

Generation mean analysis for Turcicum Leaf Blight in Ugandan sorghum

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Abstract

Sorghum (*Sorghum bicolor* (L.) Moench) is frequently devastated by Turcicum leaf blight, caused by *Exserohilum turcicum*, leading to considerable grain and fodder yield losses. However, the mechanism of inheritance of resistance to *E. turcicum* in sorghum is poorly understood. Studies were carried out in Uganda to determine the mode of inheritance of *E. turcicum* resistance in sorghum. Segregating families derived from a cross of MUC007/009 (resistant) and *Epuripuri* (susceptible, an elite sorghum variety) were used along with the two parents. This was done together with four checks, namely GAO6/106 (moderately resistant), *Lulud* (susceptible), MUC007/010 (resistant) and GAO6/18 (moderately susceptible). The results of this study suggest that resistance in sorghum to *E. turcicum* is quantitative, with some contribution of additive, dominance and epistatic effects. It also highlights that the effect of the environment on the disease response of specific sorghum genotypes can be major.

Key words: Generation mean analysis, inheritance, *Exserohilum turcicum*, *Sorghum bicolor*, Uganda

Résumé

Le sorgho (*Sorghum bicolor* (L.) Moench) est fréquemment ravagé par la rouille foliaire *Turcicum*, causé par l'*Exserohilum turcicum*, conduisant à des pertes considérables de rendement en graines et en fourrage. Cependant, le mécanisme de l'hérédité de la résistance à *E. turcicum* dans le sorgho est mal compris. Des études ont été menées en Ouganda pour déterminer le mode de transmission de la résistance de *E. turcicum* dans le sorgho. Des familles en isolement, dérivées d'un croisement de MUC007/009 (résistant) et d'*Epuripuri* (sensible, une variété de sorgho élite) ont été utilisées avec les deux parents. Cela a été fait ensemble avec quatre contrôles, à savoir GAO6/106 (moyennement résistant), *Lulud* (sensible), MUC007/010 (résistant) et GAO6/18 (modérément sensible). Les résultats de cette étude suggèrent que la résistance du sorgho à *E. turcicum* est quantitative, avec une certaine contribution des effets additifs, de dominance et épistatiques.

On souligne également que l'effet de l'environnement sur la réponse à la maladie des génotypes spécifiques de sorgho peut être majeur.

Mots clés: Analyse des moyens de génération, héritage, *Exserohilum turcicum*, *Sorghum bicolor*, Ouganda

Background

Sorghum (*Sorghum bicolor* L. Moench) is a tropical C4 monocotyledonous plant and a subject of plant genomics research (Paterson, 2008). It has a relatively small genome of about 750 million base pairs (Arumuganathan and Earle, 1991). It also has a small amount of repetitive DNA and has co-linearity with other cereal genomes (Kong *et al.*, 2000). Sorghum is frequently devastated by Turcicum leaf blight (TLB) leading to considerable grain and fodder yield losses (Ogliaril *et al.*, 2007). TLB is caused by the ascomycete fungus *Exserohilum turcicum* (Pass) K.J. Leonard and E.G. Suggs (teliomorph: *Setosphaeria turcica* (Luttrell) Leonard and Suggs. (Carson, 1995). Yield losses of up to 50% is attributed to TLB if the disease is established on susceptible varieties before panicle emergence (Mittal and Boora, 2005).

Literature Summary

The most observed symptom of *Exserohilum turcicum* is long elliptic lesions that develop first on the lower leaves and progress upward. TLB express itself as small cigar – shaped lesions that may expand and coalesce and destroy the entire foliage (Welz, 2000; Ramathani *et al.*, 2011). In Uganda, studies have shown that the disease epidemics are largely due to amounts of infested maize residues in farm fields (Adipala *et al.*, 1993). Fungal isolates from maize could infect sorghum (Ramathani *et al.*, 2011).

Upon cross inoculation on maize differential lines harbouring different *Ht* genes, four *E. turcicum* isolates were identified as race 1, two as race 2, and one isolate corresponded to race 0 and race 3, respectively, whereas 10 isolates were unclassified (Ramathani *et al.*, 2011). The disease epidemics are favoured by high rainfall and relative humidity, moderate temperatures, and presence of large amounts of inoculum (Hennessy *et al.*, 1990). On the maize – *E. turcicum* pathosystem, a gene-for-gene relation has been reported (Carson, 1995). The best approach to control TLB in sorghum is by breeding and deploying genotypes with stable resistant to the disease. The objective of this work was to determine the mode of inheritance of resistance

Study Description

to TLB on sorghum basing on disease response of segregating population and using generation mean analysis.

Study site and genetic material. This study was carried out at Makerere University Agricultural Research Institute Kabanyolo (MUARIK) in Uganda. MUARIK (O°28'N and 32°37'E) is located 1200 m above sea level (Tenywa *et al.*, 2001). Segregating families were derived from a cross of MUC007/009 (resistant to TLB) and *Epuripuri* (elite but susceptible). Experiment were established in the greenhouse and in the field during the first rainy season of 2011. Generation mean analysis was used to determine the contribution of additive (a), dominant (d) and additive x additive epistatic (aa) genetic effects and also to confirm the ratio analysis for the population distribution. The populations included parents, F₁, BC₁F₁(*Epuripuri*), F₂, F_{2:3} and F_{2:4} generations. The genetic parameters listed in Table 1 were used to compute additive, dominance and epistatic genetic effects on inheritance of resistance to leaf blight among the developed sorghum populations (Bernardo, 2002).

Table 1. Genetic ratios of additive (a) and dominance (d) effects and epistatic (aa) (Bernardo, 2002).

Population type	Mean	a	d	aa
MUC007/009	1	-1	0	1
<i>Epuripuri</i>	1	1	0	1
F ₁	1	0	1	0
BC ₁ F ₁ (<i>Epuripuri</i>)	1	0.5	0.5	0.25
F ₂	1	0	0.5	0
F _{2:3}	1	0	0.25	0
F _{2:4}	1	0	0.125	0

Inoculum preparation and inoculation techniques. Lesions were cut from infected sorghum leaves in the field and placed on moist paper towels in petri dishes for 48 hours to allow sporulation (Carson, 1995). Single spores were picked from the lesions and placed on potato dextrose agar (PDA) plates and incubated at room temperature in the dark. Individual spores of *E. turcicum* were put on fresh PDA plates, and the resulting mycelia used to inoculate and colonize autoclaved sorghum kernels for about 14 days (Carson, 1995). The colonised sorghum kernels were air-dried prior to field inoculation. Inoculation was done at the five leaf stage (Vanderlip, 1993) by placing 20 to 30 colonised sorghum kernels into the leaf whorls. Inoculation was done in the evening when dew and

ambient temperature are optimal for successful infection (Carson, 1995).

Disease assessment and analysis. Disease severity was assessed using a scale of 0 to 75 where 0= no lesions identifiable on any of the leaves and 75 = 45 - 75% of leaf surface diseased (Adipala *et al.*, 1993). Assessment commenced at stage 4 (the growing point differentiation) (Vanderlip, 1993). Weekly assessments of disease severity were used to compute relative area under disease progress curves (AUPDC) as described by Campbell and Madden (1990) and Adipala *et al.* (1993). All data were subjected to generation mean analysis.

Research Application

Area under disease progress curve (AUDPC). The overall genetic effects on AUDPC and dates to 50% flowering among different generations showed few significant effects in either the greenhouse and field conditions (Tables 2 and 3). Under the greenhouse conditions, there were only slight and non-significant differences between the generations.

Table 2. Means of area under disease progress curve (AUDPC), initial and final TLB severity ratings under greenhouse conditions (First rains, 2010).

Population type	Disease reaction	No. of plants	^a AUDPC	^b Initial severity	^c Final severity
Generation					
BC ₁ F ₁ (<i>Epuripuri</i>)		34	2.7	0.1	5.3
F ₁		101	2.4	0.1	4.6
F ₂		81	2.2	0.1	4.2
F _{2:3}		68	2.6	0.2	4.4
F _{2:4}		62	2.5	0.2	4.7
Parents					
MUC007/009	Resistance	38	2.5	0.1	4.7
<i>Epuripuri</i>	Susceptible	36	2.6	0.2	4.9
Checks					
GA06/106	Moderately resistant	16	3.1	0.5	5.6
GA06/18	Moderately susceptible	14	4.6	0.5	7.7
LSD <0.05			0.64	0.41	0.98
CV%			48.1	119.1	39.9
SED			0.33	0.21	0.49

^a = AUDPC computed as described by Campbell and Madden (1990).

^b = Initial severity was taken 14 days after inoculation based on scale 0 - 75% (Adipala *et al.*, 1993).

^c = Final severity was taken 40 days after inoculation based on scale 0 - 75% (Adipala *et al.*, 1993).

Table 3. Means of area under disease progress curve (AUDPC), initial and final TLB severity ratings under field conditions (First rains, 2010).

Population type	Disease reaction	^a AUDPC	^b Initial severity	^c Final severity
Generation				
BC ₁ F ₁ (<i>Epuripuri</i>)		6.7	0.6	7.8
F ₁		6.7	0.6	6.2
F ₂		8.8	0.6	8.6
F _{2:3}		7.2	0.4	7.5
F _{2:4}		8.8	0.4	10.0
Parents				
MUC007/009	Resistant	4.3	0.3	5.3
<i>Epuripuri</i>	Susceptible	9.8	0.4	9.9
Checks				
GA06/106	Moderate resistant	5.8	0.7	6.2
GA06/18	Moderate susceptible	4.6	0.3	6.0
LSD<0.05		2.38	0.40	2.86
CV%		32.1	89.1	36.1
SED		1.20	0.20	1.44

^a = AUDPC computed as described by Campbell and Madden (1990).

^b = Initial severity was taken 14 days after inoculation based on scale 0 - 75%, (Adipala *et al.*, 1993).

^c = Final severity was taken 40 days after inoculation based on scale 0 - 75%, (Adipala *et al.*, 1993).

In the field, the effects of the two parents were significant (Table 3). Both F₁ and BC₁F₁(*Epuripuri*) had the same mean AUDPC of 6.7%, which was higher than the corresponding value from MUC007/009 the resistant parent (Table 3). The F₂ and F_{2:4} populations had the same mean AUDPC of 8.8% (Table 3). The AUDPC value (7.2%) for the F_{2:3} progeny was lower than in the susceptible parent. The mean AUDPC of GA06/18 (susceptible check) was much lower (4.6%) than the value from *Epuripuri* the susceptible parent. On the other hand, the mean AUDPC value for GA06/106 (moderately resistant) was slightly higher than for the resistant parent (5.8%) (Table 3). Both parents had similar days to flowering (81) under the greenhouse condition, with a one day difference under field conditions (Tables 2 and 3). The F₁, BC₁F₁(*Epuripuri*) and F_{2:4} progenies flowered earlier than the resistant parent while F_{2:3} progeny flowered at the same as the resistant parent.

The mode of inheritance of resistance in sorghum to leaf blight. Generation Mean Analysis was used to investigate the contribution of additive (a), dominant (d) and epistatic (aa)

effects on resistance in sorghum to TLB. Generation means were observed on seven populations, the resistant parent MUC007/009, the susceptible parent *Epuripuri*, F₁, F₂ and BC₁F₁(*Epuripuri*) plus F_{2:3} and F_{2:4} progeny. The overall genetic effects on AUDPC among different generations showed no significant effects in either the greenhouse and field conditions. Although partitioning of genetic effects into additive, epistatic and dominance components in this study did not reveal significant effects, studies in maize also indicate that resistance to *E. turcicum* is quantitative in nature. Overall, results of this study suggest that resistance in sorghum to *E. turcicum* is quantitative, with some contribution of additive, dominance and epistatic effects. It also highlights that environment can have major effects on the response of specific sorghum genotypes to infection by TLB.

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