes	55	LLS
2014	ABAC Farm, Tifton, GA	Fung	GA06G	Conv	6/3/2014	2	No	65	LLS
2014	ABAC Farm, Tifton, GA	Fung	GA12Y	Conv	6/3/2014	2	No	60	LLS
2014	Attapulgus, GA	Culbreat	GA06G	Conv	5/29/2013	1	Yes	80	None
2014	Attapulgus, GA	Planting	GA06G	Conv	4/28/2014	0	Yes	75	ELS
2014	Attapulgus, GA	Planting	GA06G	Conv	5/14/2014	0	Yes	85	ELS
2014	Attapulgus, GA	Planting	GA06G	Conv	6/6/2014	0	Yes	90	ELS
2014	Blackshank Farm, Tifton, GA	Irr	GA06G	Conv	6/8/2014	1	Yes	80	ELS
2014	Blackshank Farm, Tifton, GA	Dry	GA06G	Conv	6/10/2014	0	Yes	90	ELS
2014	Citra, FL	None	GA06G	Conv	4/23/2014	0	Yes	75	ELS
2014	Citra, FL	Long	GA06G	Conv	4/23/2014	3	Yes	55	ELS
2014	Citra, FL	PD1	GA06G	Conv	4/30/2014	0	Yes	75	ELS
2014	Citra, FL	PD2	GA06G	Conv	4/30/2014	0	Yes	75	ELS
2014	Citra, FL	None	GA06G	Conv	5/14/2014	0	Yes	85	ELS
2014	Citra, FL	Long	GA06G	Conv	5/14/2014	3	Yes	65	ELS
2014	Citra, FL	None	GA06G	Conv	6/4/2014	0	Yes	90	ELS
2014	Citra, FL	Long	GA06G	Conv	6/4/2014	3	Yes	70	ELS
2014	FishPond, Tifton, GA	Dry	GA06G	Conv	6/3/2014	5	No	60	None
2014	Gibbs Farm, Tifton, GA	Kemerait	GA06G	Conv	6/10/2014	0	Yes	90	None
2014	Marianna, FL	Planting	GA06G	Conv	5/22/2013	2	Yes	70	LLS
2014	Marianna, FL	Planting	GA06G	Conv	4/28/2014	2	Yes	60	LLS
2014	Marianna, FL	Planting	GA06G	Conv	6/1/2014	2	Yes	75	LLS
2014	Marianna, FL	LatePlan	GA06G	Conv	6/4/2014	2	Yes	75	LLS
2014	NESPAL, Tifton, GA	Fung	GA06G	Conv	5/21/2014	0	Yes	85	None
2014	NESPAL, Tifton, GA	Fung	GA12Y	Conv	5/21/2014	0	Yes	80	None

2014	Plains, GA	Fung	GA06G	Conv	5/8/2014	0	Yes	80	None
2014	Ponder Farm, Tifton, GA	USDA	GA06G	Conv	6/10/2014	1	Yes	80	ELS
2014	Quincy, FL	5_8Plant	GA06G	Conv	5/8/2014	0	Yes	80	LLS
2014	Railroad Field, Tifton, GA	Tubbs	GA06G	Conv	6/3/2014	5	No	60	LLS
2014	RDC, Tifton, GA	Conv	GA06G	Conv	5/13/2014	3	Yes	65	LLS
2014	RDC, Tifton, GA	Strip	GA06G	Strip	5/13/2014	3	Yes	55	LLS
2014	Strippling, GA	Dry	GA06G	Conv	5/20/2010	2	No	60	LLS
2014	Strippling, GA	Irr	GA06G	Conv	5/20/2010	2	Yes	70	LLS
2014	Strippling, GA	Dry	GA12Y	Conv	5/20/2010	2	No	55	LLS
2014	Strippling, GA	Irr	GA12Y	Conv	5/20/2010	2	Yes	65	LLS
2015	ABAC Farm, Tifton, GA	Fungicid	GA06G	Conv	5/12/2015	2	Yes	60	LLS
2015	ABAC Farm, Tifton, GA	Fungicid	GA12Y	Conv	5/12/2015	2	Yes	55	LLS
2015	Attapulgus, GA	Culbreat	Bailey	Conv	6/4/2014	1	Yes	75	None
2015	Attapulgus, GA	Culbreat	FL511	Conv	6/4/2014	1	Yes	80	None
2015	Attapulgus, GA	Planting	GA06G	Conv	5/21/2014	0	Yes	85	ELS
2015	Attapulgus, GA	Planting	GA06G	Conv	6/4/2014	0	Yes	90	ELS
2015	Attapulgus, GA	Culbreat	GA06G	Conv	6/4/2014	1	Yes	80	None
2015	Attapulgus, GA	Planting	GA06G	Conv	4/27/2015	0	Yes	75	ELS
2015	Attapulgus, GA	Irr	GA06G	Conv	5/5/2015	0	Yes	80	ELS
2015	Blackshank Farm, Tifton, GA	Irr	FL511	Conv	5/19/2015	1	Yes	75	ELS
2015	Blackshank Farm, Tifton, GA	Culbreat	FL511	Conv	8/11/2015	1	Yes	80	ELS
2015	Blackshank Farm, Tifton, GA	Irr	GA06G	Conv	6/6/2014	1	Yes	80	ELS
2015	Expo, Moultrie, GA	Long	GA06G	Conv	5/5/2015	5	Yes	60	None
2015	Expo, Moultrie, GA	HayField	GA06G	Conv	5/11/2015	5	Yes	65	None
2015	Expo, Moultrie, GA	Short	GA06G	Conv	5/11/2015	5	Yes	65	None
2015	FishPond, Tifton, GA	Border	GA06G	Conv	5/27/2015	5	Yes	70	None
2015	Gibbs Farm, Tifton, GA	DrBranch	GA06G	Conv	4/10/2015	2	Yes	60	None
2015	Jones Farm, Tifton, GA	Field25	GA06G	Conv	5/7/2015	5	Yes	50	None
2015	Lang Farm, Tifton, GA	Brennema	GA06G	Conv	5/22/2013	0	Yes	85	ELS
2015	Lang Farm, Tifton, GA	Tubbs	GA06G	Conv	5/13/2014	5	Yes	65	None
2015	Lang Farm, Tifton, GA	Abney	GA06G	Conv	6/8/2014	4	Yes	70	None
2015	Lang Farm, Tifton, GA	Lateral	GA06G	Conv	5/11/2015	2	Yes	70	None

2015	Lang Farm, Tifton, GA	Culbreat	GA06G	Conv	5/11/2015	5	Yes	65	None
2015	Marianna, FL	Planting	GA06G	Conv	5/14/2014	2	Yes	70	LLS
2015	Marianna, FL	Fungicid	GA06G	Conv	5/14/2014	2	Yes	70	LLS
2015	Marianna, FL	Planting	GA06G	Conv	5/5/2015	2	Yes	65	LLS
2015	Marianna, FL	Planting	GA06G	Conv	6/2/2015	2	Yes	75	LLS
2015	Midville, GA	Kemerait	GA06G	Conv	5/14/2014	3	Yes	65	None
2015	Midville, GA	HayField	GA06G	Conv	5/7/2015	5	Yes	50	None
2015	Plains, GA	Blueberr	GA06G	Conv	5/22/2013	0	Yes	85	ELS
2015	Plains, GA	Culbreat	GA06G	Conv	5/22/2013	3	Yes	65	None
2015	Plains, GA	Irr	GA06G	Conv	5/22/2013	3	Yes	65	LLS
2015	Plains, GA	Dry	GA06G	Conv	5/22/2013	5	No	45	None
2015	Ponder Farm, Tifton, GA	Paulk	GA06G	Conv	6/1/2014	2	Yes	75	None
2015	Ponder Farm, Tifton, GA	Paulk	GA09B	Conv	6/1/2014	2	Yes	80	None
2015	Quincy	Second	GA06G	Conv	6/8/2014	0	Yes	90	LLS
2015	Quincy	First	GA06G	Conv	5/1/2015	0	Yes	80	LLS
2015	RDC, Tifton, GA	Lateral	FL107	Conv	5/15/2015	4	Yes	70	LLS
2015	RDC, Tifton, GA	Conv	GA06G	Conv	5/11/2015	3	Yes	65	LLS
2015	RDC, Tifton, GA	Strip	GA06G	Strip	5/11/2015	3	Yes	55	LLS
2015	Strippling, GA	CottonFi	FL511	Conv	5/18/2011	2	Yes	70	LLS
2015	Strippling, GA	CottonFi	FL727	Conv	5/18/2011	2	Yes	65	LLS
2015	Strippling, GA	CottonFi	GA06G	Conv	5/18/2011	2	Yes	70	LLS
2015	Strippling, GA	CottonFi	GA12Y	Conv	5/18/2011	2	Yes	65	LLS
2016	Attapulgus, GA	Irr	GA06G	Conv	4/23/2016	2	Yes	60	ELS
2016	Attapulgus, GA	Irr	GA06G	Conv	4/23/2016	2	Yes	60	ELS
2016	Attapulgus, GA	Irr	GA06G	Conv	4/23/2016	2	Yes	60	ELS
2016	Blackshank Farm, Tifton, GA	Irr	GA12Y	Conv	6/12/2016	1	Yes	75	ELS
2016	FireTower Field, Tifton, GA	Variety	C99R	Conv	5/15/2015	0	Yes	80	Both
2016	FireTower Field, Tifton, GA	Variety	C99R	Conv	6/10/2016	0	Yes	85	Both
2016	FireTower Field, Tifton, GA	Variety	Carver	Conv	5/15/2015	0	Yes	95	Both
2016	FireTower Field, Tifton, GA	Variety	Carver	Conv	6/10/2016	0	Yes	100	Both
2016	FireTower Field, Tifton, GA	Variety	FloRnr	Conv	5/15/2015	0	Yes	95	Both
2016	FireTower Field, Tifton, GA	Variety	FloRnr	Conv	6/10/2016	0	Yes	100	Both

2016	FireTower Field, Tifton, GA	Variety	GA06G	Conv	5/15/2015	0	Yes	85	Both
2016	FireTower Field, Tifton, GA	Variety	GA06G	Conv	6/10/2016	0	Yes	90	Both
2016	FireTower Field, Tifton, GA	Variety	GaGreen	Conv	5/15/2015	0	Yes	85	Both
2016	FireTower Field, Tifton, GA	Variety	GaGreen	Conv	6/10/2016	0	Yes	90	Both
2016	FireTower Field, Tifton, GA	Variety	GaVal	Conv	5/15/2015	0	Yes	95	Both
2016	FireTower Field, Tifton, GA	Variety	GaVal	Conv	6/10/2016	0	Yes	100	Both
2016	FireTower Field, Tifton, GA	Variety	NMValC	Conv	5/15/2015	0	Yes	95	Both
2016	FireTower Field, Tifton, GA	Variety	NMValC	Conv	6/10/2016	0	Yes	100	Both
2016	FireTower Field, Tifton, GA	Variety	SthrnR	Conv	5/15/2015	0	Yes	80	Both
2016	FireTower Field, Tifton, GA	Variety	SthrnR	Conv	6/10/2016	0	Yes	85	Both
2016	Lang Farm, Tifton, GA	Fungicide	GA06G	Conv	6/2/2015	2	Yes	75	None
2016	Plains, GA	Fungicide	GA06G	Conv	5/17/2010	3	Yes	65	None
2016	Ponder Farm, Tifton, GA	Variety	C99R	Conv	5/13/2014	0	Yes	80	Both
2016	Ponder Farm, Tifton, GA	Variety	C99R	Conv	6/10/2016	0	Yes	85	Both
2016	Ponder Farm, Tifton, GA	Variety	Carver	Conv	5/13/2014	0	Yes	95	Both
2016	Ponder Farm, Tifton, GA	Variety	Carver	Conv	6/10/2016	0	Yes	100	Both
2016	Ponder Farm, Tifton, GA	Variety	FloRnr	Conv	5/13/2014	0	Yes	95	Both
2016	Ponder Farm, Tifton, GA	Variety	FloRnr	Conv	6/10/2016	0	Yes	100	Both
2016	Ponder Farm, Tifton, GA	Variety	GA06G	Conv	5/13/2014	0	Yes	85	Both
2016	Ponder Farm, Tifton, GA	Variety	GA06G	Conv	6/10/2016	0	Yes	90	Both
2016	Ponder Farm, Tifton, GA	Variety	GaGreen	Conv	5/13/2014	0	Yes	85	Both
2016	Ponder Farm, Tifton, GA	Variety	GaGreen	Conv	6/10/2016	0	Yes	90	Both
2016	Ponder Farm, Tifton, GA	Variety	GaVal	Conv	5/13/2014	0	Yes	95	Both
2016	Ponder Farm, Tifton, GA	Variety	GaVal	Conv	6/10/2016	0	Yes	100	Both
2016	Ponder Farm, Tifton, GA	Variety	NMValC	Conv	5/13/2014	0	Yes	95	Both
2016	Ponder Farm, Tifton, GA	Variety	NMValC	Conv	6/10/2016	0	Yes	100	Both
2016	Ponder Farm, Tifton, GA	Variety	SthrnR	Conv	5/13/2014	0	Yes	80	Both
2016	Ponder Farm, Tifton, GA	Variety	SthrnR	Conv	6/10/2016	0	Yes	85	Both
2016	RDC, Tifton, GA	Strip	GA06G	Strip	5/17/2010	3	Yes	55	LLS
2016	Rigdon Farm, Tifton GA	MidMay	11J	Conv	5/16/2013	3	Yes	65	None
2016	Rigdon Farm, Tifton GA	BrianBre	11J	Conv	5/7/2015	0	Yes	80	ELS
2016	Rigdon Farm, Tifton GA	Culbreat	FL511	Conv	5/26/2010	1	Yes	75	None

2016	Rigdon Farm, Tifton GA	MidMay	GA06G	Conv	5/16/2013	3	Yes	65	None
2016	Rigdon Farm, Tifton GA	BrianBre	GA06G	Conv	5/7/2015	0	Yes	80	ELS
2016	Rigdon Farm, Tifton GA	MidApril	GA06G	Conv	4/11/2016	3	Yes	55	None
2016	Rigdon Farm, Tifton GA	MidMay	GA12Y	Conv	5/16/2013	3	Yes	60	None
2016	Rigdon Farm, Tifton GA	BrianBre	GA12Y	Conv	5/7/2015	0	Yes	75	ELS

CHAPTER 9

EFFECT OF LOW-INPUT FUNGICIDE PROGRAMS ON REDUCING FOLIAR DISEASES AND YIELD LOSS ON RUNNER AND VALENCIA TYPE PEANUTS IN HAITI[1]

[1]Fulmer, A.M., Kemerait, R.C., Brenneman, T.B., Scherm, H., Cantonwine, E.G., Culbreath, A.K., and Stevenson, K.L. 2017. To be submitted to *Plant Disease*.

ABSTRACT

Peanut yields in Haiti are often severely reduced due to peanut rust and late leaf spot, (caused by *Puccinia arachidis* and *Cercosporidium personatum*, respectively). Multiple field trials were conducted from the spring of 2015 to winter of 2017 in the North and Central Plateau regions of Haiti to evaluate the effectiveness of low-input fungicide programs and improved peanut cultivars from the United States to reduce disease intensity and increase yield. Separate trials were conducted for runner (local Haitian runner and the variety Georgia-06G) and Valencia market types (local Haitian Valencia and the variety New Mexico Valencia A). Fungicide regimes for runner types ranged from two to six applications of tebuconazole + chlorothalonil, with initial sprays ranging from 30-60 days after planting (DAP). Valencia types received one to four applications with initial sprays ranging from 30 to 45 DAP. Application intervals for both market types ranged from 14 to 28 days. In general, disease severity and yield loss increased with decreasing fungicide applications. For runner types, two applications made at 60 and 88 DAP significantly reduced leaf spot severity and improved yield; four applications were comparable to six, but generally not significantly different from three. For Valencia types, at least two applications were generally required to significantly reduce rust and leaf spot and increase yield; regardless of timing, three or more applications gave the highest yields. For both market types, the number of applications appeared to be more important than particular timings. Compared with the local Haitian runner, Georgia-06G had less late leaf spot, higher rust severity and at least 500 kg/ha higher yield in every trial. Disease severity was similar for both Valencia types, but the local Valencia out-yielded the New Mexico Valencia A by > 400 kg/ha in both trials conducted in the Central Plateau, but yields did not differ in the North. Percent yield loss averaged 83 and 43% for the local Haitian runner and Georgia-06G, but was reduced by 64 and 43%, respectively, by recovering the pods from the soil that were lost when uprooting the plants at harvest. Percent yield loss for the local Haitian Valencia and New Mexico Valencia A ranged from 28 to 34%. These results demonstrate that late leaf spot and rust are major yield limiting factors on local and improved runner and Valencia peanut varieties in Haiti, but that these diseases can be effectively managed with minimum fungicide inputs.

INTRODUCTION

Peanut has been cultivated in Haiti since pre-Columbian times and continues to be an important source of nutrition and a cash crop for smallholder farmers in the present day (Hammons, 1982; Nelson et al., 2003). Peanut production represents less than 3% of the total agricultural land use in Haiti and yields are extremely low, averaging 870.9 kg/ha (FAOSTAT, 2016). Similar to other agroecosystems in the tropics (Subrahmanyam et al., 1997), peanut production in Haiti is characterized by rain-fed systems on small parcels of land (<1 ha) with very limited inputs (Kostandini et al., 2017). Soil preparation, planting, weeding and harvesting is almost always done by hand. Peanut is grown throughout Haiti, but the areas of highest concentrated production are in the Northeast and the Central Plateau regions during the main rainy season (April to June) (Kostandini et al., 2017). In the Northeast, a second, more sporadic rainy season generally occurs from October to November which often allows for a second crop during the winter months. The local Haitian runner peanut is mainly planted in the Northeast and the local Haitian Valencia is planted in the Central Plateau (Kostandini et al., 2017). The runner requires 120 to 130 days to reach maturity and the Valencia requires 80 to 90 days. Both market types are local landraces of unknown origin.

A number of issues limit the quality and quantity of peanuts produced in Haiti. Quality issues are related to the widespread distribution of *Aspergillus flavus*, which often results in aflatoxin concentrations ranging from 100 to over a 1000 ppb in locally produced Haitian peanut butter (Filbert and Brown, 2012; Schwartzbord and Brown, 2015). Production constraints include poor soil fertility (Bargout and Raizada, 2013; Nelson et al., 2003), variable seed quality (Nelson et al., 2003) and foliar diseases (Fulmer et al., 2014; Fulmer et al., 2012). Peanut rust caused by *Puccinia arachidis* and early and late leaf spot caused by *Cercospora arachidicola* and *Cercosporidium personatum*, respectively, often result in the complete defoliation of the peanut plant (Fulmer et al., 2012). Due to limited studies, exact yield losses in Haiti caused by these diseases are unknown, but in other tropical areas, 50 to 86% yield losses have been reported (Subrahmanyam et al., 1997). Because there is a general lack of knowledge about these diseases and necessary resources for control, most growers do not manage foliar diseases in Haiti. Many Haitian

farmers are unaware of the negative effects that foliar diseases have on yield, and often confuse complete defoliation caused by fungi as a normal indication of maturity. As a result, the peanut crop is often harvested prior to reaching optimum maturity.

Tactics for managing rust and leaf spot in tropical, developing countries include cultural practices to reduce the initial inoculum such as destruction of volunteer peanut plants, removal of infested peanut residue, and crop rotation (McDonald et al., 1985; Subrahmanyam et al., 1997). In Haiti, while livestock are often allowed to graze on the peanut residue after harvest, most growers do not practice rotation due to limited land holdings. Furthermore, the efficacy of these tactics is uncertain due to the close proximity of neighboring fields in small landholding systems (McDonald et al., 1985; Subrahmanyam et al., 1997). Later planting dates are also recommended, but most growers in rain-fed systems do not generally have flexible planting dates (Subrahmanyam et al., 1997). Disease resistant varieties developed by the International Crops Research Institute for the Semi-Arid-Tropics (ICRISAT), the University of Georgia, New Mexico State University and the University of Florida have been evaluated in Haiti, and some have demonstrated the potential for reducing disease and increasing yields (Fulmer et al., 2012). However, a successful variety for widespread adoption in Haiti has yet to be identified.

Studies in other tropical countries have demonstrated that limited fungicide applications can significantly increase yield and potentially prove economically advantageous in some situations (Naab et al., 2009b; Naab et al., 2009a; Naab et al., 2009c; Naab et al., 2005). Studies in Malawi have shown that one or two applications of chlorothalonil can be economically advantageous for controlling early leaf spot and increasing yield in situations with normal rainfall (Subrahmanyam and Hildebrand, 1997). In West Africa, a wide range of fungicide timings and intervals have been evaluated for control of early and late leaf spot and rust for short- (<100 days) and long- (>120) maturing varieties (Waliyar et al., 2000). While results showed that two or three fungicide applications significantly reduced disease and increased net gains for all varieties, but the benefits depend on application at an appropriate time (Waliyar et al., 2000).

Haiti was selected as part of the USAID Feed the Future program to increase food security, poverty and malnutrition in developing countries (PMIL, 2017). A peanut-based Ready-to-Use Therapeutic Food (RUTF) has been accepted by the WHO as the gold standard treatment of Severe Acute Malnutrition (WHO, 2017) and has been proven successful and accepted for use with Haitian children (Iannotti et al., 2015). Meds and Foods for Kids (MFK), a non-profit organization, has been making Medika Mamba, the local name for RUTF, in Haiti for over 10 years (MFK, 2017). In 2012 a new factory was constructed with the capacity to produce large volumes of RUTF (MFK, 2017). As part of effort to develop the Haitian economy, the vision of MFK is to purchase high quality, locally grown peanuts for production of RUTF. However, low quality and quantity of yields and high market prices limit the quantity of peanuts that MFK is able to source locally.

In 2014, Acceso Peanut Enterprise Corporation, a private, for-profit company, aided by the Clinton Guistra Enterprise Partnership, was established in Haiti. Designed to encompass the entire peanut supply chain, Acceso not only provides growers in their program with basic inputs, e.g., seed, tillage, fertilizer, and fungicides on credit, but other services such as aflatoxin testing, sorting, and competitive purchase pricing at a fixed rate. These locally produced peanuts are directly sold by Acceso to Haitian businesses in need of high quality peanuts in small or large quantities, including MFK and Partners in Health. Some growers in the Acceso program currently make one or two fungicide applications at varying timings and intervals, but the efficacy of these treatments is unknown. Adoption of fungicide is not yet widespread due to the lack of understanding of impact and return on investment and Acceso considers the current limited investment by farmers a result of inconsistent improvements of productivity from the technology package.

Given the new opportunities for peanut growers in Haiti to capitalize on the demand for locally produced peanuts through increased productivity, there is a need for research-based recommendations for the technology package, including use of fungicides. However, before incorporating fungicides into their production system, there is a need to determine an appropriate, low-input, fungicide regime that would result in consistently profitable utilization of this technology. Because of economic and practical

constraints, the number of fungicide applications must be reduced to the absolute minimum. Critical to this concept is a better understanding of the general development and severity of foliar diseases and their relation to yield loss on the different market types grown in Haiti. Therefore, as part of the USAID supported Peanut and Mycotoxin Innovation Lab (Project UF-204) the objectives of these studies were to 1) determine the optimum number and timing of fungicide applications on runner and Valencia peanut market types, 2) evaluate the suitability of improved cultivars from the United States, and 3) characterize the behavior of foliar diseases and quantify the associated yield loss.

MATERIALS AND METHODS

Field sites. Field trials were conducted in research plots located in the Central Plateau and in the North of Haiti. In the Central Plateau, research plots were conducted at the Acceso research farm located in Coupe Gorge (part of the Mirebalais commune) (18°50'21.05"N latitude, 72° 3'29.33"W longitude), Haiti in the spring of 2015 and 2016. In the North, research was conducted at the Meds and Food for Kids factory located outside of Cap Haitien (Quartier Morin), Haiti (19°41'32.17"N latitude, 72° 9'16.91"W longitude) in 2015, 2016 and 2017. All fields used in these studies were located within a ~150-km radius and had a previous history of peanut. Prior to planting, fields had either been fallow or planted to sorghum, but all had been planted to peanut within the previous 6 months. Throughout the season, contiguous fields always hosted older planted peanuts as part of additional unsprayed research trials, thus ensuring a perpetual inoculum source. The soil type in fields used in the Central Plateau was a sandy clay loam comprised of 25% sand, 25% silt, and 50% clay with a pH of 7.1 ($CaCl2_2$), and calcium levels were over 3,000 kg/ha (unpublished data). At MFK, the average field is 42% sand, 27% silt, and 30% clay with a pH of 7.4, and calcium levels were also over 3,000 kg/ha (unpublished data).

Four trials with runner market type peanuts were conducted at the MFK research site from 2015 to 2017. All trials were laid out in a split plot design with four replications. Variety was the main plot and was planted to the local Haitian runner or Georgia-06G, the predominant cultivar planted in the southeastern United States. Fungicide treatment was the subplot and consisted of six different application regimes plus an untreated check (see Table 9.1 for details). Dates from planting to harvest for each trial

were as follows: 23 March to 13 August 2015; 9 November 2015 to 8 March, 2016; 19 February to 23 June 2016; 16 October 2016 to 19 February 2017.

Two trials with Valencia market type peanuts were conducted in the Central Plateau at the Acceso research site in the spring of 2015 and 2016, and two trials were conducted in the North at the MFK research site in the spring and fall of 2016. All trials were laid out in a split plot design with four replications. Variety was the main plot and was planted to the local Haitian Valencia or New Mexico Valencia A, an improved cultivar planted in the southwestern United States. Fungicide treatment was the subplot and consisted of six different application regimes plus an untreated check (see Table 9.1 for details). Dates from planting to harvest in the Central Plateau for each trial were as follows: 31 March to 30 June 2015; 8 April to 16 July 2016. Dates from planting to harvest in North for each trial were as follows: 14 March to 16 June 2015; 29 August to 28 November, 2016.

For all experiments, fungicide treatments consisted of a combination of tebuconazole (0.23 kg/ha) + chlorothalonil (0.84 a.i. kg/ha) (Muscle ADV, Sipcam Agro USA, Durham, NC) sprayed at the appropriate timings as shown in Table 9.1. Applications were in the morning (after the dew had dried from the foliage) with a hand pumped backpack sprayer calibrated to deliver 188 liters per hectare. The boom had two nozzles (8002VS flat tip nozzles from Teejet Technologies, Springfield, IL) spaced 30.4 cm apart.

For all trials, plots were 1.2 m wide and 4.6 m in length, and consisted of two rows of peanuts bordered by a single untreated row of peanuts. Blocks were separated by a 1.5-m alley. Target rows were first marked with stakes and string, and hoes were used to create furrows with an average depth of 3.8 cm. Plots were planted by hand and uniformity was ensured by placing PVC pipes (marked with the appropriate seeding rate) within the furrow while planting. Runner trials were planted at 3 seed per 30.5 cm in the first two trials and 6 seed per 30.5 cm in the last two trials. Valencia trials were planted at a rate of 3 seed per 30.5 cm in the Central Plateau and 6 seed per 30.5 cm in the North. Prior to planting, seeds were treated with azoxystrobin, fludioxonil and mefenoxam (Dynasty PD, Syngenta Crop Protection, Greensboro, NC) at a rate of 85 g of product per 45.4 kg of seed. Fields were disked two to three times

prior to planting, and rototilled within 2 days prior to planting. A few days prior to planting, fields in the Central Plateau were fertilized with diammonium phosphate at a rate of 45 kg/ha; at MFK, the same rate of diammonium phosphate was used in all trials conducted in 2015. All other trials were fertilized with 20-20-10 at a rate of 67 kg/ha. Manual weeding occurred at 4, 6 and 8 weeks after planting. A hoe was used between rows and weeds closest to the peanuts were physically removed by hand. No herbicides or insecticides were applied before or after the plots were established. At the MFK research site, plots were irrigated with a rotary sprinkler system as needed (twice a week in the absence of rain) with approximately 1.3 cm of water per irrigation event. At the Acceso research site, plots were irrigated with flood irrigation similar to the local grower standard. Weather data was recorded at the MFK research site with a Decagon ECRN-50 rain gauge (Decagon Devices, Inc., Pullman, WA).

Data collection. Two to three weeks after planting, stand counts were made for each plot, and across trials averaged > 90 % for all varieties. Beginning approximately 30 days after planting (DAP) and continued every two weeks, five leaves per plot were arbitrarily selected for destructive sampling from the lower canopy of the peanut plants in each plot. Leaves were returned to the lab and the number of present and missing leaflets, and the number of early and late leaf spot lesions per leaflet were counted. The percentage of area each leaflet covered with rust pustules was visually estimated on a 0 to 100 % scale. For each assessment date, incidence of each disease was calculated by dividing the number of leaflets with one or more lesions/pustules by the total number of leaflets collected for each plot. Area under the disease progress curves (AUDPC) were calculated for each leaf spot and rust based on the mean number of lesions per leaflet, and the mean percent rust severity per leaflet, respectively. AUDPC values were standardized (stAUDPC) by dividing AUDPC values by the number of days between the first and final evaluation.

In Valencia trials in the Central Plateau, destructive samples were collected in the spring of 2015 at 70 DAP, and in the spring of 2016 at 69 DAP. In runner trials, samples were collected at 35, 58, 78, 91 and 121 DAP in the spring of 2015; in the fall/winter of 2015/2016 samples were collected at 78, 94, 104, 115 DAP. In the spring of 2016, samples were collected at 30, 45, 60, 73, 87, 101 and 115 DAP. In the

fall/winter of 2016/2017, samples were taken at 30, 45, 60, 75, 90 and 105 DAP. In Valencia trials conducted in the North in the spring and fall of 2016, samples were taken at 30, 45, 60, 75 and 90 DAP. In Valencia trials conducted in the Central Plateau in the spring of 2015, samples were taken at 28, 42, 56, and 70 DAP, and at 68 DAP in the spring of 2016.

Final leaf spot and rust severity assessments were made immediately prior to digging. Leaf spot severity was assessed with the Florida 1 to 10 scale, where 1= no leaf spot; 2 = very few lesions on the leaves, none on the upper canopy; 3 = few lesions on the leaves, very few on the upper canopy; 4 = some lesions with more on the upper canopy, approximately 5% defoliation; 5 = lesions noticeable even on upper canopy, approximately 20% defoliation; 6 = lesions numerous and very evident on upper canopy, approximately 50% defoliation; 7 = lesions numerous on upper canopy, approximately 75% defoliation; 8 = upper canopy covered with lesions, approximately 90% defoliation; 9 = very few leaves remaining and those covered with lesions, some plants completely defoliated; and 10 = plants completely defoliated and killed by leafspot (Chiteka et al., 1988).

Rust severity was assessed using a modified 1 to 9 scale where 1 = no disease; 2 = pustules sparsely distributed, largely on lower leaves; 1-5% severity; 3 = many pustules on lower leaves, necrosis evident; very few pustules on middle leaves; 6-10 % severity; 4 = numerous pustules on lower and middle leaves; severe necrosis on lower leaves; 11-20% severity; 5 = severe necrosis of lower and middle leaves; pustules may be present on top leaves, but less severe; 21-30% severity; 6 = extensive damage to lower leaves; middle leaves necrotic, with dense distribution of pustules; pustules on top leaves; 31-40% severity; 7 = severe damage to lower and middle leaves; pustules densely distributed on top leaves; 41-60% severity; 8 = 100% damage to lower and middle leaves; pustules on top leaves, which are severely necrotic; 61-80% severity; 9 = almost all leaves withered; bare stems seen; 81-100% severity (Subrahmanyam et al., 1995).

Peanuts were manually harvested by first pulling the entire plant from the ground and removing all the attached pods. In runner trials, this was followed by excavating the soil on both sides of each row (approximately 30 cm on each side) with a spade and breaking it into fine clumps. The soil was then

filtered through by hand to recover the remaining pods left in the ground. For each plot, pods picked from the plants and those recovered from the soil were separately placed in large, green, mesh cabbage bags (Cady Bag Company, LLC., Pearson, GA). For Valencia trials, only pods remaining on the surface of the soil were recovered, and were included in the same bags as the pods picked from the plants; the exception was the trial at MFK in the fall of 2016, in which the harvest method was the same as described for the runner market types. For all market types, pods were washed in water immediately after harvest to remove any remaining soil, and then placed on a large concrete pad to dry in the sun. The bagged pods were allowed to dry for a minimum of 3 days and were moved under a shelter each night. After drying, bags were weighed, and immediately afterwards, a 100-pod sample was shelled to obtain the moisture content of the kernels. Final weights were adjusted to 10% pod moisture. A post-harvest test was made with the same 100 pod sample to evaluate the percentage of sound mature kernels (SMK). Percent SMK was calculated by dividing the weight of the sound mature kernels by the total weight of the unshelled sample. For each trial, Romer Agra-Strip test kits (Romer Labs, Getzersdorf, Austria) were used to test for aflatoxin (>10 ppb) levels from representative samples taken from plots treated with 6 or 4 fungicide applications for runner and Valencia market type peanuts, respectively, and from the non-fungicide treated plots of both peanut market types.

Statistical analysis. Final severity of leaf spot and rust, yield and %SMK were subjected to analysis of variance with PROC GLIMMIX (SAS 9.4 Institute, Cary, NC). Due to differences in seed spacing between trials, and to better understand the effect of each factor in a given environment, each trial was analyzed separately. For all trials, the model included variety and fungicide treatment considered as fixed effects, with replication and replication × variety as random effects. In all analyses the Kenward-Roger option was used to adjust the denominator degrees of freedom, and differences in the least square means were tested by Tukey's multiple comparisons test. When data violated the assumptions of normality, transformations were used. SMK was arcsin square root transformed and the natural log transformation was used for all yield variables. Back-transformed means of all transformed variables are presented in the results.

To better understand the yield loss caused by leaf spot and rust in untreated peanuts, yields were converted to percent yield loss, and the means and standard errors were calculated for each trial. This was calculated as the difference between plots sprayed six times (runner types) or four times (Valencia types) and the corresponding untreated plots. Data from across trials were used to explore the relationships between leaf spot/rust and percent yield loss for each runner variety with regression analysis (linear and quadratic models), in which case, percent yield loss was calculated for each treatment [100 × (6-spray yield – treatment yield) / 6-spray yield] for each replication in each trial.

RESULTS

Runner trials. Late leaf spot was the predominant (responsible for >99% of lesions counted) leaf spot disease in all trials conducted at MFK (data not shown). The development of rust was similar on both runner varieties, but late leaf spot developed much more rapidly on the local Haitian runner (Fig. 9.1). The onset of rust occurred from 45 to 60 DAP in most trials, but in the spring of 2016, it was already at approximately 0.4 incidence at 30 DAP (Fig. 9.1). In almost all trials, the onset of rust occurred 10 to 20 days prior to late leaf spot (Fig. 9.1). Differences in rainfall were likely responsible for some of the variation in the onset and development of each disease (Table 9.2). Although these fields were irrigated, it generally did not provide prolonged leaf wetness due to the rapid drying in the sun. In runner trials, rust and leaf spot onset tended to be earlier in the spring than in the winter trials, but did not always relate to differences in rainfall during the first 60 DAP. The least amount of rainfall during 30-60 DAP occurred in the winter 2015/2016 trial (Table 9.2), and corresponded to a delay in the development of both diseases (Fig. 9.1). However, intense rainfall within the first 30 and 60 days does not fully explain the onset of each disease. The most intense rainfall occurred in the winter of 2016/2017 (Table 9.2), but rust was not detected until 60 DAP (Table 9.2 and Fig. 9.1). Haiti tends to have cooler temperatures during the winter months, and this could have been responsible for some of the variability observed in these trials. Nevertheless, there was heavy leaf spot and rust pressure in all trials conducted at MFK, but late leaf spot was most severe on the local Haitian runner, and rust was more severe on Georgia-06G compare with the local Haitian runner (Table 9.3).

Because the standardized AUDPC of leaf spot and rust resulted in identical treatment separations as those made with the Florida 1-10 scale and the Rust 1-9 scale, only data from the latter two assessment methods are presented here. There were no interactions between variety and fungicide treatment for any of the variables assessed in this study (Table 9.4). Fungicide treatment had a significant effect on leaf spot and rust severity and yield (Table 9.4). Overall, leaf spot and rust severity increased with decreasing fungicide applications (Fig. 9.2), and yield tended to increase with increasing fungicide applications (Fig. 9.3). All fungicide treatments resulted in significantly lower disease, and in most cases, significantly higher yields compared to the untreated control (Figs. 9.2 and 9.3). With the exception of the winter 2016/2017 trial, severity of foliar diseases was lowest and yields were highest in plots treated with the six-spray program (Figs. 9.2 and 9.3). In almost every case, 3 or 4 applications offered comparable levels of control to plots sprayed with 6 applications (Figs. 9.2 and 9.3). Four applications tended to be numerically better than 3 applications and significantly better than 2 applications (Figs. 9.2 and 9.3). There were generally no significant differences in disease severity or yield between 4- and 3-spray programs initiated at 37 or 45 DAP (Figs. 9.2 and 9.3). Two applications made 60 and 88 DAP generally did not perform as well as 3 applications, but in almost every case significantly reduced leaf spot and rust severity and increased yield when compared to the untreated check (Figs. 9.2 and 9.3). Percent SMK did not differ ($P > 0.05$) between fungicide treatments in any of the trials conducted in these studies (data not shown).

With the exception of the winter 2015/2016 trial, variety had a significant effect on late leaf spot and rust severity in every trial (Table 9.4). In all cases, late leaf spot severity was numerically, and often significantly higher in the local Haitian runner than Georgia-06G, but the rust severity was higher in the latter (Tables 9.4 and 9.5). In all trials, pod yield from the plant alone was significantly higher for Georgia-06G (Tables 9.4 and 9.5). When the pods recovered from the soil were combined with those from the plant, yield differences between the two varieties was substantially reduced, resulting in only one trial with significant differences between the two varieties (Tables 9.4 and 9.5). Overall, SMK values from pods on the plant or plant + soil, were similar for both varieties, but was significantly lower on the

local Haitian runner in both the 2015 and 2016 spring trials (Table 9.5). In untreated plots, percent yield loss was more consistent for the local Haitian runner compared with Georgia-06G (Table 9.6). Based on pods from the plant in untreated plots, percent yield loss ranged from 75 to 90% and 47 to 85% for the local Haitian runner and Georgia-06, respectively (Table 9.7). When those recovered from the soil were added to those from the plant, there was a decrease in percent yield loss that ranged from 39 to 56% and 26 to 63% for the local Haitian runner and Georgia-06, respectively (Table 9.7). The highest yield loss for Georgia-06G (63%) occurred during the extremely wet 2016/2017 winter season (Tables 9.2 and 9.7).

Across trials and fungicide treatments, there was a significant linear relationship ($R^2 = 0.42$, $P = <0.0001$) between late leaf spot (assessed with the Florida 1-10 scale) and percent yield loss for the local Haitian runner, but there was a slightly lower R^2 for the total pod yield (plant + soil) (Fig. 9.4). The linear relationship for late leaf spot and percent yield loss for Georgia-06G was weak for both the pods from the plant ($R^2 = 0.25$, $P = <0.0001$) and from plant + soil ($R^2 = 0.17$, $P = <0.0001$). For Georgia-06G, the quadratic model provided the best fit for the relationship between rust (assessed with the 1-9 scale) and percent yield loss, but there was a higher R^2 for pods recovered from the plant compared with the combined pod yield from the plant + pods recovered from the soil (Fig. 9.4). For the local runner, neither the linear nor the quadratic model provided a good fit between rust and percent yield loss on pods from the plant or plant + soil, but significant *P*-values indicated a significant relationship ($R^2=0.25$, $P = <0.0001$) and ($R^2=0.26$, $P = <0.0001$), respectively.

Valencia trials. Both leaf spot and rust were present in all Valencia trials, but rust was the most damaging disease (Table 9.3). Late leaf spot was the predominant (>95%) leaf spot disease in all trials conducted at MFK (data not shown). In the Central Plateau, early leaf spot was the predominant leaf spot disease (>70%) in the spring of 2015 but late leaf spot was predominant (>90%) in the spring of 2016 (data not shown). The development of rust and leaf spot was similar on both Valencia varieties, and both diseases had a similar behavior for each trial at the MFK research site (Fig. 9.5). At MFK, final rust and leaf spot severity were high in both trials (Table 9.3). In 2015 at the Central Plateau, rust was present by 30 DAP, and increased to 90% incidence by 60 DAP (Fig. 9.5). However, final severity values were

several points below the highest possible rating indicating that the epidemic did not fully develop (Table 9.3). Overall, disease pressure was lower in 2016 than in 2015 in the Central Plateau (Table 9.3).

Fungicide applications had a significant effect on leaf spot and rust severity and yield in all trials at both locations, and there were no fungicide × variety interactions (Table 9.4). Overall, leaf spot and rust severity increased with decreasing fungicide applications (Fig. 9.6), and yield tended to increase with increasing fungicide applications (Fig. 9.7). In all trials, 2 or more applications resulted in significantly lower rust, compared with the untreated control (Fig. 9.6). At both locations, 3 applications initiated at 45 DAP and timed at 14-day intervals provided similar results to four applications initiated at 30 DAP, and resulted in numerically or statically lower leaf spot and rust severity than 3 applications initiated at 30 days and timed at 21-day intervals or 2 applications (Fig. 9.6). Two applications at 21-day intervals generally provided better disease control, and numerically or statistically higher yields than a single application made at 45 DAP. In the North, all fungicide treatments provided significantly better yield compared with the untreated check, and there were no significant differences among treatments (Fig. 9.7). In the Central Plateau, 4 or 3 applications resulted in the highest yields, but only differed significantly from a single application made at 45 DAP (Fig. 9.7).

Variety had a significant effect on leaf spot at MFK in the spring of 2016, but did not have a significant effect on either disease in any other trial (Tables 9.4 and 9.6). Pod yield (including pods from the plant and from the surface of the soil) was significantly higher on the local Haitian Valencia in the Central Plateau in both trials, but there were no differences between varieties at MFK (Tables 9.4 and 9.6). SMK did not differ between varieties at either location (Tables 9.4 and 9.6). Across years and locations, percent yield loss did not differ between varieties ($P > 0.05$). In the Central Plateau, percent yield loss for the local Haitian Valencia and New Mexico Valencia A was 31 and 34%, and 34 and 28% during 2015 and 2016, respectively. Percent yield loss for the local Haitian Valencia and New Mexico Valencia A was 31 and 34% in 2015, and 34 and 28% in 2016 in the Central Plateau, and 34 and 37% in the spring of 2016 and 48 and 43% in the fall of 2016 at MFK, respectively.

DISCUSSION

To the best of our knowledge, this is the first report to quantify the importance of yield losses caused by leaf spot and rust on runner and Valencia market types in Haiti, and to demonstrate the need to manage these diseases under a wide range of environmental conditions. Furthermore, we found that leaf spot and rust can be effectively managed with low-input fungicide applications. For both market types, our results suggest that the number of applications is generally more important than the specific timings included in these studies. This was evident based on the general inverse relationship between disease severity and the number of fungicide applications and associated yield losses. However, because we did not specifically compare a fixed number of applications with a fixed number of application timings for runner market types, more research would be needed to substantiate this conclusion. We also found that the improved runner variety, Georgia-06G, consistently had higher yields compared to the local Haitian runner, but that the improved Valencia variety, New Mexico Valencia A, offered no advantage compared to the local Haitian Valencia.

A key principle in disease control is to initiate applications prior to or at disease onset. In most of the trials conducted in these studies, the onset of rust occurred from 45 to 60 DAP with leaf spot appearing simultaneously or 10 to 20 days later. Theoretically, then, initial applications in reduced programs should be made around 45 DAP rather than 30 DAP. However, in runner trials, we did not find any consistent difference in disease control or yield in treatments initiated at 37 vs. 45 DAP in either the 3-spray or 4-spray program. The main exception was during the spring of 2016, when four applications beginning at 37 DAP provided less disease control than applications initiated at 45 DAP. This was particularly interesting given that the onset of rust occurred at 30 DAP. This suggests that as long as the initial application is made soon after onset, there is a greater advantage of later season applications for reducing disease severity rather than early season applications, even though rust onset occurs earlier in the season. In Valencia trials, the date of initiation cannot be directly compared due to treatment design. However, three applications initiated at 45 DAP and sprayed at 14-day intervals tended to provide better disease control than three applications initiated at 30 DAP and sprayed at 21-day intervals. However, this

did not generally translate into a yield difference between the treatments. Taken together, our results indicate that there is a degree of flexibility in the timing of the first application across a range of environmental conditions, but less flexibility in the number of applications

The general trend from these studies indicates that the yield benefit from fungicide increases with increasing number of applications (up to 6 for runner market types and up to 4 for Valencia market types), which is similar to previous reports in West Africa (Waliyar et al., 2000). Similarly, studies in the southeastern United States have demonstrated an inverse relationship with leaf spot and yield with incremental delays in the initial application (Culbreath et al., 2006; Shokes et al., 1982). Notwithstanding the obvious impact of fungicide in these studies, we observed a degree of variability within treatments across trials, and because there was not always a clear statistical significant separation between treatments, final treatment recommendations are not straight forward. This is not an unusual circumstance when attempting to discern optimum treatment recommendations. Waliyar et al. (2000) compared all combinations of applications made at 40, 55, 70 and 85 DAP on short- (<100 days) and medium-maturing (>120 days) peanut varieties for control of late leaf spot in West Africa, and found that one or more applications made at a particular timing (actual timing depended on variety and location) could increase yield and net gains. Similar to our studies, they found that the fungicide benefit tended to increase with increasing applications (Waliyar et al., 2000). Their results also showed that the number of possible timings that resulted in positive net gains generally increased with increasing application numbers, regardless of length of variety maturation (Waliyar et al., 2000). The general conclusion from their work was that multiple combinations of 2 or 3 applications were the best choices for both short- and medium-maturing varieties, and many of the best timing combinations included initial applications made at 40 or 55 DAP with 14- to 28-day intervals (Waliyar et al., 2000). Similarly, our results suggest that 3 or 4 applications and 2 or 3 applications at any of the timings evaluated for runner and Valencia market types in Haiti, respectively, would provide yield benefits comparable to the full season program (6 applications for the runner varieties and 4 applications for the Valencia varieties).

Final treatment recommendations must also include practical and economic considerations (Naab et al., 2009c; Waliyar et al., 2000). Most peanut growers in Haiti do not have the equipment to make the applications themselves, and depend on a service provider, such as Acceso, to make the applications for them. Therefore implementing more than 2 or 3 fungicide applications may not be practical due to logistical restraints. A simple cost-benefit analysis is usually helpful in deciphering treatment recommendations (Naab et al., 2009c), but in this case may not be very informative due to the complexity of the situation. Firstly, in our studies, peanuts were grown under best management practices that included optimum soil fertility, seed stands and populations (treated seed planted at optimum seed and row spacings with excellent germination), kept weed free and irrigated on a regular basis, and therefore may not reflect the yields and expected gains from a fungicide application in an actual grower situation in Haiti (Kostandini et al., 2017). While all of these practices are recommended as part of Acceso's technology package, actual yields in grower fields may vary because growers may not fully incorporate the recommended management practices (Kostandini et al., 2017). Another level of complexity results from the instability of purchase price depending on whether a grower attempts to sell peanuts at a potentially higher price on the open market or at a fixed price offered by Acceso and similar marketers. Due to these challenges, the data obtained from this study are currently being used as part of a larger, more in-depth economic analysis conducted by researchers at the University of Georgia in collaboration with Acceso in order to help determine the most appropriate recommendation for growers in Haiti.

Treatment recommendations must also be understood in context of mitigating practices that promote fungicide resistance. Theoretically, if only three applications were made due to logistical or economic constraints, it would be better to spray prior to or at the onset of disease, with subsequent applications made at shorter intervals (Brent, 2007). In these studies, most of the intervals for reduced programs were 21 or 28 days, and both seemed to provide good control given the intensity of the disease pressure. The benefit of the longer interval was likely conferred by combining chlorothalonil with the demethylation inhibitor fungicide, tebuconazole. However, there have been reports of reduced sensitivity to tebuconazole in populations of both the early and late leaf spot pathogens in the United States

(Stevenson and Culbreath, 2006). To the best of our knowledge, there has been no report of tebuconazole resistance in populations of the peanut rust pathogen (*Puccinia arachidis*), but there have been reports of reduced sensitivity to tebuconazole in *Puccinia striiformis* in the UK (Bayles et al., 2000). In Haiti, there are currently few options for different fungicide chemistries other than those used in these studies. Therefore, it will be important to instruct peanut growers of the need for proper rates, good coverage and applications prior to heavy disease pressure as well as to encourage service providers, such as Acceso, to seek to diversify their fungicide chemistries (Brent, 2007).

In these studies, we mimicked local grower practices by sifting through the soil to recover pods that were detached from the plant in the harvesting process. We found that, ½ to ¾ of the peanut pods of the local Haitian runner remained in the soil after pulling the plants from the ground, but that the majority of the pods remained attached to the variety Georgia-06G during the same process. Few pods were left in the soil for the Valencia varieties, likely due to a heavier concentration of pods around the perimeter of the plant base. Therefore, we did not attempt to differentiate the two methods in Valencia trials (except in the fall of 2016 when extremely high rainfall complicated harvesting). The clay content in the soils where these studies were conducted was fairly high, and therefore it was easy for the pods to snap off in the ground when plants were physically pulled from the ground. In modern mechanized harvesting operations, peanuts are inverted with a peanut digger, but are also subject to digging losses, i.e., pods left on/in the surface of the ground due to weakened pegs (Colvin, 2015). In one study, disease-free peanuts were shown to have 500 to 1,500 kg/ha pod loss in soils with heavier clay content (16% clay), and 265 to 319 kg/ha loss in sandier soils (2% clay) (Jackson et al., 2011). Colvin (2015) showed that digging losses could range from 83 to 968 kg/ha with increasing losses with later harvest dates. The cited studies were conducted with little or no leaf spot pressure, and therefore it is likely that even higher digging losses could occur under extreme disease pressure (Colvin, 2015). Therefore, yield losses due to leaf spot and rust in Haiti may be less than in more mechanized production systems due to the ability to recover pods from the soil.

Our results indicated that late leaf spot was the most yield-limiting disease for the local Haitian runner, which resulted in a linear yield loss relationship. Rust was the most important disease on Georgia-06G, and resulted in quadratic relationship with percent yield loss. These results indicate that the local Haitian runner has a moderate level of resistance to rust, while Georgia-06G has more resistance to late leaf spot. Differing levels of resistance to the rust and late leaf spot pathogens has been documented by others (Subrahmanyam et al., 1995). Also, in recent greenhouse and field trials conducted in the southeastern United States, we found that Georgia-06G is more resistant to late leaf spot when compared to more susceptible varieties such as Florunner (Chapters 6 and 7), which may partially explain the lower levels of late leaf spot observed on this cultivar in Haiti. Furthermore, C99R, one of the parental lines of Georgia-06G, is known to have a moderate levels of resistance to late leaf spot (Branch, 2007).

By differentiating pods picked from the plant vs. the pods recovered from the soil, we were able to provide a more in-depth understanding of yield loss on runner market types. To the best of our knowledge, this is a unique report of the quantitative relationship between yield loss and foliar diseases under manual harvesting conditions. We found that the average percent yield loss due to foliar diseases was reduced from 83 to 64% on the local Haitian runner by recovering the pods from the soil, but only from 51 to 43% on the variety Georgia-06G. [It should also be noted that we found that the quality of the kernels (percent SMK) was similar on pods from the soils and pods from the plant. This was an encouraging find, and in like manner it should be noted that no aflatoxin was detected with Romer strips (>10 ppb) in any of the peanuts collected in these studies (data not shown).] In studies conducted in Malawi, yield losses ranging from 50 to 65% were associated with early leaf spot on moderate maturing varieties(>110 days) (Subrahmanyam and Hildebrand, 1997). Losses associated with late leaf spot have been reported at approximately 50% in east Africa, and 25 to 50% combined losses due to rust and leaf spot (Subrahmanyam et al., 1997). While these trials were harvested by hand, it does not mention whether pods were recovered from the soil during harvest. Overall, we found less foliar-disease related yield loss (approximately 30%) on the Valencia varieties. Similarly, yield loss on short-maturing (<90 days) varieties in Ghana ranged from 20 to 40% (Naab et al., 2009a). It is likely that lower yield loss on

these Valencia varieties results to some degree of the phenomenon of escape, in which the complete destruction of the plant canopy was avoided during the majority of the season.

While not the main purpose of these trials, we also observed yield differences between trials and locations. For runner types, the highest yields were obtained in the spring of 2015. It should be noted that in this trial, the harvest was delayed to approximately 140 DAP, whereas in the other trials, peanuts were harvested between 120 and 130 DAP. Although maturity tests were conducted to determine harvest dates, delaying harvest could allow for a younger crop to reach maturity. Interestingly, the highest-yielding runner trial was planted at 3 seed per 30.5 cm and the lowest-yielding runner trial was planted at 6 seed per 30.5 cm. However, it should be pointed out that the lowest-yielding runner trial was negatively impacted by heavy rainfalls that resulted in periodic flooding of the field. Recent studies in the United States demonstrated a linear yield increase with increased seeding rates (Sarver et al., 2016), but the overall yield gain of runner market types between 3 and 6 seed per 30.4 cm was less than 9% (Sorensen et al., 2004). For Valencia varieties, our results suggest that lower seeding rates result in lower yields. The yield at MFK was twice as high as that in the Central Plateau, and seed were planted at 6 seed per 30.4 cm and 3 seed per 30.4 cm at each location, respectively. While the differences could be due to an environmental effect, we have recently conducted seeding rate trials in Haiti that corroborate these results (PMIL, unpublished data).

There was heavy disease pressure in all trials conducted in the North. Even though there were differences in the environmental conditions among trials, we did not find a consistent association between rainfall and disease pressure. This may be due to the fact these trials were irrigated. In Haiti, the main rainy season in both the North and the Central Plateau generally occurs in the spring (April to June), but the Northeast often has a second, more sporadic rainy season that generally occurs from October to November but can also extend or shift into December, January and February (Kostandini et al., 2017). These trials were generally conducted to coincide with the spring rainy season or the winter rainy season. We found that almost every trial conducted in the North had at least 2 months of heavy rainfall, with the majority either falling in the beginning or towards the end of the season. Because of similar trends across

trials, variations in rainfall between trials suggest that the fungicide programs evaluated in these studies are appropriate for a wide range of rainfall patterns.

In conclusion, data presented here illustrate that late leaf spot and rust are major yield limiting factors of peanut in Haiti. The data demonstrates the potential to manage these diseases with low input fungicide programs. These studies indicate that each increase in the number of fungicide applications tends to provide a sequential increase in disease control and yield gains, but that the particular timing evaluated for treatments with the same number of applications seem to be less critical. Final recommendations for fungicide applications will depend on a more in-depth analysis of practical and economic considerations, but will most likely result in 3 or 4 and 2 or 3 applications for runner and Valencia market types, respectively. We found that yield loss depends on harvest method, and that the local grower standard of recovering pods from the soil still resulted in foliar disease related yield loss of 51 and 43% on the local Haitian runner and the improved variety, Georgia-06G, respectively. Yield losses on Valencia market types were consistently near 30%. Our results suggest that Georgia-06G could provide superior yields compared with the local Haitian runner, but that that the local Haitian Valencia is as good or better under local conditions than the improved Valencia variety, New Mexico Valencia A. While growers may have yet to adapt the full technology package used in these studies, our results clearly demonstrate the potential to increase peanut yields in Haiti by 4 to 5-fold. Continued research is needed to confirm these findings in grower situations, and to evaluate more resistant varieties with similar fungicide programs.

ACKNOWLEDGMENTS

We thank personnel at Meds and Foods for Kids and the Acceso Peanut Enterprise Corporation for providing research facilities and assistance in research activities. Particularly, we thank Will Sheard, Patrick Dorzan, Alex Carroll, Georgy Faroutine, Rick Macajoux, and Dominique Laforest for technical support and research assistance. We would also like to acknowledge the valuable assistance of student interns from the Université Roi Henri Christophe and the Université Chrétienne du Nord d'Haïti who were responsible for implementing, managing, assessing and harvesting many of these trials as part of

MFK/PMIL internship program. In particular, we thank Daphenie Jean, Kinson Pierre, Fedeline Charles, Exan Desamours, Gary Benoit, Elizé Léandre, Marilène Saint-Juste, Galeine Queranor and Rolcky Butois. We also thank Xuelin Luo at the University of Georgia for statistical assistance. Funding for this project was provided by the USAID supported Peanut and Mycotoxin Innovation Lab (Project UF-204). A brief summary of part of this research has previously been published (Fulmer et al., 2016).

LITERATURE CITED

Bargout, R. N., and Raizada, M. N. 2013. Soil nutrient management in Haiti, pre-Columbus to the present day: lessons for future agricultural interventions. Agriculture & Food Security 2:11.

Bayles, R., Stigwood, P., and Clarkson, J. 2000. Shifts in sensitivity of Puccinia striiformis to DMI fungicides in the UK. Acta Phytopathol. Entomol. Hung. 35:381-382.

Branch, W. 2007. Registration of 'Georgia-06G' peanut. J Plant Regist 1:120-120.

Brent, K. 2007. Fungicide resistance in crop pathogens: how can it be managed? FRAC Monogr. No. 1. Global Crop Protection Federation, Brussels.

Chiteka, Z., Gorbet, D., Shokes, F., Kucharek, T., and Knauft, D. 1988. Components of resistance to late leafspot in peanut. I. Levels and variability-implications for selection. Peanut Sci. 15:25-30.

Colvin, B. C. 2015. Influence of canopy health and maturity on peanut peg strength, yield, and grade. M.S. thesis. University of Florida, Gainsville.

Culbreath, A., Kemerait Jr, R., and Brenneman, T. 2006. Management of early leaf spot of peanut as affected by fungicide and date of spray program initiation. Plant Health Progress doi:10.1094/PHP-2006-0214-01-RS.

FAOSTAT. 2016. Food and Agriculture Organization of the United Nations. Statistics Division. http://www.fao.org/faostat.

Filbert, M. E., and Brown, D. L. 2012. Aflatoxin contamination in Haitian and Kenyan peanut butter and two solutions for reducing such contamination. Journal of Hunger & Environmental Nutrition 7:321-332.

Fulmer, A., Kemerait, R., and Brenneman, T. 2014. Mountains beyond mountains: Challenges and opportunities for managing peanut diseases in Haiti. Phytopathology 104 (Suppl.)S:155.

Fulmer, A., Kemerait, R., and Brenneman, T. 2016. Effect of fungicide timing and frequency on reducing foliar diseases and yield loss on runner and valencia type peanuts in Haiti. Phytopathology 106 (Suppl.)S:66.

Fulmer, A., Kemerait, R., Sherwood, J., Jordan, D., Rhoads, J., and Brenneman, T. 2012. Evaluation of ICRISAT varieties for resistance to foliar peanut diseases in Haiti. Phytopathology 102 (Suppl.)S2:4.

Hammons, R. 1982. Origin and early history of the peanut. Pages 1-20 in: Peanut Science and Technology H. Pattee and C. Young, eds. American Peanut Research and Education Society, Yoakum, TX.

Iannotti, L. L., Henretty, N. M., Delnatus, J. R., Previl, W., Stehl, T., Vorkoper, S., Bodden, J., Maust, A., Smidt, R., and Nash, M. L. 2015. Ready-to-use supplementary food increases fat mass and BMI in haitian school-aged children. The Journal of nutrition 145:813-822.

Jackson, J., Beasley Jr, J., Tubbs, R., Lee, R., and Grey, T. 2011. Fall-bedding for reduced digging losses and improved yield in strip-till peanut. Peanut Sci. 38:31-40.

Kostandini, G., Rhoads, J., Johnson, R., and Schwartzbord, J. R. 2017. Gender differences in peanut productivity and post-harvest practices: Evidence from Haiti. *In Review*.

McDonald, D., Subrahmanyam, P., Gibbons, R., and Smith, D. 1985. Early and Late Leaf Spots of Groundnut. Information Bull. No. 21. ICRISAT, Patancheru.

MFK. 2017. Meds and Food for Kids. https://mfkhaiti.org.

Naab, J., Prasad, P., Boote, K., and Jones, J. 2009a. Response of peanut to fungicide and phosphorus in on-station and on-farm tests in Ghana. Peanut Sci. 36:157-164.

Naab, J., Boote, K., Prasad, P., Seini, S., and Jones, J. 2009b. Influence of fungicide and sowing density on the growth and yield of two groundnut cultivars. The Journal of Agricultural Science 147:179-191.

Naab, J., Tsigbey, F., Prasad, P., Boote, K., Bailey, J., and Brandenburg, R. 2005. Effects of sowing date and fungicide application on yield of early and late maturing peanut cultivars grown under rainfed conditions in Ghana. Crop Protect. 24:325-332.

Naab, J., Seini, S., Gyasi, K., Mahama, G., Prasad, P., Boote, K., and Jones, J. 2009c. Groundnut yield response and economic benefits of fungicide and phosphorus application in farmer-managed trials in Northern Ghana. Exp. Agric. 45:385-399.

Nelson, R., Jolly, C., Hinds, M., Donis, Y., and Prophete, E. 2003. Consumer preferences for peanut butter (mamba) products in Haiti: A conjoint analysis. Peanut Sci. 30:99-103.

PMIL. 2017. Peanut Mycotoxin Innovation Lab. http://www.caes.uga.edu/global/feed-the-future-innovation-labs/peanut-mycotoxin-innovation-lab.html.

Sarver, J. M., Tubbs, R. S., Jr., J. P. B., Culbreath, A. K., Grey, T. L., Rowland, D. L., and Smith, N. B. 2016. Plant population and replant method effects on peanut seeded in single rows. Peanut Sci. 43:126-132.

Schwartzbord, J. R., and Brown, D. L. 2015. Aflatoxin contamination in Haitian peanut products and maize and the safety of oil processed from contaminated peanuts. Food Control 56:114-118.

Shokes, F., Gorbet, D., and Sanden, G. 1982. Effect of planting date and date of spray initiation on control of peanut leaf spots in Florida. Plant Dis. 66:574-575.

Sorensen, R. B., Sconyers, L. E., Lamb, M. C., and Sternitzke, D. A. 2004. Row orientation and seeding rate on yield, grade, and stem rot incidence of peanut with subsurface drip irrigation. Peanut Sci. 31:54-58.

Stevenson, K. L., and Culbreath, A. 2006. Evidence for reduced sensitivity to tebuconazole in leaf spot pathogens. Proc. Amer. Peanut Res. Ed. Soc. 38:62 (Abstr.).

Subrahmanyam, P., and Hildebrand, G. 1997. Responses of peanut genotypes to fungicidal control of early leaf spot in Malawi. Peanut Sci. 24:73-77.

Subrahmanyam, P., Van Wyk, P., Kisyombe, C., Cole, D., Hildebrand, G., Chiyembekeza, A., and Van der Merwe, P. 1997. Diseases of groundnut in the Southern African Development Community (SADC) region and their management. Int. J. Pest Manage. 43:261-273.

Subrahmanyam, P., McDonald, D., Waliyar, F., Reddy, L., Nigam, S., Gibbons, R., Rao, V. R., Singh, A., Pande, S., and Reddy, P. 1995. Screening methods and sources of resistance to rust and late leaf spot of groundnut. Information Bulletin no. 47. International Crops Research Institute for the Semi-Arid Tropics.

Waliyar, F., Adamou, M., and Traoré, A. 2000. Rational use of fungicide applications to maximize peanut yield under foliar disease pressure in West Africa. Plant Dis. 84:1203-1211.

WHO. 2017. World Health Organization. http://www.who.int/maternal_child_adolescent/topics/child/malnutrition/en/.

Table 9.1. Fungicide treatments for runner and Valencia market type peanuts treated with Muscle ADV (tebuconazole + chlorothalonil) in trials conducted in multiple locations in Haiti from 2015 to 2017.

Market type	Total applications	Initiation[y]	Spray interval[z]	Application timings
Runner	6	30	14	30, 44, 58, 72, 86, 100
	4	37	21	37, 58, 79, 100
	4	45	21	45, 66, 87, 108
	3	37	28	37, 65, 93
	3	45	28	45, 73, 101
	2	60	28	60, 88
	0	-	-	-
Valencia	4	30	14	30, 44, 58, 72
	3	30	21	30, 51, 72
	3	45	14	45, 59, 73
	2	45	21	45, 66,
	2	45	28	45, 73
	1	45	-	45
	0	-	-	-

[y] Days after planting when the first application was made.
[z] Days between applications.

Table 9.2. Averages of environmental variables from the time of planting to the time of harvest for each runner and Valencia field trial conducted near Cap Haitian (MFK), Haiti from 2015 to 2017.

Market type, year, season	Day after planting	Rain events[x] > 0.254 mm	Rain events[x] > 0.63 mm	Rain events[x] > 12.7 mm	Rainfall[z] mm
Runner					
2015 spring	0 - 30	5	2	2	67
	30 - 60	8	4	2	65
	60 - 90	12	5	2	122
	90 - 120	20	11	4	156
2015/2016 winter	0 - 30	4	3	2	72
	30 - 60	2	0	0	4
	60 - 90	6	2	2	38
	90 - 120	15	9	6	472
2016 spring	0 - 30	9	3	2	213
	30 - 60	10	5	3	115
	60 - 90	6	5	3	164
	90 - 120	13	6	1	128
2016/2017 winter	0 - 30	23	15	10	869
	30 - 60	11	6	6	152
	60 - 90	8	2	2	58
	90 - 120	4	1	0	16
Valencia					
2016 spring	0 - 30	8	5	3	110
	30 - 60	8	5	3	169
	60 - 90	12	5	0	75
2016 fall	0 - 30	7	5	3	107
	30 - 60	13	9	4	203
	60 - 90	23	15	12	840

[x] Number of days that received > 0.25, > 0.63 or > 12.7 mm of rain during each interval day after planting interval. Rainfall data was measured with two on-station Decagon rain gauges set to record at hourly intervals.

[z] Total rainfall that occurred during each interval.

Table 9.3. Mean final disease severity of leaf spot and rust on untreated runner and Valencia peanut varieties for each field trial conducted near Cap Haitien (MFK) and Mirebalais (CP), Haiti from 2015 to 2017.

Runner market type,	Leaf spot[t]		Rust[u]	
trial (year, season)	Local runner	Georgia-06G	Local runner	Georgia-06G
2015 spring	8.5 ±1.1	6.9 ±1.3	8.1 ±1.0	8.3 ±0.7
2015/2016 winter	8.0 ±1.1	6.0 ±0.8	6.8 ±1.0	8.5 ±0.4
2016 spring	8.1 ±0.9	3.5 ±0.9	2.8 ±1.2	8.3 ±0.6
2016/2017 winter	9.1 ±0.3	4.0 ±0.7	6.4 ±1.9	9.0 ±0.0
Valencia market type, trial	Leaf spot[t]		Rust[u]	
(location, year, season)	Local Valencia	NM Val A	Local Valencia	NM Val A
CP, 2015 spring	5.9 ±0.9	5.4 ±0.8	6.6 ±1.2	6.0 ±0.9
CP, 2016 spring	3.0 ±0.7	2.8 ±0.3	3.3 ±0.9	3.0 ±0.4
MFK, 2016 spring	4.6 ±0.3	4.2 ±0.6	8.5 ±0.4	8.0 ±0.4
MFK, 2016 fall	7.8 ±0.6	7.5 ±1.0	8.8 ±0.3	8.8 ±0.3

[t] Florida 1 to 10 scale, where 1= no leaf spot and 10 = complete defoliation/death of plant.
[u] Rust 1 to 9 scale where 1 = no disease and 9 = almost all leaves withered; bare stems seen; 81-100% severity.
[z] Means of each trial; ± values are the standard error of the mean.

Table 9.4. *P* values from the analysis of variance of the final leaf spot and rust severity and yield attributes of runner and Valencia market types in field trials conducted near Cap Haitien (MFK) and Mirebalais (CP), Haiti from 2015 to 2017.

Location, year, season source of variation	Final severity		Yield[v]		SMK[w]	
	Leaf spot	Rust	Plant[x]	Plant + Soil[y]	Plant[x]	Plant + Soil[y]
Cap Haitien (MFK)	Runner market type					
2015 spring						
Variety (V)	0.0426	<0.0001	<0.0001	0.0052	0.0083	0.0012
Fungicide (F)	<0.0001	<0.0001	0.0002	0.0188	0.3639	0.7613
V x F	0.8203	0.3040	0.0789	0.4013	0.9415	0.7992
2015/2016 winter						
Variety (V)	0.9471	0.0167	0.0156	0.1977	0.2717	0.0814
Fungicide (F)	0.0003	<0.0001	<0.0001	<0.0001	0.6304	0.8018
V x F	0.1381	0.8941	0.0941	0.9483	0.4655	0.5742
2016 spring						
Variety (V)	<0.0001	0.0106	0.0020	0.1485	0.0013	0.0072
Fungicide (F)	<0.0001	<0.0001	<0.0001	<0.0001	0.8746	0.9666
V x F	0.0602	0.4405	0.5663	0.9972	0.5578	0.0920
2016/2017 winter						
Variety (V)	0.0028	0.0435	0.0452	0.2735	0.0583	0.0410
Fungicide (F)	0.0002	<0.0001	<0.0001	<0.0001	0.3992	0.6206
V x F	0.1160	0.1119	0.1445	0.2596	0.896	0.5208
Mirebalais (CP)	Valencia market types					
2015 spring						
Variety (V)	0.2677	0.1367	-[z]	0.0230	-	-
Fungicide (F)	<0.0001	<0.0001	-	<0.0001	-	-
V x F	0.1455	0.6608	-	0.9981	-	-
2016 spring						
Variety (V)	0.4828	0.4135	-	<0.0001	0.2183	-
Fungicide (F)	<0.0001	0.0005	-	<0.0001	0.3388	-
V x F	0.1260	0.3700	-	0.6128	0.5868	-
Cap Haitien (MFK)						
2016 spring						
Variety (V)	0.0700	0.0812	-	0.4976	0.1725	-
Fungicide (F)	<0.0001	<0.0001	-	<0.0001	0.5194	-
V x F	0.3550	0.5776	-	0.9624	0.0751	-
2016 fall						
Variety (V)	0.2635	0.3869	0.8131	0.9233	0.9811	0.4487
Fungicide (F)	<0.0001	<0.0001	<.0001	0.0013	0.2346	0.3634
V x F	0.9497	0.9023	0.8152	0.8770	0.5901	0.5728

[v] Final pod weights were adjusted to 10% pod moisture.

[w] Percent SMK was calculated by dividing the weight of the sound mature kernels by the total weight ofunshelled pods in a 100 pod sample.

[x] Based on pods picked from plants after being manually pulled from the ground.

[y] For runner types, this was based on total of pods from plants + pods recovered from manually excavating the soil. For Valencia types, this was based on pods recovered from the surface of the soil or manually excavating the soil (latter method only done in the fall of 2016).

[z] Data not taken for these trials.

Table 9.5. Effect of variety on final leaf spot and rust severity and yield attributes of runner market types pooled across fungicide treatments for each trial conducted near Cap Haitien (MFK) from 2015 to 2017.

Year, season, variety	Final severity		Yield (kg/ha)[t]		SMK[u]	
	Leaf spot[v]	Rust[w]	Plant[x]	Plant + Soil[y]	Plant[x]	Plant + Soil[y]
2015 spring						
Local runner	5.9 a	4.4 a	923 b	3698 b	75.6 b	75.0 b
Georgia-06G	4.4 b	4.9 a	4840 a	6220 a	80.0 a	79.3 a
2015/2016 winter						
Local runner	4.4 a	3.9 a	1556 b	2912 a	73.0 a	73.3 a
Georgia-06G	3.2 a	4.6 a	2777 a	3252 a	75.6 a	75.7 a
2016 spring						
Local runner	5.0 a	2.4 b	1607 b	3594 a	64.5 b	61.8 b
Georgia-06G	3.3 b	4.9 a	3377 a	4079 a	73.4 a	67.7 a
2016/2017 winter						
Local runner	3.6 a	3.0 b	1474 b	2254 a	64.0 a	62.5 b
Georgia-06G	2.3 b	4.1 a	2037 a	2711 a	71.7 a	69.2 a

[t] Final pod weights were adjusted to 10% pod moisture.

[u] Percent SMK was calculated by dividing the weight of the sound mature kernels by the total weight ofunshelled pods in a 100 pod sample.

[v] Florida 1 to 10 scale, where 1= no leaf spot and 10 = complete defoliation/death of plant.

[w] Rust 1 to 9 scale where 1 = no disease and 9 = almost all leaves withered; bare stems seen; 81-100% severity.

[x] Based on pods picked from plants after being manually pulled from the ground.

[y] Based on total of pods from plants + pods recovered from manually excavating the soil.

[z] For each trial, means within the same column with the same letters are not significantly different based upon Tukey's honestly significant difference test.

Table 9.6. Effect of variety on final leaf spot and rust severity and yield attributes of Valencia market types pooled across fungicide treatments for trials conducted near Cap Haitien (MFK) and Mirebalais (CP), Haiti from 2015 to 2017.

Location, year, season, variety	Final severity		Yield (kg/ha)[v]	SMK[w]
	Leaf spot[x]	Rust[y]	Plant + Soil	Plant + Soil[y]
Mirebalais (CP)				
2015 spring				
Local Valencia	3.7 a[z]	4.9 a	1510 a	-
NM Valencia A	3.5 a	4.3 a	1067 b	-
2016 spring				
Local Valencia	2.3 a	2.5 a	2899 a	69.3 a
NM Valencia A	2.3 a	2.4 a	2392 b	68.4 a
Cap Haitien (MFK)				
2016 spring				
Local Valencia	3.5 a	5.4 a	4256 a	71.3 a
NM Valencia A	2.7 b	4.7 a	4069 a	69.6 a
2016 fall				
Local Valencia	5.4 a	6.6 a	3084 a	58.5 a
NM Valencia A	5.1 a	6.2 a	3065 a	58.5 a

[v] Final pod weights were adjusted to 10% pod moisture. Pods from plants + pods recovered from the surface of the soil or manually excavating the soil (latter method only done in the fall of 2016).

[w] Percent SMK was calculated by dividing the weight of the sound mature kernels by the total weight of unshelled pods in a 100 pod sample.

[x] Florida 1 to 10 scale, where 1= no leaf spot and 10 = complete defoliation/death of plant.

[y] Rust 1 to 9 scale where 1 = no disease and 9 = almost all leaves withered; bare stems seen; 81-100% severity.

[z] For each trial means within the same column with the same letters are not significantly different based upon Tukey's honestly significant difference test

Table 9.7. Effect of trial on final disease severity and percent yield loss on untreated runner peanut varieties in field trials conducted near Cap Haitien (MFK), Haiti from 2015 to 2017.

	% Yield loss (plant)[v] [w]		% Yield loss (plant + soil)[x]	
Trial (year, season)	Local runner	Georgia-06G	Local runner	Georgia-06G
2015 spring	90 (6)	47 (16)	56 (12)	26 (9)
2015/2016 winter	85 (6)	67 (11)	55 (6)	48 (8)
2016 spring	75 (6)	56 (29)	39 (3)	37 (17)
2016/2017 winter	85 (7)	90 (5)	56 (12)	63 (4)
Average (std error)[z]	83 (8)	64 (23)	51 (11)	43 (17)

[t] Florida 1 to 10 scale, where 1= no leaf spot and 10 = complete defoliation/death of plant.
[u] Rust 1 to 9 scale where 1 = no disease and 9 = 9 = almost all leaves withered; bare stems seen; 81-100% severity.
[v] Based on pods picked from plants after being manually pulled from the ground.
[w] Percent loss calculated as [100 × (6 spray yield - untreated yield) / 6 spray yield].
[x] Based on total of pods from plants + pods recovered from the soil.
[y] Means within the same column with the same letters are not significantly different based upon Tukey's honestly significant difference test.
[z] Means averaged across all trials; values in parentheses are the standard error of the mean.

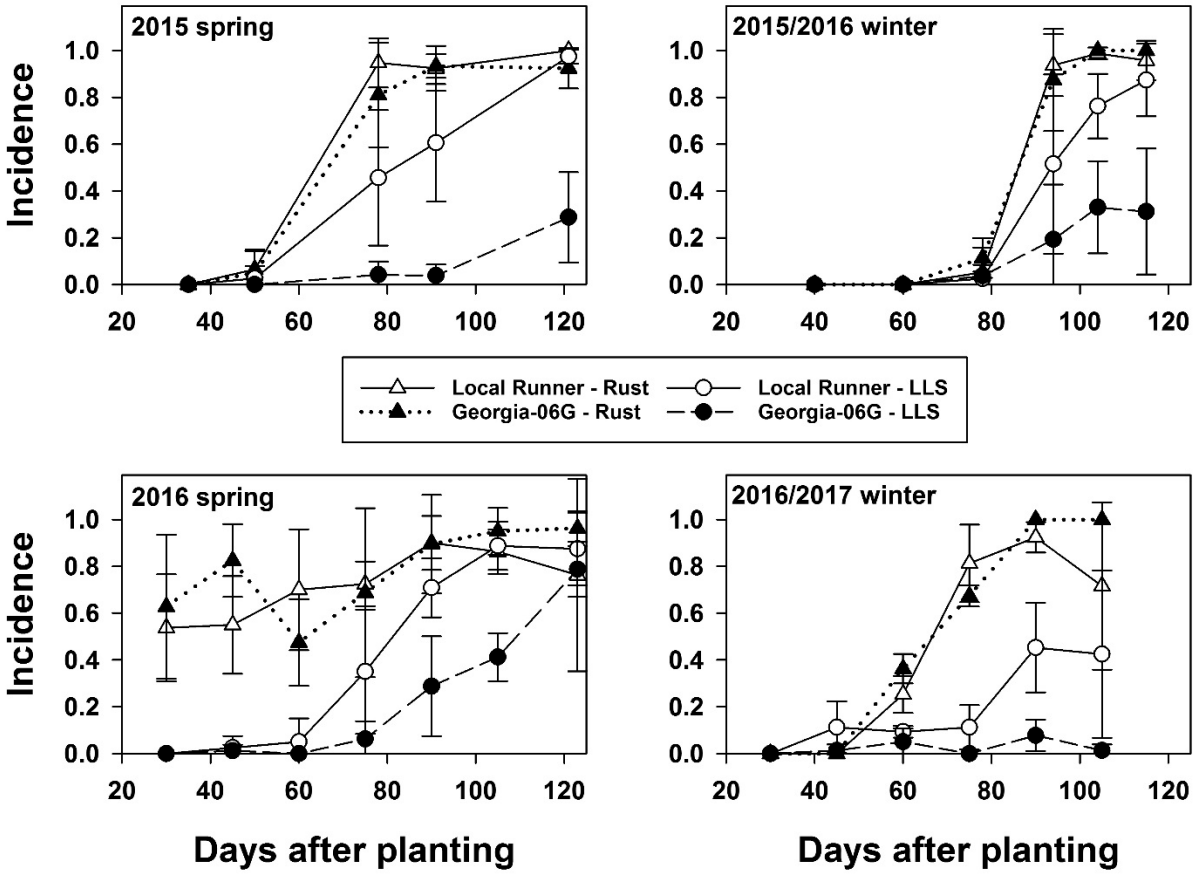


Fig. 9.1. Development of late leaf spot (LLS) and rust incidence in untreated plots planted to two varieties (local Haitian runner and Georgia-06G) of runner market type peanuts. Field trials were conducted in research plots near Cap Haitien, Haiti. Data points represent the mean of four replications and error bars represent the standard error of the mean.

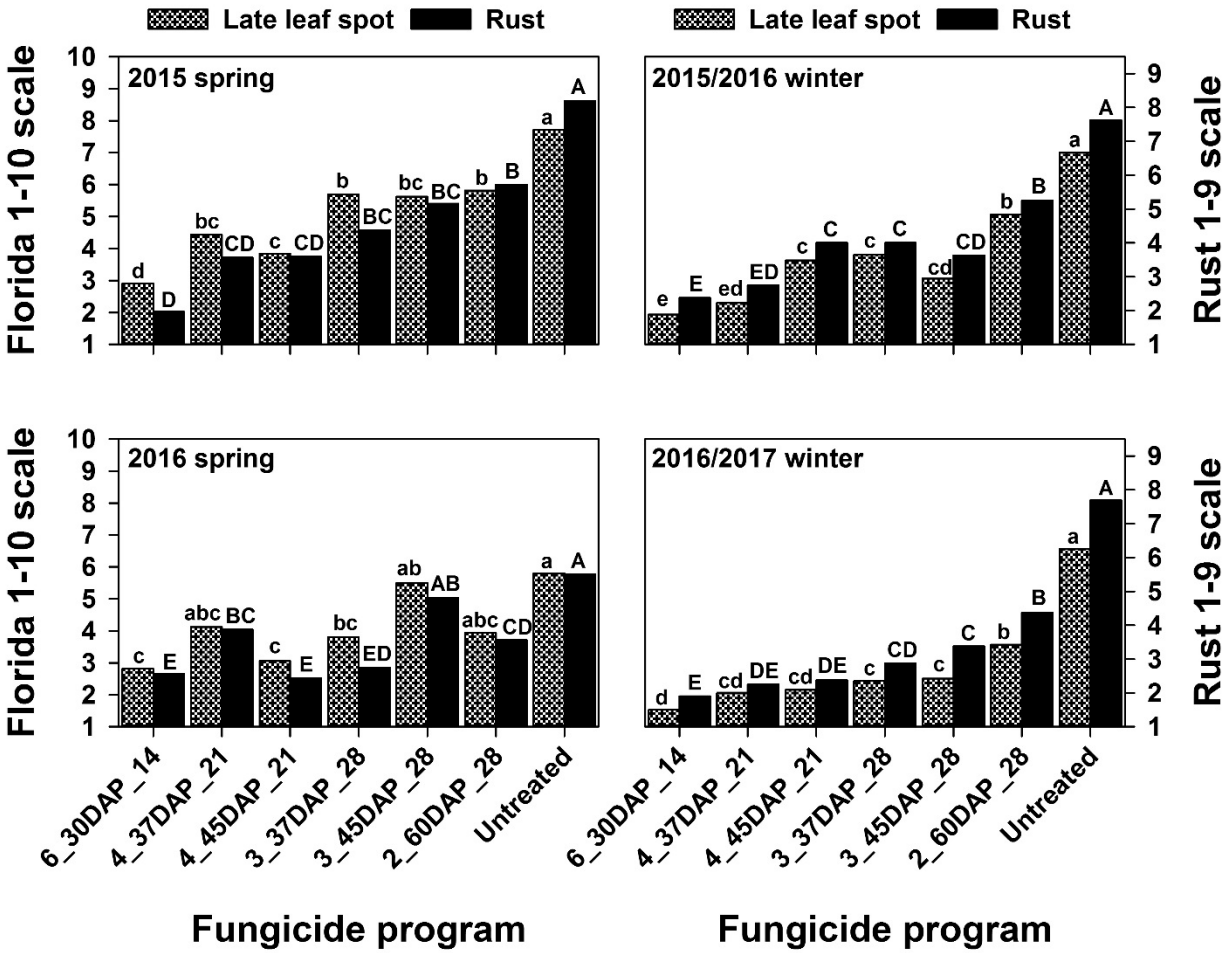


Fig. 9.2. Effect of fungicide treatment on the final severity of late leaf spot and rust on runner market types peanuts. Data are pooled across the local Haitian runner and the improved variety Georgia-06G. Trials were conducted in research plots near Cap Haitien, Haiti. Fungicide treatments are labeled as follows: number of applications _ day after planting of first application _ subsequent spray interval (in days). Late leaf spot was assessed with the Florida 1-10 scale, where 1= no leaf spot and 10 = complete defoliation/death of plant. Rust was assessed with a 1-9 scale, where 1 = no disease and 9 = almost all leaves withered; bare stems seen; 81-100% severity. Grouped bars with the same lower case or upper case letters represent no significant differences between fungicide treatments for leaf spot and rust, respectively, and are based upon Tukey's honestly significant difference test.

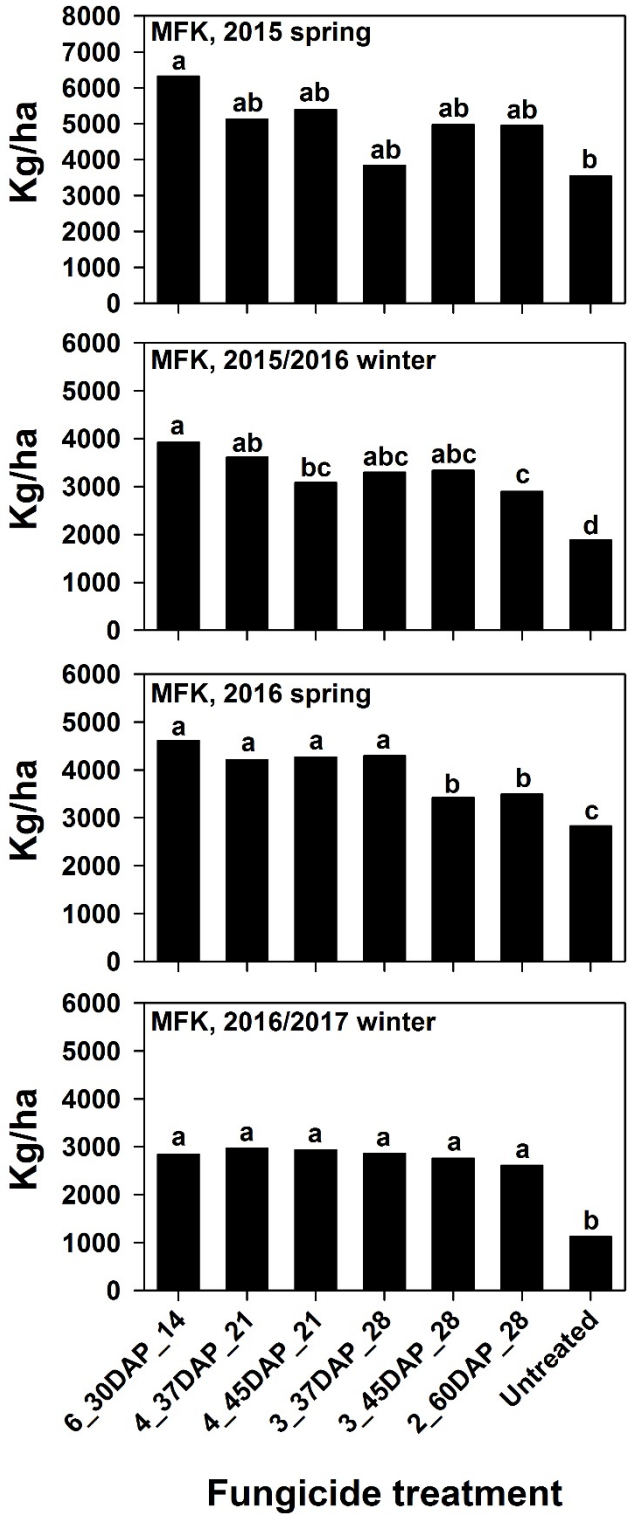


Fig. 9.3. Effect of fungicide program on total pod yield (kg/ha) pooled across two runner peanut varieties. Fungicide treatments are labeled as follows: number of applications _ day after planting of first application _ subsequent spray interval (in days). Grouped bars for each fungicide treatment with the same letters are not significantly different based upon Tukey's honestly significant difference test.

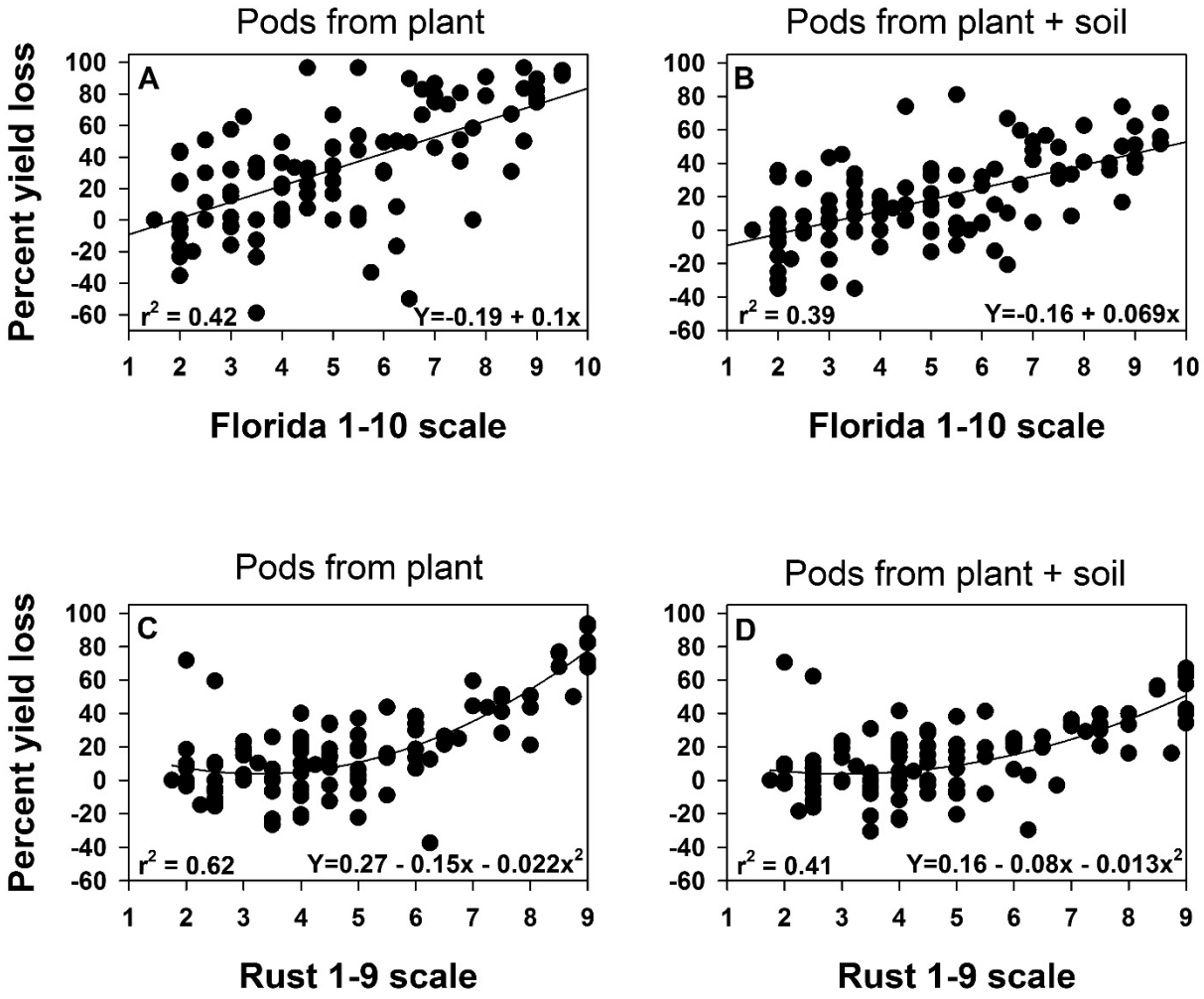


Fig. 9.4. Relationships of percent yield loss and foliar disease severity on runner type peanuts across four trials conducted in research plots near Cap Haitien, Haiti during 2015 to 2017. Percent loss was calculated as [100 × (6 spray yield – treatment yield) / 6 spray yield]. **A** and **B**. Percent yield loss in relation to late leaf spot severity on the local Haitian runner, where 1= no leaf spot and 10 = complete defoliation/death of plant. **C** and **D**. Percent yield loss in relation to rust severity on the variety Georgia-06G, where 1 = no disease and 9 = almost all leaves withered; bare stems seen; 81-100% severity. Regression analyses were conducted separately for (**A** and **C**) pods picked from plants after physically pulling from the ground, and (**B** and **D**) the combination from those picked from the plant and those recovered from the soil.

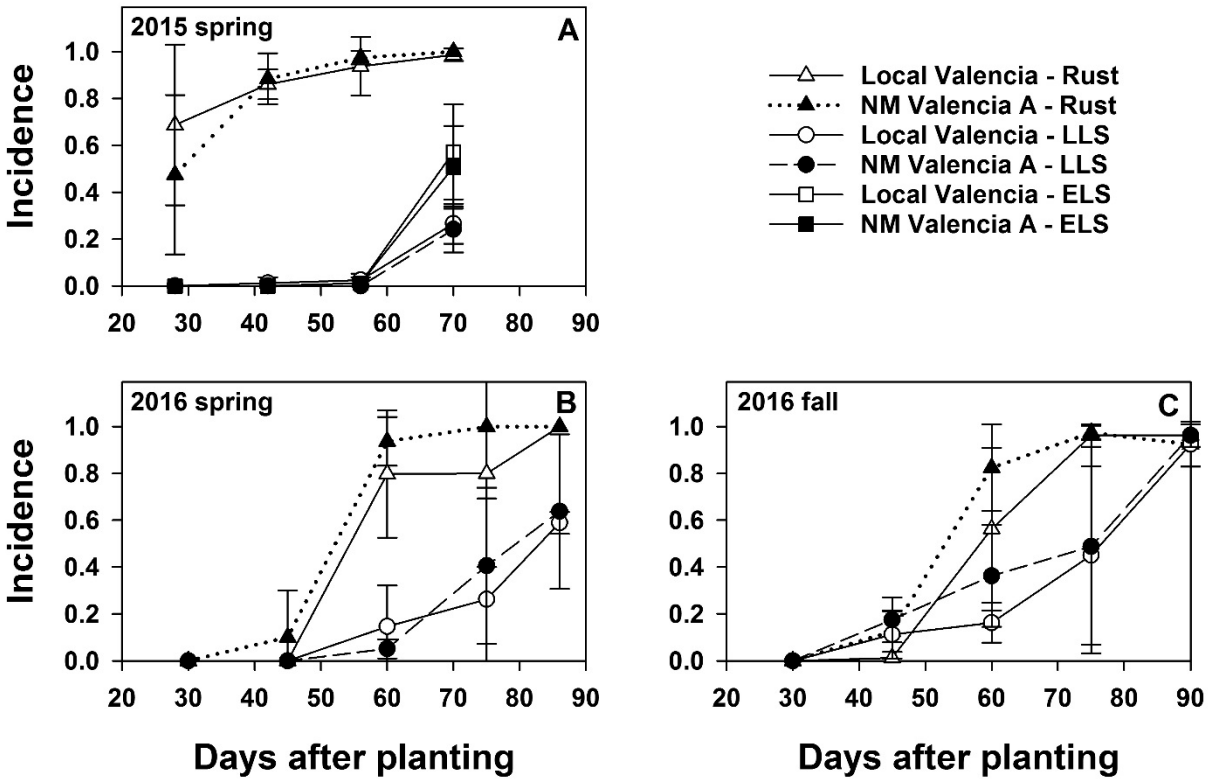


Fig. 9.5. Development of early (ELS) and late leaf spot (LLS) and rust incidence in untreated plots planted to two varieties (local Haitian Valencia and New Mexico Valencia A) of Valencia peanuts. **A.** Field trials were conducted in research plots near the town of Mirebalais, Haiti in the spring of 2015 and 2016 (ratings from the latter trial were not taken throughout the season, and thus are not displayed). **B** and **C**. Field trials were conducted in research plots near Cap Haitien, Haiti. Data points represent means of four replications and error bars represent the standard error of the mean.

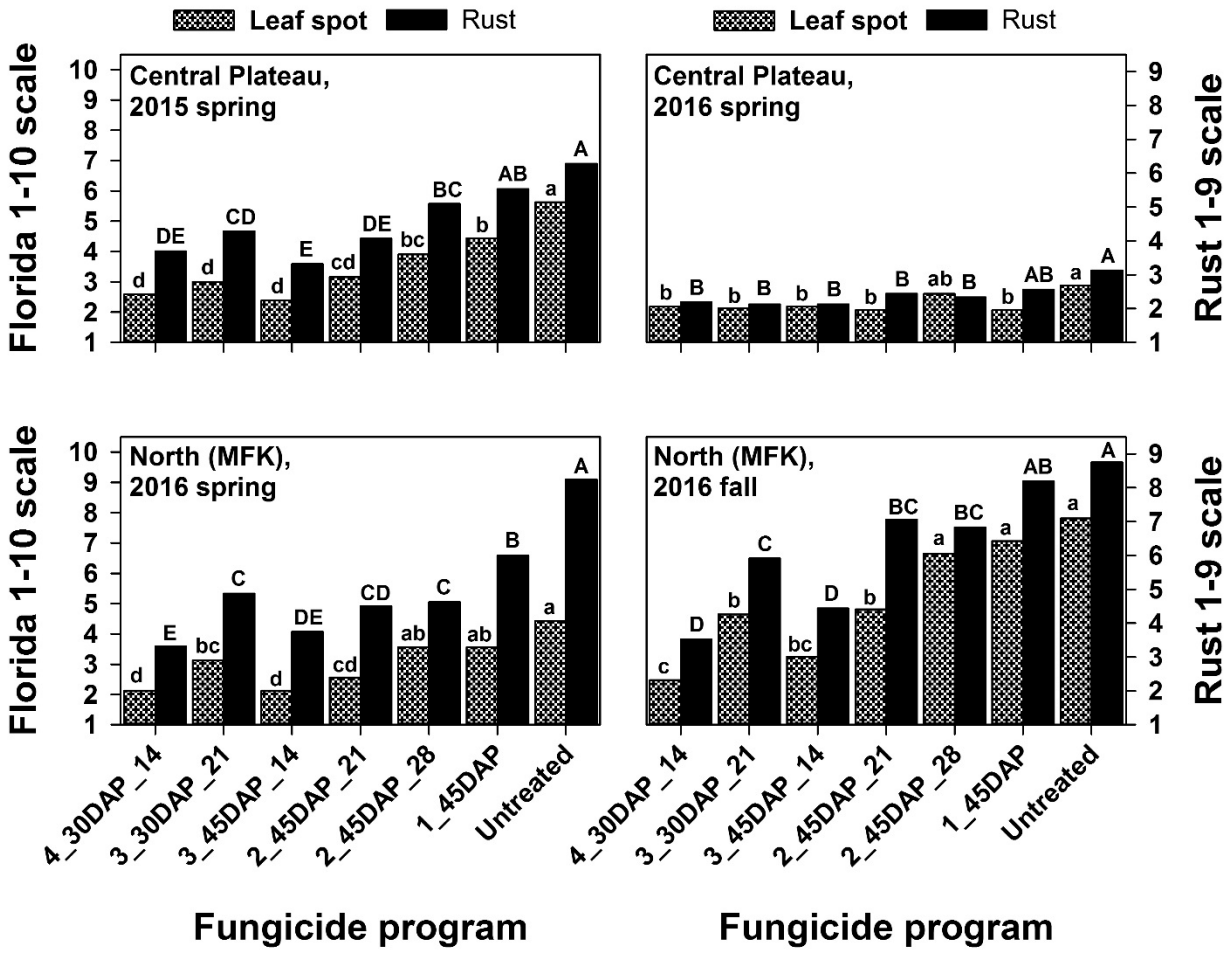


Fig. 9.6. Effect of fungicide treatment on the final severity of late leaf spot and rust on Valencia market types. Data are pooled across the local Haitian runner and New Mexico Valencia A. Fungicide treatments are labeled as follows: number of applications _ day after planting of first application _ subsequent spray interval (in days). Late leaf spot was assessed with the Florida 1-10 scale, where 1= no leaf spot and 10 = complete defoliation/death of plant. Rust was assessed with a 1-9 scale, where 1 = no disease and 9 = almost all leaves withered; bare stems seen; 81-100% severity. Grouped bars with the same lower case or upper case letters represent no significant differences between fungicide treatments for leaf spot and rust, respectively, and are based upon Tukey's honestly significant difference test.

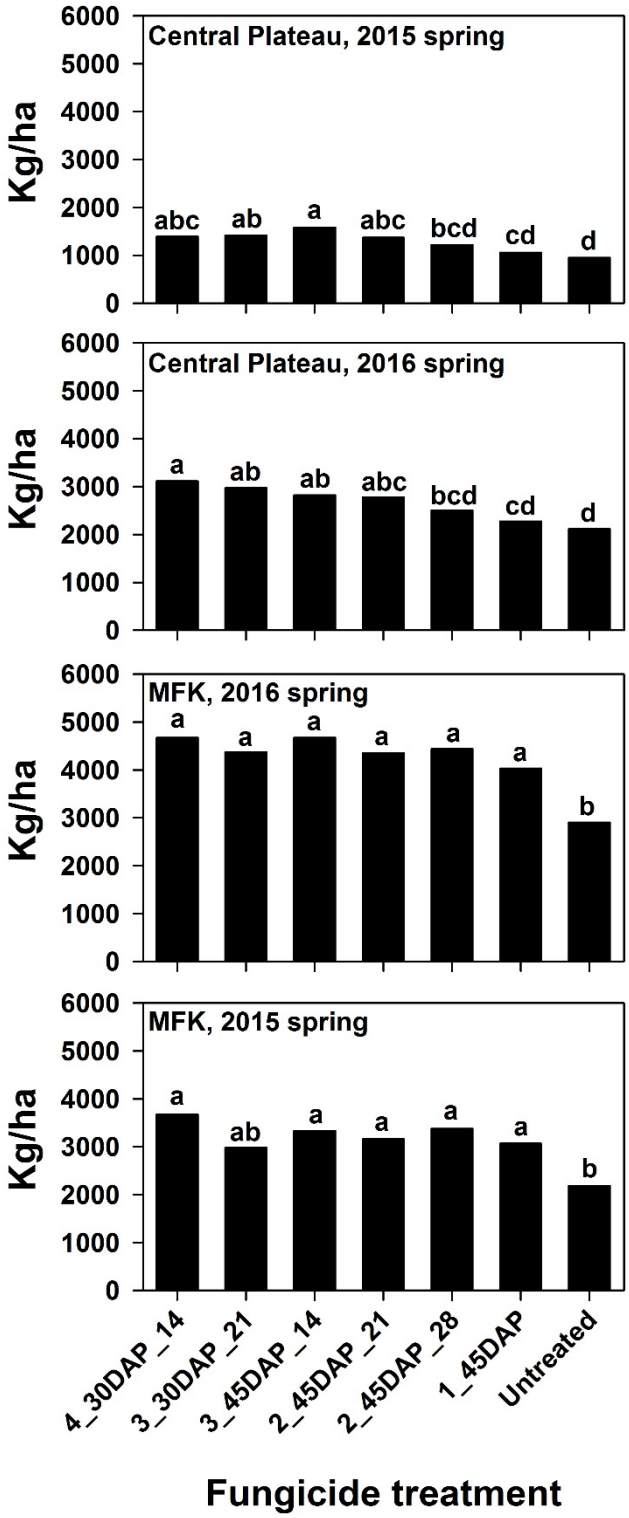


Fig. 9.7. Effect of fungicide program on total pod yield (kg/ha) pooled across two varieties of Valencia market type peanuts. Fungicide treatments are labeled as follows: number of applications _ day after planting of first application _ subsequent spray interval. Grouped bars for each fungicide treatment with the same letters are not significantly different based upon Tukey's honestly significant difference test.

CHAPTER 10

CONCLUSIONS

The primary objective of this research was to differentially understand the epidemics of early (ELS) and late (LLS) leaf spot of peanut (caused by *Cercospora arachidicola* (Ca) and *Cercosporidium personatum* (Cp), respectively). This was to characterize the differences and similarities in their epidemic behavior, to identify factors responsible for the differential development of the two diseases, and to determine whether current forecasting systems and reduced-input fungicide programs were equally applicable for each disease. This required a destructive sampling method in which more than 50,000 main stems were sampled over a 6-year period. Additionally, hundreds of thousands of leaflets were assessed in order to determine the onset, rate and abundance of each leaf spot over the course of each growing season. This research focused on two primary differences in the development of ELS and LLS: relative predominance of one compared with the other and time of disease onset. While in previous studies, researchers hypothesized putative factors responsible for some of the variability in these two response variables, the relative effect for each disease has generally not been documented. Hence, the studies reported here are the first to document, specifically, how each disease responds to multiple, interacting factors across a wide range of environments. More importantly, we have provided data to support the mechanisms responsible for much of the variability in the behavior of the two diseases.

Based on these data, we now have greater certainty as to why ELS onset is generally 2 to 4 weeks prior to LLS onset. More importantly, we can demonstrate why the onset of LLS can be at the same time or prior to ELS onset. The factors that affect disease onset are closely linked to our ability to predict which disease will be prevalent in a given field, and include rotation, inoculum level, planting date, and cultivar. However, some factors did not affect both leaf spots equally, and when considered alone, none of these factors fully explained the variability in prevalence and onset of ELS and LLS. However, when taken together, they provide a deeper framework to understand and predict the prevalence and onset of

each disease. For example, the onset of ELS was consistently earlier than LLS in fields treated with peanut residue infested with both pathogens, and this was not influenced by cultivar nor by the presence of the LLS pathogen. In contrast, the onset of LLS was significantly delayed by the presence of the ELS pathogen, and cultivar significantly affected the onset of LLS by as much as 25 days. However, the onset of LLS was prior to or at the same time as ELS in fields with a history of LLS regardless of rotation, cultivar or planting date. Initial inoculum source, based on use of infested peanut residue or knowledge of historical prevalence, was the most important determinant of which disease was predominant. However, the combination of later-planted peanuts and cultivars more susceptible to LLS (e.g., Florunner and Georgia Valencia), generally resulted in more LLS lesions in plots inoculated with Ca alone or Ca + Cp, and this tended to also occur in fields with a history of ELS. Furthermore, in field plots treated with peanut residue infested with both pathogens and greenhouse studies co-inoculations co-inoculated with Ca + Cp indicate that there was a negative association between the two diseases, but that the development of LLS was more negatively affected by the presence of ELS than ELS by the presence of LLS. We hypothesize that there is a degree of competitive exclusion in which the ELS pathogen outcompetes the LLS pathogen early in the season, and that this may be an additional factor that influences the prevalence and the delay in the onset of LLS.

Initial studies conducted from 2010 to 2012 to evaluate prescription fungicide programs confirmed that fields had primarily ELS or LLS, and that this was linked, in every case, to the historical prevalence at each location. An important finding from these trials was that the predominance and onset of each disease was greatly dependent on the historical prevalence of each disease for a given location, and not necessarily the disease risk overall. This strongly suggested that the initial inoculum is generally from a local source and is one of the greatest contributors to the differences in the epidemiological behavior of each disease. Furthermore, although inoculum appeared to be the overwhelming factor in these trials, plots planted to the cultivar New Mexico Valencia C, in addition to the use of fewer fungicide applications, tended to result in more ELS lesions than LLS lesions in some situations, suggesting that a differential level of resistance to each leaf spot, and the intensity of the fungicide program (but not the

active ingredients evaluated), could be additional factors that influence the temporal dynamics of each disease.

With this in mind, multiple field trials were conducted from 2014 to 2016 to better understand the effect of inoculum, planting date and cultivar and associated interactions on the development of ELS and LLS. Inoculum studies were planted to the cultivar Georgia-06G and plots were treated with peanut residue infested with Ca alone, Cp alone, a 50/50 mix of Ca + Cp and a non-treated control. Three planting dates (April, May and June) were evaluated across the same inoculum levels. Among others, variety evaluations included three of the most important cultivars grown in the southeastern US: Florunner (1970-1996), Georgia Green (1996-2008), and Georgia-06G (2006-present). Inoculum source was found to be the most important factor regulating the differences between the predominance and onset of each disease, but in situations where there was more Ca inoculum, there was an additive effect with the addition of planting date and cultivar. This was observed in that fields with a history of ELS, or plots treated with peanut residue infested with Ca or Ca + Cp, the prevalence of LLS tended to increase with later planting dates and in plots planted to cultivars with more susceptibility to LLS (e.g., Florunner and Georgia Valencia). Similarly, the onset of ELS was always prior to that of LLS in plots planted to Georgia-06G, but the difference in the time of onset of the two diseases decreased when plots were treated with peanut residue infested with Cp as well as with later planting dates and on cultivars more susceptible to LLS. In fields that had a prior history of LLS, the onset of LLS was at the same time or prior to that of ELS onset. The development of LLS tended to be more suppressed than the development of ELS when plots were treated with peanut residue infested with Ca + Cp in that there was a delay in the onset of LLS but not of ELS, and plants in these plots tended to have more ELS lesions than LLS lesions.

Ca and Cp were inoculated separately or together in two greenhouse trials on the historically dominant peanut cultivars mentioned above. Across genotypes, components of resistance were similar for Ca, but Georgia Green and Georgia-06G had significantly less sporulation on LLS lesions compared with Florunner and Georgia Valencia. Taken together with the field data, this suggests that the switch from Florunner to Georgia Green could have been partially responsible for the shift from LLS to ELS at

the regional scale. Co-inoculations of Ca + Cp resulted in a 33% reduction in the number of ELS lesions and a 73% reduction in the number of LLS when compared with Ca alone and Cp alone, respectively. Similar to the results from the field plots treated with peanut residue infested with Ca + Cp, this further indicated that Cp is more suppressed in the presence of Ca, but that Ca is less suppress by the presence of Cp. Field data collected form the inoculum trials were used to further elucidate the ecological association ELS and LLS. For both leaf spots, incidence progressed from the bottom to the top of the canopy, and the greatest number of lesions occurred in the mid-to-upper part of the canopy. LLS tended to be more numerous in the mid- to -upper part of the canopy and ELS was more numerous in the bottom and top part of the canopy. Frequency of co-occurrence increased over time, indicating a neutral to positive association at the leaflet level. Covariance between the number of ELS and LLS lesions per leaflet indicated a weak, negative ecological association. Taken together, these data suggest that there is a negative ecological association between ELS and LLS, but that the development of ELS is less affected by LLS than vice versa.

From a management perspective, this work provides novel insight into the utility of Peanut Rx to predict the onset of ELS and LLS. The predictive potential of Peanut Rx was demonstrated based on 168 cases (unique combinations of Peanut Rx factors). When regressed against risk points, leaf spot onset [time in days after planting (DAP) when either leaf spot reached 1% lesion incidence] was the best response variable with the best fit compared to final defoliation and stAUDPC of lesions per leaflet. However, risk points did not predict LLS onset as well as ELS onset. Results from multiple regression analysis confirmed that all Peanut Rx risk factors/predictor variables contribute to the variability of at least one component of the combined or separate epidemic of ELS and LLS. However, not all factors affected ELS and LLS equally. Historical leaf spot prevalence significantly contributed to the multiple regression model for predicting the onset and proportion of the epidemic attributed to LLS, and we suggest that it may be a useful pre-plant risk factor for predicting LLS epidemics.

By evaluating prescription fungicide programs in fields with differing levels of ELS and LLS, this work provides additional data to support the efficacy of these programs on runner and Valencia

peanut market types. More importantly, we demonstrated the potential to increase the efficacy of low-input fungicide regimes by adjusting the timing of fungicide applications to better align with the expected onset of each disease. As the onset of LLS was observed to be > 100 days after planting (DAP) in moderate risk fields with a history of LLS in prior studies conducted in 2011 and 2012, additional field trials were conducted in 2013 and 2014 to determine if leaf spot control could be enhanced by delaying fungicide timings to better match expected disease onset and provide better late season coverage. The delayed moderate and low-risk prescription fungicide programs based on Peanut Rx provided statistically better LLS control than their normal counterparts, but did not significantly improve yield. Additional studies in dryland fields with a history of LLS offered further proof that fungicide timings could be delayed up to 86 DAP without compromising disease control and yield. Although delayed applications did not result in a gain in yield, these results suggest that suppression of LLS with prescription fungicide programs can be enhanced by modifying initial fungicide applications to correspond with actual disease onset.

An additional aspect of this research was to characterize the development of both leaf spot diseases and peanut rust (caused by *Puccinia arachidis*) on runner and Valencia peanut market types in Haiti, to quantify associated yield losses, and to determine the most appropriate low-input fungicide regime for smallholder farmers in Haiti. While all three diseases were present in Haiti, LLS and rust were the most consistent yield-limiting factors in these studies. In general, disease severity and yield loss increased with decreasing fungicide applications. For runner types, two applications made at 60 and 88 DAP significantly reduced leaf spot severity and improved yield; four applications were comparable to six, but generally not significantly different from three. For Valencia types, at least two applications were generally required to significantly reduce rust and leaf spot and increase yield; regardless of timing, three or more applications resulted in the highest yields. Percent yield loss averaged 83 and 43% for the local Haitian runner and Georgia-06G, but was reduced to 64 and 43%, respectively, by recovering the pods from the soil that were lost when uprooting the plants at harvest. Relative yield loss for the local Haitian Valencia and New Mexico Valencia A ranged from 28 to 34%. These results document that limited

fungicide applications have the ability to significantly decrease disease and increase yield; however, final recommendations will depend on a more in-depth analysis of practical and economic considerations.

APPENDIX 1 (CHAPTER 11):

WITHIN-CANOPY DISTRIBUTION AND ECOLOGICAL ASSOCIATION OF EARLY AND LATE LEAF SPOT OF PEANUT[1]

[1]Fulmer, A.M., Kemerait, R.C., Brenneman, T.B., Scherm, H., Cantonwine, E.G., Culbreath, A.K., and Stevenson, K.L. 2017. To be submitted to *Peanut Science*.

ABSTRACT

Studies comparing the epidemiology of early (ELS) and late leaf spot (LLS) of peanut, caused by *Cercospora arachidicola* (Ca) and *Cercosporidium personatum* (Cp), respectively, have generally focused on the onset, rate and final severity of each disease. However, the distribution of each disease within the canopy and ecological association between the two diseases has not been characterized. Detailed within-canopy disease assessments were made in multiple field trials during 2014 and 2015 featured untreated plots with differing initial inoculum sources and multiple assessment dates where lesion counts were made for every leaflet at each node position on five main stems sampled per plot. This resulted in a total of 3,317 main stems and 272,518 leaflets, of which 28,380 were infected with one or both diseases. For both leaf spots, incidence progressed from the bottom to the top of the canopy, and the greatest of number of lesions occurred in the mid- to upper part of the canopy. LLS tended to be more prevalent in the mid- to upper part of the canopy and ELS was more prevalent in the bottom and top part of the canopy. Jaccard index values (where 0 = negative association and 1 = positive association), increased with each assessment date, and values at the final date ranged from 0.39 to 0.72 (0.56 average) across trials and inoculum treatments. There was a weak, negative association between the number of ELS and LLS lesions at the leaflet level. When analyzed across all trials, inoculum treatments and assessment dates, the correlation coefficient was -0.08 ($P < 0.0001$). Taken together, the results from this study suggest that although there was a similar distribution of each disease in the peanut canopy, a degree of competitive exclusion between ELS and LLS may occur at the leaflet level.

INTRODUCTION

Early (ELS) and late (LLS) leaf spot are two of the most important foliar diseases of peanut worldwide, and are caused by *Cercospora arachidicola* Hori (*Ca*) and *Cercosporidium personatum* (Berk & M.A. Curtis) Deighton (*Cp*) (Shokes and Culbreath, 1997). Both are globally distributed, polycyclic diseases that can coexist in the same field and even on the same leaflet and result in yield losses greater than 50% (Backman and Crawford, 1984; McDonald et al., 1985). Although they are similar in many respects, there are two unique characteristics about this disease complex that have yet to be fully explained. Firstly, the onset of ELS generally precedes that of late leaf spot by 2 to 4 weeks, although both can range from as early as 30 days after planting (DAP) to as late as the end of the season (~140 DAP) (Chapters 3, 4, 5, 6 and 8). Secondly, the relative abundance of each leaf spot tends to vary in time and space. In Georgia, LLS was predominant from 1976 to the early 1990s; otherwise ELS has generally been the more prevalent (Cantonwine et al., 2008; Smith and Littrell, 1980). In recent years, the prevalence of LLS has increased in Georgia, and it is common to find fields that are predominantly ELS or LLS (Chapter 3).

We have recently demonstrated that there are several key factors that can affect differences in the prevalence and onset of each disease under irrigated field conditions. In order of decreasing importance, these include, inoculum source of each pathogen, differential levels of host resistance, and planting date (Chapter 4, 5, 6 and 7). While fungicide has an obvious effect on the onset of each disease, we have also shown that untreated plots tend to have more ELS than LLS when the inoculum source of each pathogen is present (Chapters 3, 4 and 5). However, because these factors have not fully explained the differences in the behavior of these diseases, it is likely that there are additional mechanisms involved.

Differences in the onset and the prevalence of each disease may result from a negative ecological association between the two pathogens. In ecological theory, there are three basic associations between pathogens occupying the same niche: negative, neutral or positive effects on co-occurring species (Ludwig and Reynolds, 1988). Negative associations are a result of interspecific competition between pathogens when resources are in short supply, and according to the competitive exclusion principle,

species sharing the same niche will eventually drive competitors to extinction, or exclusion from a given area (Fitt et al., 2006). From a plant pathology standpoint, occurrence and covariance are concepts that can be employed to characterize ecological associations between coexisting species (Turechek and Madden, 2000). The former refers to the frequency of association at a given scale such that a negative association would result in few cases in which both diseases co-occur at the same unit of scale, e.g., leaflet. The latter refers to the degree to which the increase or decrease of each disease is correlated. In the case of leaf spot, if an increase in the number of ELS lesions corresponds to a decrease in the number of LLS lesions, or vice versa, this would be considered a negative association (Turechek and Madden, 2000).

While much of the epidemiology of ELS and LLS is understood at the field scale, there is little information regarding the distribution of each disease within the peanut canopy, nor the resulting association of each disease at the individual leaflet scale. Researchers generally agree that each disease first appears in the lower part of the canopy and progresses up the canopy over the course of the season (Plaut and Berger, 1980). This is further illustrated by the fact the most common visual rating system (i.e., the Florida 1-10 scale) for leaf spot is based on this expectation (Chiteka et al., 1988). However, there has only been one study that has characterized the progression of leaf spot within the peanut canopy, and it was limited to three layers (bottom, middle and top) and to LLS alone (Plaut and Berger, 1980). Exactly how each disease is distributed within the canopy over time is unknown, particularly when the inoculum source of each pathogen may not be equally distributed in the field

Results from several greenhouse studies indicate that there could be a negative association between the two leaf spot pathogens at the leaflet scale. Monasterios de La Torre (1980) reported that when co-inoculations of *C. arachidicola* and *C. personatum* were compared with single inoculations with each pathogen alone on multiple genotypes, the number of ELS lesions was reduced on average by 30% compared with inoculations with *C. arachidicola* alone. In contrast, the number of LLS lesions was reduced on average by 94% compared with inoculations of *C. personatum* alone (Monasterios de La Torre, 1980). In similar studies, we recently reported that co-inoculations of *C. arachidicola* + *C.*

personatum on four different peanut cultivars resulted in an average reduction of 33% in number of ELS lesions and a 73% reduction in the number of LLS when compared with *C. arachidicola* alone and *C. personatum* alone (Chapter 7).

A better understanding of the mechanisms involved in the shifts in prevalence would contribute a broader understanding to the nature of co-existing diseases in general, but may also have real practical value for peanut growers. The ability to predict which disease is prevalent in a field may be a potential management tool to better understand risk. For example, if a field is known to be predominantly ELS, it is possible that a cultivar that is more LLS susceptible and ELS resistant could be selected as a tool to reduce the disease risk. Also, we have recently shown that the onset of both diseases tends to be later in fields with a history of LLS, and this knowledge can be used to increase the efficacy of reduced fungicide programs by delaying the initial application date (Chapter 11). Lastly, confirmation of the location of each leaf spot within the canopy overtime may provide better insight into potential spray coverage.

Therefore, the objectives of this study were to characterize i) the within-canopy distribution of each leaf spot; ii) the frequency of association at the leaflet and plant level; and iii) the covariance of the number of lesions at the leaflet and plant scale. Because the within-field initial inoculum source is a critical factor in the occurrence and severity of each disease, these studies were based on field trials intentionally treated infested peanut residue harboring each leaf spot pathogen.

MATERIALS AND METHODS

Data collection. Data used for these analyses were previously collected from field trials designed to compare the temporal dynamics of ELS and LLS at the plot level (Chapters 4 and 5). These trials were conducted in 2014 and 2015 at the University of Georgia Coastal Plain Experiment Station in Tifton and the Vidalia Onion and Vegetable Research Center located in Reidsville, Georgia. At the Coastal Plain Experiment Station main campus farm in Tifton, studies were conducted at the FireTower field during 2014 and 2015 and the Ponder farm in TyTy, Georgia in 2015. All trials were set up as a split-block design with four replications, and included two treatment factors: inoculum source (referred to as inoculum) and tillage, or inoculum and planting date (see Chapters 4 and 5 for details). This resulted

in a total of four trials used from studies conducted at Reidsville (each year two separate trials inoculum/tillage and inoculum/planting date), and three trials from Tifton (inoculum/tillage).

Inoculum treatments consisted of four combinations of peanut residue infested with *C. arachidicla* (*Ca*) alone, *C. personatum* (*Cp*) alone, a 50/50 mix of *Ca* + *Cp* and a non-treated control. Peanut residue was collected from fields in Tifton, with severe epidemics of either ELS or LLS (>90% of the lesions counted attributed to either disease), and stored separately. In most cases, the residue came from untreated plots that had nearly 100% severity (a "10" on the Florida 1-10 scale). However, in 2014 in Reidsville, the *Cp* inoculum came from a field with ~ 80 % LLS severity, and in 2015, the *Ca* inoculum at Reidsville came from a trial with ~ 70 % ELS severity. Peanut residue including leaflets, petioles and stems, was collected within a few days after harvesting peanuts (late October or November) and stored in a large peanut wagon under an open shed for 4 to 6 weeks. In December, plots (1.8 × 7.6 m) were harrowed and rototilled, and dry peanut residue (~4.5 kg) for each treatment was spread evenly across the middle (4.5 m) section of each plot. For the spring and strip tillage plots, the peanut residue applied to the plots was allowed to overwinter on the surface of the soil. Each plot was bordered by two residue free buffer rows of the peanut cultivar Georgia-06G on each side, and separated by 3 m between each block.

In order to compare treatments across studies, only plots that were strip tilled and planted in May were selected. For the strip tillage treatment, a KMC (Kelley Manufacturing Co., Tifton, GA) rip/strip rig with two shanks with a dragging depth of 45.7 cm. was used to prepare two rows (~ 10 to 15 cm wide) for seeding. The strip tillage treatment was meant to serve as a positive control since it would leave the greatest amount of residue on the soil surface, and for that reason, no cover crop was planted during the winter months. All plots consisted of two rows spaced 0.91 m apart and were planted to the cultivar Georgia-06G at a rate of 6 seed per 30.5 cm. Plots were planted at Reidsville on 22 May 2014 and 21 May 2015, at the FireTower field on 19 May 2014 and 13 May 2015 and at the Ponder farm on 29 May 2015. At the time of planting, all plots received an in-furrow application of phorate (Thimet 20 G, AMVAC Chemical Corporation, Los Angeles, CA) at rate of 1.12 kg a.i./ha.

Disease assessment. Beginning approximately 30 days after planting (DAP), plots were visually inspected for leaf spot symptoms. Upon detecting the first leaf spot symptoms, a more intensive evaluation was initiated and continued approximately on a bi-weekly schedule until the end of the peanut season (harvest). Five main stems per plot were arbitrarily selected for destructive sampling and transported to the lab where the number of present and missing leaflets, and the number of ELS and LLS lesions per leaflet were counted for each node. ELS and LLS were differentiated by symptoms and signs associated with each causal pathogen. In 2014, main stems were sampled at the following days after planting (DAP): FireTower – 42, 56, 70, 89, 99, 113, 126, 143; Reidsville – 32, 42, 60, 74, 89, 103, 116, 132, 144. In 2015, locations were sampled at the following DAP: FireTower – 40, 54, 66, 78, 93, 103, 117, 131, 146; Ponder – 42, 53, 61, 76, 94, 108, 122, 140; Reidsville – 41, 53, 67, 82, 96, 111, 124, 148.

Characterizing within-canopy distribution. Because the number of nodes on the main stem changes over time and varies among plants, the distribution of each leaf spot on the main stem was compared over time by standardizing the position of each node on the main stem. Briefly, as part of the disease assessment methodology, the node position of each leaf was labeled starting from the bottom to the top of the main stem in numerical order, i.e., 1, 2, 3…such that the highest integer was at the top of the main stem. The relative node position was calculated as a proportion where each node on each main stem was divided by the greatest integer (the node position at the top of the main stem), such that the value closest to 0 represented the lowest part of the canopy and the value of 1 represented the highest part of the canopy.

Based on the mean number of nodes that observed on the main stem of a mature Georgia-06G peanut plant, 20 categories of node positions were calculated in order to make relative comparisons within the canopy over time. These included increments of 0.05 from 0 to 1 such that any proportion falling within each specified range was included. The reason for the creating the 20 node positons was in order to graph and analyze discrete positions on the main stem. For each inoculum treatment and assessment date (DAP), scatter plots were created to display the number of ELS and LLS lesions per leaflet at each node position. Other plots were created to display the number of lesions per leaflet at each node position

across years, locations, inoculum treatments and assessment dates. To compare the progression of ELS and LLS incidence over time across years and locations, rating dates were segregated into five categories based on the number of days after planting (DAP) as follows: 1 = 40 to 60 DAP, 2 = 60 to 80 DAP, 3 = 80 to 100 DAP, 4 = 100 to 120 DAP and 5 = 120 to 140 DAP.

To determine the effect of node position on LLS predominance (proportion of the total leaf spot epidemic attributed to LLS calculated by dividing the number of LLS lesions per leaflet by the combined number of ELS and LLS lesions per leaflet), data were subjected to linear mixed model analysis of variance using PROC GLIMMIX (SAS v.9.4, SAS Institute, Cary, NC). The model was analyzed as a split plot design, with inoculum treatment as the main plots and node positon as the sub-plots, and both were considered fixed effects. Replication, year, location and replication × inoculum were included as random effects. The Kenward-Roger option was used to adjust the denominator degrees of freedom, and differences in the least square means were tested by Tukey's multiple comparisons test.

Interspecific association. The frequency at which the diseases were found together on the same leaflet or on the same plant was analyzed with the Jaccard index (Ludwig and Reynolds, 1988). This index represents the proportion of leaflets where both diseases occur together relative to the number of leaflets where at least one of the leaf spot diseases is found. This calculation requires binary data (incidence) and does not include leaflets where neither disease occurred (Ludwig and Reynolds, 1988). Numerically, the calculation for each leaflet was as follows: $J=a/(a + b + c)$, where a = leaflets with the co-occurrence of ELS and LLS, b = leaflets with ELS alone, and c = leaflets with LLS alone. The general interpretation of the values (range 0 to 1) calculated by the Jaccard index is that smaller proportions suggest a negative association and higher proportions suggest a positive association (Turechek and Madden, 2000).

Jaccard associations between ELS and LLS were calculated by year, location, inoculum treatment and assessment date. Due to the later onset of LLS, dates prior to 75 DAP were not utilized in these analyses as they would have always resulted in a Jaccard value of 0. Jaccard values were not calculated for each node position due to several reasons. First, the within-canopy distribution indicated no obvious

clustering pattern between diseases (see Supplementary Tables S11.1 to S11.20). Second, because the onset of LLS was 2 to 4 weeks later than ELS onset, the assessment date was a very obvious factor that had to be included. Thirdly, for the assessment dates made later in the season, heavy defoliation resulted in many node positions where neither leaf spot occurred. Taken together, it was not possible to uniformly compare each node position for each assessment date.

Interspecific covariance. To determine if the intensity of each disease tended to increase/decrease with the corresponding increase/decrease of the other leaf spot disease, lesion counts per leaflet were analyzed with Spearman's rank correlation statistic using PROC CORR in SAS (SAS v.9.4) (Ludwig and Reynolds, 1988; Turechek and Madden, 2000). The covariance between ELS and LLS lesions per leaflet were first analyzed by year, location, inoculum treatment and assessment date. Analyses were also conducted across years, locations and rating dates for each inoculum treatment, and across years, locations, inoculum levels and assessment dates. For all analyses, PROC SGPANEL was used to generate scatter plots to display the association between the lesion counts for each disease. Due to the later onset of LLS, data collected prior to 75 DAP were not included in these analyses.

RESULTS

Out of a total of 3,317 main stems sampled across years, locations, inoculum treatments and assessment dates, 2,345 had one or more lesions of ELS and/or LLS. Out of 272,518 leaflets evaluated, 31,637 leaflets had one or more lesions of ELS and/or LLS. After removing data collected prior to 75 DAP, a total of 28,380 leaflets, obtained from 1,519 main stems, were utilized for the frequency and correlation analyses.

Within-canopy distribution. Overall, the mean incidence of ELS and LLS increased from the lower part of the canopy with each increasing time frame (Fig. 11.1). For both diseases, lesions did not begin to progress up the main stem until 80 to 100 DAP. Progress of disease up the main stem was similar for all plots treated with infested peanut residue, but non-inoculated plots tended to be located slightly lower in the canopy for each time point (Fig. 11.1).

Based on the visual inspection of graphs, the greatest number of lesions per leaflet of each disease were observed in the upper region of the canopy (node position 0.6 to 0.8) regardless of inoculum treatment (Fig. 11.2). The number of lesions per leaflet tended to decline at the top of the canopy, which was more apparent for LLS. For ELS, there were no major differences in distribution among inoculum treatments, but the fewest lesions were observed on plants from plots treated with peanut residue infested with *Cp* (Fig. 11.2). For LLS, the least number of lesions occurred in plots treated with peanut residue infested with *Ca*, and the greatest number of lesions occurred in plots treated peanut residue infested with *Cp* or *Ca* + *Cp*. Residue-free plots tended to have more LLS in the lower portion of the canopy than plots treated with infested peanut residue, but this did not appear to be the case for ELS (Fig. 11.2).

Predominance was significantly affected ($P < 0.05$) by inoculum treatment and node positon on the main stem, and there was no significant interaction between inoculum treatment and node position ($P = 0.9387$). Generally, the proportion of LLS increased with increasing node position up to node position 0.7 and began to decline after that (Fig. 11.3). Based on Tukey's HSD means separation, the node positions 0.6 to 0.8 had significantly higher proportions of LLS, and the node positions 0.1 to 0.35 had significantly lower proportions of LLS (data not shown). All other node positions were not significantly different. The effect of inoculum treatment on predominance was the same as previously described at the plot level (Chapter 4). Briefly, LLS predomance and Tukey mean separations (letters in brackets) were as follows: plots treated with peanut residue infested with *Cp* had significantly higher LLS predominance (0.56 [a]) compared with plots treated with peanut residue infested with *Ca* + *Cp* (0.38 [b]) and *Ca* (0.19 [c]); the latter two inoculum treatments had numerically or statistically lower LLS predomance compared to residue-free plots (0.50 [ab]).

Interspecific association. Across all years and locations, the association of ELS and LLS at the leaflet level increased over time regardless of inoculum treatment (Table 11.1), indicating a neutral to positive association towards the end of the season. Leaflets from plots treated peanut residue infested with *Ca* generally had the lowest Jaccard index value prior to 100 DAP, but afterwards were comparable to other inoculum treatments. There was a much stronger association between ELS and LLS at the plant

level. When averaged across all years, locations, inoculum treatments and assessement dates (i.e., DAP>70), the average Jaccard index value was 0.83 (0.26 standard deviation) across 1,519 main stems (1,271 stems with both diseases, 235 stems with ELS alone, 13 stems with LLS alone).

Interspecific covariance. Overall, there tended to be a weak, negative association between the number of ELS and LLS lesions at the leaflet level. Although there was some variability among years, locations, inoculum treatments and assessment dates, the general trend was that the highest lesion counts for each disease occurred when the counts for the other leaf spot were lowest (Figs. 11.4 to 11.8). For plots treated with peanut residue infested with *Ca*, there were three assessment dates with a significant positive association, and one assessment date for plots treated with peanut residue infested with *Ca* + *Cp* (Table 11.2). Otherwise, all significant associations at the leaflet level were negative (Table 11.2). Plots treated with peanut residue infested with *Ca* had the fewest number (7) of negative associations and plots treated with *Cp* had the greatest number (17) of assessment dates with significant negative associations (Table 11.2). Untreated plots were similar to those treated with *Cp*, and resulted in 16 rating dates with significant negative associations. (Table 11.2). Plots treated with peanut residue infested with *Ca* + *Cp* were between the extreme of *Ca* and *Cp*, and resulted in 13 assessment dates with significant negative associations (Table 11.2). When analyzed across years, locations and assessment dates, the correlation coefficients for plots treated with peanut residue infested with *Ca*, *Ca* + *Cp*, *Cp* and the untreated control (none) were -0.05, -0.14, -0.18 and -0.24, respectively, and all had a *P* value of <0.0001 (Fig. 11.9). When the data were analyzed across all years, locations, inoculum treatments and assessment dates the association between ELS and LLS was still negative, but the correlation coefficient was closer to zero (r = -0.08, $P < 0.0001$) (Fig. 11.10), indicating more of a neutral association.

DISCUSSION

Results from this study demonstrate that ELS and LLS have a similar distribution within the peanut canopy over time, and that both pathogens were capable of infecting leaflets at all node positions. Moreover, the two leaf spots tended to co-exist on the same leaflet more frequently than when either disease occurred alone. The only exception was during early assessment dates, but much of this is likely

explained by the later onset of LLS (89 to 100 DAP) compared with ELS onset (58 to 78 DAP) (Chapters 4 and 5). As a result, this was also one likely reason why the proportion of LLS was greater in the mid- to upper canopy. However, later onset cannot explain the lower proportion of LLS in the upper part of the canopy. Taken together, these results suggest that there was no niche differentiation between the two diseases with respect to the node position within the lower to mid part of the peanut canopy, but that top part of the canopy may favor ELS more than LLS. Across node positions there was strong evidence for a neutral to positive frequency of association at the leaflet level. However, while the two diseases tend to co-occur more often than not, our results indicate that there is a weak to moderate negative association between the number of ELS and LLS lesions at the leaflet level, suggesting that there may be a degree of competitive exclusion in regards to resource utilization (i.e., the leaflet). There is very limited published data regarding the distribution of either disease within the peanut canopy, and to the best of our knowledge this the first study to evaluate the ecological association between ELS and LLS.

Most field studies rely upon naturally occurring epidemics. In the present study we were able to ensure that the inoculum of each leaf spot pathogen was in the field by treating plots with infested peanut residue. Because was applied in the late fall and allowed to overwinter in the field, this method provided a more realistic situation in which to study the progression within the peanut canopy compared with inoculating young peanut plants with a spore suspension. As such, we found that lesions of both diseases initially appeared on leaflets in the lower canopy, and over time, symptoms progressed up the main stem. This partially corroborates previous work that demonstrated that LLS generally progress from the bottom to the top of the canopy (Plaut and Berger, 1980). It has been assumed that both diseases have a similar distribution within the canopy. While our results confirm that this was indeed the case in regards to incidence, the distribution of the number of lesions per leaflet within the canopy is somewhat different for the two diseases, and contradicts previous findings for LLS.

Plaut and Berger (1980) reported the greatest LLS severity in the lower canopy, slightly less in the middle canopy, and the least in the upper canopy. In contrast, we found the highest density of lesions of both ELS and LLS in the mid- to upper canopy, slightly less in the top part of the canopy, and the

fewest in the lowest part of the canopy. We also report that the number of LLS lesions and the proportion of LLS lesions is much greater in the mid- to upper part of the canopy compared with ELS. However, it should be noted that the highly susceptible cultivar Florunner was utilized in the study conducted by Plaut and Berger (1980), whereas in our studies, we used the slightly more resistant (Chapters 6 and 7) cultivar Georgia-06G. Furthermore, our studies included the use of phorate (Thimet 20 G) to reduce thrips related feeding damage. There is some evidence to suggest that phorate can lead to premature defoliation in the lower canopy (A. Culbreath, personal communication). While this has yet to be substantiated, this could be one reason for lower lesion numbers observed in the lower part of the peanut canopy.

The fact that LLS predominance increased with higher node positions, reached a peak at the 0.7 node position, and then began to decline towards the upper part of the canopy (Fig. 11.3), suggests that there may be an advantage for ELS at the top of the canopy. While differences in microenvironment or leaf age susceptibility are two possible explanations to account for this, there is little evidence to substantiate either hypothesis. It is certain that leaflets at the top of the canopy would have been the youngest on the plant. As such, these leaflets are exposed to more sunlight as well as other environmental effects than leaflets within the canopy. More likely however, as previously pointed out in Chapter 7, the ELS pathogen may have had an advantage due to differences in host resistance. However, more research would be needed to determine whether these factors had an effect on LLS predominance within the peanut canopy.

Our results indicate that the frequency of association between the two leaf spot diseases tends to increase over time, regardless of inoculum treatment (Table 11.1). Lower Jaccard values signify that there is no association (dissociation), higher values (closer to 1) signify that the diseases co-occur more frequently (Turechek and Madden, 2000). In our study, Jaccard values at the final assessment dates at the leaflet level ranged from 0.39 to 0.72 across years, locations, inoculum treatments, with an average of 0.56. When compared with the plant level, at which there was a strong positive association (average Jaccard value of 0.86), the lower Jaccard value of 0.56 suggests that there may be more of a neutral to moderately positive association at the leaflet level.

Further evidence of competitive exclusion was provided by the results of the analysis of the covariance between the abundance of ELS vs. LLS lesions. Although the two diseases tended to coexist on the same leaflet, the number of lesions per leaflet of each leaf spot was generally the greatest when the lesions of the other disease were the lowest. These results strongly suggest that there is some degree of competitive exclusion between the two diseases (Fitt et al., 2006). Monasterios de La Torre (1980) reported that when co-inoculation of *C. arachidicola* and *C. personatum* was compared to single inoculations with each pathogen alone on multiple genotypes, the number of ELS lesions was reduced on average by 30% compared with inoculations with *C. arachidicola* alone. In contrast, the number of LLS lesions was reduced on average by 94% compared with inoculations of *C. personatum* alone (Monasterios de La Torre, 1980). In similar studies, we recently reported that co-inoculations of *C. arachidicola* + *C. personatum* on four different peanut cultivars resulted in an average reduction of 33% in number of ELS lesions and a 73% reduction in the number of LLS lesions when compared with *C. arachidicola* alone and *C. personatum* alone (Chapter 7). Co-occurrence has also been documented to inhibit the development of the ascochyta blight complex on pea. Le May et al. (2009) showed that simultaneous inoculations of pea plant with *Mycosphaerella pinodes* and *Phoma medicaginis* var. *pinodella* resulted in reduced lesion size and fewer pycnidia for each disease than when each pathogen was inoculated alone. The authors proposed that this was due to direct competition for space, antibiosis or induced plant resistance (Le May et al., 2009).

We hypothesize that a degree of competitive exclusion at the leaflet level may help explain some of the differences in the temporal dynamics of ELS and LLS. First, we briefly proposed that the increase in the frequency of ELS and LLS over time may be partially explained by the later onset of LLS. However, this does not address the question of why LLS onset is later. In these studies, the onset of ELS was 20 to 40 days prior to that of LLS, and therefore prior colonization by the ELS pathogen may inhibit the initial development of LLS. Therefore, it is possible that the later onset of LLS was delayed due a degree of competitive exclusion. Secondly, competitive exclusion could affect the prevalence of each

disease in a given field. However, additional research in more controlled conditions would needed to test these hypotheses.

In conclusion, this is the first study to report the dynamics of ELS and LLS within the peanut canopy and their association at the leaflet and plant level. Our results confirm that both diseases progress from the bottom to the top of the canopy and provide new information in that the greatest of number of lesions of both diseases occurred in the middle part of the canopy. Within the canopy, LLS tended to be more prevalent in the mid- to upper part of the canopy and ELS was more prevalent in the bottom and top part of the canopy. The data suggest that there was a neutral to slightly positive association between ELS and LLS at the leaflet level, but that there was a negative association with respect to the number of lesions per leaflet. Taken together, the results suggest the presence of competitive exclusion between ELS and LLS at the leaflet level. We hypothesize that this ecological association is an additional factor responsible for the later onset of LLS on the cultivar Georgia-06G when the inoculum source of both pathogens is present in the field, and may also contribute to differences in the prevalence of each leaf spot among fields.

ACKNOWLEDGMENTS

We thank personnel at the University of Georgia research stations for the assistance provided. Particularly, we thank John Paulk, Dewayne Dales, Andy Carter, Cliff Riner, Randy Hill, Denny Thigpin, Caleb Clements, Lina Flor Young, Jeannette Mixon, Jhen Bennett and Edna Shannon for technical support; and Xuelin Luo for statistical assistance.

LITERATURE CITED

Backman, P., and Crawford, M. 1984. Relationship between yield loss and severity of early and late leafspot diseases of peanut. Phytopathology 74:1101-1103.

Cantonwine, E., Culbreath, A., Holbrook, C., and Gorbet, D. 2008. Disease progress of early leaf spot and components of resistance to *Cercospora arachidicola* and *Cercosporidium personatum* in runner-type peanut cultivars. Peanut Sci. 35:1-10.

Chiteka, Z., Gorbet, D., Shokes, F., Kucharek, T., and Knauft, D. 1988. Components of resistance to late leafspot in peanut. I. Levels and variability-implications for selection. Peanut Sci. 15:25-30.

Fitt, B. D., Huang, Y.-J., van den Bosch, F., and West, J. S. 2006. Coexistence of related pathogen species on arable crops in space and time. Annu. Rev. Phytopathol. 44:163-182.

Le May, C., Potage, G., Andrivon, D., Tivoli, B., and Outreman, Y. 2009. Plant disease complex: antagonism and synergism between pathogens of the Ascochyta blight complex on pea. J. Phytopathol. 157:715-721.

Ludwig, J. A., and Reynolds, J. F. 1988. Statistical Ecology: A Primer in Methods and Computing. John Wiley & Sons, New York.

McDonald, D., Subrahmanyam, P., Gibbons, R., and Smith, D. 1985. Early and Late Leaf Spots of Groundnut. Information Bull. No. 21. ICRISAT, Patancheru.

Monasterios de La Torre, T. 1980. Genetic resistance to Cercospora leafspot diseases in peanut (*Arachis hypogaea* L.). Ph.D. Dissertation. University of Florida, Gainesville.

Plaut, J., and Berger, R. 1980. Development of *Cercosporidium personatum* in three peanut canopy layers. Peanut Sci. 7:46-49.

Shokes, F., and Culbreath, A. K. 1997. Early and late leaf spots. Pages 17-20 in: Compendium of Peanut Diseases, 2nd Ed. N. Kokalis-Burelle, D. Porter, R. Rodriguez-Kabana, D. Smith and P. Subrahmanyam, eds. American Phytopathological Society Press, St. Paul, MN.

Smith, D. H., and Littrell, R. H. 1980. Management of peanut foliar diseases with fungicides. Plant Dis. 64:356-361.

Turechek, W., and Madden, L. 2000. Analysis of the association between the incidence of two spatially aggregated foliar diseases of strawberry. Phytopathology 90:157-170.

Table 11.1. Frequency of early (ELS) and late (LLS) leaf spot and Jaccard association index values at the leaflet level for each year, location, inoculum level and day after planting (DAP).

Year	Location	Inoculum[v]	DAP	*n*[w]	ELS+LLS[x]	ELS[y]	LLS[y]	Jaccard[z]	SE[z]
2014	FireTower	Ca	89	98	2	95	1	0.02	0.03
			99	256	18	234	4	0.07	0.02
			113	323	112	197	14	0.35	0.04
			126	339	204	117	18	0.60	0.08
		Ca+Cp	89	115	9	99	7	0.08	0.02
			99	285	45	212	28	0.16	0.05
			113	398	230	130	38	0.58	0.05
			126	360	235	67	58	0.65	0.07
		Cp	89	113	11	56	46	0.10	0.02
			99	311	88	97	126	0.28	0.04
			113	421	253	43	125	0.60	0.06
			126	335	213	25	97	0.64	0.09
		None	89	28	1	21	6	0.04	0.02
			99	239	42	140	57	0.18	0.03
			113	435	208	107	120	0.48	0.06
			126	446	270	71	105	0.61	0.13
2014	Reidsville	Ca	89	205	6	196	3	0.03	0.02
			103	436	9	418	9	0.02	0.01
			116	811	87	711	13	0.11	0.03
			133	560	379	174	7	0.68	0.03
		Ca+Cp	89	201	1	191	9	0.00	0.00
			103	377	21	344	12	0.06	0.01
			116	856	173	654	29	0.20	0.04
			133	593	385	187	21	0.65	0.08
		Cp	89	165	19	119	27	0.12	0.06
			103	295	41	179	75	0.14	0.03
			116	874	221	535	118	0.25	0.02
			133	636	454	113	69	0.71	0.06
		None	89	70	1	63	6	0.01	0.03
			103	195	21	125	49	0.11	0.02
			116	801	166	492	143	0.21	0.04
			133	955	586	187	182	0.61	0.04
2015	FireTower	Ca	93	207	14	190	3	0.07	0.04
			103	324	108	181	35	0.33	0.12
			117	269	127	114	28	0.47	0.11
			131	212	142	59	11	0.67	0.05
		Ca+Cp	93	202	46	108	48	0.23	0.07
			103	370	189	54	127	0.51	0.08
			117	340	236	56	48	0.69	0.14
			131	224	135	47	42	0.60	0.08

		Cp	93	232	53	95	84	0.23	0.03
			103	333	169	50	114	0.51	0.07
			117	294	181	34	79	0.62	0.09
			131	151	99	29	23	0.66	0.05
		None	93	153	20	121	12	0.13	0.04
			103	368	137	106	125	0.37	0.10
			117	395	250	45	100	0.63	0.16
			131	255	183	36	36	0.72	0.03
2015	Ponder	Ca	76	97	2	93	2	0.02	0.02
			94	253	65	182	6	0.26	0.05
			108	383	237	129	17	0.62	0.05
			122	64	26	35	3	0.41	0.14
		Ca+Cp	76	81	2	69	10	0.02	0.02
			94	249	106	123	20	0.43	0.10
			108	351	224	78	49	0.64	0.02
			122	70	31	35	4	0.44	0.10
		Cp	76	93	6	55	32	0.06	0.03
			94	234	107	48	79	0.46	0.02
			108	299	182	17	100	0.61	0.10
			122	93	38	38	17	0.41	0.08
		None	76	64	2	58	4	0.03	0.01
			94	219	79	121	19	0.36	0.14
			108	440	266	82	92	0.60	0.08
			122	120	67	41	12	0.56	0.04
2015	Reidsville	Ca	82	139	3	133	3	0.02	0.02
			96	295	55	144	96	0.19	0.03
			111	739	323	160	256	0.44	0.08
			124	835	468	129	238	0.56	0.04
		Ca+Cp	82	143	14	113	16	0.10	0.04
			96	409	82	103	224	0.20	0.06
			111	686	259	111	316	0.38	0.06
			124	639	334	75	230	0.52	0.05
		Cp	82	173	23	96	54	0.13	0.05
			96	563	108	77	378	0.19	0.03
			111	824	262	68	494	0.32	0.05
			124	718	372	46	300	0.52	0.06
		None	82	98	6	78	14	0.06	0.01
			96	236	32	85	119	0.14	0.04
			111	920	294	77	549	0.32	0.06
			124	992	385	38	569	0.39	0.08

[v] Inoculum treatments consisted of plots amended with peanut residue infested with *Cercospora arachidicola* (Ca), *Cercosporidium personatum* (Cp), 50/50 mix of Ca + Cp or none.

[w] Number of leaflets used in the analysis.

[x] Number of leaflets with one or more lesions of early (ELS) and late (LLS) leaf spot (co-occurrence).

[y] Number of leaflets with ELS or LLS only.

[z] Jaccard index = proportion of leaflets where ELS and LLS co-occurred in relation to the number of leaflets where at least ELS or LLS is found. Calculated as follows: Both/(Both + ELS +LLS). SE = standard error of the mean for the Jaccard index.

Table 11.2. Spearman's correlation coefficients between early (ELS) and late (LLS) leaf spot lesions per leaflet in plots inoculated with differing combinations of peanut residue infested with each leaf spot pathogen in field trials conducted in Tifton and Reidsville, Georgia, during 2014 and 2015.

			Inoculum (infested peanut residue)[w]							
			Ca		Ca + Cp		Cp		None	
Year	Location[x]	DAP[y]	r^z	*P*-value	*r*	*P*-value	*r*	*P*-value	*r*	*P*-value
2014	FireTower	89	-0.20	0.0462	-0.29	0.0016	-0.72	<0.0001	-0.74	<0.0001
		99	0.05	0.3954	-0.22	0.0002	-0.42	<0.0001	-0.47	<0.0001
		113	0.11	0.0390	0.04	0.4148	0.02	0.6340	-0.12	0.0135
		126	-0.19	0.0006	-0.41	<0.0001	-0.24	<0.0001	-0.23	<0.0001
2014	Reidsville	89	-0.08	0.2265	-0.41	<0.0001	-0.45	<0.0001	-0.53	<0.0001
		103	-0.11	0.0209	-0.11	0.0386	-0.53	<0.0001	-0.62	<0.0001
		116	-0.05	0.1611	-0.06	0.0813	-0.26	<0.0001	-0.42	<0.0001
		133	0.07	0.1206	-0.02	0.6316	-0.13	0.0014	-0.19	<0.0001
2015	FireTower	93	0.01	0.8467	-0.45	<0.0001	-0.51	<0.0001	-0.10	0.2046
		103	-0.09	0.0891	0.06	0.2707	-0.14	0.0126	-0.22	<0.0001
		117	-0.08	0.2140	0.12	0.0301	-0.11	0.0643	-0.01	0.8429
		131	0.00	0.9658	-0.15	0.0281	-0.31	0.0001	-0.10	0.1041
2015	Ponder	76	-0.28	0.0064	-0.50	<0.0001	-0.72	<0.0001	-0.45	0.0002
		94	0.08	0.2051	-0.06	0.3695	-0.25	0.0001	0.12	0.0817
		108	0.14	0.0049	0.10	0.0514	-0.06	0.2953	-0.16	0.0007
		122	0.53	<0.0001	0.38	0.0011	-0.33	0.0012	-0.21	0.0213
2015	Reidsville	82	-0.29	0.0005	-0.37	<0.0001	-0.61	<0.0001	-0.65	<0.0001
		96	-0.58	<0.0001	-0.51	<0.0001	-0.37	<0.0001	-0.62	<0.0001
		111	-0.19	<0.0001	-0.22	<0.0001	-0.21	<0.0001	-0.13	<0.0001
		124	-0.21	<0.0001	-0.27	<0.0001	-0.21	<0.0001	-0.12	0.0002

[w] Inoculum treatments consisted of plots amended with peanut residue infested with *Cercospora arachidicola* (Ca), *Cercosporidium personatum* (Cp), 50/50 mix of Ca + Cp or none.
[x] FireTower field and Ponder farm located on the Tifton Coastal Plain Experiment station in Tifton, Georgia.
[y] DAP = Day after planting.
[z] Spearman rank correlation coefficient.

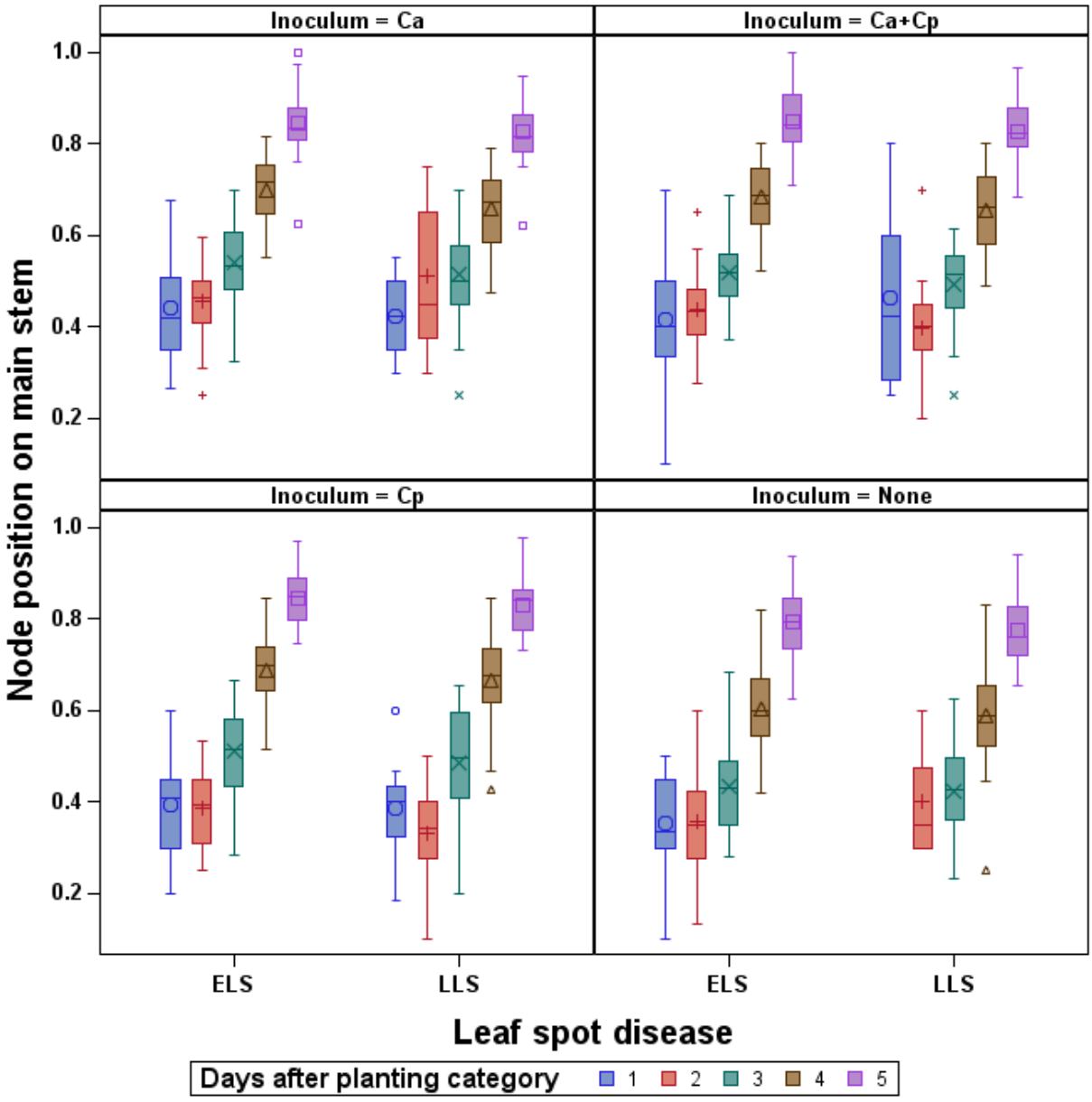


Fig. 11.1. Distribution of the incidence of early (ELS) and late (LLS) leaf spot on the main stem in plots treated with peanut residue infested with *Cercospora arachidicola* (Ca), *Cercosporidium personatum* (Cp), a 50/50 mixture of Ca + Cp and none (untreated control). Box plots were created with data across all years and locations. Node position values closest to 0 represent the lowest part of the canopy and the value of 1 represents the highest part of the canopy. Days after planting categories are labeled as follows: 1 = 40 to 60 DAP, 2 = 60 to 80 DAP, 3 = 80 to 100 DAP, 4 = 100 to 120 DAP and 5 = 120 to 140 DAP. Incidence data based on the average of five main stems per plot and four replications.

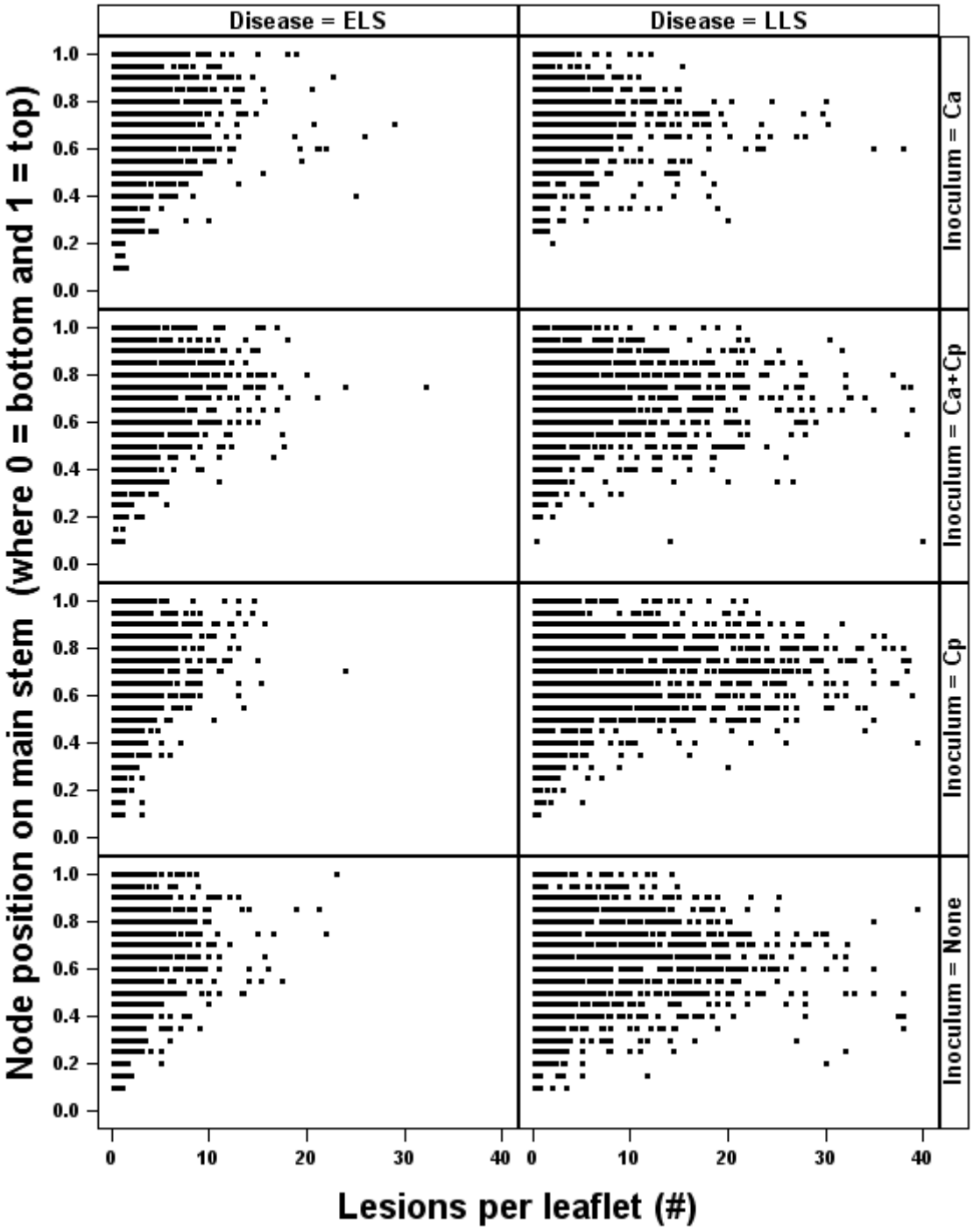


Fig. 11.2. Distribution of early (ELS) and late (LLS) leaf spot lesions on the main stem in plots treated with peanut residue infested with *Cercospora arachidicola* (Ca), *Cercosporidium personatum* (Cp), a 50/50 mixture of Ca + Cp and none (untreated control). Node position values closest to 0 represent the lowest part of the canopy and the value of 1 represents the highest part of the canopy. Data are averaged across all years, locations, and sampling dates.

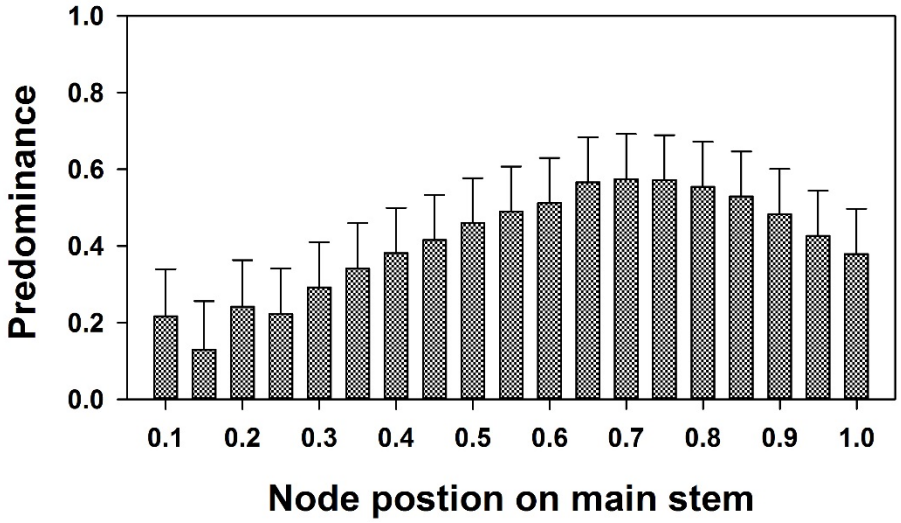


Fig. 11.3. Distribution of predominance (proportion of the total leaf spot epidemic attributed to late leaf spot (LLS)) for each node positon on the main stem. Node position values are based on the proportion of the total number of nodes; values closest to 0 represent the lowest part of the canopy and the value of 1 represents the highest part of the canopy. Data pooled across all years, locations, inoculum treatments and assessment dates. Error bars represent the standard error of the mean.

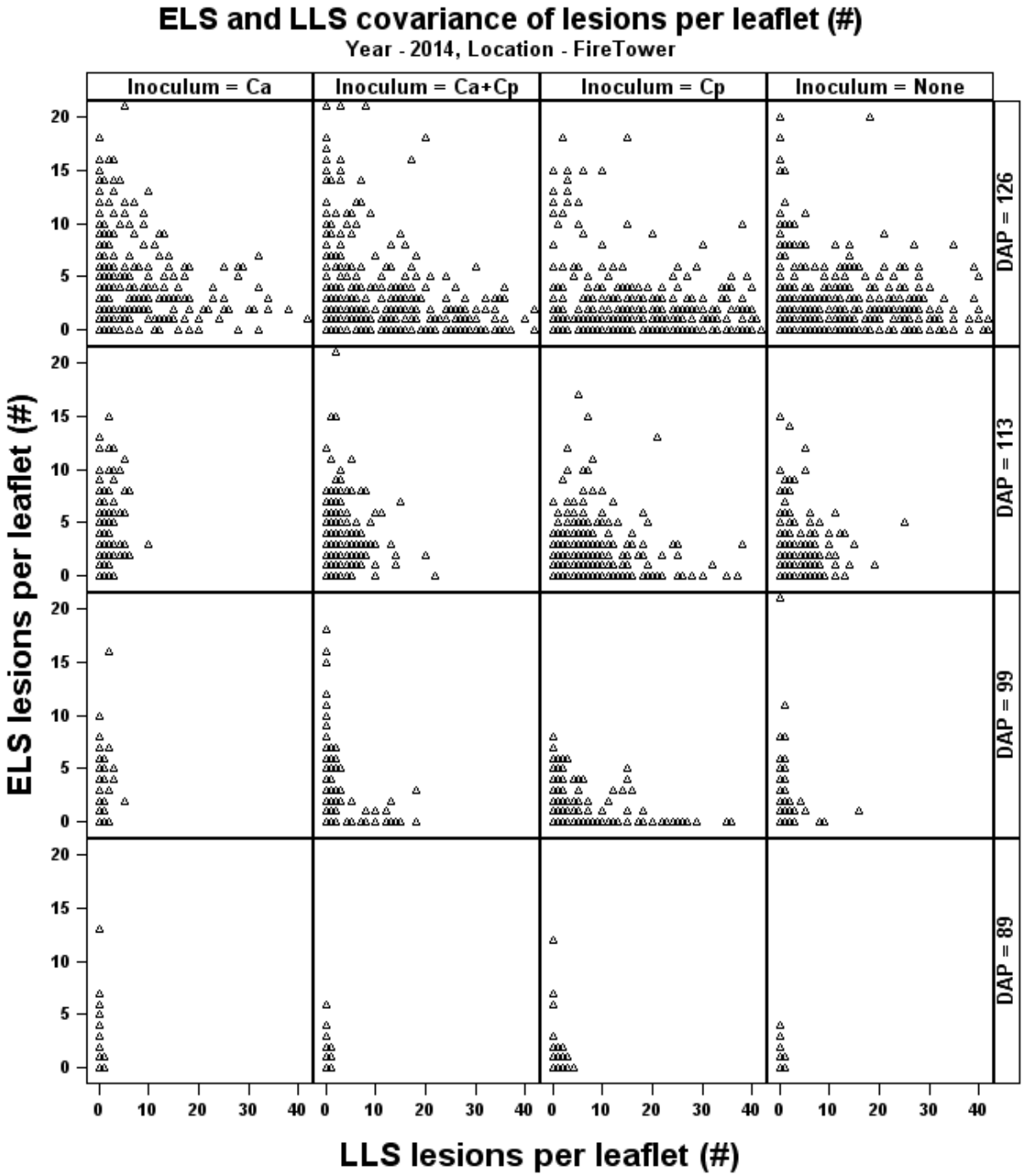


Fig. 11.4. Covariance of early (ELS) and late (LLS) leaf spot lesions per leaflet for each assessment date (DAP = days after planting) in plots treated with peanut residue infested with *Cercospora arachidicola* (Ca), *Cercosporidium personatum* (Cp), a 50/50 mixture of Ca + Cp and none (untreated control). Data from a trial in the FireTower field located at the Coastal Plain Experiment station in Tifton, Georgia, 2014.

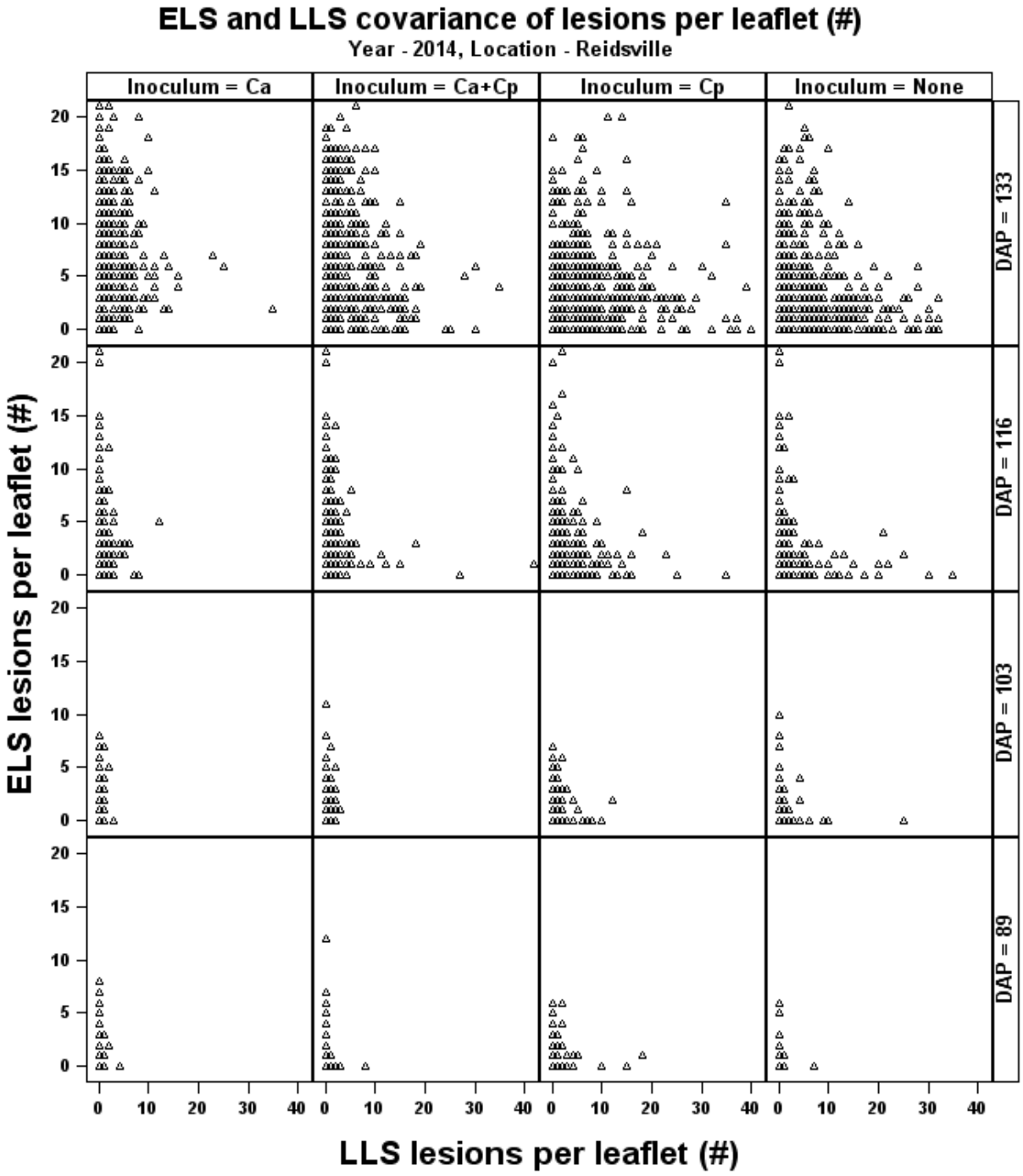


Fig. 11.5. Covariance of early (ELS) and late (LLS) leaf spot lesions per leaflet for each assessment date (DAP = days after planting) in plots treated with peanut residue infested with *Cercospora arachidicola* (Ca), *Cercosporidium personatum* (Cp), a 50/50 mixture of Ca + Cp and none (untreated control). Data pooled from two trials conducted at Reidsville, Georgia, 2014.

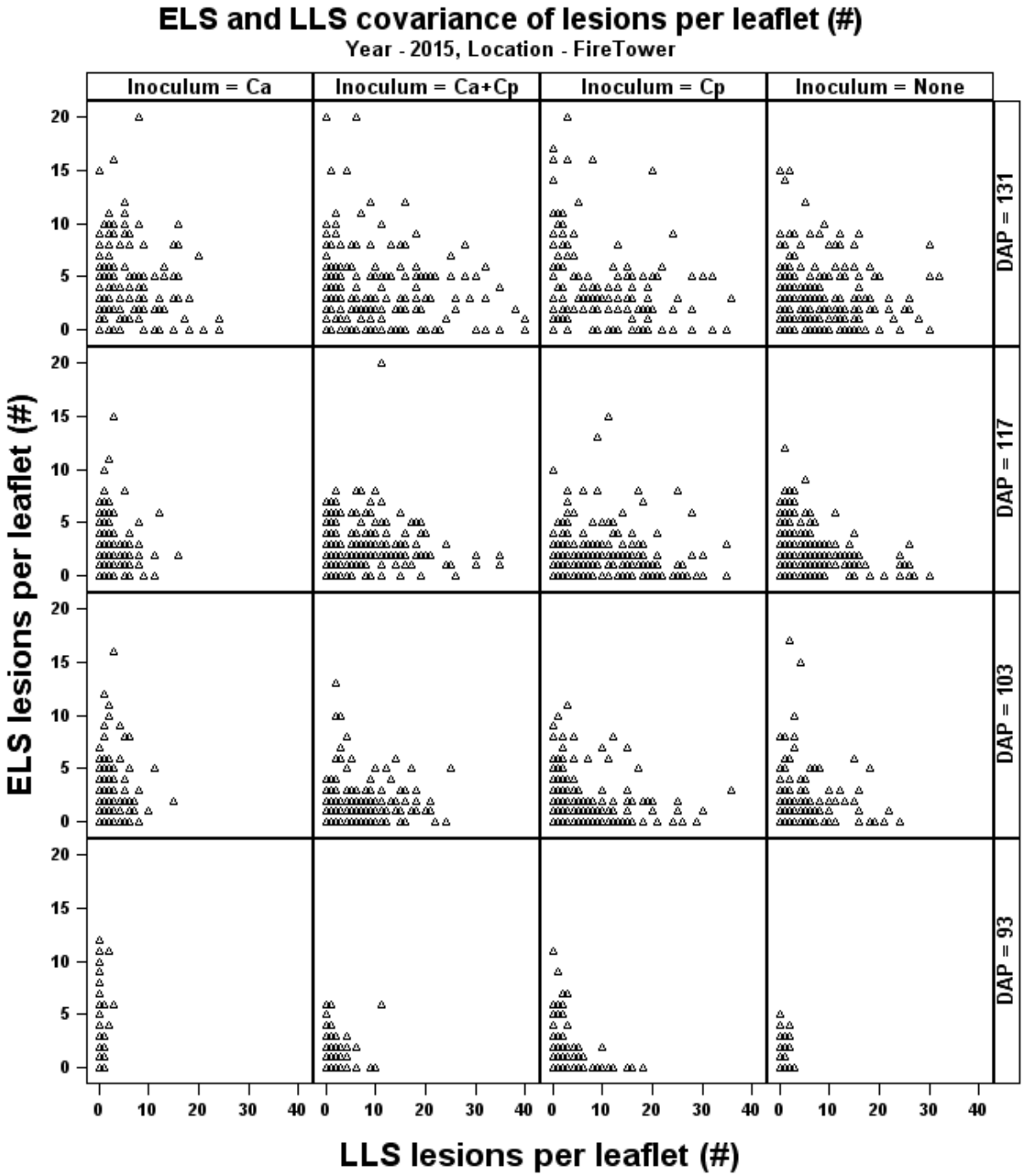


Fig. 11.6. Covariance of early (ELS) and late (LLS) leaf spot lesions per leaflet for each assessment date (DAP = days after planting) in plots treated with peanut residue infested with *Cercospora arachidicola* (Ca), *Cercosporidium personatum* (Cp), a 50/50 mixture of Ca + Cp and none (untreated control). Data were from a trial in the FireTower field located at the Coastal Plain Experiment station in Tifton, Georgia, 2015.

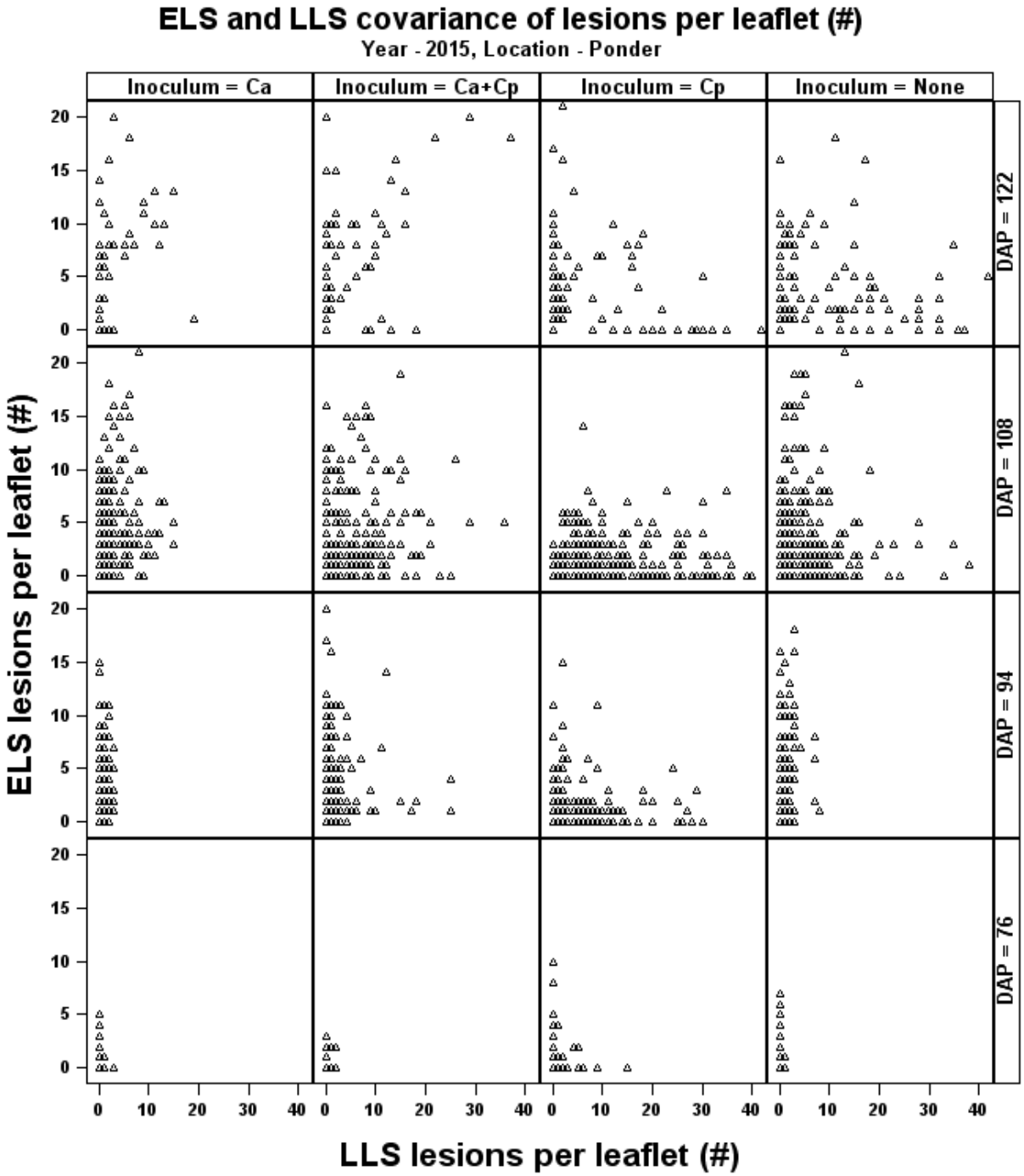


Fig. 11.7. Covariance of early (ELS) and late (LLS) leaf spot lesions per leaflet for each assessment date (DAP = days after planting) in plots treated with peanut residue infested with *Cercospora arachidicola* (Ca), *Cercosporidium personatum* (Cp), a 50/50 mixture of Ca + Cp and none (untreated control). Data were from a trial in a field at the Ponder farm located in Tifton, Georgia, 2015.

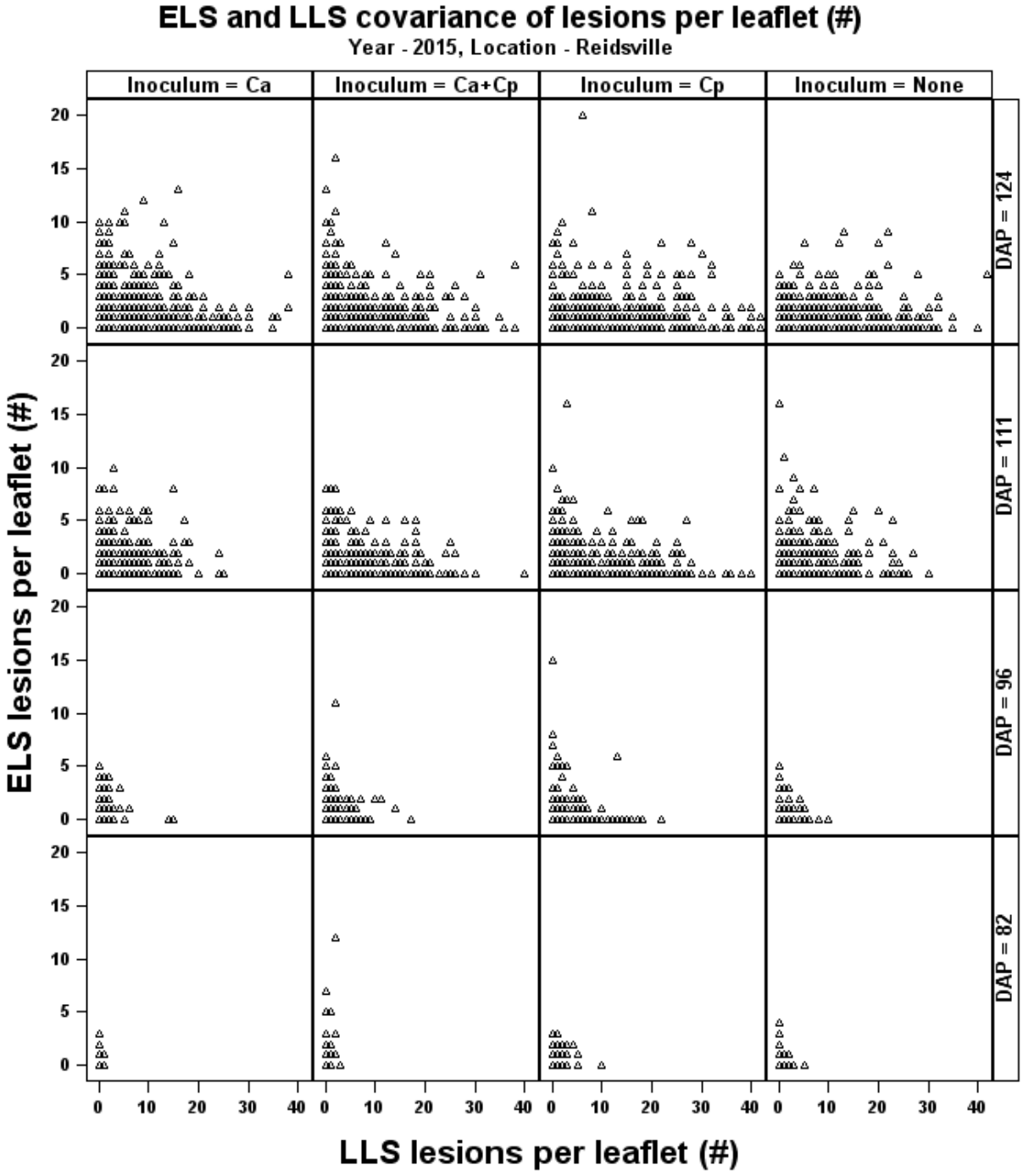


Fig. 11.8. Covariance of early (ELS) and late (LLS) leaf spot lesions per leaflet for each assessment date (DAP = days after planting) in plots treated with peanut residue infested with *Cercospora arachidicola* (Ca), *Cercosporidium personatum* (Cp), a 50/50 mixture of Ca + Cp and none (untreated control). Data pooled from two trials conducted at Reidsville, Georgia, 2015.

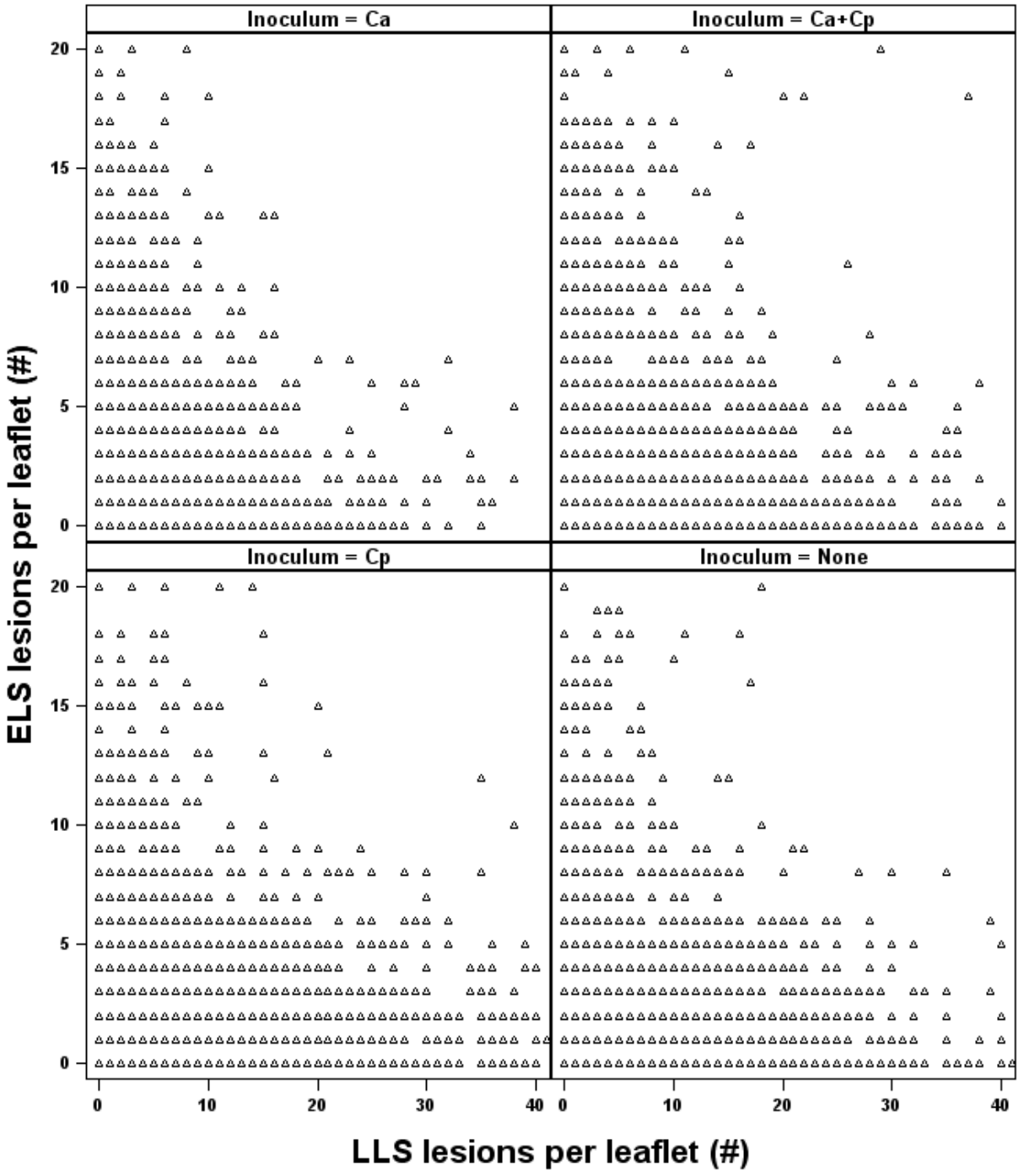


Fig. 11.9. Covariance of early (ELS) and late (LLS) leaf spot lesions per leaflet for all assessment dates in plots treated with peanut residue infested with *Cercospora arachidicola* (Ca), *Cercosporidium personatum* (Cp), a 50/50 mixture of Ca + Cp and none (untreated control). Data averaged across all years, locations, and sampling dates.

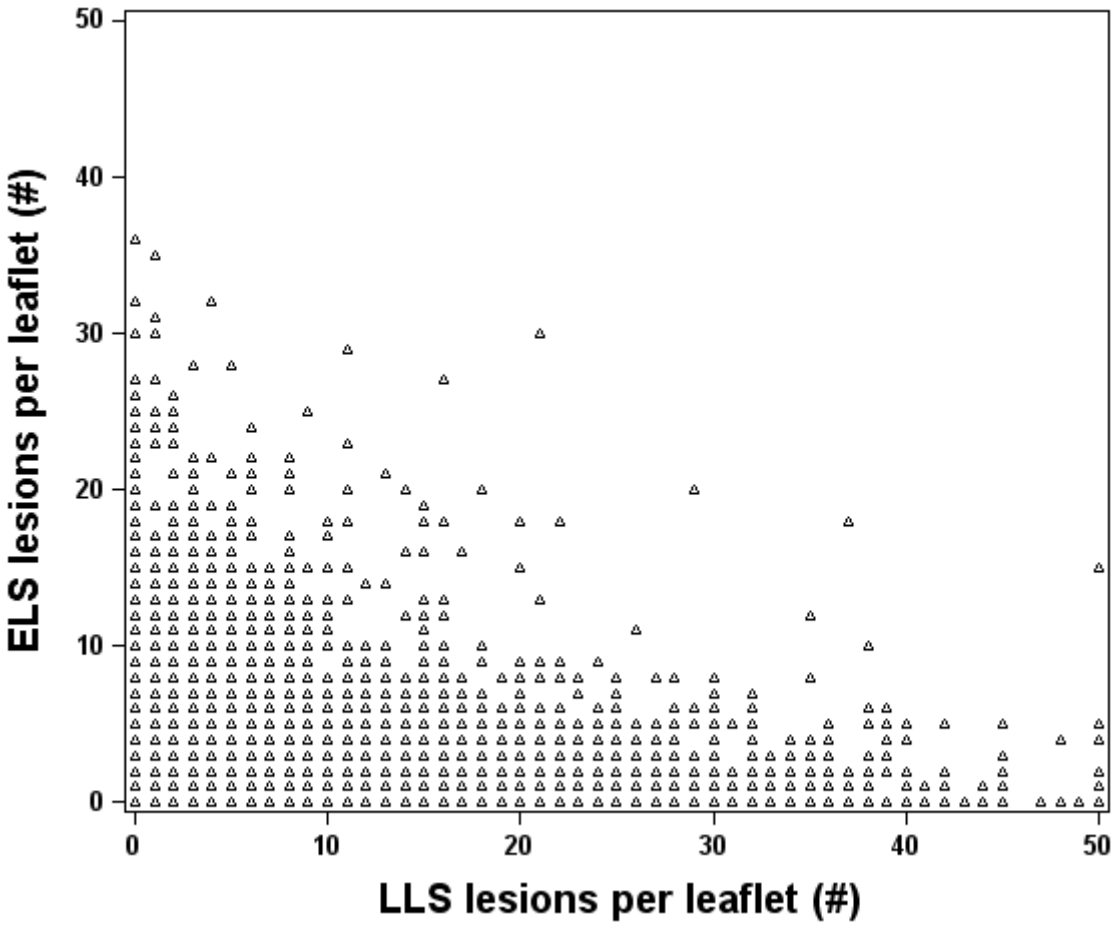
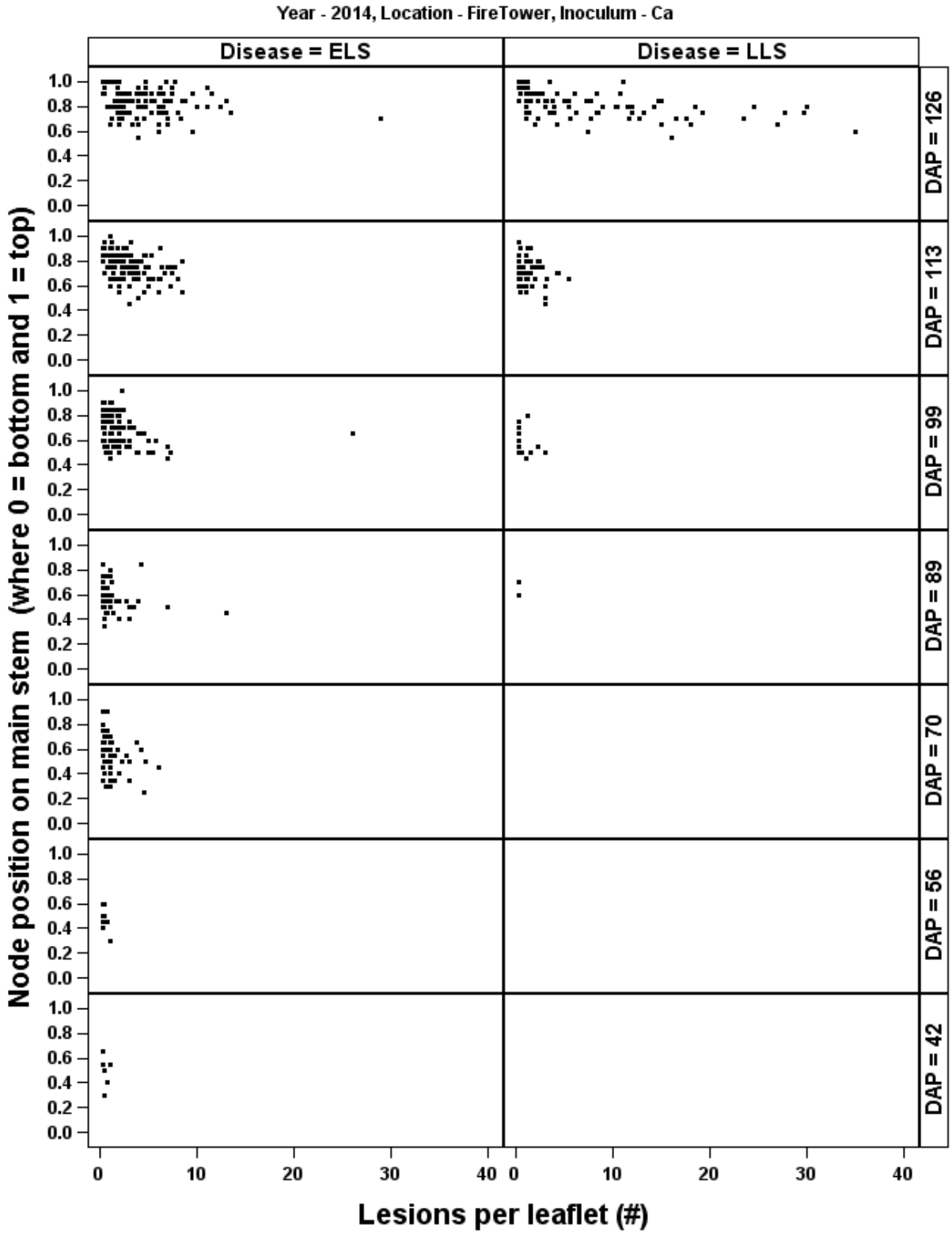
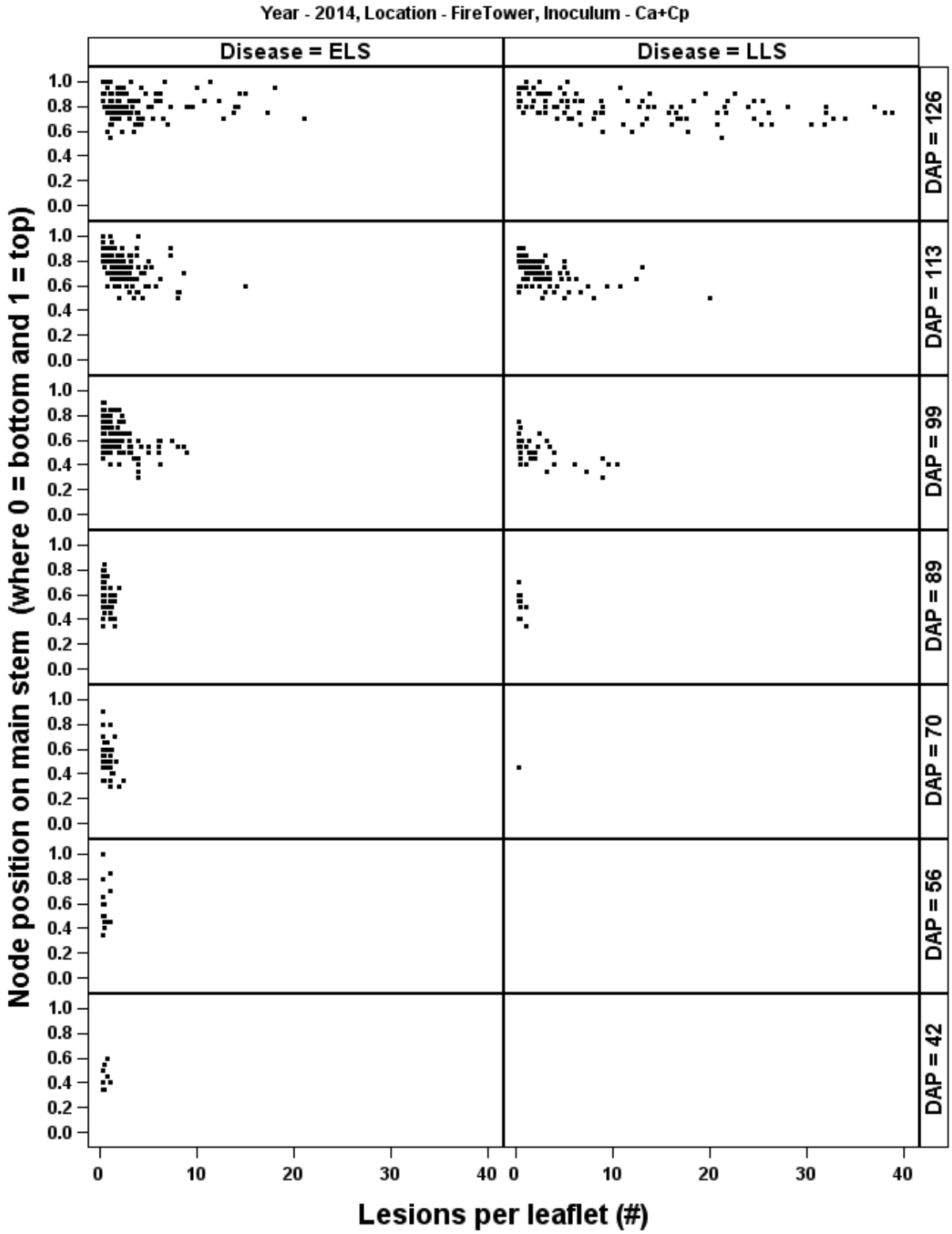


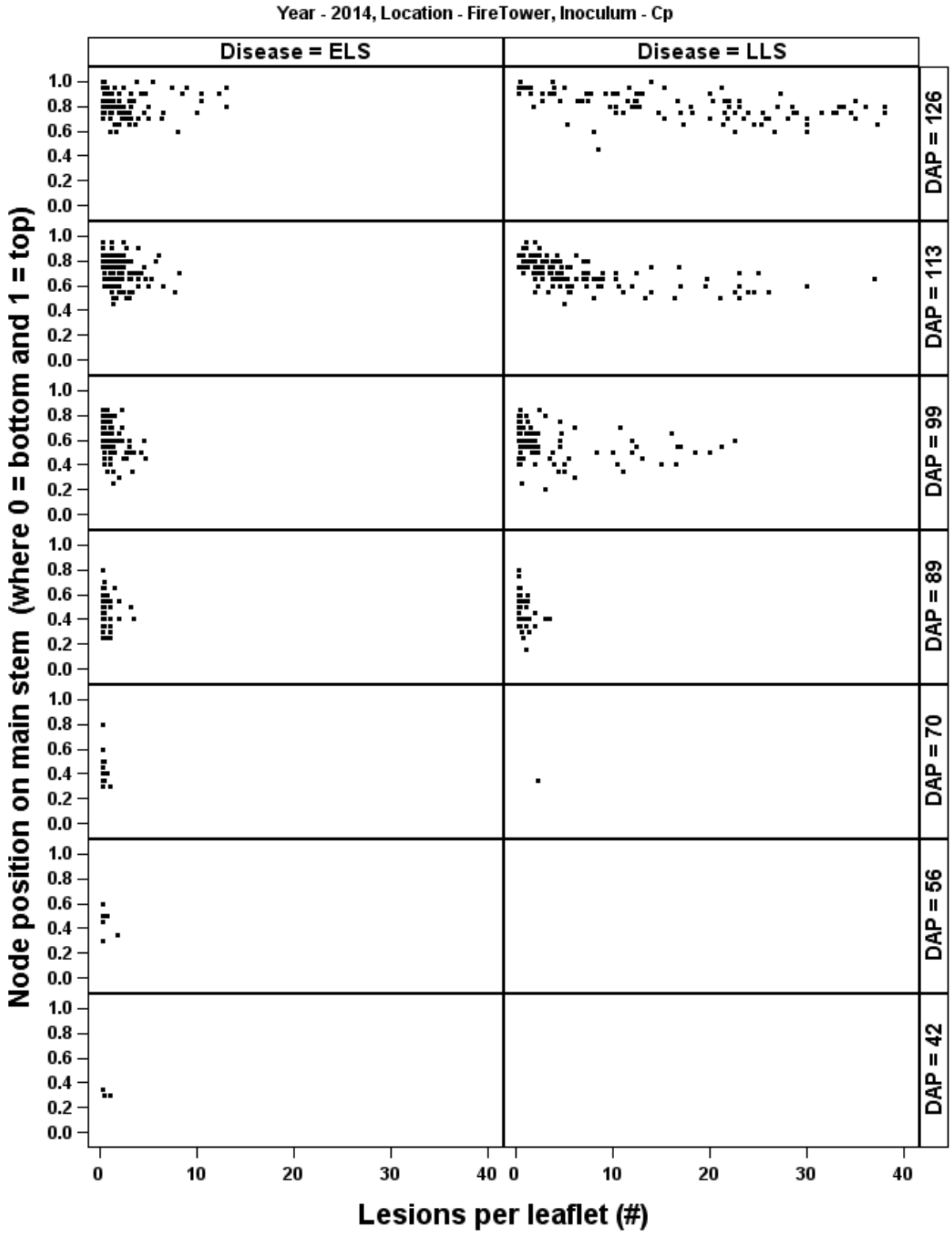
Fig. 11.10. Covariance of early (ELS) and late (LLS) leaf spot lesions per leaflet. Data averaged across all years, locations, inoculum treatments and sampling dates (*n* = 28,380).



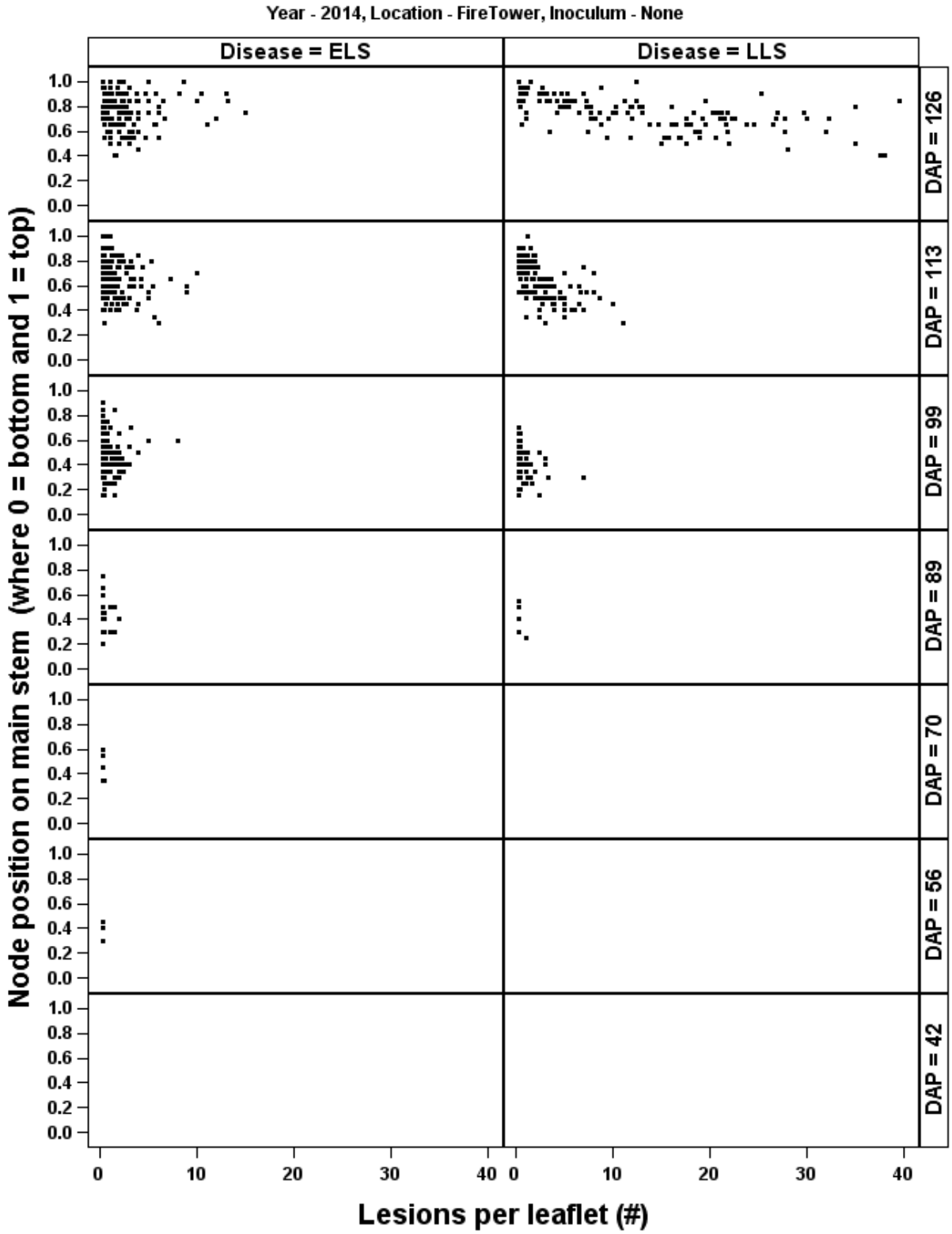
Supplementary Fig. S11.1. Distribution of early (ELS) and late (LLS) leaf spot lesions on the main stem in plots treated with peanut residue infested with *Cercospora arachidicola* (Ca) at the FireTower field in 2014.



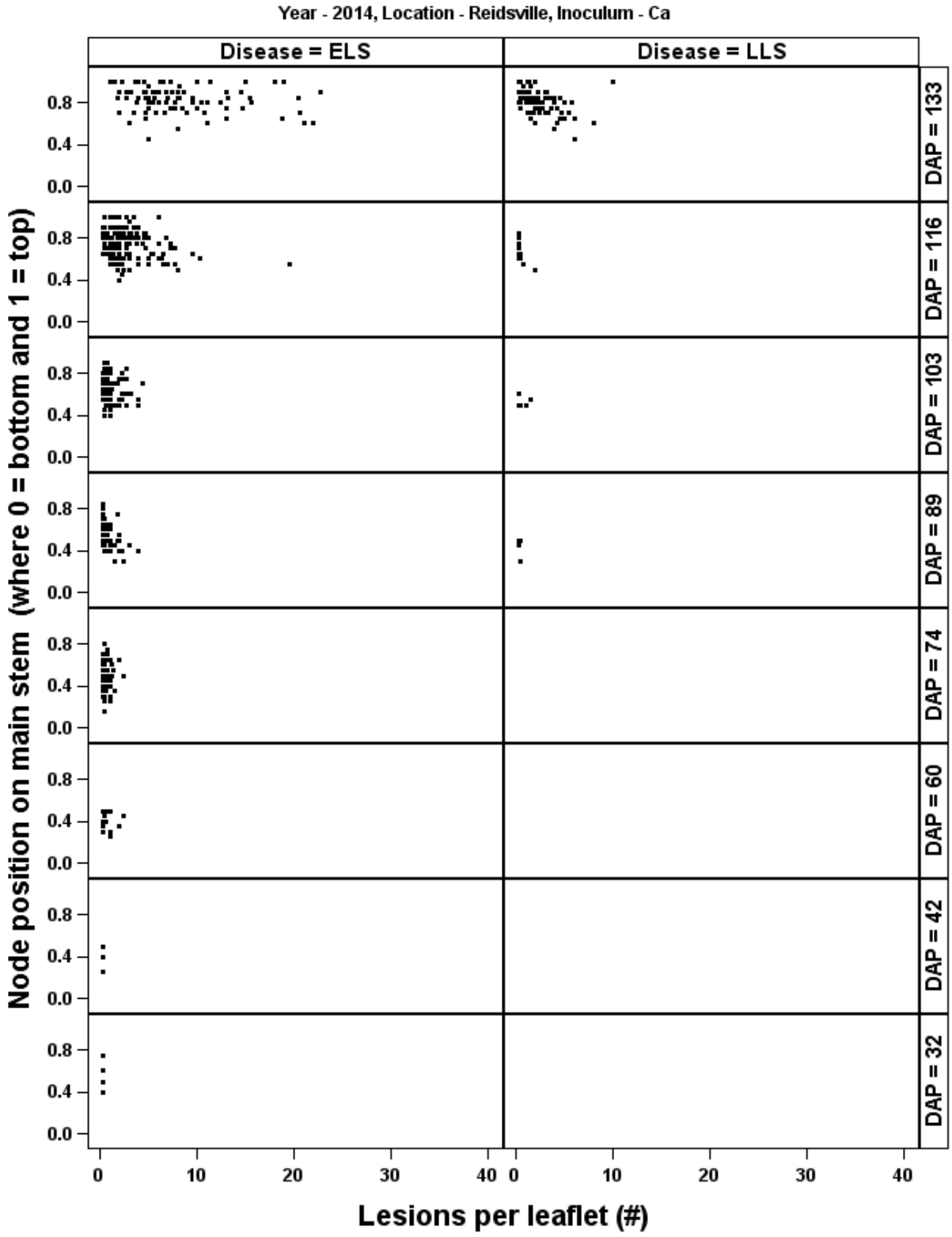
Supplementary Fig. S11.2. Distribution of early (ELS) and late (LLS) leaf spot lesions on the main stem in plots treated with peanut residue infested with *Cercospora arachidicola* (Ca) and *Cercosporidium personatum* (Cp) at the FireTower field in 2014.



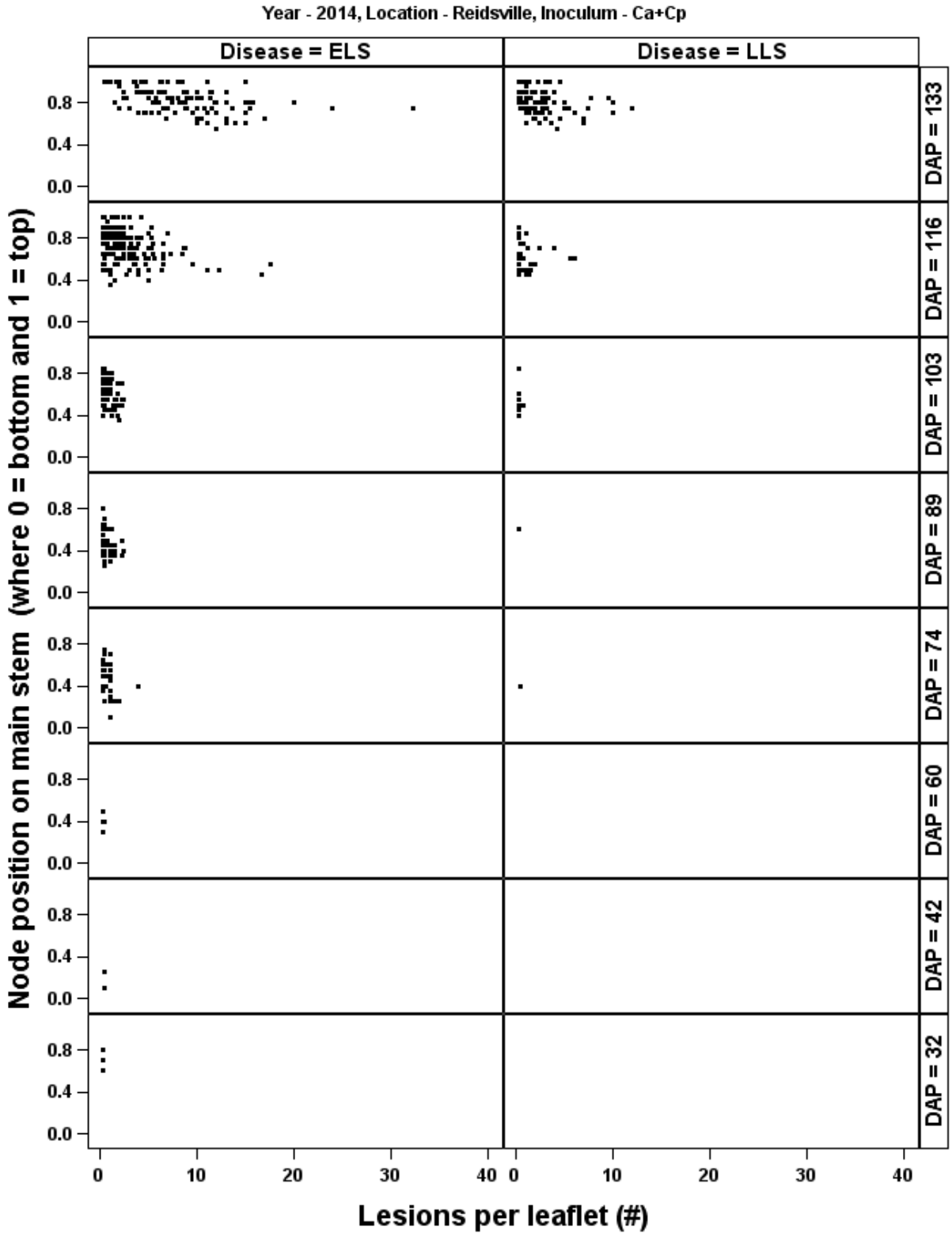
Supplementary Fig. S11.3. Distribution of early (ELS) and late (LLS) leaf spot lesions on the main stem in plots treated with peanut residue infested with *Cercosporidium personatum* (Cp) at the FireTower field in 2014.



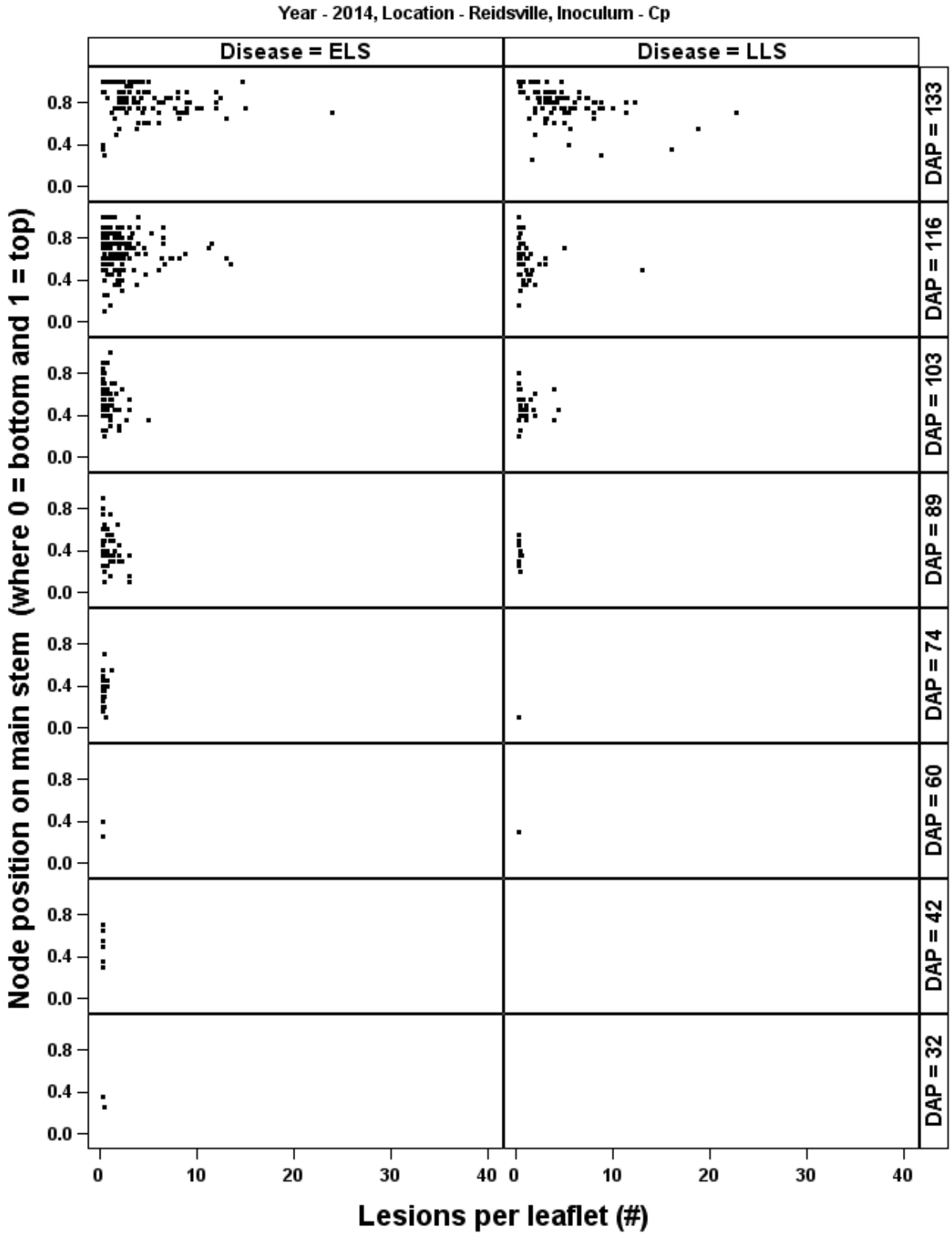
Supplementary Fig. S11.4. Distribution of early (ELS) and late (LLS) leaf spot lesions on the main stem in residue-free plots (none) at the FireTower field in 2014.



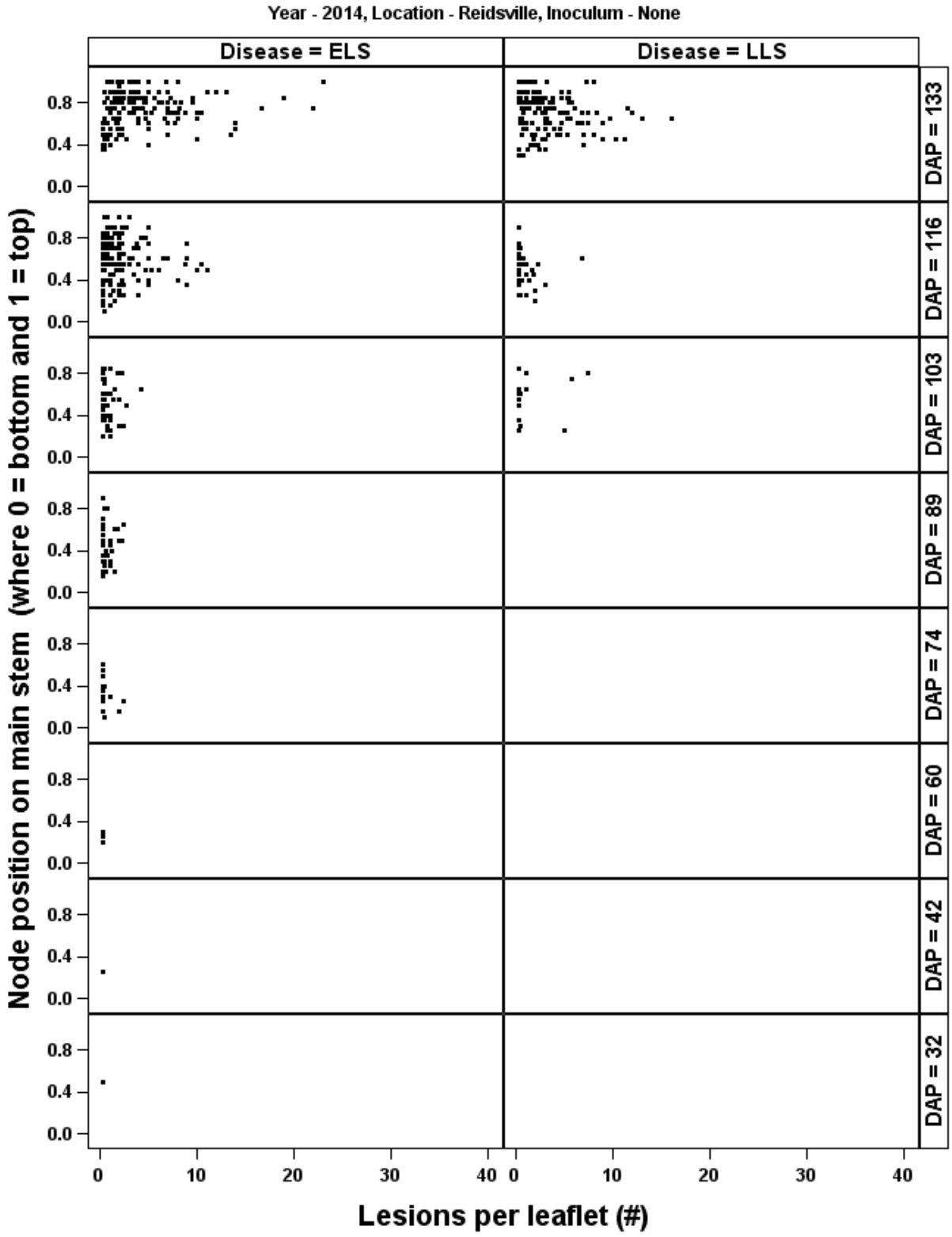
Supplementary Fig. S11.5. Distribution of early (ELS) and late (LLS) leaf spot lesions on the main stem in plots treated with peanut residue infested with *Cercospora arachidicola* (Ca) at Reidsville in 2014.



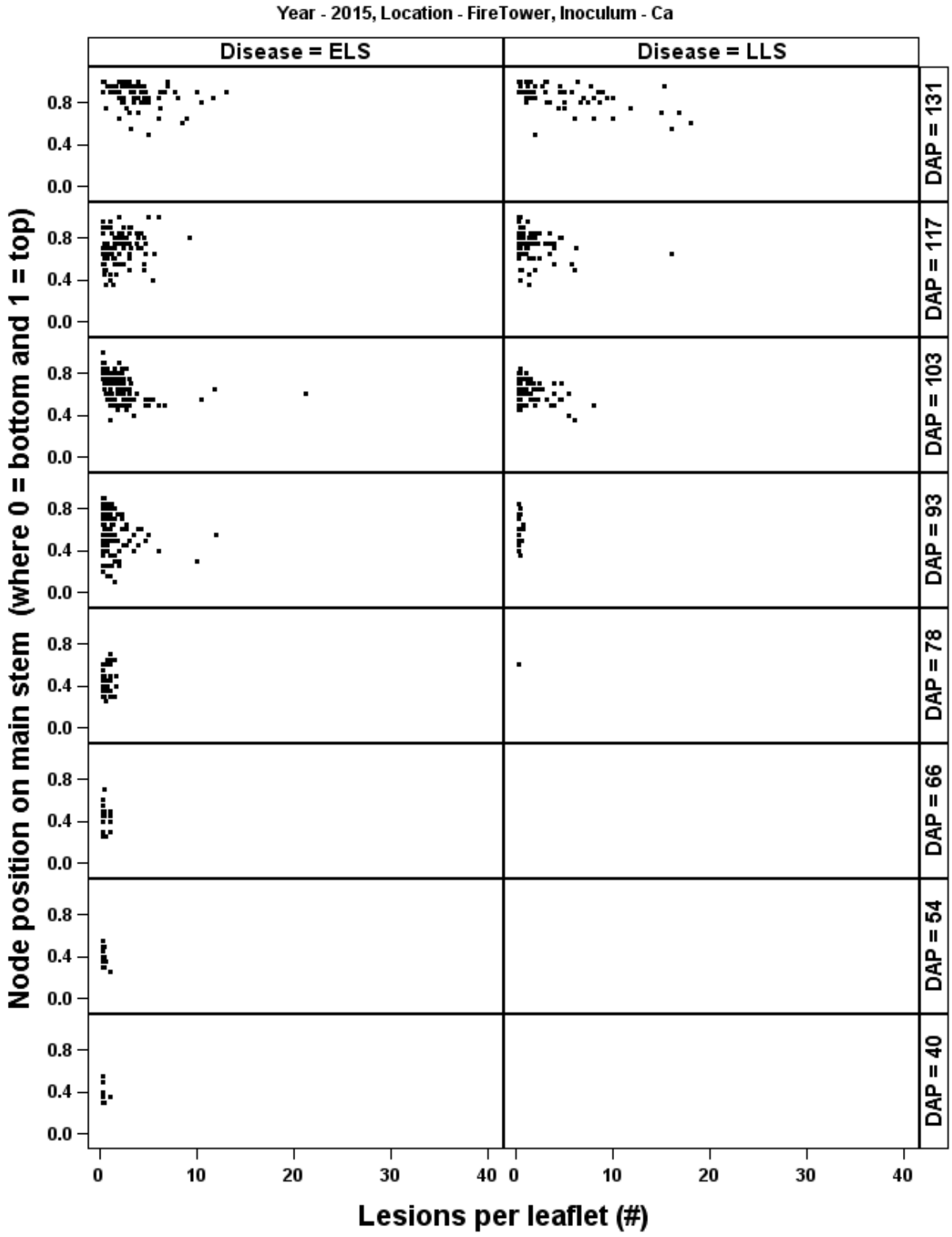
Supplementary Fig. S11.6. Distribution of early (ELS) and late (LLS) leaf spot lesions on the main stem in plots treated with peanut residue infested with *Cercospora arachidicola* (Ca) and *Cercosporidium personatum* (Cp) at Reidsville in 2014.



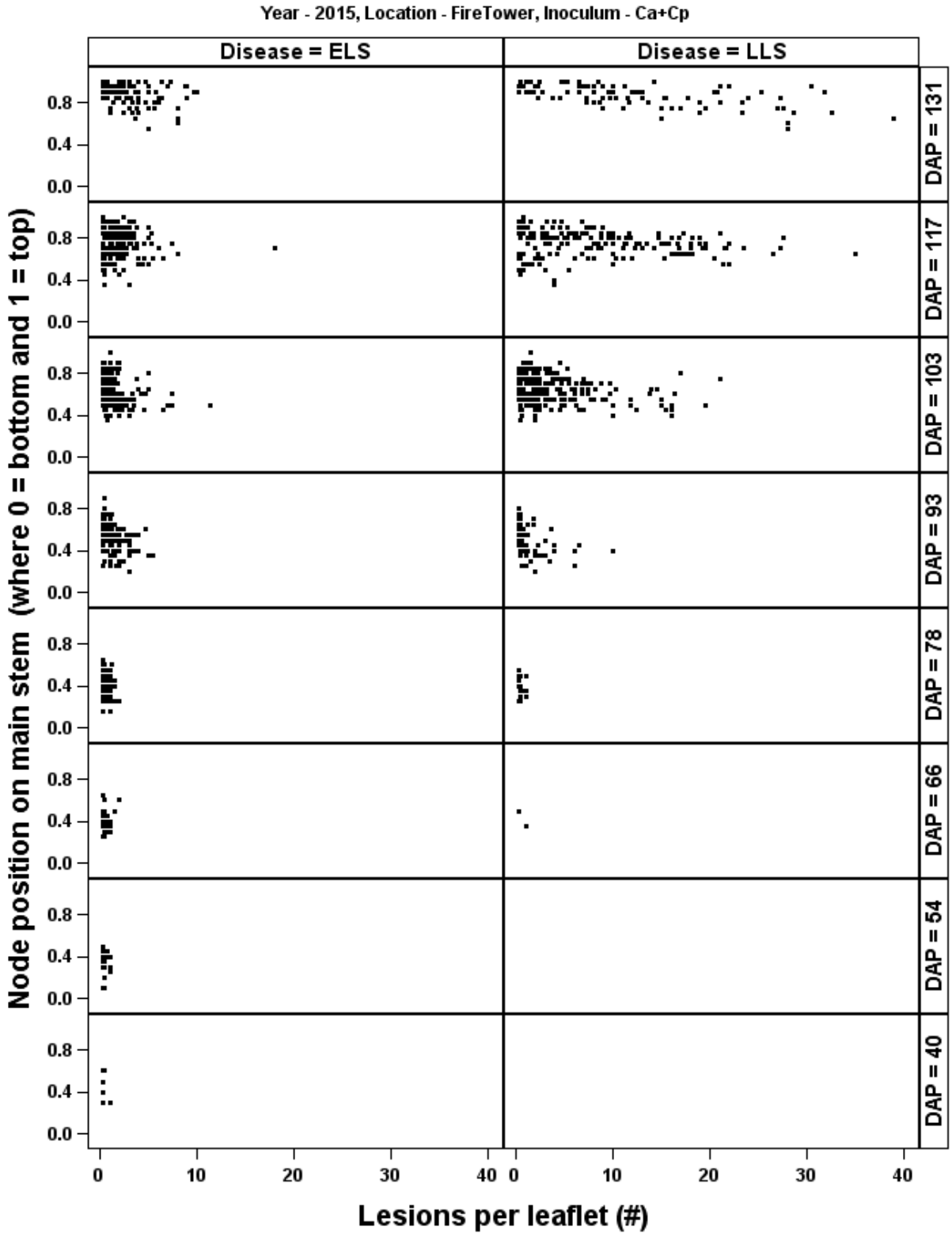
Supplementary Fig. S11.7. Distribution of early (ELS) and late (LLS) leaf spot lesions on the main stem in plots treated with peanut residue infested with *Cercosporidium personatum* (Cp) at Reidsville in 2014.



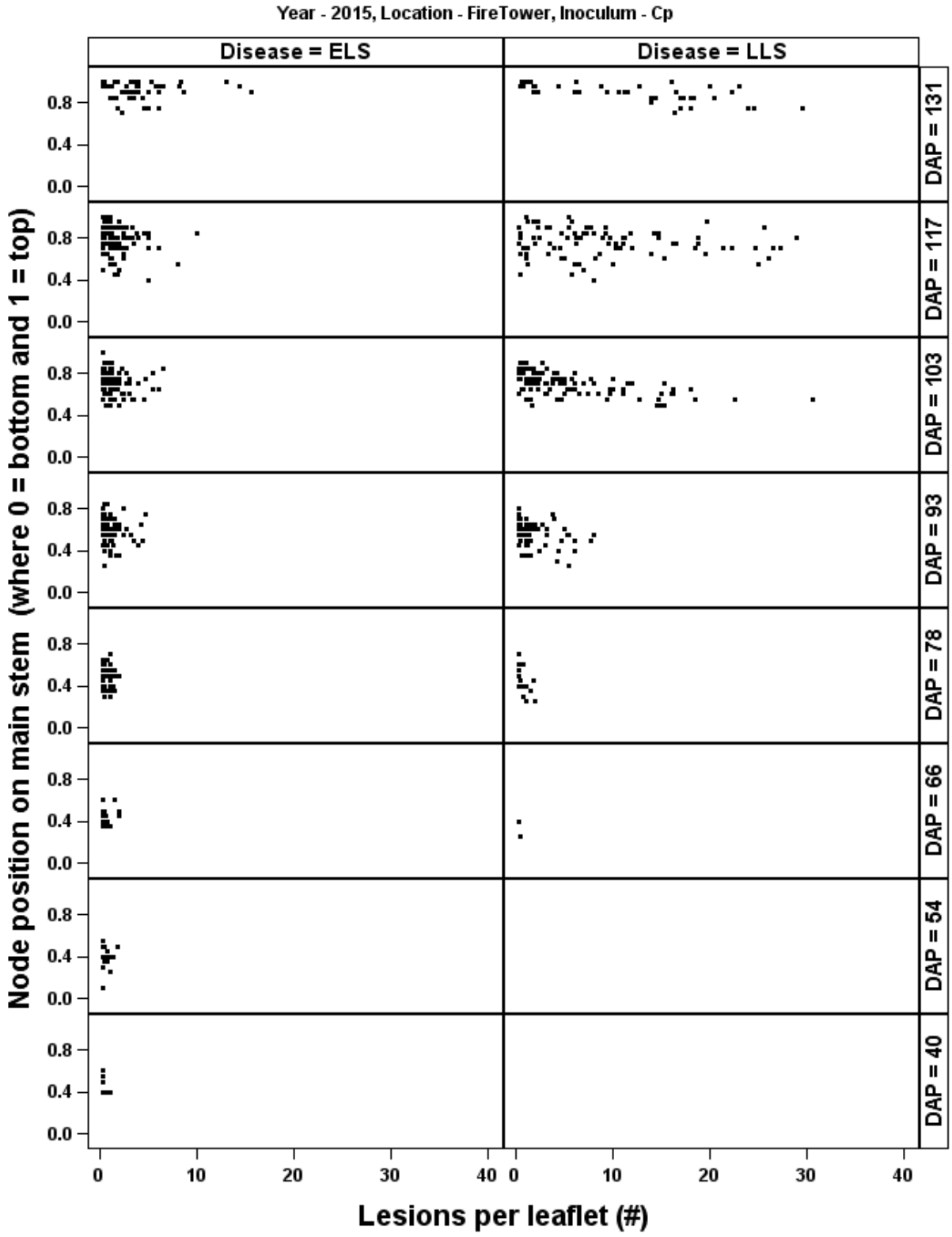
Supplementary Fig. S11.8. Distribution of early (ELS) and late (LLS) leaf spot lesions on the main stem in residue-free (none) plots at Reidsville in 2014.



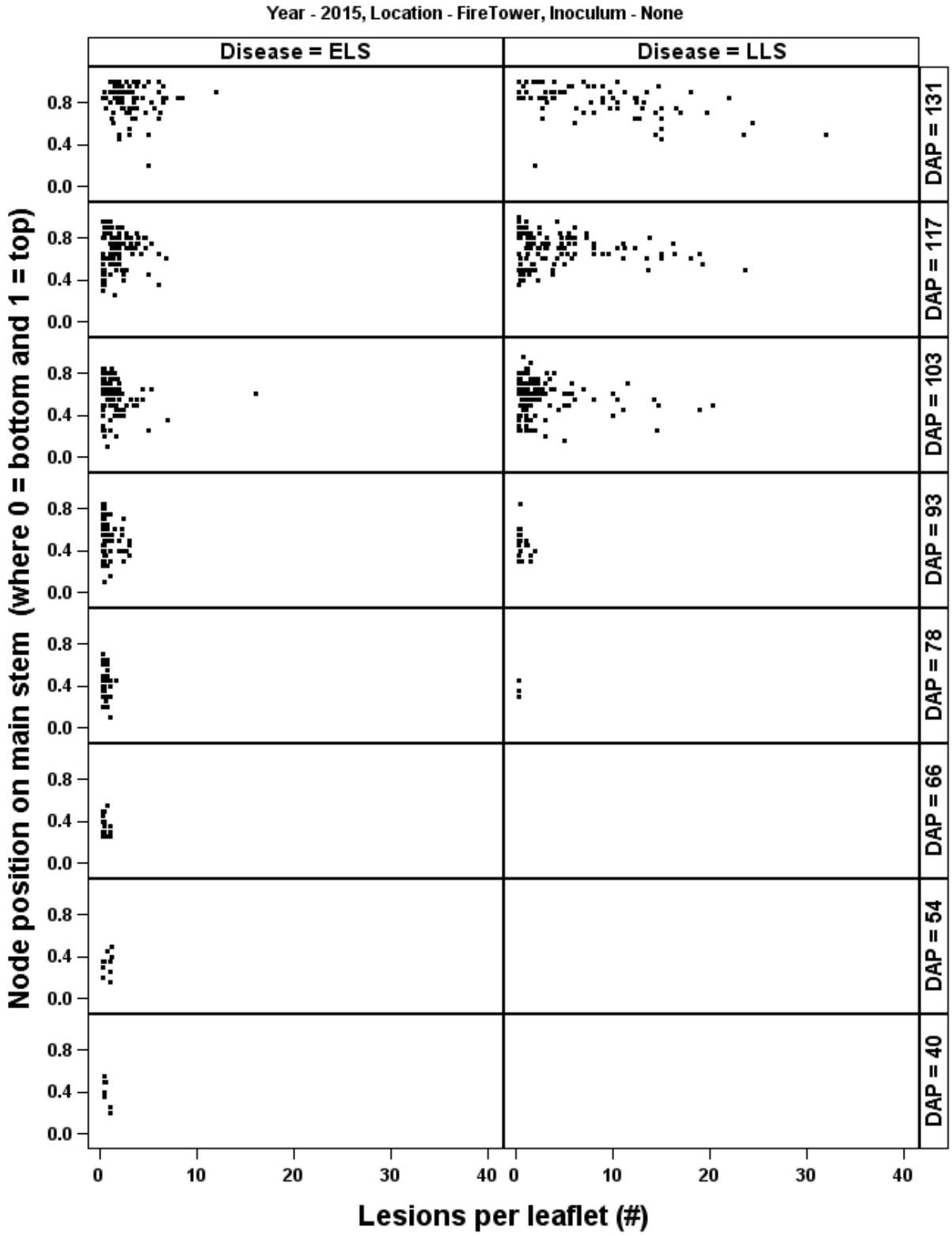
Supplementary Fig. S11.9. Distribution of early (ELS) and late (LLS) leaf spot lesions on the main stem in plots treated with peanut residue infested with *Cercospora arachidicola* (Ca) at the FireTower field in 2015.



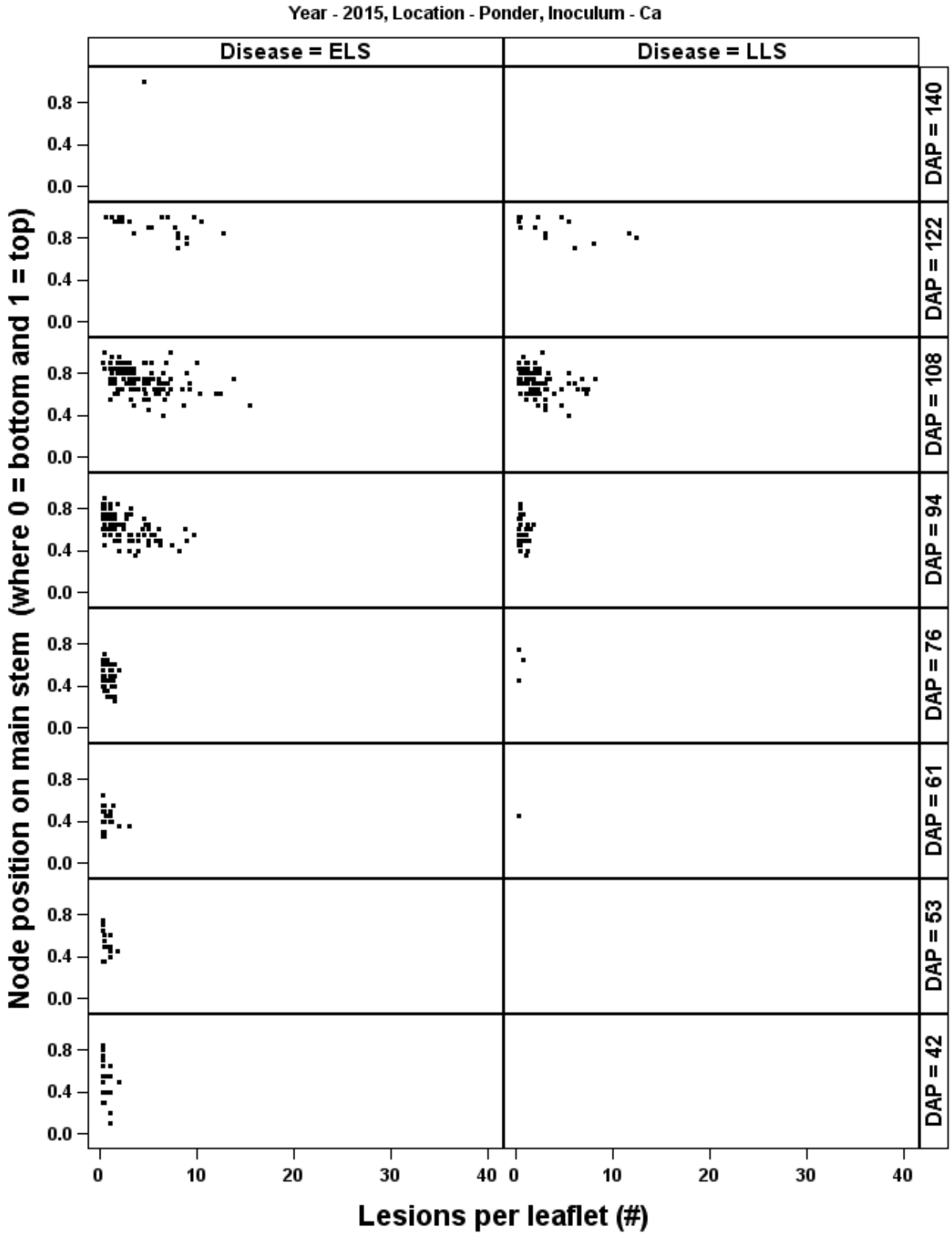
Supplementary Fig. S11.10. Distribution of early (ELS) and late (LLS) leaf spot lesions on the main stem in plots treated with peanut residue infested with *Cercospora arachidicola* (Ca) and *Cercosporidium personatum* (Cp) at the FireTower field in 2015.



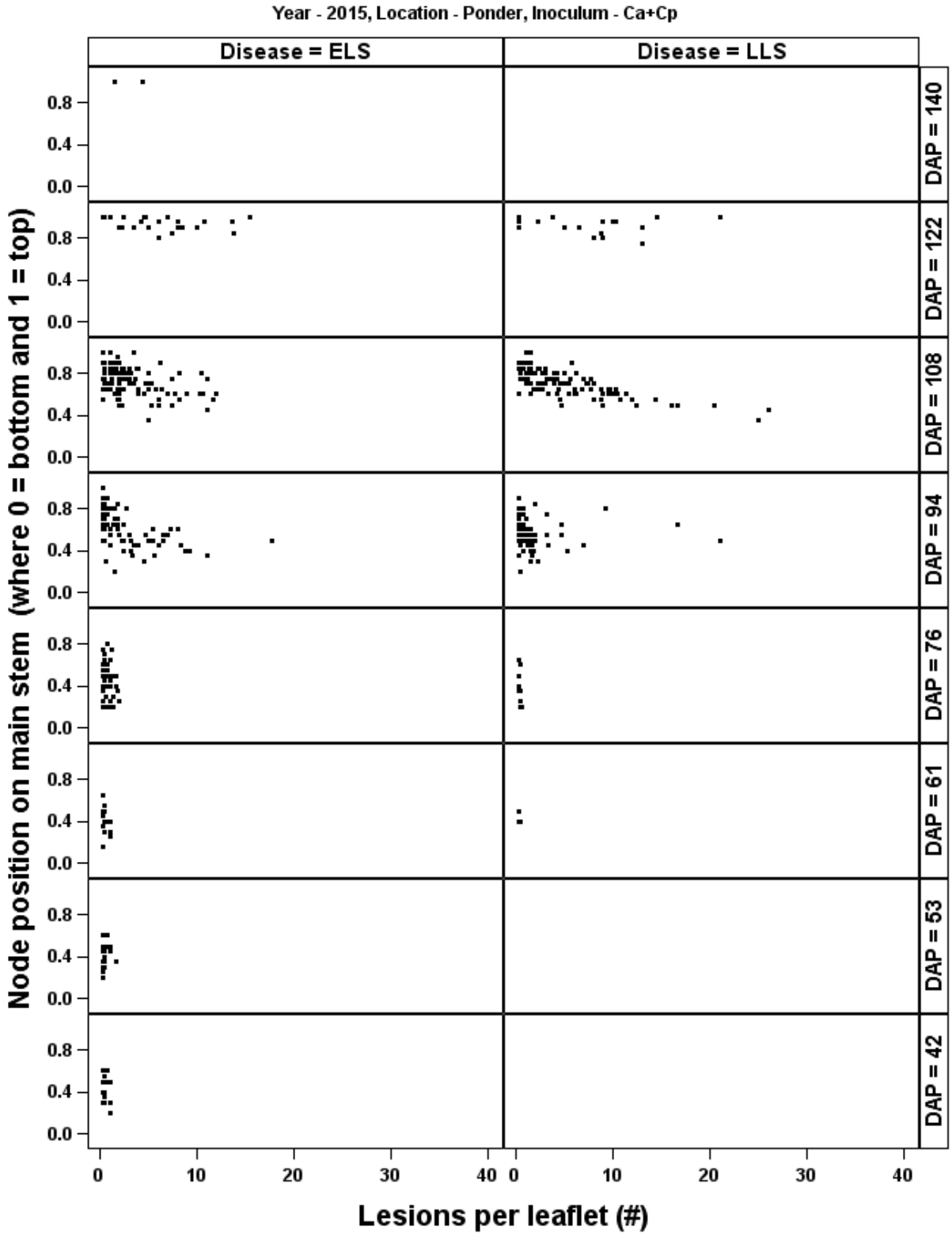
Supplementary Fig. S11.11. Distribution of early (ELS) and late (LLS) leaf spot lesions on the main stem in plots treated with peanut residue infested with *Cercosporidium personatum* (Cp) at the FireTower field in 2015.



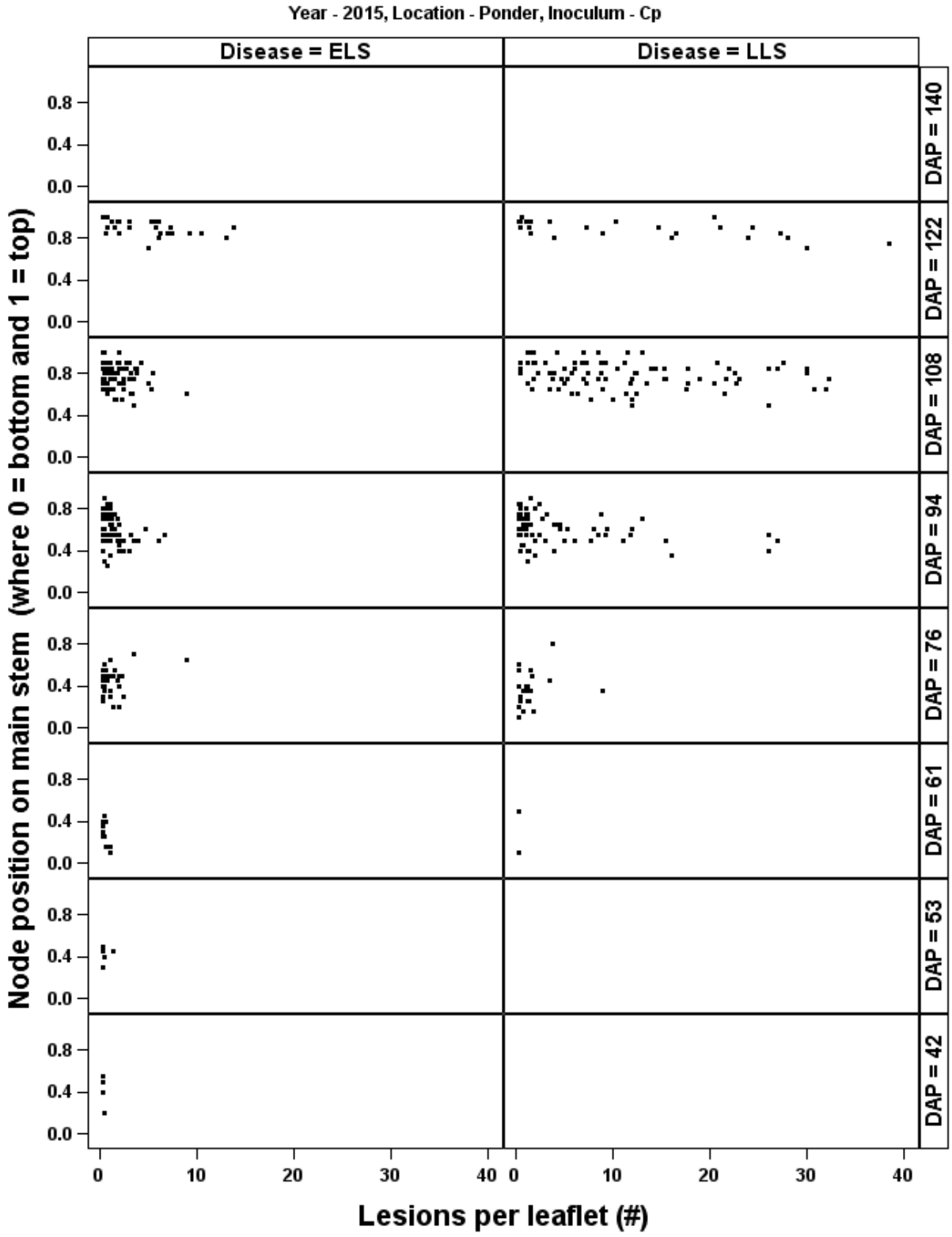
Supplementary Fig. S11.12. Distribution of early (ELS) and late (LLS) leaf spot lesions on the main stem in residue free plots (none) at the FireTower field in 2015.



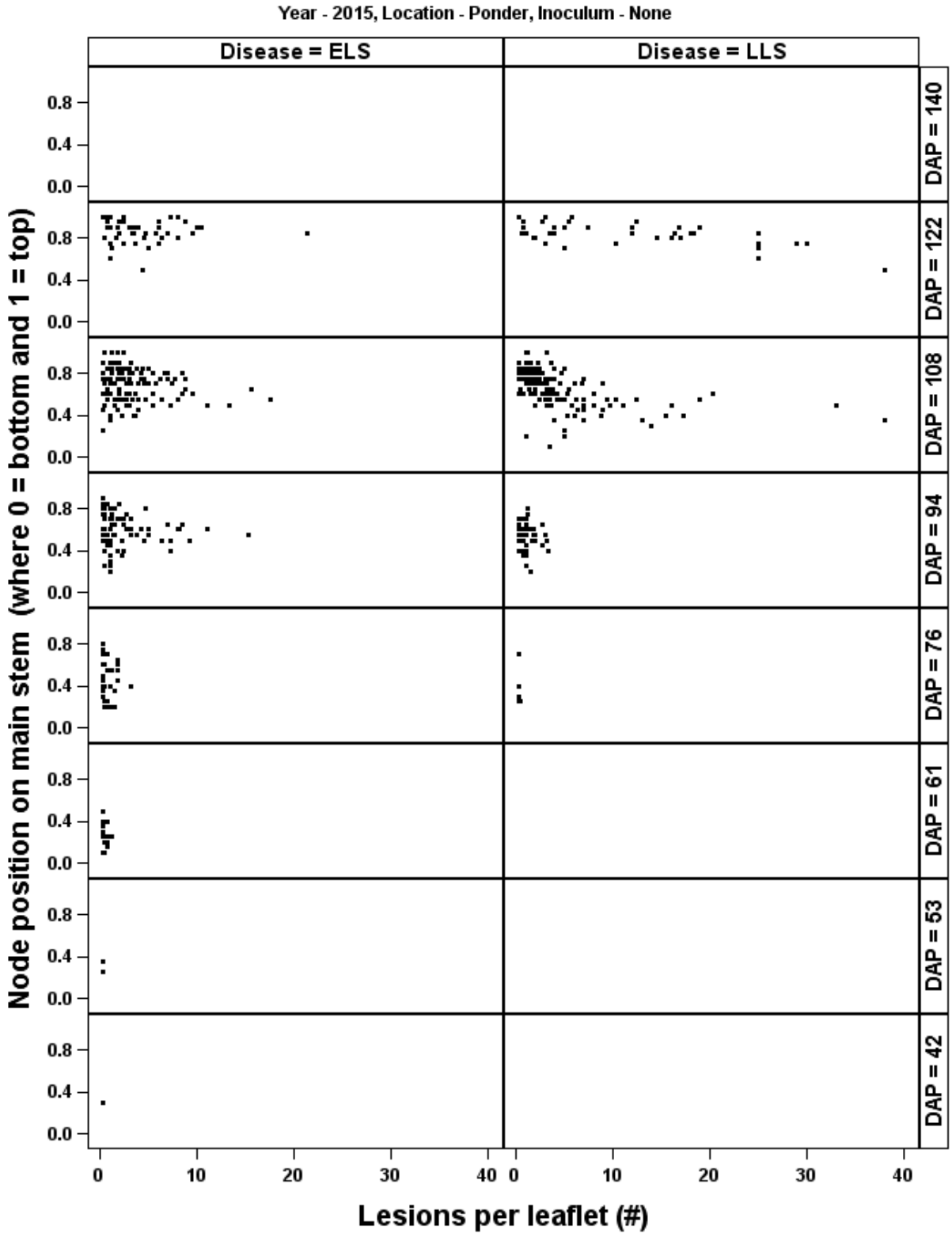
Supplementary Fig. S11.13. Distribution of early (ELS) and late (LLS) leaf spot lesions on the main stem in plots treated with peanut residue infested with *Cercospora arachidicola* (Ca) at the Ponder farm field in 2015.



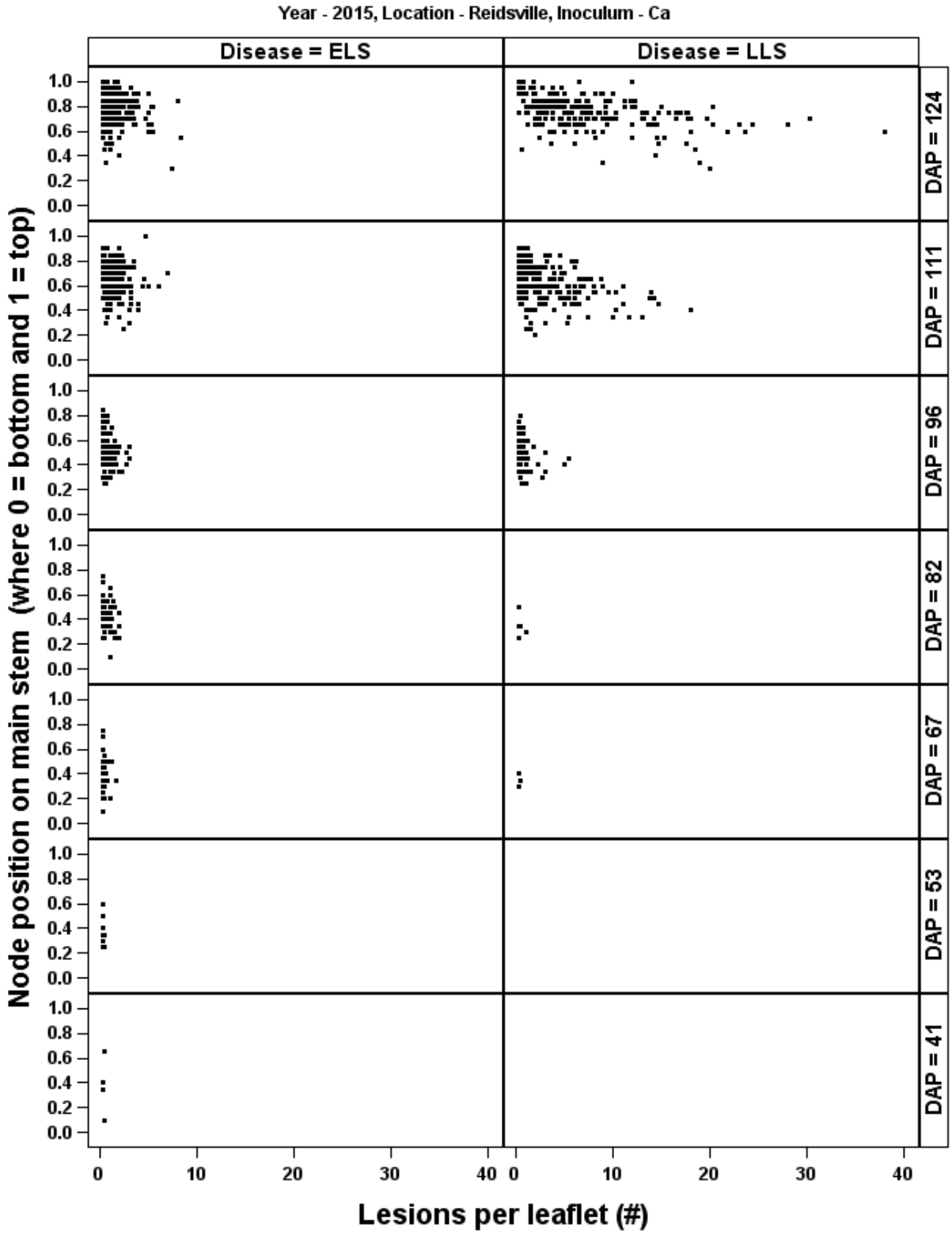
Supplementary Fig. S11.14. Distribution of early (ELS) and late (LLS) leaf spot lesions on the main stem in plots treated with peanut residue infested with *Cercospora arachidicola* (Ca) and *Cercosporidium personatum* (Cp) at the Ponder farm field in 2015.



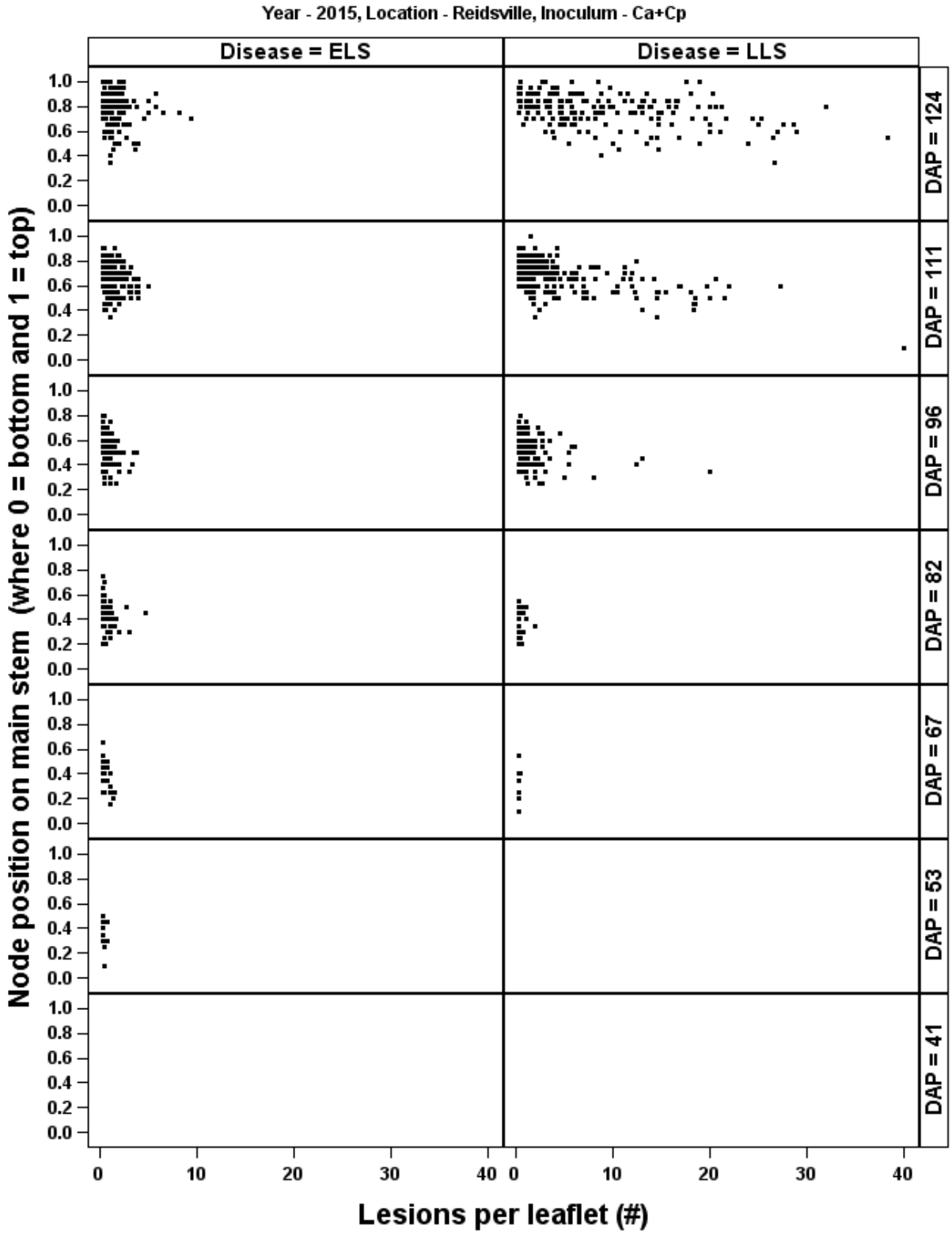
Supplementary Fig. S11.15. Distribution of early (ELS) and late (LLS) leaf spot lesions on the main stem in plots treated with peanut residue infested with *Cercosporidium personatum* (Cp) at the Ponder farm field in 2015.



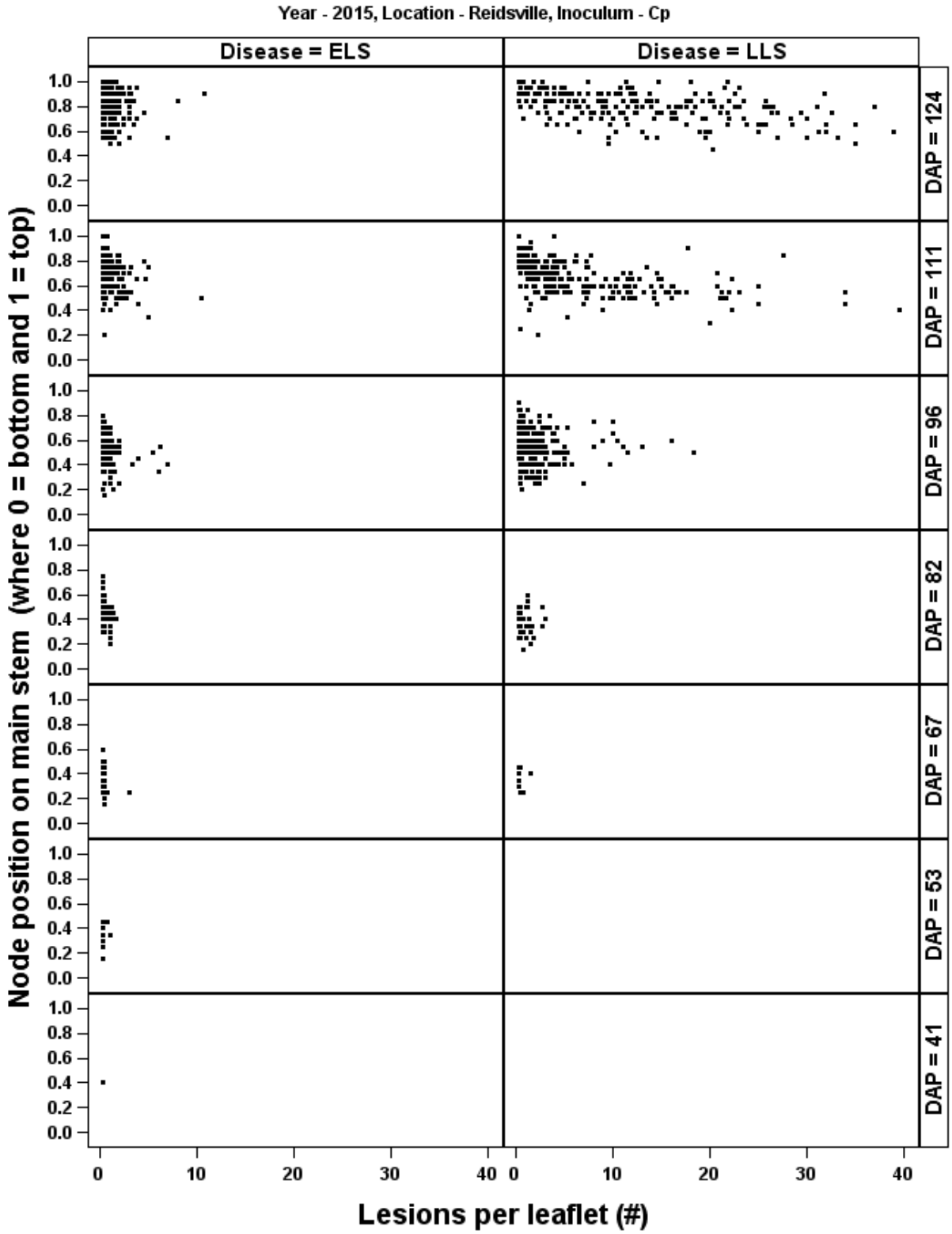
Supplementary Fig. S11.16. Distribution of early (ELS) and late (LLS) leaf spot lesions on the main stem in residue free plots (none) at the Ponder farm field in 2015.



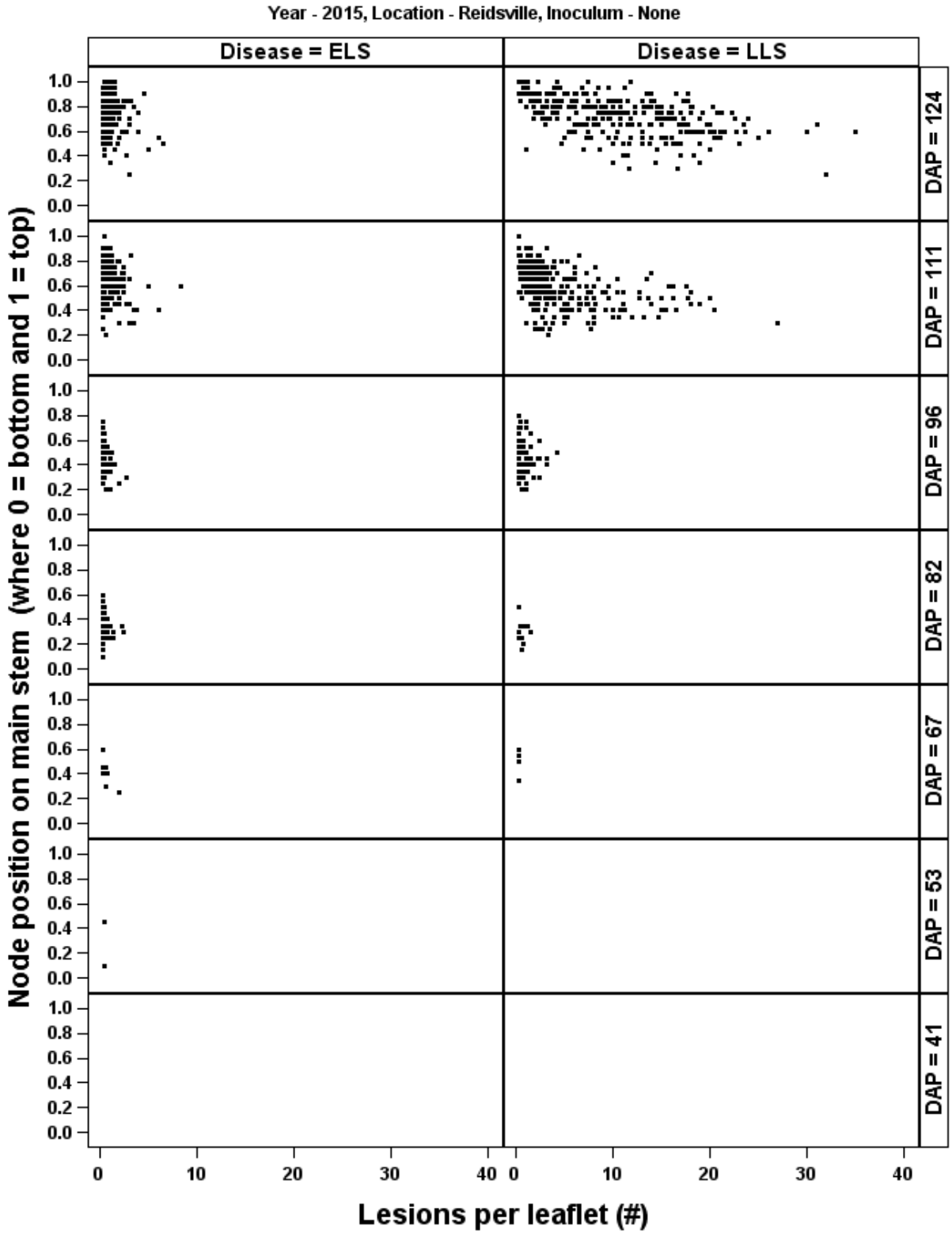
Supplementary Fig. S11.17. Distribution of early (ELS) and late (LLS) leaf spot lesions on the main stem in plots treated with peanut residue infested with *Cercospora arachidicola* (Ca) at Reidsville in 2015.



Supplementary Fig. S11.18. Distribution of early (ELS) and late (LLS) leaf spot lesions on the main stem in plots treated with peanut residue infested with *Cercospora arachidicola* (Ca) and *Cercosporidium personatum* (Cp) at Reidsville in 2015.



Supplementary Fig. S11.19. Distribution of early (ELS) and late (LLS) leaf spot lesions on the main stem in plots treated with peanut residue infested with *Cercosporidium personatum* (Cp) at Reidsville in 2015.



Supplementary Fig. S11.20. Distribution of early (ELS) and late (LLS) leaf spot lesions on the main stem in residue free plots (none) at Reidsville in 2015.

APPENDIX 2 (CHAPTER 12):

PRESCRIPTION FUNGICIDE PROGRAMS VIA PEANUT RX: REASSESSING APPLICATION TIMINGS FOR BETTER CONTROL OF LATE LEAF SPOT OF PEANUT[1]

[1]Fulmer, A.M., Kemerait, R.C., Brenneman, T.B., Scherm, H., Cantonwine, E.G., Culbreath, A.K., and Stevenson, K.L. 2017. To be submitted to *Peanut Science*.

ABSTRACT

The initiation and subsequent application interval of prescription fungicide programs are a function of the expected onset and final intensity of leaf spot for a given risk level predicted by Peanut Rx. However, recent epidemiological studies on early (ELS) and late leaf spot (LLS) of peanut, caused by *Cercospora arachidicola* and *Cercosporidium personatum*, respectively, have demonstrated consistent differences in their development. In prior studies conducted in 2011 and 2012, the onset of LLS was observed to be >100 days after planting (DAP) in moderate-risk fields in, therefore, field trials were conducted in 2013 and 2014 to determine if its control could be enhanced by delaying fungicide timings to better align with expected disease development. Conventional and/or strip-tilled plots in a moderate-risk field were planted to the runner cultivar Georgia-06G. Applications of azoxystrobin, propiconazole, or chlorothalonil were initiated at 30, 37 or 44 DAP for regular or at 44, 58 or 65 DAP for delayed high, moderate, and low-risk programs, respectively. In 2013 and 2014, the average onset of LLS in untreated plots was 90 and 100 DAP, respectively. Statistically, there was no difference in the regular and delayed high-risk programs, but both had significantly lower LLS severity than all other treatments. The delayed moderate and low-risk programs provided statistically better LLS control than their regular counterparts, but no differences in yield were observed. Additional trials were conducted in 2014 and 2015 to further assess the effect of late season applications in dryland fields with a history of LLS. Plots were planted to the cultivars Georgia-06G and Georgia-12Y, and applications of chlorothalonil were applied at timings appropriate for full, early, mid-season and late-season applications. In these trials, onset of ELS and LLS ranged from 95 to 112 DAP across years for both cultivars. Across years, the full spray program for both cultivars averaged 1.8 on the Florida 1-10 leaf spot intensity scale and increased by ~1, 2 and 3 points for mid-season, late-season and early-season programs, respectively. All treatments had significantly higher yields compared with the control, but there were no differences among treatments receiving fungicides. This results from this study suggest that suppression of LLS with prescription fungicide programs can be enhanced by modifying initial fungicide applications to correspond with actual disease onset.

INTRODUCTION

A major component of peanut (*Arachis hypogaea* L.) production is focused on protecting the crop from early leaf spot (ELS), caused by *Cercospora arachidicola* Hori, and late leaf spot (LLS), caused by *Cercosporidium personatum* (M.A. Berk & Curtis) Deighton (Shokes and Culbreath, 1997). These are two of the most important diseases of peanut worldwide, and in untreated situations, this can result in complete defoliation/death of the peanut plant and associated yield losses as high as 70% (Backman and Crawford, 1984; McDonald et al., 1985; Shokes and Culbreath, 1997). Management relies heavily on fungicide inputs (Culbreath et al., 2006), which represent one of the highest input costs for peanut growers. In Georgia, the average annual damage plus cost of control from 2012 to 2014 was estimated at $37.9 million (Kemerait, 2012, 2013, 2014).

A key premise for effective management of peanut leaf spot with fungicides is to initiate applications prior to or at disease onset, and make subsequent applications at close enough intervals to ensure protection through the course of the season (Culbreath et al., 2006). Historically, fungicides have been applied on a calendar-based schedule, beginning at 30 days after planting (DAP), and every 14 days until ~ weeks prior to harvest (Culbreath et al., 2006). However, growers have a number of options to vary the timing of these applications. Weather-based advisories such was AU-Pnut allow growers to time initial and subsequent applications based on expected disease occurrence in relation to the probability of future rain events (Jacobi et al., 1995). Depending on the season, this theoretically allows for a wide range of initial applications dates and subsequent spray intervals.

A more recent alternative is to utilize prescription fungicide programs based on pre-plant field risk as predicted with Peanut Rx (Woodward et al., 2014). Peanut Rx is a risk index that acts as an educational tool for peanut producers in the southeastern United States to estimate the relative intensity of the most important foliar and soilborne diseases in a given field prior to planting (Kemerait et al., 2017). For leaf spot, pre-plant risk factors include cultivar, planting date, rotation, field history, and irrigation vs dryland (Kemerait et al., 2017). Risk points are assigned to each factor and after calculation of total risk points, fields are assigned a high, moderate or low level of risk. Prescription fungicide programs feature

later initial applications and greater spray intervals with decreasing risk values. High-risk fields are treated according to the standard 14-day calendar-based program (7 applications initiated at 30 DAP), whereas in moderate- and low-risk fields fungicide applications are initiated at 37 and 44 DAP, with subsequent applications made at 14 and/or 21 and 21-day intervals, for a total of 5 and 4 applications, respectively (Kemerait et al., 2017). These schedules are based on the expected development of the general leaf spot epidemic as predicted by risk. The assumption is that, depending on risk, the infection process will begin between 30 and 45 DAP, and that early- and mid- to mid-late season control are the most critical protection windows. However, timing of fungicide applications does not always align with actual development of ELS and LLS in the field, and could therefore lead to sub-optima control.

Generally, the onset of each disease is earlier with increasing field risk (Chapter 8). In high risk situations, where sources of inoculum of both leaf spot pathogens are present in the field, the onset of ELS typically occurs 30 to 40 days after planting (DAP), but LLS does not occur until 2 to 4 weeks later (Smith and Littrell, 1980). Onset also depend on which leaf spot is predominant. Fifty years ago, in Georgia, ELS was the most prevalent disease until a shift took place in 1976 and LLS prevailed until the mid-1990s (Cantonwine et al., 2008; Smith and Littrell, 1980). In recent years, there has been a resurgence of LLS in Georgia (Cantonwine et al., 2008), and now it is not unusual to have fields in close proximity that are predominantly one disease or the other (Chapter 3). We have recently shown that both diseases generally do not appear until > 100 DAP in moderate-risk fields that have a history of LLS, but this can still result in heavy epidemics toward the end of the season (Chapters 3 and 8).

The general efficacy of prescription fungicide programs has been well established in fields with varying risk levels and in fields with predominantly ELS or LLS (Woodward et al., 2014; Woodward et al., 2010). Previous studies demonstrates that although disease severity may be marginally higher in low- and moderate-risk programs, yield is not compromised in prescription fungicide programs (Woodward et al., 2014; Woodward et al., 2010). Similarly, in more recent trials, moderate- and low-risk fungicide programs resulted in higher disease severity than the high-risk, 7-spray program when evaluated in moderate-risk fields with a history of LLS, and although yield was not statistically different, there was a

consistent numerically trend toward lower yields in the plots with reduced fungicide programs (Chapter 3).

Given the consistency of delayed onset of ELS and LLS observed in moderate-risk fields with a history of LLS, we hypothesize that delaying the initial fungicide applications will better align with the expected disease onset and provide better disease control at the end of the season that will further increase the efficacy of reduced-fungicide programs. Therefore, the primary objective was to compare the efficacy of regular and delayed prescription fungicide programs in moderate-risk fields with a history of LLS. To further investigate the effect of delaying initial applications in-full season, calendar-based programs, a secondary objective was to evaluate early-season control vs. late-season control on two peanut cultivars in moderate-risk, dryland fields with a history of LLS.

MATERIALS AND METHODS

Delayed prescription fungicide programs. Three field trials were conducted at the RDC Pivot located on the University of Georgia Coastal Plain Experiment Station in Tifton, Georgia, during 2013 and 2014. The field was divided into four quadrants under a center pivot irrigation system; in each year, peanut was rotated to a new quadrant. In both years, each quadrant had the same previous history of cotton, soybean, corn and peanut. Each year, a winter cover crop of wheat (*Triticum aestivum*) was planted, and killed with an application of glyphosate (Roundup 4 EC, Monsanto, Kansas City, MO) at 1.2 kg a.i./ha approximately 2 weeks prior to planting. In 2013, one experiment was conducted in the portion of the field with conventional tillage that included deep-turning with a moldboard plow, and another trial was conducted in the strip-till section. Only the strip-till section was used in the trial conducted in 2014. Strip till plots were prepared for planting using a strip-till implement (as described by Monfort et al. (2004)). All plots were 1.8 m wide and 12.2 m long. The cultivar Georgia-06G was planted at 19.2 seeds/m in two single rows spaced 91-cm apart. Plots were planted on 16 May and 13 May and inverted at 145 and 160 DAP in 2013 and 2014, respectively. The late digging in 2014 resulted from a combination of late maturity and unconducive weather conditions. All herbicides, insecticides and

fertilizers were applied following recommendations of the University of Georgia Cooperative Extension Service (Monfort, 2017).

All trials were arranged in a randomized complete block design with four replications. Six fungicide treatments were evaluated, and all treatments included the following fungicides: propiconazole + chlorothalonil (Tilt Bravo, Syngenta Crop Protection, Greensboro, NC), azoxystrobin (Abound, Syngenta) and chlorothalonil (Bravo Weather Stik, Syngenta). All trials included an untreated control. Three of the fungicide treatments were comprised of high-, moderate- and low-risk fungicide programs initiated at 30, 37 and 44 days after planting (DAP), and subsequent sprays were made at 14- or 21-day intervals, depending on the program (see Table 12.1 for details). The other three treatments were modifications to each of the regular prescription programs. The modification involved delaying the first treatment to 44, 58 and 65 DAP for modified high-, moderate- and low-risk programs, respectively, and each program included an additional application 14 days after the last scheduled application in the regular programs (see Table 12.1 for details). All applications were made with a CO_2-pressurized backpack sprayer with four 8002VS flat-tip nozzles (Teejet Technologies, Springfield, IL) calibrated at 188 liters/ha.

Early vs late-season applications. Two field trials were conducted at the ABAC farm located at the University of Georgia Coastal Plain Experiment Station in Tifton, Georgia in 2014 and 2015. In each year, peanut followed a previous cropping sequence of cotton, corn and peanut. Each year, a winter cover crop of wheat was planted, and killed with an application of glyphosate (Roundup 4 EC, Monsanto) at 1.2 kg a.i./ha approximately 2 weeks prior to planting. Both trials had conventional tillage including deep-turning with a moldboard plow. All plots were 1.8 m wide and 12.2 m long. Peanuts were planted at 19.2 seeds/m in two single rows spaced 91 cm apart for all trials. Plots were planted on 3 June and 12 May and inverted at 153 and 154 DAP in 2014 and 2015, respectively. All herbicides, insecticides and fertilizers were applied following recommendations of the University of Georgia Cooperative Extension Service.

Both trials were arranged in a split-plot design with four replications. Fungicide program was the main plot, and cultivar (Georgia-06G and Georgia-12Y) was the subplot. Fungicide programs consisted of five different application schedules of chlorothalonil (Bravo Weather Stik, Syngenta), and an untreated control. Chlorothalonil was applied at a rate of 1.26 kg/ha according to the schedules: i) full-season program applied at 30, 44, 58, 72, 86, 100, 114 and 128 DAP; ii) early-season program applied at 30, 44, 58 and 72 DAP; iii) mid-late season program applied at 72, 86, 100, 114 and 128 DAP; iv) late-season program applied at 100, 114 and 128 DAP. At 60 and 90 DAP, flutolanil (Convoy, Nichino America, Wilmington, DE) was applied to the plots at a rate of 0.91 kg/ha to suppress stem rot caused by *Sclerotium rolfsii*. Applications were made with a CO_2-pressurized backpack sprayer, and a boom equipped with four 8002 flat-tip nozzles (Teejet Technologies, Springfield, IL) calibrated to deliver 188 liters/ha. Plots were supplemented with irrigation as needed, and all herbicides, insecticides and fertilizers were applied following recommendations of the University of Georgia Cooperative Extension Service.

Data collection. Beginning approximately 30 days after planting (DAP), plots were visually inspected for initial leaf spot symptoms. Upon detecting the first symptoms of leaf spot, a more intensive evaluation was initiated. In the prescription program trials, five main stems per plot were arbitrarily selected for destructive sampling. Samples were collected approximately every other week up to approximately 1 to 2 weeks prior to inverting the peanuts. In the early vs. late season application trials, four untreated border rows were arbitrarily selected within the field and monitored all season. All plots were destructively sampled one time at the end of the season. Samples were returned to the lab where the number of present and missing leaflets, and the number of ELS and LLS lesions per leaf were counted. ELS and LLS were differentiated by symptoms and signs associated with each causal pathogen.

Incidence of each disease at each assessment date for each plots was calculated by dividing the number of leaflets with at least one lesion by the total number of leaflets collected from the five main stems. Area under the disease progress curves (AUDPC) were calculated for each leaf spot based on the number of lesions per leaflet. AUDPC values were standardized (stAUDPC) by dividing AUDPC values

by the duration of the epidemic (Campbell and Madden, 1990). For the early- vs. late-season trials, severity was calculated as the average number of ELS and LLS lesions per leaflet. The day prior to inverting peanuts, final leaf spot intensity was assessed for all treatments with the Florida 1-10 scale, where 1 = no spots, and 10 = a completely defoliated plant (Chiteka et al., 1988). Immediately after inverting the peanuts, incidence of southern stem rot was estimated by counting the number of 30.5-cm sections ("hits") with at least one disease locus divided by the total number of possible 30.5-cm sections of linear row per plot; this was expressed as a percentage by multiplying by 100. After drying to approximately 10% moisture, pods from each plot were weighed.

Statistical analysis. Data were subjected to linear mixed model analysis of variance using PROC GLIMMIX (SAS v.9.4 Institute, Cary, NC). For the delayed prescription programs, the model was analyzed as a split-plot design with trial as the whole plots and fungicide program as the sub plots. Trial and fungicide program were included in the model as fixed effects, and replication and replication × trial were included as random effects. Contrast statements were used to compare regular programs vs. delayed programs for each risk level and across all risk levels. For the early- vs. late-season trials, the model was analyzed as a split-split-plot design with year as the whole plots, cultivar as the split plot and fungicide regime as the split-split plots. Trial, cultivar and fungicide program were included as fixed effects and replication, replication × trial and replication × variety were included as random effects. In all analyses, the SLICE option was used to explore all two-way interactions, and the Kenward-Roger option was used to adjust the denominator degrees of freedom, and differences in the least square means were tested by Tukey's multiple comparisons test. When data violated the assumptions of regularity, transformations were used. As such, standardized AUDPC (stAUDPC) was natural log-transformed and lesions per leaflet were square-root transformed prior to statistical analysis. Back-transformed means of all transformed variables are presented in the results.

RESULTS

Delayed prescription fungicide programs. Overall, weather conditions were more favorable for leaf spot disease in 2013 than in 2014 (Fig. 12.1). In 2013, rainfall was greater or equal to the 30-year

average in every month of the growing season, but in 2014, rainfall in all months except May was well below the 30-year average (Fig. 12.1). Hence, it is not surprising that disease onset was earlier and severity greater in 2013 than 2014 (Fig. 12.2). LLS was the predominant disease in both years across trials. In untreated plots, the onset of LLS either preceded or was equal to that of ELS, and ranged from 95 to 110 time after planting (Fig 12.2). Disease progress was slower in 2014 than in 2013, and at 145 DAP defoliation was ~ 40% less than in untreated plots in 2013 at the same DAP (Fig. 12.2). However, due to delayed maturity in 2014, defoliation in untreated plots did reach >60% by 160 DAP (Fig. 12.2).

Apart from a significant trial × fungicide program interaction for stAUDPC (standardized area under the disease progress curve), there were no other interactions for other variables analyzed in these studies (Table 12.2). For each trial, disease severity tended to increase with fewer fungicide applications, but more so in the regular prescription programs (Fig. 12.3). Values of stAUDPC were numerically lower in all treated plots than the untreated check, but did not differ significantly in the conventionally tilled field in 2013 (Fig. 12.3). Overall, stAUDPC values for each fungicide treatment for were similar among trials, but were near identical in the strip-tillage trials in 2013 and 2014 (Fig. 12.2). Final percent defoliation at the end of the season did not differ significantly among fungicide treatments, but all were significantly lower than the untreated check (Table 12.3). Final severity values based on the Florida 1-10 scale showed similar treatment differences as stAUDPC values (Table 12.3). Stem rot incidence was minimum (<10%) across trials, but was significantly lower in all treated plots than the untreated check. Yield was significantly higher in all treated plots compared to the untreated check, but there were no significant differences among fungicide treatments (Table 12.3). Due to a significant ($P < 0.05$) interaction between trial and fungicide program (Table 12.2), results for contrast statements for stAUDPC is not reported. For leaf spot severity, contrast statements showed that the lower Florida 1-10 values across the delayed programs was significantly different from the regular programs, but there was no significant difference for percent defoliation on the main stem (Table 12.4). Further results from the contrast statements showed that the moderate- and low-risk delayed programs were significantly better than the corresponding regular programs for leaf spot severity based on the Florida 1-10 scale (but not for

main stem defoliation), but there were no differences between each version of the high risk programs (Table 12.4). There were not significant differences among fungicide programs in regard to southern stem rot or yield (Table 12.4).

Early vs late season applications. Weather conditions were similar in June of 2014 and 2015, but July and August had substantially more rainfall in 2015 than in 2014 (Fig. 12.1). May and June were extremely dry in 2015, but rainfall in both months exceeded the 30-year average in 2014 (Fig. 12.1). However, disease onset of ELS and LLS was similar on both cultivars in both years and ranged from 95 to 110 DAP in untreated plots (Fig. 12.4). Both ELS and LLS were present, but LLS was the predominant disease in both years (Fig. 12.4). Disease progress was slow in both years and defoliation did not begin to increase substantially until >120 DAP (Fig. 12.4). Final defoliation was generally lower in 2014 than in 2015, and was lower on Georgia-12Y than on Georgia-06G in both years (Fig. 12.4).

Fungicide, cultivar and fungicide × variety had a significant effect on the number of lesions per leaflet (Table 12.5). For both cultivars, the number of lesions per leaflet was greatest in the untreated check, followed by the early application regime (Table 12.6). The number of lesions per leaflet in the mid-late season program was significantly lower than in the late-season program for Georgia-06G, but not for Georgia-12Y (Table 12.6), but did not differ significantly from the full-season program for either cultivar (Table 12.6). Georgia-06G had more lesions per leaflet for every treatment than Georgia-12Y (Table 12.6). Final severity values based on the Florida 1-10 scale resulted in similar interactions (Table 12.5) and treatment differences (Table 12.6) as obtained for the number of lesions per leaflet, but there were no differences between cultivars for disease severity assessed with the Florida 1-10 scale at P =0.05. Final percent defoliation was significantly affected by year, fungicide, year × fungicide and cultivar (Table 12.5). The effect of fungicide on percent defoliation resulted in similar trends as that of the other disease variables previously described, but generally was not as significant statistically (Table 12.6). Incidence of southern stem rot was near zero for every plot in both years, including the untreated control (Table 12.6). Yield was significantly higher in treated plots compared to the untreated check (Table 12.6). There were numerical differences in yield that corresponded to disease severity values, but

differences among fungicide treatments were not significant (Table 12.6). There was a significant year × cultivar interaction for yield (Table 12.5). In 2014, the mean yield across treatments was 6,111 kg/ha for Georgia-12Y and 5,764 kg/ha for Georgia-06G. In 2015, the mean yield across treatments was 8376 kg/ha for Georgia-06G yields and 7938 kg/ha for Georgian-12Y. The differences in yield between cultivars were significant in both years (Table 12.5).

DISCUSSION

Results presented here illustrate that delaying fungicide applications in order to coincide with the expected onset of LLS can provide similar levels of control to that of full-season programs. Furthermore, we showed that delayed prescription fungicide programs can provide similar or superior LLS control compared to corresponding programs initiated earlier in the season. While this did not result in a significant gain in yield, it does demonstrate the potential to delay fungicide timings up to 65 DAP without compromising yield. Furthermore, our results indicate that applications can be delayed up to 86 DAP in moderate-risk, dryland fields with a history of LLS. Overall, these data are contrary to similar studies (Culbreath et al., 2006; Shokes et al., 1982). Shokes et al. (1982) evaluated seven initial fungicide timings (34 to 118 DAP) and reported that with each delay in fungicide initiation date, there was a corresponding increase in LLS severity. In a similar study that evaluated three fungicide groups with six initiation dates (30 to 100 DAP) for the control of ELS, there was a linear increase in ELS severity with each delay in initiation date for two chemistries (chlorothalonil and tebuconazole), and a curvilinear increase for pyraclostrobin. In both studies, increased severity did not significantly affect yield until applications were delayed to ~ 70 DAP (Culbreath et al., 2006; Shokes et al., 1982). However, in our studies, we did not observe a significant reduction in yield when the initial application was delayed to 65 DAP in prescription programs (Table 12.3), or when the initial application was delayed to 86 or 100 DAP for calendar-based programs (Table 12.6). It should be noted that fields in the previously mentioned studies (Culbreath et al., 2006; Shokes et al., 1982) would have been considered high-risk based on Peanut Rx (Kemerait et al., 2017), whereas all fields in our studies were moderate risk

fields. This is likely one factor that may explain the lower disease severity and lack of yield loss across all delayed programs.

Results from these trials provide additional evidence that the onset of ELS and LLS is consistently later (>95 DAP) in moderate-risk fields with a history of LLS compared to moderate- or high-risk fields with a history of ELS. Furthermore, because the overall development of disease was similar in untreated plots across irrigated and dryland fields over the course of 3 years with very different weather patterns, these results demonstrate that calculating pre-plant risk via Peanut Rx, with additional knowledge of historical ELS or LLS predominance in a given field, is an excellent predictor of the onset and progress of the leaf spot diseases. These findings are consistent with recent work conducted by the authors (Chapter 3), where we reported that ELS and LLS onset was >100 DAP in 2011 and 2012 in moderate-risk fields with a history of LLS. Although there was a significant delay in disease onset, LLS developed rapidly toward the end of the season, particularly in the irrigated trials conducted in 2013 and 2014. This resulted in average final severity values of 8.4 (Table 12.5) on the Florida1-10 scale which confirms previous reports of the explosive nature of the LLS epidemic (Backman and Crawford, 1984; Hemingway, 1955). As a result, we found that the delayed prescription fungicide programs provided better disease suppression compared with their corresponding regular programs.

Moderate- and low-risk programs tended to have more foliar disease than high-risk programs, but there was no decrease in yield, which is consistent with our previous findings (Chapter 3). This supports the hypothesis by Gine (2012) that yield loss due to leaf spot in modern cultivars may not result in as steep a linear decline as previously reported for older, more susceptible cultivars like Florunner (Backman and Crawford, 1984). However, it should be noted that, similar to the trials conducted in our studies, Gine (2012) also pointed out that his studies were conducted in fields where both ELS and LLS onset were not observed until >90 DAP. This suggests that modern cultivars such as Georgia-06G and Georgia-12Y have a degree of tolerance to leaf spot that enables a higher level of defoliation to occur prior to leading to yield loss.

According to this hypothesis, plants that are disease-free during the critical pod-fill /early maturity growth stages are capable of losing a large portion of the canopy toward the end of the season without suffering yield loss. For Florunner, full pod fill occurred at the R6 growth stage (~74 DAP) and harvest maturity occurred at the R8 growth stage (129 DAP) (Boote, 1982). These growth stages are dependent on environmental conditions (Boote, 1982), and are likely different for each cultivar. Boote (1982) also reported that LLS compromised the peg strength at the R9 stage (123 DAP), and led to premature over ripening. Knauft et al. (1988) suggested that Florunner peg strength was weakened by severe epidemics of LLS which resulted in more severe yield losses associated with harvesting operations. According to Colvin (2015), Georgia-06G peg strength was not compromised by moderate leaf spot epidemics. Therefore, it is possible that modern cultivars such as Georgia-06G and Georgia-12Y can withstand more leaf spot defoliation later in the season due to stronger pegs. Our results seem to corroborate this hypothesis, particularly the effect of the early season application of chlorothalonil alone in 2014 and 2015. While this treatment had the highest disease values (Table 12.6), it did not result in a significant yield loss compared to the full-season program. Early applications ended at 72 DAP thus ensuring protection up to 86 DAP. The leaf spot epidemic did not really get under way until >100 DAP in both years. Taken together, this strongly indicates that the majority of the yield reduction due to leaf spot occurred on plants that were > 100 DAP. We suggest that more research is needed on the relationship between yield loss and leaf spot when severe epidemics are delayed until later in the season.

For the 2014 and 2015 trials designed to evaluated late-season applications of chlorothalonil alone, onset of leaf spot was not detected until 111 and 95 DAP, respectively. However, since disease onset did not occur until > 90 DAP, why was there was more disease in plots where fungicide applications were initiated at 86 DAP compared with those at 30 DAP? Firstly, this can be partially explained by considering the time required for infection prior to the development of symptoms and signs. The incubation and latent periods for both ELS and LLS is ~ 7 to 14 and ~14 to 21 days, respectively, depending on genotype and environment (Cantonwine et al., 2008; Shew et al., 1988; Shokes and Culbreath, 1997). Here, onset was defined as the first sporulating lesion, and thus the infection process

for an onset of 95 and 110 DAP would have commenced at ~90 to 95 and ~ 75 to 80 DAP, respectively. Applications made at 86 DAP would have been made prior to or immediately after infection occurred in 2014 and 2015 respectively, and those made at 100 DAP would have been after infection occurred in both years. Because chlorothalonil is strictly a contact fungicide, this could explain why there was more disease in the delayed initiation programs.

Secondly, although applications initiated at 30 and 86 DAP were both prior to disease onset, significantly higher levels of disease in the latter treatment could also be linked to higher residual levels of chlorothalonil in the lower canopy of the peanut plant of the former treatment. Chlorothalonil is known to be fairly stable compound with a 65-day aqueous photolysis half-life (NCBI), and several researchers have examined the persistence of chlorothalonil residues on a range of crops (Bruhn and Fry, 1982a, b; Latin, 2006; Moorman and Lease, 1992), including peanut (Brenneman et al., 1990; Elliott and Spurr, 1993). For peanut, Elliott and Spurr (1993) reported an average half-life of 13.6 days, and that the residues levels declined with increasing time intervals between applications. Twenty days after a single application, the residue levels on the foliage were reduced more than half (Elliott and Spurr, 1993). They also reported that fungicide persisted longer in the lower layer of the canopy. Brenneman et al. (1990) also reported higher residue levels on the top than the bottom part of the canopy, but they reported a half-life of 3.8 days in the top and 4.8 days in the middle and lower levels of the canopy. Bruhn and Fry (1982b) reported a higher half-life of chlorothalonil in the bottom of the potato canopy (21.3 days) compared to the top part of the canopy (3.6 days). Rainfall also had a major effect on residue levels on peanut (Elliott and Spurr, 1993), and in studies on potato canopies, it was shown that chlorothalonil can be re-distributed to the lower part of the canopy (Bruhn and Fry, 1982a).

It should be noted that the persistence of chlorothalonil residue is likely dependent on the formulation (e.g., Bravo Ultra vs. Bravo WeatherStik), and therefore, the chlorothalonil formulation used in our studies may not allow for strong inferences based on the previously mentioned studies. However, assuming that a higher half-life in the lower part of the peanut canopy could provide a degree of extended protection on previously treated peanut plants, it is possible that there may also be a higher residue level

in the bottom part of the canopy if chlorothalonil is redistributed from the upper canopy. Applications started at 86 or 100 DAP would likely not have completely penetrated the interior of the canopy (Brenneman et al., 1990), and could therefore result in less uniform coverage of the plant. It is also possible that infections had already occurred by 86 DAP, but were not visible until later in the season or not detected by our sampling method. Further evidence of residual activity of chlorothalonil comes from the early season applications of chlorothalonil that ended at 72 DAP. If chlorothalonil residues were washed off the plant by 14 DAP, protection would have ended at 86 DAP – which was prior to symptom development. However, the early treatment had significantly less disease pressure than the untreated check. More research is needed to determine whether the actual level of chlorothalonil residues on the foliage would be high enough to inhibit the leaf spot pathogens.

In conclusion, these results suggest that the management of LLS with prescription fungicide programs can be enhanced by modifying initial fungicide applications to correspond with actual disease onset. By doing so, applications are shifted to focus on the latter part of the season when the disease is of greatest impact on yield. Our results showed that delayed prescription fungicide programs significantly improved leaf spot control compared to the regular programs without compromising yield. Similarly, results from the studies conducted in 2014 and 2015 indicate that initial applications can be delayed up to 100 DAP on two modern runner cultivars without a significant yield reduction compared to the full-season program. Taken together, these results indicate that, in addition to overall leaf spot risk predicted with Peanut Rx, optima fungicide application timings may depend on whether ELS or LLS is predominant in a field. Although modifications to prescription programs did not provide a significant gain in yield on the cultivar Georgia-06G, we suggest that managing LLS the same way as ELS in moderate-risk fields with a previous history of LLS may increase risk to the overall leaf spot epidemic and decrease the efficacy of reduced-input fungicide programs on a more susceptible cultivar.

ACKNOWLEDGMENTS

We thank the personnel at the University of Georgia research stations for all of the assistance provided. Particularly, we thank John Paulk, Andy Carter, Lina Flor Young, Jeannette Mixon, Jhen Bennett and

Edna Shannon for technical support; and Xuelin Luo for statistical assistance. Weather data provided by the University of Georgia Automated Environmental Monitoring Network.

LITERATURE CITED

Backman, P., and Crawford, M. 1984. Relationship between yield loss and severity of early and late leafspot diseases of peanut. Phytopathology 74:1101-1103.

Boote, K. 1982. Growth stages of peanut (*Arachis hypogaea* L.) Peanut Sci. 9:35-40.

Brenneman, T. B., Sumner, H. R., and Harrison, G. W. 1990. Deposition and retention of chlorothalonil applied to peanut foliage: effects of application methods, fungicide formulations and oil additive. Peanut Sci. 17:80-84.

Bruhn, J., and Fry, W. 1982a. A statistical model of fungicide deposition on potato foliage. Phytopathology 72:1301-1305.

Bruhn, J., and Fry, W. 1982b. A mathematical model of the spatial and temporal dynamics of chlorothalonil residues on potato foliage [*Solanum tuberosum*, fungicides, redistribution and loss of residues]. Phytopathology.

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

Cantonwine, E., Culbreath, A., Holbrook, C., and Gorbet, D. 2008. Disease progress of early leaf spot and components of resistance to *Cercospora arachidicola* and *Cercosporidium personatum* in runner-type peanut cultivars. Peanut Sci. 35:1-10.

Colvin, B. C. 2015. Influence of canopy health and maturity on peanut peg strength, yield, and grade. M.S. thesis. University of Florida, Gainsville.

Culbreath, A., Kemerait Jr, R., and Brenneman, T. 2006. Management of early leaf spot of peanut as affected by fungicide and date of spray program initiation. Plant Health Progress doi:10.1094/PHP-2006-0214-01-RS.

Elliott, V., and Spurr, H. 1993. Temporal dynamics of chlorothalonil residues on peanut foliage and the influence of weather factors and plant growth. Plant Dis. 77:455-460.

Gine, P. A. N. 2012. Characterization of the Relationship Between Leaf Spot Severity and Yield in New Peanut Runner-type Cultivars and Effects of New Peanut Genotypes on Leaf Spot Epidemics. M.S. thesis. University of Georgia, Athens.

Hemingway, J. 1955. The prevalence of two species of Cercospora on groundnuts. Trans. Brit. Mycol. Soc. 38:243-246.

Jacobi, J., Backman, P., Davis, D., and Brannen, P. 1995. AU-Pnuts advisory I: Development of a rule-based system for scheduling peanut leaf spot fungicide applications. Plant Dis. 79:666-671.

Kemerait, R. C., Jr. 2012. Georgia Plant Disease Loss Estimates. Coop. Ext. Ser. Bull. 102-5, University of Georgia, Athens.

Kemerait, R. C., Jr. 2013. Georgia Plant Disease Loss Estimates. Coop. Ext. Ser. Bull. 102-6, University of Georgia, Athens.

Kemerait, R. C., Jr. 2014. Georgia Plant Disease Loss Estimates. Coop. Ext. Ser. Bull. 102-7, University of Georgia, Athens.

Kemerait, R. C., Jr., Brenneman, T. B., and Culbreath, A. K. 2017. Peanut disease update. Pages 23-62 in: 2017 peanut update. W. Monfort, ed. Coop. Ext. Ser. College of Agric. Environ. Sci. University of Georgia., Athens.

Knauft, D., Gorbet, D., and Norden, A. 1988. Yield and market quality of seven peanut genotypes as affected by leafspot disease and harvest date. Peanut Sci. 15:9-13.

Latin, R. 2006. Residual efficacy of fungicides for control of dollar spot on creeping bentgrass. Plant Dis. 90:571-575.

McDonald, D., Subrahmanyam, P., Gibbons, R., and Smith, D. 1985. Early and Late Leaf Spots of Groundnut. Information Bull. No. 21. ICRISAT, Patancheru.

Monfort, W., Culbreath, A., Stevenson, K., Brenneman, T., Gorbet, D., and Phatak, S. 2004. Effects of reduced tillage, resistant cultivars, and reduced fungicide inputs on progress of early leaf spot of peanut *(Arachis hypogaea*). Plant Dis. 88:858-864.

Monfort, W., ed. 2017. 2017 Peanut Update. Coop. Ext. Ser. College of Agric. Environ. Sci. University of Georgia., Athens.

Moorman, G., and Lease, R. 1992. Residual efficacy of fungicides used in the management of Botrytis cinerea on greenhouse-grown geraniums. Plant Dis. 76:374-376.

NCBI. National Center for Biotechnology Information. PubChem Compound Database AID=12.2.3., Source=HSDB, http://toxnet.nlm.nih.gov/cgi-bin/sis/search/r?dbs+hsdb:@term+@rn+@rel+1897-45-6 (accessed May 21, 2017).

Shew, B., Beute, M., and Wynne, J. 1988. Effects of temperature and relative humidity on expression of resistance to *Cercosporidium personatum* in peanut. Phytopathology 78:493-498.

Shokes, F., and Culbreath, A. K. 1997. Early and late leaf spots. Pages 17-20 in: Compendium of Peanut Diseases, 2nd Ed. N. Kokalis-Burelle, D. Porter, R. Rodriguez-Kabana, D. Smith and P. Subrahmanyam, eds. American Phytopathological Society Press, St. Paul, MN.

Shokes, F., Gorbet, D., and Sanden, G. 1982. Effect of planting date and date of spray initiation on control of peanut leaf spots in Florida. Plant Dis. 66:574-575.

Smith, D. H., and Littrell, R. H. 1980. Management of peanut foliar diseases with fungicides. Plant Dis. 64:356-361.

Woodward, J., Brenneman, T., Kemerait, R., Culbreath, A., and Smith, N. 2010. Management of peanut diseases with reduced input fungicide programs in fields with varying levels of disease risk. Crop Protect. 29:222-229.

Woodward, J., Brenneman, T., Kemerait Jr, R., Culbreath, A., and Smith, N. 2014. On-farm evaluations of reduced input fungicide programs in peanut fields with low, moderate, or high levels of disease risk. Peanut Sci. 41:50-57.

Table 12.1. Regular and delayed prescription peanut leaf spot fungicide programs used in 2013 and 2014.

Program[v]	Total[w]	Timing[x]	Fungicide	Formulation	Rate (kg a.i./ha)[y]
High	7	30, 44, 72	Propiconazole	Tilt Bravo™	0.06
			+ Chlorothalonil		0.84
		58, 86	Azoxystrobin	Abound®	0.33
		100, 114	Chlorothalonil	Bravo Weather Stik®	1.26
Mod	6	37	Propiconazole	Tilt Bravo™	0.09
			+ Chlorothalonil		1.26
		58, 86	Azoxystrobin	Abound®	0.33
		72	Propiconazole	Tilt Bravo™	0.06
			+ Chlorothalonil		0.84
		107	Chlorothalonil	Bravo Weather Stik®	1.26
Low	4	44,	Propiconazole	Tilt Bravo™	0.06
			+ Chlorothalonil		1.26
		65, 86	Azoxystrobin	Abound®	0.22
			+ Chlorothalonil	Bravo Weather Stik®	0.84
		107	Chlorothalonil	Bravo Weather Stik®	1.26
High (delayed)	7	44, 72, 128	Propiconazole	Tilt Bravo™	0.06
			+ Chlorothalonil		0.84
		58, 86	Azoxystrobin	Abound®	0.33
		100, 114	Chlorothalonil	Bravo Weather Stik®	1.26
Mod (delayed)	6	58, 86	Azoxystrobin	Abound®	0.33
		72	Propiconazole	Tilt Bravo™	0.06
			+ Chlorothalonil		0.84
		107	Chlorothalonil	Bravo Weather Stik®	1.26
		121	Propiconazole	Tilt Bravo™	0.09
			+ Chlorothalonil		1.26
Low (delayed)	4	65, 86	Azoxystrobin	Abound®	0.22
			+ Chlorothalonil	Bravo Weather Stik®	0.84
		107	Chlorothalonil	Bravo Weather Stik®	1.26
		121	Propiconazole	Tilt Bravo™	0.09
			+ Chlorothalonil		1.26
Untreated		-	-	-	-

[v] Prescription fungicide programs based upon Peanut Rx are labeled High, Mod (Moderate) and Low; regular programs were modified (delayed) by removing the first application date of each program, and inserting this application 2 weeks after the last application of the regular program.

[w] Total number of applications made each season for each fungicide program.

[x] Days after planting at which each application was made.

[y] Rate of each fungicide application is expressed as kg a.i./ha.

Table 12.2. P-values from the linear mixed model analysis of variance results for all leaf spot related variables, stem rot and yield for trials conducted in 2013 and 2014.

Source of variation	stAUDPC[v]	Pdefol[w]	FL 1-10[x]	Stem rot[y]	Yield[z]
Trial (T)	0.0051	0.0028	<0.0001	<0.0001	0.0150
Fungicide program (F)	<0.0001	<.0001	<0.0001	0.0004	<0.0001
T x F	0.0110	0.4977	0.0641	0.3829	0.7868

[v] The total standardized AUDPC of the combined number of early and late leaf spot lesions.
[w] Percent defoliation on the main stem.
[x] Florida 1 to 10 leaf spot rating scale where 1 = no disease and 10 = a dead plant.
[y] Percent of 30.5-cm sections per linear row with at least one disease locus.
[z] Pod yield measured in kg/ha and adjusted to 10% moisture.

Table 12.3. Effect of regular and delayed prescription fungicide program on percent defoliation, leaf spot severity, stem rot incidence and peanut yield pooled across three trials conducted in 2013 and 2014.

Fungicide program[t]		Pdefol[u]	FL 1-10[w]	Stem rot[x]	Yield[y]
Regular	High	45.2 b[z]	4.2 d	2.1 b	7544.8 a
	Mod	53.7 b	5.6 b	3.3 b	7633.7 a
	Low	48.3 b	5.1 bc	3.0 b	7486.5 a
Delayed	High	43.5 b	3.9 d	2.5 b	7503.3 a
	Mod	46.3 b	4.4 cd	2.7 b	7473.0 a
	Low	46.1 b	4.4 cd	3.4 b	7764.3 a
Untreated	-	79.4 a	8.4 a	8.1 a	5837.4 b

[t] Regular prescription programs and modified prescription programs (i.e., first application was delayed).
[u] The total standardized AUDPC of the combined number of early and late leaf spot lesions.
[v] Percent defoliation on the main stem.
[w] Florida 1 to 10 leaf spot rating scale where 1 = no disease and 10 = a dead plant.
[x] Percent of 30.5-cm sections per linear row with at least one disease locus.
[y] Pod yield measured in kg/ha and adjusted to 10% moisture.
[z] Means within columns with the same letters are not significantly different based upon Tukey's honestly significant difference test.

Table 12.4. *P* values from contrast statements comparing the effect of regular versus delayed prescription fungicide programs against peanut leaf spot.

Fungicide program[t]	Pdefol[v]	FL 1-10[w]	Stem rot[x]	Yield[y]
Regular vs delayed	0.1344	0.0058	0.3156	0.5596
High vs High delayed	0.6417	0.2555	0.6890	0.9014
Mod vs Mod delayed	0.0392	<0.0001	0.5769	0.6221
Low vs Low delayed	0.5391	0.0057	0.6953	0.3996

[t] Regular prescription programs vs modified prescription programs (i.e., first application was delayed).
[u] The total standardized AUDPC of the combined number of early and late leaf spot lesions.
[v] Percent defoliation on the main stem.
[w] Florida 1 to 10 leaf spot rating scale where 1 = no disease and 10 = a dead plant.
[x] Percent of 30.5-cm sections per linear row with at least one disease locus.
[y] Pod yield measured in kg/ha and adjusted to 10% moisture.
[z] *P* values represent the combined analysis of all trials conducted during 2013 and 2014.

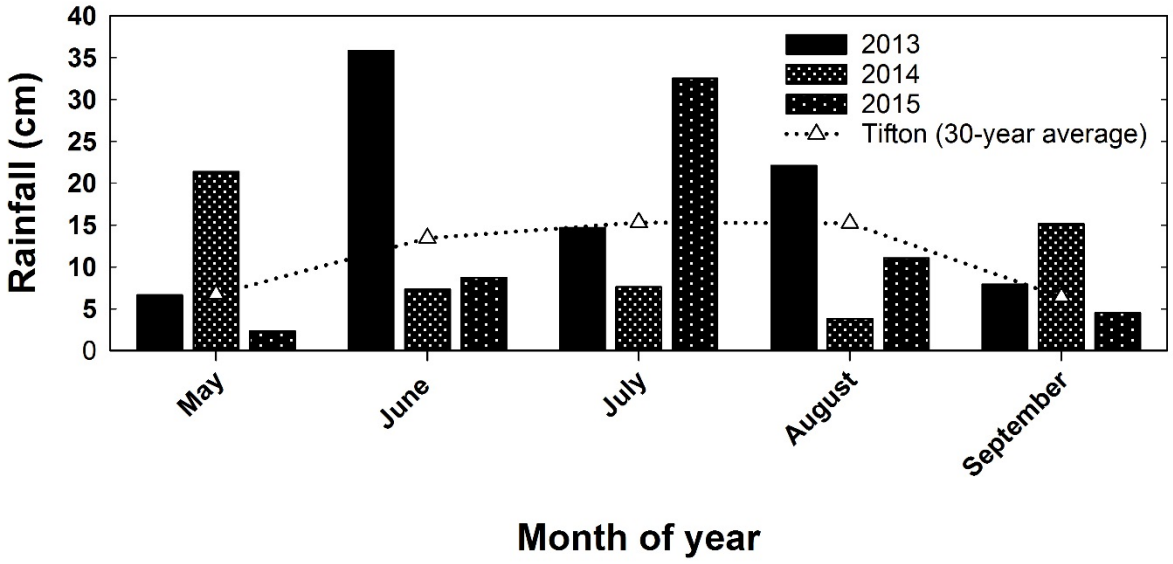


Fig. 12.1. Rainfall (cm) during the primary months that trials were conducted in 2014, 2015 and 2016 in Tifton, Georgia.

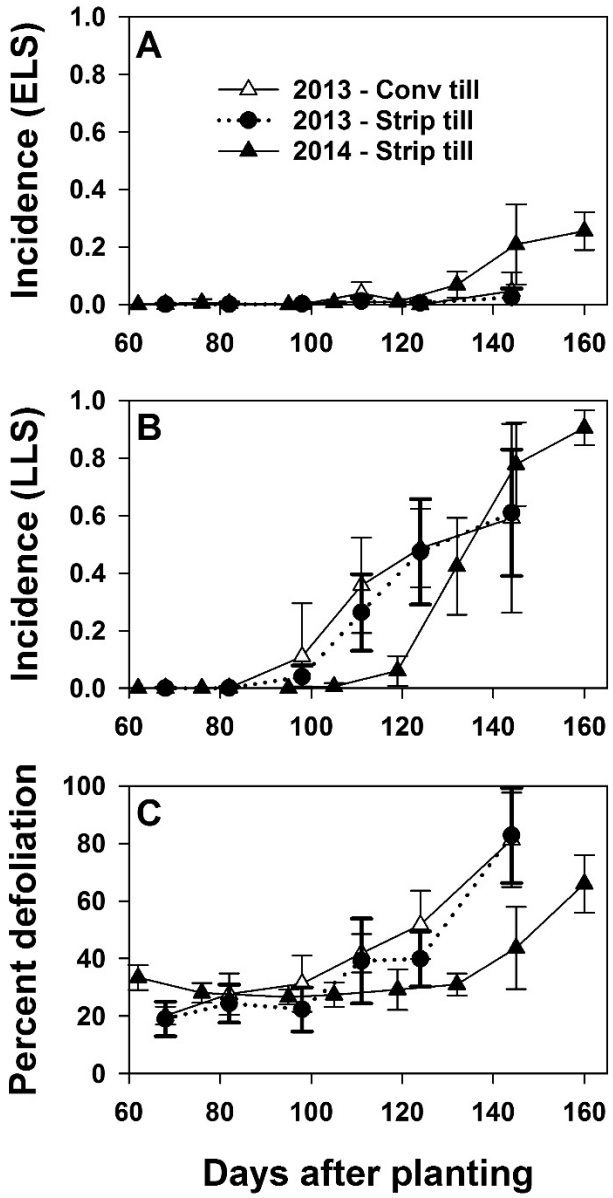


Fig. 12.2. Development of early (**A**) and late (**B**) leaf spot incidence and percent defoliation (**C**) in untreated plots planted to the peanut cultivar Georgia-06G. Trials were conducted in 2013 and 2014 in irrigated fields located in Tifton, Georgia. Error bars indicate the standard error of the mean (n = 4).

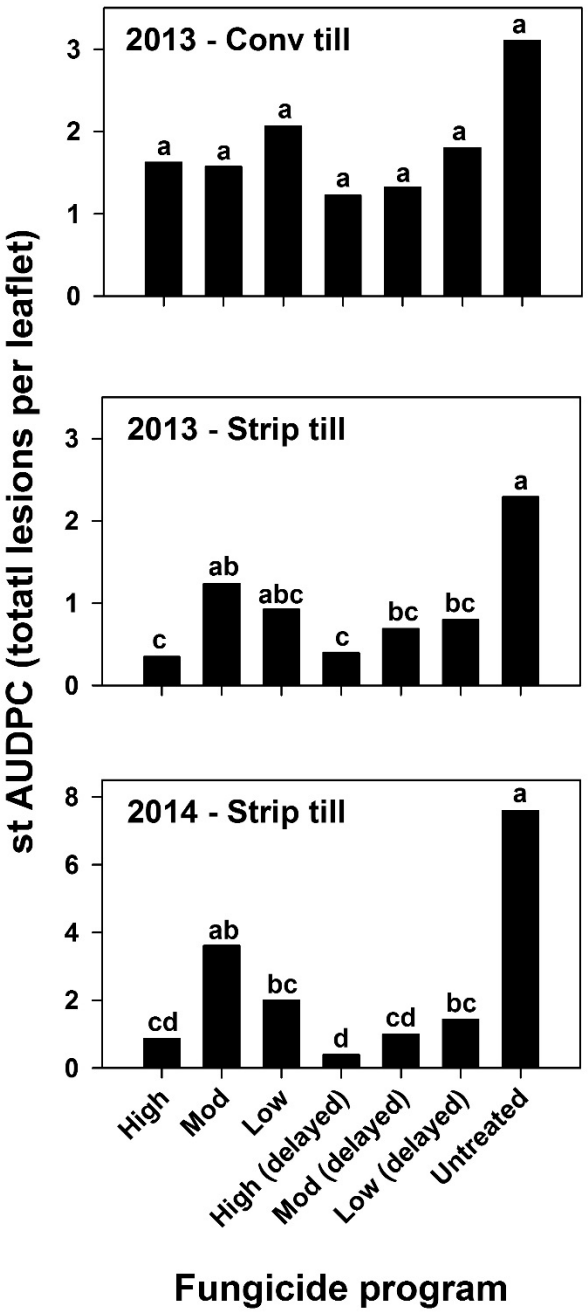


Fig. 12.3. Effect of regular and delayed prescription fungicide programs on the standardized AUDPC (area under the disease progress curve) of the combined number of early and late leaf spot lesions per leaflet. Bars with the same letters are not significantly different based upon Tukey's honestly significant difference test ($\alpha = 0.05$).

.

Table 12.5. P-values from the linear mixed model analysis of variance results for all leaf spot related variables, stem rot and yield for trials conducted in 2013 and 2014.

Source of variation	Lesions/leaflet	Pdefol[w]	FL 1-10[x]	Stem rot[y]	Yield[z]
Year (Y)	0.8385	0.0002	0.0006	0.1491	0.0004
Fungicide (F)	<0.0001	<0.0001	<0.0001	0.3241	0.0006
Y x F	0.2263	<0.0001	<0.0001	0.1624	0.4767
Variety (V)	<0.0001	<0.0001	<0.0001	0.9245	0.0026
Y x V	0.9086	0.7102	0.5887	0.7796	0.0002
F x V	0.0157	0.6757	0.0181	0.9189	0.8837
Y x F x V	0.0917	0.8140	0.3284	0.2560	0.3034

[v] The combined number of early and late leaf spot lesions per leaflet counted at the end of the season.
[w] Percent defoliation on the main stem.
[x] Florida 1 to 10 leaf spot rating scale where 1 = no disease and 10 = a dead plant.
[y] Percent of 30.5-cm sections per linear row with at least one disease locus.
[z] Pod yield measured in kg/ha and adjusted to 10% moisture.

Table 12.6. Effect of regular and delayed prescription fungicide program on percent defoliation, leaf spot severity, stem rot incidence and yield pooled across three trials conducted in 2013 and 2014.

	Lesions per leaflet			FL 1-10[w]			
Fungicide applications	Georgia-06G	Georgia-12Y	Pdefol[u]	Georgia-06G	Georgia-12Y	Stem rot[x]	Yield[y]
30, 44, 58, 72, 86, 100, 114, 128	0.6 d	0.2 c	23.8 c	1.8 e	1.8 d	0.3 a	7190.5 a
30, 44, 58, 72	6.4 b	4.5 b	27.2 bc	3.9 b	3.8 b	0.5 a	6960.2 a
86, 100, 114, 128	1.4 d	0.6 c	25.0 bc	2.5 d	2.3 c	0.3 a	7154.5 a
100, 114, 128	3.3 c	0.9 c	29.6 b	3.1 c	2.7 c	0.4 a	6974.8 a
Untreated	11.5 a	8.0 a	64.0 a	6.9 a	6.3 a	1.0 a	6429.8 b

[t] Day after planting that applications were made for each treatment. All fungicide treatments consisted of chlorothalonil (Bravo Weather Stik) at a rate of 1.26 kg/ha

[u] The combined number of early and late leaf spot lesions per leaflet counted at the end of the season.

[v] Percent defoliation on the main stem.

[w] Florida 1 to 10 leaf spot rating scale where 1 = no disease and 10 = a dead plant.

[x] Percent of 30.5-cm sections per linear row with at least one disease locus.

[y] Pod yield measured in kg/ha and adjusted to 10% moisture.

[z] Means within columns with the same letters are not significantly different based upon Tukey's honestly significant difference test.

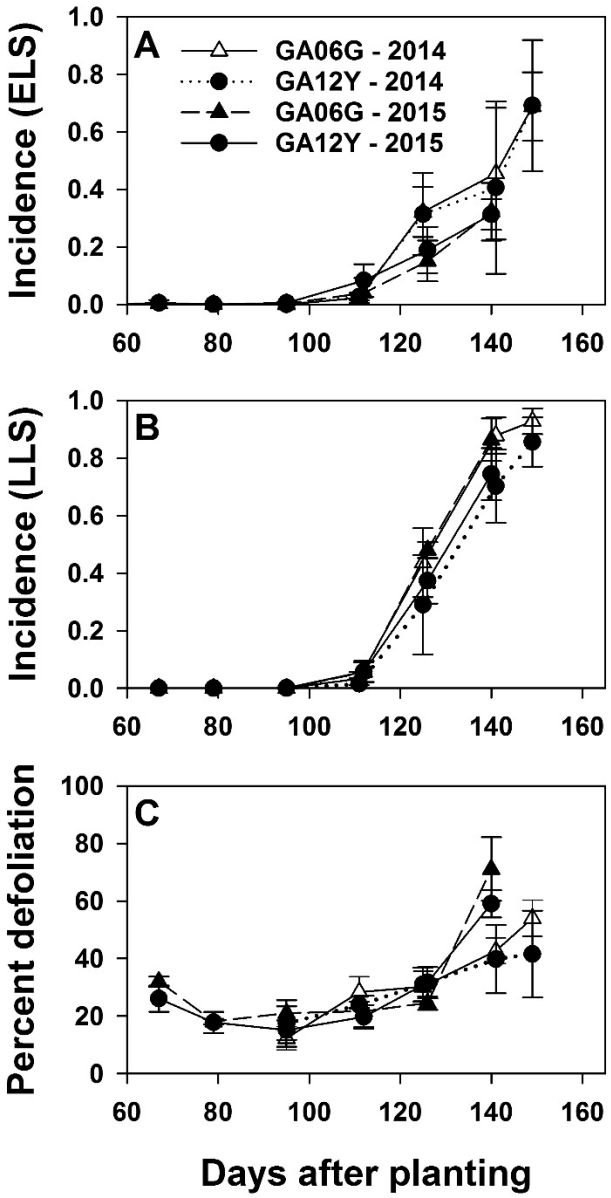


Fig. 12.4. Development of early (**A**) and late (**B**) leaf spot and percent defoliation (**C**) in untreated peanut plots planted to the cultivars Georgia-06G and Georgia-12Y. Studies were conducted in 2014 and 2015 in non-irrigated fields located in Tifton, Georgia. Error bars indicate the standard error of the mean (n = 4).