

Research Application Summary

Temporal development of sorghum anthracnose in Uganda

Akwero, A.¹, Edema, R.¹ & Okori, P.^{1,2}

¹Department of Agricultural Production, School of Agricultural Studies, College of Agricultural and Environmental Sciences, Makerere University, P. O. Box 7062, Kampala, Uganda

²International Crops Research Institute for the Semi-Arid Tropics (ICRISAT), Lilongwe, Malawi

Corresponding author: akwero@gmail.com

Abstract

The temporal characterization of sorghum anthracnose spread from an inoculum source and selection of models to describe the disease progress overtime was the focus of this study. The development of sorghum anthracnose in time on three cultivars was investigated in field plots inoculated with mixtures of strains of *Colletotrichum sublineolum* at two locations in Uganda in 2007 and 2008. The effect of genotype was used to estimate the infection rate, amount of inoculum at the start of epidemics, final severity of disease and the area under disease progress curves (AUDPC). The linearised logistic was used to describe the temporal disease development from inoculum source. Host genotype significantly affected the progress of foliar anthracnose from the point of inoculation with the increase in anthracnose severity limited to susceptible and moderately resistant cultivars. The rates of disease progress were higher at Makerere University Agricultural Research Institute Kanbanyolo (MUARIK) than at the National Semi Arid Resources Research Institute (NaSARRI). The rate of disease progress was also higher in 2008 but the intercept was lower in 2008 compared to 2007. Host resistance was consistently associated with lower AUDPC values, lower rates of disease progress and with reduced disease severity at crop maturity. This study identified accession MUC007/162 with lower rates of disease progress and low disease severity that can be used a parent in sorghum breeding programs for development of anthracnose resistant varieties.

Key words: *Colletotrichum sublineolum*, *Sorghum bicolor*, spread, temporal, Uganda

Résumé

Cette étude a fait l'objet de la caractérisation temporelle de la propagation de l'anthracnose du sorgho à partir d'une source d'inoculum et de la sélection de modèles pour décrire l'évolution temporelle de la pathologie. Le développement temporel de l'anthracnose du sorgho au sein de trois cultivars a été étudié dans des parcelles inoculées avec des mélanges de souches de *Colletotrichum sublineolum* dans deux localités en Ouganda en 2007 et 2008. L'effet du génotype a été utilisé pour estimer le taux d'infection, la quantité d'inoculum au début des épidémies, la sévérité finale de la pathologie et l'aire sous les courbes de

progression de la pathologie. La logistique linéarisée a été utilisée pour décrire le développement temporel de la pathologie à partir de la source de l'inoculum. Le génotype de l'hôte a significativement influencé le développement de l'anthracnose foliaire à partir du temps d'inoculation avec une augmentation de la sévérité de l'anthracnose limitée aux cultivars sensibles et modérément résistants. Les taux de progression de la pathologie étaient plus élevés à l'Institut de recherche agricole de l'Université de Makerere à Kanbanyolo qu'à l'Institut national de recherche sur les ressources semi-arides. Le taux de progression était aussi plus élevé en 2008, mais la valeur à l'ordonnée était plus faible en 2008 qu'en 2007. La résistance de l'hôte était constamment associée aux valeurs faibles des aires sous les courbes de progression, aux taux de progression plus faibles et à une réduction de la pathologie. Cette étude a identifié l'élément MUC007/162 avec des taux bas de progression et une faible sévérité de la pathologie qui peuvent être utilisés comme parent dans les programmes de sélection du sorgho pour le développement de variétés résistantes à l'anthracnose.

Mots clés: *Colletotrichum sublineolum*, *Sorghum bicolor*, dissémination, temporel, Ouganda

Introduction

Sorghum anthracnose caused by *Colletotrichum sublineolum* poses a serious threat to sorghum [*Sorghum bicolor* (L.) Moench] production and profitability (Thakur and Mathur, 2000). The pathogen affects all above ground parts of the plant with infection more commonly observed on the leaves (Prom *et al.*, 2012). The extent of damage is related to the degree of host susceptibility, the environment, the aggressiveness of the strains and the physiological status of the crop (Agrios, 2005). The disease is particularly damaging under warm and humid conditions (Ali and Warren, 1987). The pathogen survives as mycelium, conidia and microsclerotia in crop debris which act as a primary source of inoculum (Ali and Warren, 1992). Sserumaga *et al.* (2013) attributed the high anthracnose incidence in Uganda to the farming system where farmers grow sorghum on a given piece of land year after year resulting in inoculum build-up. Secondary inoculum is dispersed by rain splash to susceptible host tissues (Nicholson and Moraes, 1980).

To enable efficient control of a disease of an agricultural crop, it is desirable to understand the mechanisms that underlie its progress and spread-epidemics. The epidemic onset, infection rate, final amount of disease and area under disease progress curve (AUDPC) have been used to compare and evaluate disease management strategies (Madden and Campbell, 1990; Ojiambo *et al.*, 2000; Ngugi *et al.*, 2000; Li and TeBeest, 2009). Significant differences in time of inflection point, disease onset, absolute rate, disease severity and upper asymptote values for sorghum anthracnose epidemics caused by unknown field isolates was observed among sorghum cultivars in Kenya (Ngugi *et al.*, 2000). Observations from small-scale field experiments, in which disease severity is intensively mapped at several time points, may shed some light on these mechanisms (Gibson, 1996). Another approach to investigate the different factors influencing temporal disease progress is to plant cultivars with varying levels of disease expression. This provides information on the host pathogen interaction under varying environmental conditions. Such information can be quantified using mathematical

models to summarise disease progress (Madden and Campbell, 1990). There is however paucity of information in that regard on sorghum anthracnose in Uganda. Since anthracnose is endemic in sub-Saharan Africa, and is a threat to sorghum production in Uganda, it is important to characterise its epidemics so as to develop effective disease control packages, hence the focus of this study.

Materials and methods

Field plots were established in two locations; Makerere University Agricultural Research Institute, Kabanyolo (MUARIK) located in central Uganda and the National Semi-Arid Resources Research Institute (NaSARRI) in Serere district in eastern Uganda in 2007 (October to December) and 2008 (March to July). Three sorghum cultivars, a resistant (MUC007/162), a moderately resistant (MUC007/066) and MUC007/081 a susceptible line to anthracnose were used to study the epidemiology of sorghum anthracnose in the field. Where MUC007/066 seed was limiting, MUC007/153 or MUC007/033 derived from the same cluster was used. Plots of 5m x 5m were arranged in a randomised complete block design with three replications and at 60x30 cm plant spacing.

Inoculum preparation and inoculation was done using *C. sublineolum* infested sorghum kernels (Erpelding and Prom, 2006.) Twenty plants from the four middle rows were then tagged randomly. Each tagged plant was visually scored for percentage leaf area affected (PLAA) using a 1-9 visual rating scale (Hess *et al.*, 1999) 14 days after inoculation at 7 days interval. For each assessment date, severity data from each plot were averaged to give a single severity value based on a mean of 20 plants evaluated.

Severity data on PLAA was used to compute the Area under Disease Progress Curve (AUDPC) (Campbell and Madden, 1990). The formula used for computing AUDPC was given as:

$$\sum_i^{n-1} \left[\left(\frac{y_i + y_{i+1}}{2} \right) (t_{i+1} - t_1) \right]$$

In which t_i is the time at the i th observation, t_{i+1} is the time at (i+1)th observation, y_i is the disease severity (%) at i th observation, y_{i+1} is the severity at (i+1)th observation, t_1 is the time at the first observation

Disease proportion of sorghum anthracnose was analysed by genotype, location and year over time using linearised Logistic model in order to describe the temporal disease development because it provided the best fit. Goodness of fit was tested using coefficient of determination (R^2) and a plot of residuals (Campbell and Madden, 1990). The apparent infection rate (r), depicting rate of disease increase over time was computed by fitting the Logistic model:

$$\ln(y/I-y) = Y_{ox} + rt$$

Where y is the observed anthracnose severity, Y_{0x} is the logit of the amount of disease at time zero and r is rate of disease increase with time (t) to the logit transformed data. All the disease modelling was done using GenStat (Payne *et al.*, 2007).

Additionally, ANOVA was also performed on initial disease severity (Y_i), final disease severity (Y_f), the logit of the amount of disease at time zero (Y_{0x}) and rate of disease increase (r) with time and AUDPC. All the mean comparisons were conducted using Fisher's protected Least Significant Difference (LSD) test at $P < 0.05$ (Steel *et al.*, 1997)

Results

Disease progress on the selected ccessions. Severity of foliar anthracnose increased over time, suggesting that the inoculum built up over time. The disease progress curves plotted as anthracnose severity on a scale of 1-9 against days after inoculation showed that the epidemic started at varying dates depending on the genotype and location of the study. These days ranged from 32 to 50 days after inoculation. Disease severity on MUC007/162 remained consistently very low in both testing seasons at both locations (Fig. 1). The disease progress curves for accession MUC007/081 showed an initial steep rise followed by a reduction in the rate of progress while accession MUC007/066 had a steady increase of disease followed by leveling off. The disease generally developed differently across locations and seasons.

Temporal patterns of anthracnose epidemics. Analysis of disease progress curves in the two seasons in both locations revealed R^2 values for all individual accessions ranging from 0.71 and 0.99. The rates of disease progress (r), and predicted amount of disease at the start of the epidemics (Y_{0x}) were higher in MUARIK than NaSARRI and was higher for the susceptible accession. In general, the rate of disease progress was higher in 2008 than 2007 (Table 1). Although the amount of inoculum at the start of the epidemic was relatively high, the epidemics progressed slowly in 2007 and much faster in 2008. The genotype and the interaction between genotype and time had a significant ($P < 0.001$) effect on the apparent rate of infection (r) and intercept (Y_{0x}) at all locations during both seasons. Resistant accessio MUC007/162 consistently had lower AUDPC values across both location and season. The final regression model for explaining diseases progress at both locations was given as:

$$\ln(y/(1-y)) = (\text{intercept}) + \text{Time} + \text{Accession} + \text{Time} * \text{Accession}.$$

Discussion

Resistance to anthracnose in sorghum is well documented (Pande *et al.*, 1991; Erpelding and Prom, 2004, 2006; Erpelding, 2011). However, with the exception of Thomas *et al.* (1996), Ngugi *et al.* (2000) and Li and TeBeest (2009), few studies have examined how host resistance affects the dynamics of disease progress in sorghum. In the present study, host genotype significantly affected the rate of disease progress. Host resistance was consistently associated with lower rates of disease progress and with reduced disease severity

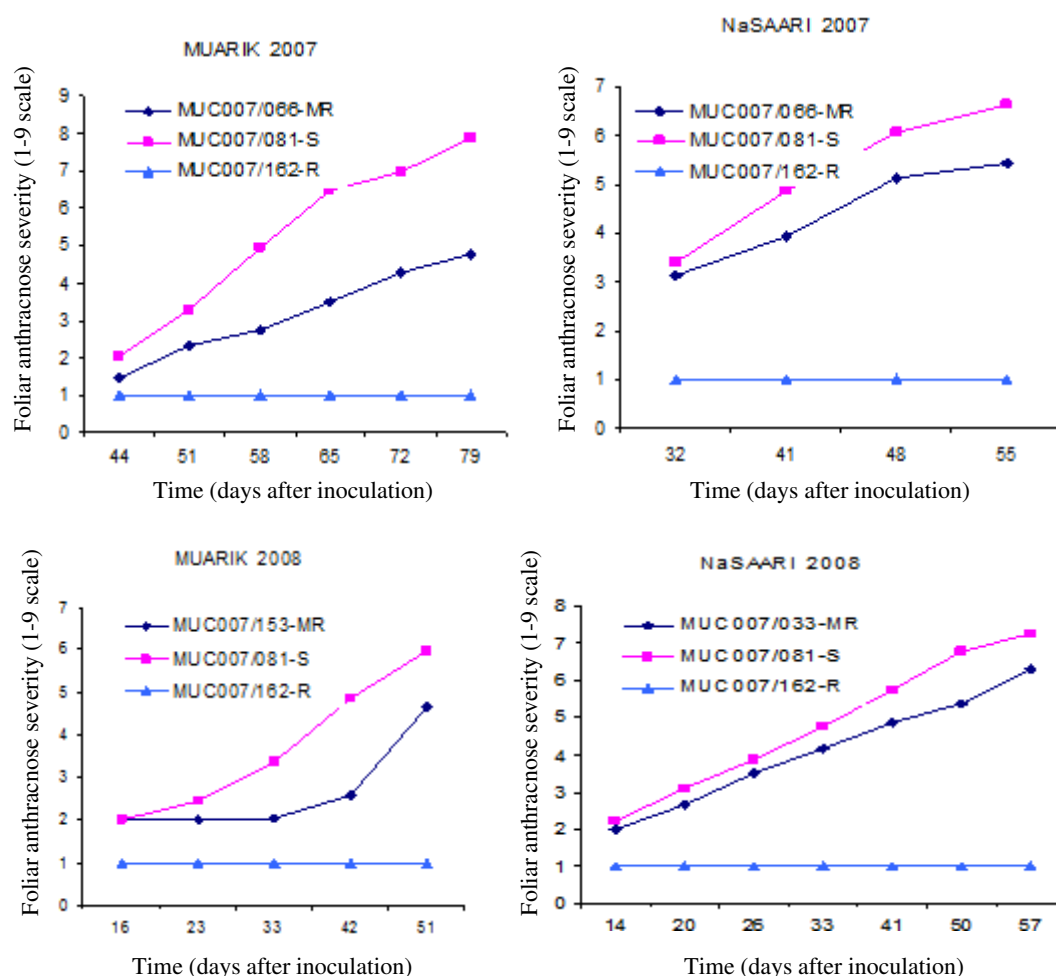


Figure 1. Foliar anthracnose disease progress curves on selected sorghum accessions during second rains (October to December) 2007 and first rains (March to July) 2008

at crop maturity. These results are in agreement with those of Ngugi *et al.* (2000) and Li and TeBeest (2009) who associated resistance to reduced rate of disease progress and lower disease levels in western Kenya. Among polycyclic diseases such as anthracnose, reduced rate of progress as that observed during the second rain season of 2008 and lower disease levels may indicate inhibition of pathogen development or host colonisation. This alludes to a quantitative form of resistance in accession MUC007/081. This form of resistance slows down the development of individual infection loci on a plant thereby slowing down the spread of the disease and the development of epidemics in the field (Agrios, 2005). In most cases, genotype significantly affected the severity of anthracnose. This study has identified accession MUC007/162 as a potential parent for breeding for resistance to sorghum anthracnose because of its consistently low anthracnose severity across location and year. This is the first study on the quantitative analysis of disease gradients of sorghum anthracnose in Uganda. In this study the ten isolates used for inoculation for the temporal analysis of

Table 1. Reaction of selected sorghum accessions to foliar anthracnose and disease epidemiology parameters obtained through linear regression analysis of disease progress of sorghum anthracnose for cultivar, year and location

Accession	^aY_i	^bY_f	AUDPC	Intercept $\hat{Y}_{0x}$	Slope r
MUARIK (2007)					
MUC007/162	1	1	35	-4.595 (0.265) ^a	0.000 (0.0043)
MUC007/066	1.43	4.75	111.2	-5.639 (0.375)	0.0351*** (0.006)
MUC007/081	2.03	7.88	186.37	-5.436 (0.375)	0.0398*** (0.006)
MUARIK (2008)					
MUC007/162	1	1	28	-0.459*** (0.145)	0.000 (0.0438)
MUC007/153	2	4.65	69.59	-4.263*** (0.206)	0.2004** (0.062)
MUC007/081	2.03	5.98	102.73	-4.218*** (0.206)	0.2959*** (0.062)
NaSARRI(2007)					
MUC007/162	1	1	21	-4.595 (0.122)	0.000 (0.0271)
MUC007/066	3.13	5.42	87.8	-4.264 (0.172)	0.0264*** (0.004)
MUC007/081	3.39	6.62	106.9	-3.954*** (0.172)	0.0243*** (0.004)
NaSARRI (2008)					
MUC007/162	1	1	42	-4.595*** (0.057)	0.000 (0.0128)
MUC007/033	2	6.3	173.4	-3.983*** (0.081)	0.1933*** (0.018)
MUC007/081	2.2	7.25	202.71	-3.888*** (0.081)	0.2070*** (0.018)

^aY_i = Initial severity was taken 44 days after inoculation (DAI) at MUARIK and 32 days after inoculation at NaSARRI (second rain 2007) and 16 days after inoculation at MUARIK and 14 days after inoculation in NaSARRI (first rain 2008)

^bY_f = Final severity was taken 79 (DAI) at MUARIK and 55 (DAI) at NaSARRI (second rains 2007) and 51 (DAI) at MUARIK and 57 DAI at NaSARRI (first rains 2008)

^astandard error is given in the parentheses. * Significant at 5%; ** Significant at 1%; *** Significant at 0.1%

$\hat{Y}_{0x}$ = intercept (amount of inoculum at the start of the epidemics), r = apparent rate of disease progress are computed according to Campbell and Madden, 1990

anthracnose epidemics in Uganda were not yet characterized into distinct pathotypes or races and yet physiological races within populations of *C. sublineolum* have been suggested to occur (Ali and Warren, 1987; Pande *et al.*, 1991; Casela *et al.*, 1995; Prom *et al.*, 2012).

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