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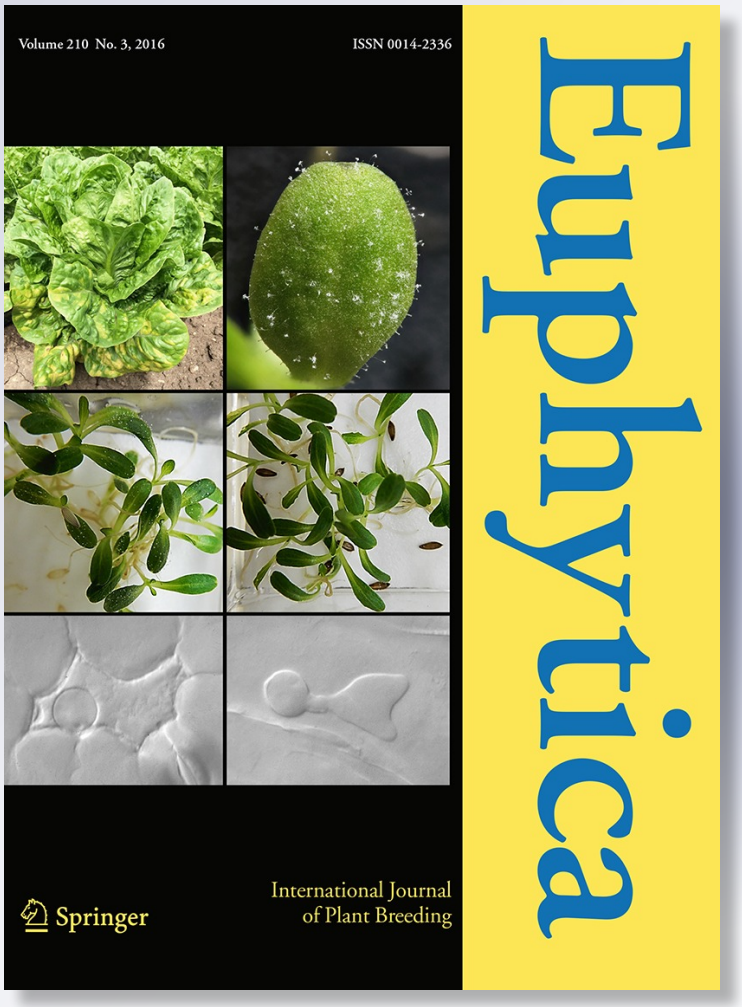

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Abstract Alternaria blight (*Alternaria* spp.) is an important sweetpotato disease in Uganda causing yield losses of over 50 % in susceptible genotypes. In Uganda, *Alternaria bataticola* and *Alternaria alternata* are the major species with A. *bataticola* the more aggressive of the two. The most effective control measure for this disease is the use of resistant genotypes. This study was conducted to determine the inheritance of resistance to Alternaria blight and the general and specific combining abilities of the available germplasm. Sixteen parental clones varying in reaction to Alternaria blight were crossed using the North Carolina II mating scheme. Due to incompatibility of some parents, two sets of compatible parents were formed. Differences among the families for Alternaria blight severity were significant while general combining ability (GCA) and specific combining ability (SCA) mean squares were highly significant ($P < 0.001$) for the disease with GCA sum of squares (SS) being more predominant at 67.4 % of the treatment SS for Set 1 and the SCA SS predominant at 54.0 % of the treatment SS for Set 2. This indicated that both additive and non-additive effects are important in controlling this trait. Some parents with high, negative GCA effects produced families with undesirable SCA effects and the reverse was also true. This implies that the best parents should not be chosen on GCA alone but also on SCA of their best crosses. The wide range in the area under disease progress curve for the families indicated that it was possible to select for highly resistant genotypes.

Keywords Alternaria blight · Area under disease progress curve · General combining ability (GCA) · Specific combining ability (SCA) · Sweetpotato

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Introduction

Alternaria leaf petiole and stem blight (*Alternaria* spp.) is an important sweetpotato disease. It is a minor disease in some parts of the world where sweetpotato is grown (Clark et al. 2009) and it has been reported in Zimbabwe (Whiteside 1966) and Nigeria (Arene and Nwankiti 1978) and more recently reported in South Africa (Narayanin et al. 2010a; Thompson et al. 2011). *Alternaria* spp (commonly referred to as Alternaria blight) is the most important fungal disease of

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sweetpotato in East Africa (Skoglund and Smit 1994). It is a serious production constraint due to the presence of aggressive *Alternaria* spp. (Lenné 1991). The major *Alternaria* species in East Africa are *Alternaria bataticola* and *A. alternata,* of which *A. bataticola* is the more aggressive species (Anginyah et al. 2001; Osiru et al. 2007b, 2008). However, studies by Woundenberg et al. (Woudenberg et al. 2014) on Alternaria samples collected from different parts of the world, revealed three other Alternaria species on sweetpotato, namely; *Alternaria. ipomoeae, Alternaria. neoipomoea* and *Alternaria. argyroxiphii.* In Uganda, *Alternaria* spp has gained importance in the last few years with resultant yield losses ranging from 25 to 54 % in some parts of the country (Osiru et al. 2007b). Several control measures can be employed against Alternaria blight. However, given the fact that sweetpotato is a low value crop and mostly grown by resource poor farmers in marginal areas, the most economic control measure is the use of resistant genotypes (Osiru et al. 2007b). Anginyah et al. (2001), van Bruggen (1984) and Narayanin et al. (2010b) reported differences in resistance levels among genotypes in Kenya, Ethiopia and South Africa, respectively. Similarly, in Uganda, Osiru et al. (2007b), Yada et al. (2011), Niringiye et al. (2014a, b) identified Alternaria blight resistant and susceptible genotypes. They attributed the differences in disease levels to inherent differences in susceptibility or resistance of the genotypes. In order to breed for resistance to the disease, whether durable or non-durable, it is essential to understand the mode of inheritance of the resistance. However, there is currently scant information about the inheritance of resistance to Alternaria blight in sweetpotato.

The mode of inheritance for resistance to several production constraints in sweetpotato has been studied by several workers. For example, Mihovilovich et al. (2000), Mwanga et al. (2002) and Okada et al. (2002) studied the mode of inheritance of sweetpotato virus disease (SPVD) and Collins (1977) investigated the inheritance of resistance to Fusarium wilt. Heritability estimates of resistance to root knot nematodes (*Meloidogyne* spp.) and soil insect pests were estimated by Jones and Dukes (1980) and Jones et al. (1979), respectively. The application of ten heritability estimates for different traits in sweetpotato breeding was reviewed by Jones (1986). Courtney et al. (2008) determined heritability estimates for micronutrient composition of sweetpotato storage roots while Gasura et al. (2008) analysed the genetic variance of root yield and quality, and severity of various virus diseases in sweetpotato germplasm in Uganda. Studies by Simon and Strandberg (1998) on *Alternaria dauci* (Kühn) on carrots (*Daucus carota* L.) and by Christ and Haynes (2001) on *Alternaria solani* of diploid potato (*Solanum tuberosum* L.) showed the additive variance to be more important than the non-additive variance. However, no such studies have been carried out to determine the mode of inheritance of Alternaria blight of sweetpotato and thus the need for this study.

Materials and methods

Germplasm source

Parental genotypes for this study comprised of six cultivars released by the National Sweetpotato Program at the National Crops Resources Research Institute (NaCRRI) and ten landraces commonly grown in different parts of Uganda. The released cultivars were NASPOT 1, NASPOT 2, NASPOT 4, Bwanjule, Tanzania, New Kawogo, and the landraces were Silk Omupya, Semanda, Kidodo, Araka Red, Dimbuka, Shock, Mbale, Budde, Magabali, and Silk Luwero. The levels of resistance of these parents to Alternaria blight were already known (Table 1). The resistant parents were used as female (seed) parents, while the moderately resistant and susceptible were used as male (pollen) parents.

Crossing block

The selected parents were planted in a crossing block at Mukono Zonal Agricultural Research and Development Institute (MUZARDI) in June 2009 and hand pollinations were made using a 7 × 9 North Carolina II mating design (Comstock and Robinson, 1948). However, as some parents were incompatible (Table 2), they were divided into two compatibility groups or sets (Table 3). Set 1 comprised the following females: Bwanjule, Silk Omupya, Semanda, Kidodo; and males: Araka Red, NASPOT 2, NASPOT 4, Dimbuka and NASPOT 1. Set 2 comprised the following females: Shock, Mbale, Tanzania; and males: Budde, Magabali, New Kawogo and Silk Luwero. A total of 32 families were generated from

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Table 1 Selected sweetpotato parents for hybridization for Alternaria blight genetic studies in Uganda

Name	District	Status	Alternaria blight	Reference
Semanda	Mpigi	Landrace	Resistant	Mwanga et al. (2009)
Silk Omupya	Palisa	Landrace	Resistant	USD
Silk Luwero	Luwero	Landrace	Moderate	Osiru et al. (2009a)
Kidodo	Kabale	Landrace	Resistant	USD
Dimbuka	Rakai	Landrace	Susceptible	Mwanga et al. (2007b)
Araka Red	Soroti	Landrace	Moderate	USD
Mbale	Mpigi	Landrace	Resistant	USD
Shock	Mbale	Landrace	Resistant	USD
Magabali	Kabale	Landrace	Susceptible	USD
Budde	Masaka	Landrace	Susceptible	USD
Bwanjule		Released	Resistant	Mwanga et al. (2001)
New Kawogo		Released	Susceptible	Mwanga et al. (2001)
NASPOT 1		Released	Susceptible	Gibson (2006) and Mwanga et al. (2003)
NASPOT 2		Released	Susceptible	Mwanga et al. (2003)
NASPOT 4		Released	Moderate	Mwanga et al. (2003)
Tanzania		Released	Resistant	Osiru et al. (2009b)

The National Sweetpotato Program collected sweetpotato landraces from all regions of Uganda in 2005 and evaluated them for SPVD, Alternaria blight and total storage root yield. The details are posted on the Uganda Sweetpotato Database (www.viazivitamu.org/ugasp_db/index.php) and partially published by (Yada et al. 2011)

USD Uganda sweetpotato database

Table 2 Cross-compatibility of sweetpotato genotypes selected as female and male parents

♀/♂	Budde	Araka Red	Magabali	New Kawogo	NASPOT 4	Dimbuka	NASPOT 2	NASPOT 1	Silk Luwero
Shock	✔	x	✔	✔	x	x	x	x	✔
Bwanjule	✔	✔	✔	x	✔	✔	✔	✔	x
Silk Omupya	x	✔	x	✔	✔	✔	✔	✔	✔
Mbale	✔	✔	✔	✔	x	x	x	x	✔
Tanzania	✔	x	✔	✔	x	x	✔	x	✔
Semanda	x	✔	x	✔	✔	✔	✔	✔	✔
Kidodo	✔	✔	✔	✔	✔	✔	✔	✔	x

✔ = Compatible, x = Incompatible

the crosses, viz. 20 families (4 × 5) from Set 1, and 12 families (3 × 4) from Set 2.

Hand pollination

Hand pollination was carried out using a modification of the method described by Wilson et al. (1989). The flower buds of the female parents to be hand pollinated the following morning were selected late in the evening, gently opened, emasculated and the corolla was held closed at the tip with a finely coiled length of aluminium foil. Similarly, unopened flowers of the male parents were held closed until the following morning. Hand pollination was carried out in the morning between 06h00 and 09h00. Each flower to be used as the source of pollen was removed from the male parent plant, the corolla opened and the anthers rubbed gently on the stigma of the reopened flower of the female parent. The corolla of the female flower was then closed again to prevent contamination by pollen carried by insects. The pollinated female flowers were inspected five to seven days later and those that had

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Table 3 Cross-compatible sweetpotato genotypes within each of the two sets with Alternaria blight resistant parents used as females and the moderately resistant and susceptible parents used as males

Set 1		Set 2	
Females	Males	Females	Males
Semanda	Dimbuka	Tanzania	New Kawogo
Silk Omupya	NASPOT 2	Mbale	Silk Luwero
Kidodo	NASPOT 1	Shock	Magabali
Bwanjule	NASPOT 4		Budde
	Araka Red		

been successfully pollinated, as evidenced by swollen ovaries, were counted and recorded.

Seedling generation

A wire file was used to mechanically scarify the seeds. The seeds were then immersed in water for 30 min and placed on moistened blotting paper overnight to allow the radical to emerge. The germinated seeds were then individually planted in the cells of plastic seedling trays containing heat sterilised soil and grouped according to family. When the seedlings were 6–10 cm in height they were transplanted to polyethylene bags, containing sterilised soil. Foliar fertilizer was applied once a week. Thirty seedlings from each family that had attained a vine length of 30–40 cm were selected for further multiplication. Side shoots were also cut and planted. In order to produce enough vine cuttings for a replicated trial at two sites, the rapid multiplication technique was used. Each vine was cut into short lengths of three nodes each to give 5–6 cuttings per F_1 genotype. Each cutting was planted in a polyethylene bag filled with sterilised soil, and watered twice daily. Foliar fertilizer was applied once a week after the cuttings had set roots. After four months the plants had produced several vines from which 30 cm long cuttings could be collected.

Field evaluation of F_1 families

The F_1 genotypes were evaluated at two sites during the first rains[1] of 2011 (2011A) namely: the National Crops Resources Research Institute (NaCRRI), located at Namulonge, 28 km from Kampala in central Uganda (0°32′N, 32°35′E; 1150 m above sea level (masl)); and Kachwekano Zonal Agricultural Research and Development Institute (KAZARDI) located 400 km from Kampala in south western Uganda (01°16′S, 29°57′E; 2200 masl). Kachwekano is a "hotspot" for Alternaria blight (Mwanga et al. 2007a; Osiru et al. 2007a), while Namulonge is located in an area of medium disease incidence (Mwanga et al. 2007a). The two trials were established in April 2011 (when the first rains had commenced) using a 5×7 row-column design (Patterson and Williams 1976) with two replications at each site. All 32 families from the two sets (without considering the sets) were randomly allocated to the plots within the design. The extra three plots at the bottom, right of each replication were planted to the 16 parents. Five cuttings from each of 30 F_1 siblings per family were planted 0.3 m apart on six ridges, each 7.5 m in length and spaced 1 m apart i.e. 150 cuttings were planted per plot. NASPOT 1, which was previously tested to be the most susceptible parent to Alternaria blight (Mwanga et al. 2003; Gibson 2006; Osiru et al. 2009b) was planted as a border row around the perimeter of the trial to act as a spreader of the disease. At one month after planting, the genotypes were inoculated with *A. bataticola* spores at an approximate concentration of 5.0×10^4 spores ml^{-1} (Lopes and Boiteux 1994). Inoculation was done by spaying the spores on the plant late in the evening to avoid spore germination being affected by heat and UV radiation. Data for each genotype were collected from the middle three plants of each single, five plant row.

Data collection

Plants were scored for Alternaria blight severity from 3 weeks after inoculation which continued at 3 week intervals until four data sets were collected. The disease severity rating was done using a subjective visual scale of 0–5 modified after van Bruggen (1984), where: 0 = no disease; 1 = ≤1 %; 2 = 1–10 %; 3 = 11–25 %; 4 = 26–50 %; and 5 = ≥50 % foliar infection. The Alternaria blight scores were used to calculate the area under disease progress curve (AUDPC) according to Shaner and Finney (1977).

[1] First rains start at the end of March up to end of June.

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$$\text{AUDPC} = \sum_{i=1}^{n}[(X_{i+1} + X_i)/2][t_{i+1} - t_i] \quad (1)$$

where X_i = infected leaf area (%) at the ith observationt, t_i = time (days) at the ith observation, n = total number of observations.

Data analysis

Genetic data analysis

Data for each site were first analysed separately and the error variances of the individual sites were tested for homogeneity using Hartley's F_{max} test (Hartley 1952). As the differences between the error variances were not significant, a combined analysis of the two sites was performed using the residual maximum likelihood (REML) procedure in GENSTAT 14th Edition (Payne et al. 2011) to obtain family means. Genetic information was determined on a family mean basis. To obtain combining abilities an analysis of variance (ANOVA) of the North Carolina II mating design was performed on the individual sets, using model 1 in SAS version 9.3 (SAS Institute SAS Institute Inc 2010) with parents considered as fixed effects and the sites as random effects. The ANOVA of the individual sets (Set 1 and Set 2) was performed to provide set specific information on the combining ability effects and the contribution of the components of the treatment SS to the gene action underlying trait expression (Hallauer and Miranda 1988).

The treatment sum of squares (SS) was partitioned into the variation due to females, males, and the female × male interaction. The main effects due to female and male parents are independent estimates of The relative importance of additive (GCA) effects while female × male interaction effects represent The relative importance of non-additive (SCA) effects. The GCA effects due to female parents are denoted as GCA_f and that due to male parents are denoted as GCA_m throughout this paper.

Standard errors for the GCA_f and GCA_m effects and standard errors for the SCA effects of the crosses were calculated separately as the number of females and males was not equal using the method described by Cox and Frey (1984).

GCA and SCA genetic effects in determining the performance of the progeny for each of the traits was determined by individually expressing the GCA_f SS, GCA_m SS, and the SCA SS as a percentage of the treatment (crosses) SS.

To obtain both broad and narrow sense heritability, the additive (σ^2A), non-additive (σ^2NA), and environmental (σ^2SE) variance were computed according to Nyadanu et al. (2012); using mean squares for GCA (MSg), SCA (MSs), and error (MSE) extracted from the analysis of variance table as follows: $\sigma^2 A = (MSg - MSs)/(P + 2)$; $\sigma^2NA = MSs - MSE$; $\sigma^2E = MSE$; P = number of parents. Broad sense heritability (H^2) and narrow sense heritability (h^2) estimates were computed as follows: $H^2 = (\sigma^2 A + \sigma^2NA)/(\sigma^2A + \sigma^2NA + \sigma^2E)$ and $h^2 = (\sigma^2A)/(\sigma^2A + \sigma^2NA + \sigma^2E)$.

Full-blown reciprocal effects could not be obtained in North Carolina mating II design, because one set of parents are female and the other male, and no female parents were reciprocally used as male parents and the male parents were not reciprocally used as female parents. To get an indication of the magnitude of the female parent contribution to additive gene action relative to the contribution of the male parent to the Alternaria blight expression, the GCA_f/GCA_m ratio of SS was used.

Results

North Carolina II ANOVA for individual sets of parents for Alternaria severity (AUDPC) evaluated at two sites

In the ANOVA for Set 1, the Site, GCA_f, GCA_m, SCA mean squares (MS) were highly significant ($P \leq 0.001$) for AUDPC (Table 4). The Site × GCA_f and Site × GCA_m MS interactions were highly significant ($P \leq 0.001$). The Site × SCA MS were non-significant.

In Set 2, the Site, GCA_m and SCA MS were highly significant ($P \leq 0.001$) for AUDPC. The GCA_f MS were not significant (Table 5). The Site × GCA_f and Site × SCA MS were not significant while the Site × GCA_m MS were very significant ($P \leq 0.001$).

The GCA_f and GCA_m SS for Set 1 contributed over 71.5 % of the treatment SS and SCA contributed 28.5 % (Table 4). On the other hand for Set 2, the GCA_f and GCA_m contributed only 46.0 % and SCA contributed 54.0 % (Table 5).

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Table 4 North Carolina II ANOVA mean squares and sum of squares for Set 1 parents for Alternaria severity (AUDPC) evaluated at Namulonge and Kachwekano (2011A)

Source	DF	AUDPC MS
Site	1	54,033.21***
Rep(Site)	2	9333.53***
GCA_f	3	22,084.00***
GCA_m	4	16,814.72***
SCA	12	4434.22***
Site × GCA_f	3	6717.50***
Site × GCA_m	4	3880.14***
Site × SCA	12	1135.04^{NS}
Error	38	620.70
Treatment SS		186,721.50
%SS due to GCA		71.5
%SS due to SCA		28.5

*** Significant at $P \leq 0.001$

NS not significant, *AUDPC* area under disease progress curve for Alternaria blight, *Site* Namulonge and Kachwekano, GCA_f female parent general combining ability, GCA_m. male parent general combining ability, *SCA* specific combing ability, *%SS* due to GCA and SCA expressed relative to the treatment SS, *MS* mean square, *DF* degrees of freedom

Table 5 North Carolina II ANOVA mean squares and sum of squares for Set 2 parents for Alternaria severity (AUDPC) evaluated at Namulonge and Kachwekano (2011A)

Source	DF	AUDPC MS
Site	1	81,394.74***
Rep(Site)	2	6729.30***
GCA_f	2	1051.97^{NS}
GCA_m	3	4833.42***
SCA	6	3249.06***
Site*GCA_f	2	706.52^{NS}
Site*GCA_m	3	3116.49**
Site*SCA	6	1098.43^{NS}
Error	22	529.68
Treatment SS		36,098.55
%SS due to GCA		46.0
%SS due to SCA		54.0

** Significant at $P \leq 0.01$, *** Significant at $P \leq 0.001$

NS not significant, *AUDPC* area under disease progress curve for Alternaria blight, *Site* Namulonge and Kachwekano, GCA_f female parent general combining ability, GCA_m male parent general combining ability, *SCA* specific combing ability, *%SS* due to GCA and SCA expressed relative to the treatment SS; *MS* mean square, *DF* degrees of freedom

General combining ability effects across two sites

A negative GCA effect for Alternaria blight for a parent indicates a contribution to increased disease resistance in its progeny (relative to the trial mean), which is desirable. Conversely, a positive GCA effect indicates an undesirable contribution to increased susceptibility in the progeny. In Set 1 (Table 6), the GCA effects for the female parents Semanda, Silk Omupya, and Kidodo and male parents Dimbuka and NASPOT 2 were significant for AUDPC, with only Silk Omupya and NASPOT 2 having very significant ($P < 0.01$), negative GCA effects. Bwanjule had high but non-significant, negative GCA effects. In Set 2 (Table 7), the GCA effects for AUDPC were not significant for all the female and male parents. However, Budde and Silk Luwero had the largest, negative GCA effects of −20.1 and −13.8, respectively.

Specific combining ability effects for individual sets across two sites

Since there were no common parents across the two sets, only the SCA effects across two sites for the

Table 6 Performance and general combining ability effects of Set 1 parents for Alternaria severity (AUDPC) across two sites

Parent	AUDPC	
	Mean	GCA
Females		
Semanda	192.5	28.11*
Silk Omupya	129.9	−34.51**
Kidodo	193.3	28.80*
Bwanjule	142.0	−22.40
SE	11.3	12.72
Males		
Dimbuka	195.7	31.24**
NASPOT 2	112.5	−51.97**
NASPOT 1	181.6	17.19
NASPOT 4	155.6	−8.84
Araka Red	176.8	12.37
SE	12.7	11.92

* Significant at $P \leq 0.05$, ** Significant at $P \leq 0.01$

AUDPC area under disease progress curve for Alternaria blight severity, *GCA* general combining ability across two sites, *SE* standard error, *GCA* effects across two sites were considered

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individual set analyses are considered. Among the 20 full-sib families in Set 1, the AUDPC value for Alternaria blight ranged from 96.9 in family Bwanjule × NASPOT 2 to 269.7 in family Kidodo × Dimbuka (Table 8). However, both families had positive SCA effects with only that of Kidodo × Dimbuka very significant ($P < 0.01$). Of the 20 families in this set, only seven had significant ($P < 0.05$) SCA effects of which three had desirable negative effects and four had undesirable positive effects. Family Kidodo × NASPOT 1 had the largest negative SCA effects (−67.12) and is therefore a more desirable family. Bwanjule × Dimbuka and Silk Omupya × Araka Red may be regarded as good families for Alternaria blight resistance with SCA effects of −30.31 and −27.16, respectively. The *per se* performance (AUDPC means) of these families was good with Kidodo × NASPOT 1, Bwanjule × Dimbuka, Silk Omupya × Araka Red having mean AUDPC values of 143.3, 142.9 and 115.2 which were low compared to the mean. Families Silk Omupya × NASPOT 2, Kidodo × NASPOT 4 and Bwanjule × NASPOT 1 had significant ($P < 0.05$), positive SCA effects, and Kidodo × Dimbuka had a very significant ($P < 0.01$), positive SCA effect and are therefore not desirable families for breeding for resistance to Alternaria blight.

Table 7 Performance and general combining ability effects of Set 2 parents for Alternaria severity (AUDPC) across two sites

Parents	AUDPC	
	Mean	GCA
Females		
Tanzania	194.6	7.59
Mbale	178.4	−8.65
Shock	185.9	1.06
SE	12.7	13.41
Males		
New Kawogo	210.8	26.74
Silk Luwero	173.2	−13.84
Magabali	194.2	7.19
Budde	167.0	−20.10
SE	14.6	14.90

AUDPC area under disease progress curve for Alternaria blight, *GCA* general combining ability, *SE* standard error, *GCA* effects across two sites were considered

Table 8 Performance and specific combining ability effects of Set 1 families for Alternaria severity (AUDPC) across two sites

Family	AUDPC	
	Mean	SCA
Semanda × Dimbuka	219.5	−4.27
Semanda × NASPOT 2	124.3	−16.28
Semanda × NASPOT 1	220.0	10.25
Semanda × NASPOT 4	173.8	−9.97
Semanda × Araka Red	225.2	20.27
Silk Omupya × Dimbuka	150.6	−10.63
Silk Omupya × NASPOT 2	106.7	28.69*
Silk Omupya × NASPOT 1	169.9	22.83
Silk Omupya × NASPOT 4	107.4	−13.73
Silk Omupya × Araka Red	115.2	−27.16*
Kidodo × Dimbuka	269.7	45.21**
Kidodo × NASPOT 2	112.1	−19.20
Kidodo × NASPOT 1	143.3	−67.12**
Kidodo × NASPOT 4	210.6	26.26*
Kidodo × Araka Red	220.5	14.85
Bwanjule × Dimbuka	142.9	−30.31*
Bwanjule × NASPOT 2	96.9	6.80
Bwanjule × NASPOT 1	193.3	34.04*
Bwanjule × NASPOT 4	130.7	−2.56
Bwanjule × Araka Red	146.5	−7.97
Mean	164.0	
SE	25.3	12.98

* Significant at $P \leq 0.05$, ** Significant at $P \leq 0.01$

AUDPC area under disease progress curve for Alternaria blight severity, *SCA* specific combining ability across two sites, *SE* standard error

Semanda was inconsistent as a parent, in some cases producing resistant families such as Semanda × NASPOT 2 (AUDPC of 124.3) with a negative SCA effect of −16.28 and in other cases producing very susceptible families such as Semanda × Dimbuka (219.5), Semanda × NASPOT 1 (220) and Semanda × Araka Red (225.2) with SCA effects of −4.27, 10.25 and −20.27, respectively. Kidodo was also inconsistent in that it produced resistant families Kidodo × NASPOT 2 (112.1) and Kidodo × NASPOT 1 (143.3) with SCA effects of −19.2 and −67.12, respectively, and susceptible families Kidodo × Dimbuka (269.7), Kidodo × NASPOT 4 (210.6), and Kidodo × Araka Red (220.5) with SCA effects of 45.21, 26.26 and 14.85, respectively. Kidodo × NASPOT 1 which had the highest

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significant ($P < 0.01$), negative SCA effect of −67.12 and AUDPC of 143.3 was therefore one of the most desirable families along with Kidodo × NASPOT 2 with a SCA effect of −19.2 and AUDPC of 112.1.

In Set 2, the SCA effects for AUDPC of families Mbale × Silk Luwero and Shock × Magabali were very significant ($P < 0.001$) but with positive effects thus these families were undesirable (Table 9). The SCA effect of Mbale × Magabali was significant ($P < 0.05$) and negative and therefore a desirable family. Shock × Silk Luwero had the lowest AUDPC value (142.4) with high but non-significant, negative SCA effects. All crosses with New Kawogo produced families with high AUDPC values (above 200) but, interestingly, only Mbale × New Kawogo had positive SCA effects. Shock performed inconsistently in crosses producing families with the highest AUDPC (231.5) for Shock × Magabali and also the lowest AUDPC value for Shock × Silk Luwero (142.4) and SCA effects of 36.15 and −22.14, respectively.

Broad and narrow sense heritability

In Set 1, the broad sense heritability (H^2) estimates for AUDPC were slightly higher for female parents (92 %) than for male parents (90 %). Similarly, the narrow sense heritability (h^2) estimates were higher for the female parents (39 %) than for the male parents (29 %) (Table 10). In Set 2, the broad sense heritability estimates were higher for the male parents (85 %) than for the female parents (81 %). Narrow sense heritability estimates for both male and female parents was low at 15 and 8 % respectively. The ratio of GCA_f/GCA_m was higher for Set 1 parents (1.3) than for Set 2 (0.22).

Table 9 Performance and specific combining ability effects of Set 2 families for Alternaria severity (AUDPC) across two sites

Family	AUDPC	
	Mean	SCA
Tanzania × New Kawogo	216.7	−4.77
Tanzania × Silk Luwero	180.2	−0.62
Tanzania × Magabali	193.0	−8.59
Tanzania × Budde	188.5	13.97
Mbale × New Kawogo	214.6	9.48
Mbale × Silk Luwero	197.0	32.46**
Mbale × Magabali	158.1	−27.55*
Mbale x Budde	143.9	−14.38
Shock x New Kawogo	201.2	−4.71
Shock x Silk Luwero	142.4	−22.14
Shock x Magabali	231.5	36.15**
Shock x Budde	168.4	0.41
Mean	186.3	
SE	25.3	11.85

* Significant at $P \leq 0.05$, ** Significant at $P \leq 0.01$

AUDPC area under disease progress curve for Alternaria blight severity *SCA* specific combining ability across two sites, *SE* standard error

Discussion

This study was carried out to understand the gene action controlling the inheritance of resistance to Alternaria blight. This information would assist future breeding programmes for resistance to the disease. Just as importantly, the F_1 progeny may be used as sources of new genetic variation in breeding for resistance to Alternaria blight and improved agronomic performance.

North Carolina II ANOVA for Alternaria severity (AUDPC) evaluated at two sites

The significance of the Site MS ($P < 0.001$) for AUDPC (Tables 4, 5) indicated that there were significant differences between the site means for Alternaria severity. That is, Alternaria blight was more severe at one of the sites. This is consistent with the findings of Osiru et al. (2007a, b) and Yada et al. (2011) who reported Alternaria blight to be more severe at Kachwekano than at Namulonge. This

Table 10 Heritability estimates and ratio of female and male combining abilities for Set 1 and Set 2 parents across two sites

Parameter	Set 1	Set 2
H_f^2	0.92	0.81
h_f^2	0.39	0.15
H_m^2	0.90	0.85
h_m^2	0.29	0.08
GCA_f/GCA_m	1.30	0.22

H_f^2 female parent broad sense heritability, h_f^2 female parent narrow sense heritability, H_m ^{2}male parent broad sense heritability, h_m^2 male parent narrow sense heritability, GCA_f female parent general combining ability, GCA_m male parent general combining ability

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implies that Alternaria blight severity greatly depends on the environment thus the need to evaluate a particular genotype in the target environment before recommending it to the farmers.

The significance ($P < 0.05$) of the GCA_f MS in Set 1 for AUDPC indicated that additive genetic variance contributed by the female parents is very important in controlling the expression of resistance to Alternaria blight (Table 4). Similarly, significance ($P < 0.05$) of the GCA_m MS for AUDPC indicated that the male parents in Set 1 contributed significant additive genetic effects to the expression of this trait. Significance ($P < 0.05$) of the SCA MS for AUDPC indicated that the non-additive gene action is important in the expression of this trait for the parents in Set 1 (Table 4). Significance ($P < 0.05$) of the Site × GCA_f MS for AUDPC indicated that the additive genetic effects for the female parents in Set 1 was not consistent across the sites for this trait. The Site × GCA_m MS was very significant ($P < 0.01$) for AUDPC indicating that the additive genetic effects of male parents in Set 1 were not consistent across the sites. The Site × SCA MS was not significant for AUDPC indicating that the effect of non-additive gene action for this trait did not vary with change in site.

Significance ($P < 0.05$) of the GCA_m for AUDPC indicated that additive genetic variance due to male parents in Set 2 was very important in the expression of this trait (Table 5). The SCA MS was highly significant ($P < 0.001$) for AUDPC indicating that the non-additive gene action was important in the expression of this trait for the parents in Set 2. Similarly, significance of Site × GCA_m for AUDPC ($P \leq 0.01$) indicated that the additive gene action due to male parents in Set 2 for AUDPC was not consistent over sites.

Mean performance, and general and specific combining ability effects for Alternaria severity

Area under disease progress curve

The parental AUDPC values for Alternaria blight ranged from 112.5 to 195.7 in Set 1 (Table 6) and 167.0 to 210.8 in Set 2 (Table 7). This wide range in AUDPC values indicates that selection of genotypes for high resistance to Alternaria blight from within the available germplasm is possible. The significant ($P < 0.05$), positive GCA effects for AUDPC in three of the Set 1 parents Semanda, Kidodo and Dimbuka of 28.11, 28.80 and 31.24, respectively implies that they are not good general combiners for Alternaria blight resistance since they contribute towards higher susceptibility. Conversely, Silk Omupya, NASPOT 2 with very significant ($P < 0.01$) and large negative GCA effects of −34.51 and −51.97, and Bwanjule with non-significant but large GCA effects of −22.4 are good general combiners when breeding for resistance to the disease.

Set 1 families exhibited considerable variation in terms of reaction to Alternaria blight. The AUDPC values ranged from 96.9 for the most resistant family (Bwanjul × NASPOT 2) to 269.7 for the most susceptible family (Kidodo × Dimbuka) (Table 8). Family Bwanjule × NASPOT 2 had a non-significant, positive SCA effect of 6.8 for AUDPC, but parent Bwanjule had a large negative GCA effect of −22.40 and parent NASPOT 2 also had the highest significant ($P < 0.01$), negative GCA effect of −51.97. Similarly, parents Silk Omupya and NASPOT 2, with significant ($P < 0.05$), negative GCA effects, produced progeny with a low AUDPC (106.7) but with significant ($P < 0.05$), positive SCA effects. The positive SCA effects of these crosses were unexpected since both parents had negative GCA effects. A similar scenario was reported by Mwanga et al. (2002) for SPVD where two very good combiners for SPVD produced susceptible progeny with undesirable SCA effects. The difference in the current study, however, is that despite the positive SCA effects, the progeny of these crosses had high levels of resistance to the disease.

The susceptible family Kidod × NASPOT 4 with an AUDPC of 210.6 and a significant ($P < 0.05$), positive SCA effect of 26.26 (Table 8), resulted from a cross between a female parent with a significant ($P < 0.05$), positive GCA effect of 28.80 and a male parent with a non-significant ($P > 0.05$), negative GCA effect of −8.84 (Table 6). Conversely, family Bwanjule × Dimbuka with a significant ($P < 0.05$), negative SCA effect of −30.31 (Table 8) was the result of a cross between a female parent with a non-significant, negative GCA effect of −22.40 and a male parent with a very significant ($P < 0.01$), positive GCA effect of 31.24 (Table 6). The implication being that parents with positive GCA effects may be of value in the development of resistant Alternaria blight genotypes and conversely, some parents with negative GCA effects may not be very useful in the

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development of Alternaria blight resistant genotypes. Therefore parents should not be eliminated from the crossing program solely on the basis of GCA alone, but after a thorough evaluation of the *per se* performance of their progeny.

Female parents Silk Omupya and Bwanjule (Table 6), across all male parents, produced families with the lowest AUDPC values (Table 8) and were therefore the best combiners for resistance to Alternaria blight. Similarly, male parent NASPOT 2 (Table 6) produced the most resistant families across all female parents (Table 8). All these parents had significant ($P < 0.05$), negative GCA effects for the disease and thus were the best at transmitting resistance to Alternaria blight to their progeny. These parents should be used as sources of resistance to the disease.

The GCA and SCA MS were both significant for AUDPC implying that both additive and non-additive gene actions were important for this trait. The GCA SS contributed 71.5 % in Set 1 and 46.0 % in Set 2 of the treatment SS for this trait indicating that additive gene action and non-additive gene action were both important and the predominance of either depends on the parents used. However, results reported by Simon and Strandberg (1998) for *A. dauci* in carrots (*Daucus carota* L.) indicated that additive gene action was more predominant. Maiero et al. (1990) also reported resistance to early blight (*A. solani*) in tomato (*Solanum lycopersicum* L.) to be predominantly controlled by additive gene action. Furthermore, Christ and Haynes (2001) reported both additive and non-additive gene action to be important in conditioning the resistance to early blight (*A. solani*) of diploid potato with the additive component predominant.

Broad and narrow sense heritability

Broad sense heritability estimates for AUDPC were high in both sets (>80 %) implying that rapid genetic gains should be expected through use of mass selection based on the knowledge of the phenotype of the parent. On the other hand, narrow sense heritability estimates were low in both sets (<40 %) implying that to a great extent, the environment plays a major role in the expression of Alternaria blight (Table 10).

The ratio of GCA_f/GCA_m was higher for Set 1 parents (1.3) than for Set 2 (0.22). This implies that female parents in Set 1 contributed more to the additive gene action than the male parents. In Set 2 the ratio was lower than 1 implying the male parents contributed more to the additive gene action than the female parents (Table 10).

Conclusion

It was apparent that both additive and non-additive gene actions were important for the phenotypic expression of the traits under consideration, although additive gene action generally predominated. With respect to Alternaria blight, the implication of both additive and non-additive gene action contributing to the expression of resistance to the disease is that improved cultivars with good resistance levels to the disease can be obtained by careful selection of progeny expressing both gene actions. Both additive and non-additive gene action will be conserved in the best performing progeny through vegetative propagation. Predominance of additive gene action for any trait generally means that the performance of the parents of the crosses can be used to predict performance of the progeny. Conversely, predominance of non-additive gene action means progeny performance may not be accurately predicted based on parental performance. There were also instances in this study that proved exceptions to the rule where resistant progeny with desirable SCA effects were obtained from parents whose GCA effects were not desirable. Therefore, before discarding any parents it is important to evaluate the *per se* performance of their progeny and to not depend entirely on the magnitude and significance of GCA effects alone.

Female parents Silk Omupya and Bwanjule produced the most Alternaria resistant families across all male parents while male parent NASPOT 2 produced the most Alternaria resistant families across all female parents (Table 8).

Since NASPOT 2 is susceptible to Alternaria (Mwanga et al. 2003), it turned out to have lower AUDPC values than most of the other parents, and an attempt should be made to cross resistant × resistant parents to determine the type of progeny that will be produced.

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