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Resistance to anthracnose and turcicum leaf blight in sorghum under dual infection

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With 5 tables

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Abstract

A half diallel mating design was used to study the inheritance of anthracnose and turcicum leaf blight (TLB) in six sorghum cultivars. Applying pathogens inoculum separately and applying both pathogens simultaneously differently affected the reaction of each genotype. GA06/18 was resistant to both pathogens. GA06/106 x Epuripuri and MUC007/009 x Epuripuri showed high heterosis for resistance to both diseases indicating that they were good materials for sorghum breeding. Additive and non-additive (dominance) variance components were almost equally reflected by equal contribution of both variances towards the anthracnose resistance suggesting that both additive and dominance gene effects were involved in anthracnose resistance. Contribution of additive gene effects towards TLB resistance was greater than non-additive gene effects suggesting that additive gene effects were more important in controlling TLB resistance. This study highlighted that deployment of resistant varieties is the most cost effective way to manage both diseases especially when integrated with appropriate agronomy practices.

Key words: *Colletotrichum sublineolum* — dual infection — *Exserohilum turcicum* — gene action — *Sorghum bicolor*

Sorghum (*Sorghum bicolor* (L.)) and its fungal pathogens have continuously confronted each other through coevolution for growth and survival (Inghelandt et al. 2010). In this process, sorghum has evolved array of structural and gene-based defence mechanisms designed to combat different pathogen (Taylor et al. 2006), and so have pathogen, by developing new pathotypes of races (Tesso et al. 2012). Turcicum leaf blight (TLB), caused by *Exserohilum turcicum* (Pass) K.J. Leonard and E.G. Suggs (teleomorph: *Setosphaeria turcica* (Luttrell) Leonard and Suggs), and anthracnose, caused by *Colletotrichum graminicola* (Ces.) Munt.-Cvetk. (anamorph *Colletotrichum graminicola*), are two major foliar diseases of sorghum that limit its productivity in sub-Saharan Africa (Reddy and Prasad 2013). Both diseases cause grain abortion of up to 70% resulting in significant reduction of grain yield (Reddy and Prasad 2013). While several disease management measures exist, host-plant resistance remains the most economical and successful management strategy (Hess et al. 2002). Thus, for smallholder agriculture, and indeed, any other scale of farming, the breeding and deployment of high yielding resistant farmer-preferred adapted varieties is key for both diseases (Ngugi et al. 2000, Prom et al. 2012).

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The genetic variability that is present in both pathogens populations and the fact that sorghum is a crop presumably domesticated in Africa, suggests that there is potential for the identification of novel disease resistance genes in the crop for deployment (Marley et al. 2001, Ramathani et al. 2011). Indeed, several sources of resistance to TLB (Reddy and Prasad 2013) and anthracnose (Ngugi et al. 2000) have been identified separately. Resistance to TLB in cereals is conditioned by genes that display qualitative and quantitative reaction (Reddy and Prasad 2013), and in some cases, such as maize partial dominance has been demonstrated (Welz and Geiger 2000). This implies that additive effects with genotypes x environment interactions account for the variation in variety reaction to *E. turcicum* infection in sorghum (Ngugi et al. 2000). Resistance to anthracnose on the other hand is conditioned by dominant genes (Singh et al. 2006). Other studies have corroborated that resistance of both leaf spots and TLB in maize is controlled by dominance and additive gene effects (Matiello et al. 2012).

Given that both maize and sorghum related and shared several genes that are conserved in location, order and orientation (Swigoňová et al. 2004), the occurrence of common resistance loci architecture cannot be precluded. The aim of this study was to investigate nature of resistance to TLB and anthracnose in sorghum. In this study, combining ability was used to elucidate gene action for resistance to both diseases and to identify parents as well as hybrids with high general combining ability (GCA) and specific combining ability (SCA), respectively.

Material and Methods

Experimental sites: Greenhouse and field experiments were conducted at Makerere University Agricultural Research Institute Kabanyolo (MUARIK) in central of Uganda during the first rains (April–August) of 2012 and at Gezira Research Station, Wad Medani, Sudan during the rainy season (August–November) of 2014. MUARIK, a disease pressure site for both diseases (Sserumaga et al. 2013) is at an elevation of 1200 m above sea level (0°28′N and 32°37′E), and Wad Medani is at an elevation of 414 m above sea level (14°41′N and 33°05′E).

Techniques for inoculation: *Colletotrichum sublineolum* and *E. turcicum* inoculum was prepared as described by Ramathani et al. (2011). Isolates for both pathogens were obtained from Sudan for use in Sudan and from Uganda for use in Uganda. In the case of simultaneous infection, the plants were first inoculated with *E. turcicum* and immediately inoculated with *C. sublineolum* at vegetative growth stage two (five-leaf stage) under greenhouse conditions and at stage three in the field (Vanderlip 1993). For greenhouse experiments, 25 seedlings of each genotype were incubated in a

humid chamber at 22°C for 48 h after which the observations were taken (Mittal and Boora 2005). For field experiments, inoculation was performed in the evening hours when dew and ambient temperature were optimal for successful infection (Ramathani et al. 2011) and was repeated three times at six-intervals to ensure successful infection (Carson 1995).

Genetic materials and experimental designs: Six sorghum cultivars from East Africa were subjected to *C. sublineolum* and *E. turcicum*. Cultivar 'HD1', farmers-preferred varieties (FPV) in Sudan and cultivars 'GA06/106', 'GA06/18', 'MUC007/009', 'Sekedo' and 'Epuripuri', FPVs in Uganda were used in the study. Genotype GA06/18 is moderately susceptible to TLB (Beshir 2011, Beshir et al. 2012), and genotype MUC007/009 is resistant to TLB (Ramathani 2009) while genotype GA06/106 is moderately resistant to TLB (Beshir et al. 2012). The screening of the six parents was conducted under greenhouse and field in a condition in a split plot design in Uganda. Split plot was not used in Sudan. Each cultivar (subplots) was inoculated with four treatments (main plots): *C. sublineolum* only; *E. turcicum* only; both *C. sublineolum* and *E. turcicum*; and the uninoculated control. All required agronomic practices for the crop were followed.

The six genotypes were crossed following 6x6 half diallel mating design using model 1 method 2 (Griffing 1956). Only 12 F_1 progeny were obtained of a possible 15 (two crosses were lost due to incompatibility between the parental lines) and were advanced to F_2. The twelve F_2 progeny, along their parents, were evaluated under greenhouse conditions at Gezira Research Station, Wad Medani, Sudan. Experiments were arrayed following a randomized complete block design (RCBD) with three replications. Each F_2 population was represented by 90 progeny while the parents considered of 25 competitive plants on which observations were recorded. The F_2 distribution and segregation pattern for the 12 F_2 crosses could be included to improve the quality of the work especially for the resistant by susceptible crosses. It would be interesting to know how many genes are controlling resistance and if there are differences in loci. Humidity conditions were maintained in the greenhouse using overhead sprinklers. The experiment was artificially inoculated with both pathogens, but all the agronomic practices were applied to ensure good crop growth.

Data collection: Cultivars and F_2 segregating populations were assessed for disease severity from five-leaf stage (stage 2) (Vanderlip 1993) till physiological maturity at a weekly interval (Dube et al. 2010). Disease severity was computed based on the scale suggested by Ramathani et al. (2011).

Data analysis: Severity data were subjected to analysis of variance using GenStat 12th Edition (VSN International Ltd, 2 Amberside House Wood Lane Hemel Hempstead, HP2 4TP UK). Means were compared using the Fisher's protected least significance difference test (LSD) at $P < 0.05$ (Steel et al. 1997). Area under disease progress curves (AUDPC) were computed using the weekly severity ratings (Madden et al. 2007).

F-values for combining ability analysis were computed according to Owolade et al. (2006). The GCA mean square was tested against SCA mean square, and SCA mean square was tested against error mean squares while the crosses mean square was tested against error mean square (Vivek et al. 2009). These components were used to decide whether GCA or SCA would account for anthracnose and TLB resistance. Variance components were estimated to determine genetic and environmental effects. Additive (σ^2A), dominance (σ^2D) and phenotypic (σ^2P) variances were calculated from expected mean squares of analysis of variance according to Singh and Chaudhary (1999) as follows:

$$\sigma^2 A = 4\sigma^2 GCA \quad (1)$$

$$\sigma^2 D = 4\sigma^2 SCA \quad (2)$$

$$\sigma^2 P = 2\sigma^2 GCA + \sigma^2 SCA + \sigma^2 E \quad (3)$$

Heritability estimates on plot and entry mean basis were determined using the fixed effects model (Baker 1978). Broad sense heritability on entry mean basis (broad sense coefficient of genetic determination) and narrow sense heritability on plot basis (narrow sense coefficient of genetic determination) were determined as follows:

$$\text{BS-CGD}(H^2) = \text{Broad sense coefficient of genetic determination} = \frac{2\sigma^2 GCA + \sigma^2 SCA}{2\sigma^2 GCA + \sigma^2 SCA + \sigma^2 E} \quad (4)$$

$$\text{NS-CGD}(h^2) = \text{Narrow sense coefficient of genetic determination} = \frac{2\sigma^2 GCA}{2\sigma^2 GCA + \sigma^2 SCA + \sigma^2 E} \quad (5)$$

where; σ^2GCA, general combining ability variance; σ^2SCA, specific combining ability variance; σ^2E, environmental error variance component.

GCA effects were calculated and tested for significance from zero using a t-test at 90 degrees of freedom for the error mean square (Singh and Chaudhary (1999)). The GCA and SCA mean squares were calculated according to fixed effects model 1 (Baker 1978). Baker's ratio was used to determine the progeny performance and is hereafter referred to as Baker's ratio (Baker 1978). Significance of variance components was determined using t-test using the standard error of means and standard error of differences according to Dabholkar (1992).

$$\text{Baker's Ratio} = \frac{2gi^2}{2gi^2 + sij^2} \quad (6)$$

where; *gi* and *sij*, GCA and SCA mean squares.

Results

Disease reaction under dual infection

Analysis of variances for reaction to anthracnose and TLB severity based on final severities of both diseases were highly significant ($P < 0.001$) under greenhouse, but not significant under field conditions (Table 1). In general, disease was more severe under greenhouse than under field conditions (Table 1). Anthracnose severity and AUDPC varied significantly ($P < 0.05$) when only *C. sublineolum* was applied under greenhouse, while severity and AUDPC were not significantly different under field condition (Table 2). TLB severity assessed using both leaf area affected and lesion numbers was in general higher under dual than under single infection. Increase in disease severity under dual infection was in part due to larger lesions especially under greenhouse conditions (Table 2). The trend in data was confirmed through correlation analysis that showed a negative but non-significant correlation between anthracnose and TLB severity and AUDPC (data not shown).

Estimates of combining ability and heritability

GCA effects estimates of anthracnose and TLB are presented in Table 3. Estimates of GCA effects showed that cultivars HD1 and Epuripuri had the lowest significant ($P < 0.01$) but negative GCA effects for anthracnose, indicating resistance to anthracnose, while the cultivars GA06/106 and MUC007/009 had the highest significant ($P < 0.05$) but positive GCA. The cultivars GA06/106 and MUC007/009 had the lowest GCA for TLB severity indicating resistance to *E. turcicum*. Contrastingly, the cultivars HD1 and Sekedo had positive and significant ($P < 0.05$) GCA effects indicating susceptibility to *E. turcicum*. Cultivar GA06/18 had non-significant and negative GCA effects for anthracnose and TLB suggesting resistance to both diseases. Estimation of SCA for anthracnose and TLB severities of F_2

Table 1: Mean square of combined means for severity of leaf anthracnose and turcicum leaf blight under greenhouse and field conditions at MUARIK (first and second rains of 2012)

	Greenhouse conditions					Field conditions				
		Anthracnose		Turcicum leaf blight			Anthracnose		Turcicum leaf blight	
Sources of variation	df	Severity[1]	AUDPC[2]	Severity[1]	AUPDC[2]	df	Severity[1]	AUDPC[2]	Severity[1]	AUDPC[2]
Rep	2	139*	8591^{+}	887***	20 079***	2	314**	489 556***	247**	184 388**
Inoculation	3	194^{+}	3564	72	4215	3	23.8	61313^{+}	39	91 663
Genotype	16	91	7875^{+}	111	3192	16	53.3	46798^{+}	34	34 846
Interaction	48	71	4594^{+}	79***	3306**	47	37.9	29 990	29	25 491
Residual	126	38	3335	49	1977	87	46.3	35 268	44	38 143

+,*,**,***significantly different at $P \leq 0.1$, $P \leq 0.05$, $P \leq 0.01$ and $P \leq 0.001$.
[1]Final severity was taken 40 days after inoculation.
[2]Area under disease progress curve.

Table 2: Reaction of sorghum genotypes for *Colletotrichum sublineolum* and *Exserohilum turcicum* inoculum evaluated under greenhouse and field conditions at MUARIK (first and second rains of 2012)

	Greenhouse conditions						Field conditions					
Trait	ANT	TLB	ANT and TLB	Control	SED ($P \leq 0.05$)	LSD ($P \leq 0.05$)	ANT	TLB	ANT and TLB	Control	SED ($P \leq 0.05$)	LSD ($P \leq 0.05$)
Final severity (%) for ANT[1]	4.16	1.38	1.74	0.74	1.54	3.06	3.66	3.73	3.43	3.75	0.42	0.82
AUDPC for ANT[2]	0.66	0.47	0.53	0.38	0.13	0.27	0.82	0.84	0.91	0.99	0.09	0.18
Final severity (%) for TLB[1]	2.11	2.17	2.94	2.03	0.61	1.20	1.94	1.89	2.08	1.75	0.45	0.89
AUDPC for TLB[2]	0.82	0.84	1.03	0.89	0.08	0.16	0.41	0.50	0.51	0.56	0.09	0.18
Large lesion number	26.2	18.4	25.1	19.4	4.50	8.94	2.95	3.04	3.23	3.28	9.40	18.6

ANT, Anthracnose inoculum; TLB, Turcicum leaf blight inoculum.
[1]Final severity was taken 40 days after inoculation.
[2]Area under disease progress curve.

Table 3: Estimates of general combining ability (GCA) effects for reactions to anthracnose and turcicum leaf blight of six cultivars evaluated in Wad Medani under greenhouse condition (rains of 2014)

	Anthracnose		Turcicum leaf blight	
Parent	Final severity[1]	GCA	Final severity[1]	GCA
Epuripuri	5.8	−2.3*	14.5	1.7
GA06/106	14.7	3.1***	17.9	−5.3
GA06/18	5.4	−0.8	9.0	−2.1
HD1	5.1	−2.7**	12.4	6.8
MUC007/009	22.2	2.4*	13.5	−3.4
Sekedo	7.5	0.3	14.4	6.7
SEij	0.6	1.1	0.7	57.1
SED ($P \leq 0.05$)	0.9		0.9	
LSD ($P \leq 0.05$)	1.7		1.8	

*,**,***Significantly different at $P \leq 0.05$, $P \leq 0.01$ and $P \leq 0.001$.
[1]Final severity was taken 40 days after inoculation.

Table 4: Estimates of specific combining ability effects to anthracnose and turcicum leaf blight of F_2 populations evaluated in Wad Medani (rains of 2014)

Segregating population (F_2)	Anthracnose severity[1]	Turcicum leaf blight severity[1]
Epuripuri x GA06/18	0.0	2.4
Epuripuri x HD1	2.1	2.8
Epuripuri x Sekedo	−1.0	0.6
GA06/106 x Epuripuri	−0.4	−4.9
GA06/106 x GA06/18	−1.9	1.2
GA06/106 x HD1	−2.9	3.7
GA06/106 x MUC007/009	4.2**	0.6
GA06/106 x Sekedo	1.0	−0.6
GA06/18 x HD1	3.2*	−5.2
MUC007/009 x Epuripuri	−0.6	−0.9
MUC007/009 x GA06/18	−1.2	1.6
MUC007/009 x HD1	−2.4	−1.3

*,**Significantly different at $P \leq 0.05$ and $P \leq 0.01$.
[1]Final severity was taken 40 days after inoculation.

segregating populations are presented in Table 4. Non-significant but negative SCA estimates among F_2 segregating populations were observed on seven populations for anthracnose severity and five populations for TLB of 12 populations studied. The populations derived from the crosses GA06/106 x MUC007/009 and GA06/18 x HD1 had significant ($P \leq 0.05$) but positive SCA for anthracnose severity indicating susceptibility to *C. sublineolum*. Based on the lowest anthracnose severity and non-significant negative SCA estimates, two superior populations GA06/106 x HD1 and MUC007/009 x HD1 were found. Relatedly for TLB, non-significant negative SCA estimates were obtained in two populations GA06/18 x HD1 and GA06/106 x Epuripuri. Overall, the F_2 populations MUC007/009 x Epuripuri and MUC007/009 x HD1 showed non-significant negative SCA estimates for both diseases severities.

Heritability estimates and Baker's ratio of anthracnose and TLB are presented in Table 5. The estimate of broad sense heritability for anthracnose (0.73) and TLB (0.88) was high. However, the narrow sense heritability for anthracnose was low (0.42) while the narrow sense heritability for TLB was moderate (0.65). High non-significant GCA and SCA mean squares were observed among crosses for TLB severity. Higher SCA variance component (σ^2SCA) among populations was observed than

Table 5: Mean squares, variance components, Baker's ratio and heritability of F_2 populations for anthracnose and Turcicum leaf blight evaluated in Wad Medani under greenhouse condition (rains of 2014)

		Anthracnose severity[1]		Turcicum leaf blight severity[1]	
Source of variation	df	Mean squares	Variance components	Mean squares	Variance components
Population	11	134.5***		266.7	
GCA	5	20.6***	3.288	72.0	13.5
SCA	6	8.9*	4.800	14.3	9.5
Residual	90	4.1	4.100	4.8	4.8
σ^2A^2		13.15		53.79	
σ^2D^3		19.17		38.16	
σ^2P^4		15.51		41.20	
BS-CGD[5]		0.733		0.884	
NS-CGD[6]		0.424		0.652	
Baker's Ratio		0.407		0.585	

*,***significantly different at $P \leq 0.05$ and $P \leq 0.001$.
[1]Final severity was taken 40 days after inoculation.
[2]Variance due to additive effects.
[3]Variance due to non-additive (Dominance) effects.
[4]Phenotypic variance.
[5]Broad sense coefficient of genetic determination.
[6]Narrow sense coefficient of genetic determination.
[2,3,4,5,6]were computed according to fixed effect model.

σ^2GCA for anthracnose severity while the opposite was observed for TLB severity. Additive variances were significant ($P < 0.001$) for anthracnose severity, and non-significant for TLB. Dominance variances for anthracnose were higher than for TLB. Baker's ratio for TLB (0.59) was higher than the one of anthracnose (0.40). Baker's ratio, and broad and narrow sense heritability for anthracnose were less than those for TLB.

Discussion

Disease reaction under dual infection

The results in general show that under dual infection there appears to be cross-protection against anthracnose and not TLB. TLB lesions were larger under dual infection and so the AUDPC. These results were more vivid when the small lesions of TLB under dual, and single infections were compared. In general, plants with small lesions also had less anthracnose. This suggests that loci conditioning resistance to both diseases could be collected together. Thus, small lesion trait could be used to characterize and select for resistance to anthracnose and TLB in sorghum. Indeed, lesion size has been used in previous studies to characterize resistance to TLB in maize (Welz and Geiger 2000) and sorghum (Reddy and Prasad 2013). Genotypes did not show significant variations for lesion colour under both conditions. However, sorghum leaves and stalks of some genotypes for example MUC007/009, GA06/106 and GA06/18 accumulated red pigments upon wounding while others did not. Correlation of lesion colour and diseases resistance did not support the role of red pigmentation or anthocyanin in these sorghum genotypes as was previously suggested by Dykes et al. (2005) and Funnell-Harris et al. (2013).

Estimates of combining ability and heritability

The study also showed that additive and non-additive nature (dominance and epistasis) gene action conditioned resistance to both diseases. The role of non-additive gene action for anthracnose resistance was further confirmed by low Baker's ratio (0.04) (Falconer and Mackay 1996). Additive gene action also played a significant ($P < 0.05$) role in the inheritance of resistance to anthracnose in this study and was more important than non-additive gene action in conditioning resistance to TLB. The large role of additive gene action in host resistance of sorghum to both diseases suggests an evolutionary adaptation to a wide array of pathotypes. TLB in Eastern Africa has been reported in the past to be predominantly due to race O (Adipala et al. 1993), and more recently suggestions of new races identified from sorghum have been reported (Ramathani et al. 2011). Race O however is non-reactive (avirulent) to the common resistance genes designated Ht (Leonard et al. 1989), and the predominance of this race or prevalence of a few races of *E. turcicum* in Africa could thus explain the predominance of genetics observed. In other studies, six orthologous resistance genes in maize, but present in a cluster of three pairs, on chromosome 5 of sorghum have been reported (Martin 2011). These genes are highly conserved among the Poaceae especially in sorghum, rice, foxtail millet, maize and brachypodium and may reflect wide adaptation to diverse fungi and or their races as is expected under additive gene action. Indeed, the role of additive gene action in TLB resistance was confirmed by the moderately high Baker's ratio for TLB (Falconer and Mackay 1996). In the case of anthracnose, *C. sublineolum* additive and non-additive (dominance) variance components were almost equal, suggesting that both additive and dominance gene effects were involved in resistance to anthracnose. This is particularly plausible given highly variable pathogen among members of this (Cannon et al. 2012). Under dual infection TLB severity increased, while anthracnose severity decreased, suggesting confounding effects of both pathogens on the crop. Both pathogens are hemibiotrophs with final stages of necrotrophic phenotypes where they kill plant tissue to access nutrients (Balint-Kurti and Johal 2009, Cannon et al. 2012). This adaptation allows benign infection and perhaps suggests the suppression of resistance mechanisms. Interestingly, both pathogens are associated with induction of phytoalexins at the point of infection (Ibraheem et al. 2010, 2015). It is thus possible that during infection, *E. turcicum* may exploit the opportunistic biotropic phase of both pathogens, during which host defence is compromised and grow faster ultimately colonizing larger leaf area. Plants that are resistant to both diseases are therefore the best candidates, as they will exploit similar resistance mechanisms. In this study, parent GA06/18 had negative GCA for anthracnose and TLB suggesting that this genotype could be used in sorghum breeding. Progeny from resistant parents including GA06/106 such as GA06/106 x Epuripuri and MUC007/009 x Epuripuri showed negative SCA effects for both diseases indicating that these two crosses would yield populations for selection for resistance to anthracnose and TLB and could be utilized for selecting dual resistant cultivars. These materials and others exhibiting multiple host resistance to the endemic foliar pathogen will underpin sorghum productivity increase especially for resource constrained agriculture.

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