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Inheritance of multiple resistance to fungal diseases in tropical maize germplasm

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Abstract. Maize (*Zea mays* L.) ear rots caused by *Aspergillus flavus, Fusarium graminearum* and *Stenocarpella maydis* affect grain quality and are associated with mycotoxins that pose precarious food and feed safety issues. The objective of this study was to determine the inheritance of multiple resistance to major fungal pathogens (*Aspergillus flavus, Fusarium graminearum* and *Stenocarpella maydis*) in maize in the tropical Africa. A total of 30 tropical inbred lines with varying resistance to *A. flavus* and both *F. graminearum* and *S. maydis,* were mated in a North Carolina II Design and the progeny consisting of single crosses, test crosses and their parents evaluated for single infection and yield performance. Resistance to the three ear rot pathogens and grain yield was found to be inherited independently. Therefore the three fungal infections had low or negligible effect on grain yield, though adversely reduced the grain quality. Multiple resistance to the three ear rot pathogens was majorly additive in nature. Some non-additive gene effects like dominance and epistasis could be contributing to multiple ear rot resistance. Maize hybrid vigour was found to enhance ear rot resistance.

Key words: *Aspergillus flavus,* Ear rots, *Fusarium graminearum*

Introduction

The impact of climate change has been identified as an emerging threat to food security and safety; and the increased incidence of mycotoxin contamination in maize over the last two decades is considered a potential emerging hazard (Lanubile *et al*., 2017). Maize ear rots caused by three major fungi, *Aspergillus flavus, Fusarium graminearum* and *Stenocarpella maydis,* are associated with mycotoxins and affect grain yield, and quality especially in the tropics. The existing control measures for the ear rots are temporary and not effective; yet the sophisticated maize storage mechanisms to control damage due to the ear rots are costly and inaccessible to resource poor farmers, especially in Africa (Afolabi *et al*., 2007).

These fungi produce mycotoxins that are consumed inadvertently in the tropics through ingesting maize possessing invisible ear rot infection symptoms (Alunga *et al*., 2016). Therefore, efforts for addressing challenges associated with maize ear rots and their related mycotoxins in the tropics are a priority.

Alunga *et al.* (2016) emphasises a need for farmers to grow resistant varieties, as well as application of joint efforts in sensitising the public about preserving grain quality through proper post-harvest handling. They further recommended the need for researchers to prioritise breeding for ear rot

resistance. Knowledge of inheritance patterns of ear rot resistance is necessary to ease their breeding efforts.

Tembo *et al*. (2022) cited that resistance to *S. maydis, S. macrospora* and *F. graminearum* was reported to be closely related. However, Mesterhazy (2012) described the possibility of multiple resistances towards more than one fungal pathogen. Earlier studies have reported that resistance to *S. maydis* is controlled by additive gene action; while resistance to *F. graminearum* comprises of both additive and non-additive gene action (Rossouw *et al.*, 2013). It is anticipated that understanding the inheritance pattern will help breeders efficiently transfer the genes controlling multiple resistance to ear rot resistance. The objective of this study was to determine the inheritance of multiple resistance to major fungal pathogens (*Aspergillus flavus, Fusarium graminearum* and *Stenocarpella maydis*) in maize in tropical Africa.

Materials and Methods

Experimental sites

The study was carried out in three sites in Uganda; namely Bulindi (1°25 N, 31°21 E; altitude 1140 masl) ; Namulonge (0°32 N, 32°35 E; altitude 1150 masl) ; and Ngetta (2°31 N, 32°93 E, altitude 1128 masl). All the three locations experience a bimodal rainfall pattern, annual means of 1,270, 1,240 and 1,361mm, respectively. The annual mean temperatures are 23.5, 22.8 and 23.4 ^{o}C, respectively

Plant materials

The germplasm materials included tropical inbred lines, selected based on their resistance to the fungal ear rot pathogens. Hybrid F1 crosses consisting of test crosses and single crosses were evaluated for yield, ear rot resistance and agronomic performance. A total of 30 inbred lines, out of which 23 resistant to *Aspergillus flavus* (male lines) were evaluated. Three inbred lines; WL118-10, CML506 and WL429-35, had dual resistance to *F. graminearum* and *S. maydis* (female lines). Line NML 89 was used as a susceptible check for both *F. graminearum* and *S. maydis*. Inbred seed multiplication and pollination were done concurrently, at a seed nursery established at Namulonge in the first rainy season.

Experimental design

Crosses were made using the North Carolina Design (NCD 2) to develop both the test and the single crosses. Three inbred lines (CML 216, CML 395 and CML 442) were included in the study as yield testers. These were crossed with the male lines, using the NCD 2, to produce test crosses. Single crosses were also developed by crossing the three dual resistant inbreds; WL118-10, CML506 and WL429-35 with the *A. flavus* resistant line (male lines) using the NCD 2 crossing design.

The experiment was laid out following an alpha lattice design, with two replications across the three trial sites. The plants were established in two row plots at a spacing of 75 cm X 30 cm, each row measured 5 meters long and had 17 plants per row; with one seed planted per hill. Both the single and test crosses were made during the first cropping season of 2013 (2013A) at NaCRRI to get F1s. In the second season (2013B), the parental inbred lines, F1s including the single and test crosses, were thereafter evaluated in the three test locations: Namulonge, Bulindi and Ngetta using an alpha lattice experimental design. The evaluation of the inbred parents, test crosses and single crosses was done in three separate setups established adjacent to each other in each of the three test sites.

Preparation of pathogen culture and inoculation

This was done by isolating the three pathogens with *F. graminearum, S. maydis* and *A. flavus* isolated from infected maize cobs. Inoculum was prepared following the modified procedure of Chambers (1998). Infected maize kernels were first sterilised in 10% commercial bleach (JIK brand) 0.39% sodium hypochlorite (NaClO) (Reckitt Benkiser East Africa Limited, Nairobi, Kenya) for three minutes and then rinsed thrice in distilled water. The seeds were subsequently blotted dry on sterilised filter paper and then 2 - 3 seeds plated on 3% potato dextrose (Becton Dickinson, Sparks, MD, USA) agar plates and incubated at 28 - 30 °C.

The fungal growths on plates were sub-cultured after four days and were ready for transfer to toothpicks after 5 - 7 days. Toothpicks were initially sterilised by boiling in water three times to wash out tannins and other fungal growth-inhibiting compounds from the wood, before air-drying. They were then placed upright in bottles measuring 6 cm in diameter and 11 cm height, containing 100 – 150 ml of potato dextrose broth, prepared by infusion of 200 g freshly unskinned potato and 20g of dextrose in 1 litre of distilled water, to coat the toothpicks. They were autoclaved for 30 minutes and left to cool to room temperature. Each sterile bottle contained 600 - 650 toothpicks.

Fungal plugs from pure cultures of either *F. graminearum* or *S. maydi*s, were placed in each bottle and allowed to colonise tooth picks for 10 days; following procedure described by (Chambers, 1988). After the toothpicks were fully colonised, they were air-dried before being using to inoculate the test genotypes. Inoculation was performed by piercing developing ears through the middle, using colonised toothpicks 20 days after mid-silking (R3) stage (Chambers, 1988). Care was taken when piercing, to only prick the developing kernels and not the underlying cob thus very deep piercing was avoided, ears were inoculated by single pathogens (Fig. 1). Five plants per row, selected from the opposite sides of the row, were inoculated by either *F. graminearum* or *S. maydis*. At maturity, the cobs in the inoculated rows were harvested separately, dehusked and assessed for fungal infection.

Figure 1. Singly tooth pick inoculated cobs (*S. maydi*s): intact (left) and unhusked (right).

Data collection

Severity rating were done using a scale of 1 - 5 for *S. maydis* where 1 = 0-25%; 2 = 26- 50%, 3 = 51 - 75%, 4 = 76 - 99% and 5 = 100% (completely rotten) (Tembo *et al*., 2013). For *F. graminearum* the scale was 1 = 1 – 3%, 2 = 3 – 10%, 3 = 11 – 25%, 4 = 26 – 50%, 5 = 51 – 75%, and 6 = 76 – 100% (Simpasa et al., 2018). For *A. flavus* the scale of 1 - 5 was used with; 1 = 0-25%; 2 = 26-50%, 3 = 51 - 75%, 4 = 76 - 99% and 5 = 100% (completely rotten) (Alunga *et al*., 2016).

Average disease scores were computed for each entry and for each pathogen. Cobs in the non-inoculated rows were also harvested separately, bulked, counted for yield data collection. The cobs were then hand shelled and bulked into paper bags where they were carefully dried to avoid direct heat on the kernels to facilitate uniform drying prior to evaluation for *A. flavus* resistance in the laboratory.

Laboratory evaluation

Dry kernels were soaked in distilled water for 1 minute to enable them gain at least 30% moisture content. This was meant to mimic the field moisture conditions of the kernels on the cob. The kernels were then removed from water, drained and placed singly in bottle caps and put on disposable aluminum foil plates lined with wet cotton wool. A total of 15 kernels were put on each plate.

Two plates were prepared for each sample, implying that a total of 30 kernels per sample were assayed. For each genotype, two replications (60 kernels) were used. Each of the kernels was then inoculated by applying a 20 µl conidial suspension of *A. flavus* inoculum (with concentration of 1.0 ×106 conidia/ml) to its surface using a micro-pipette. To enable homogeneous incubating conditions, plates were stacked and incubated at 31°C and a relative humidity of 95 - 100% for 7 days. This was made possible by closing the plate lids tightly. The wet cotton wool maintained humid incubation conditions.

On the 7th day of incubation, the plate lids were removed and the kernel infection rates recorded by counting the infected kernels; noting the number of those severely infected. A kernel was considered severely infected if fungal growth covered over 50% of its surface. Three measurements of kernel infection rate were evaluated; and the incidence of infection was expressed as Percent-of-Kernels-Infected (PKI), Incidence of Severely-Infected Kernels (ISIK), and Percent-Severely-Infected Kernels (PSIK). A genotype infection score was also computed and this was obtained by averaging scores recorded for the two plates representing each entry. The above measurements were calculated as follows:

$$PKI = \frac{\text{Number of infected kernels}}{\text{Total number of kernels incubated}} \times 100\ \% \quad \text{..... (i)}$$

$$ISIK = \frac{\text{Number of severely infected kernels}}{\text{Total number of incubated kernels}} \times 100\ \% \quad \text{..... (ii)}$$

$$PSIK = \frac{\text{Number of severely infected kernels}}{\text{Total number of infected kernels}} \times 100\% \quad \text{..... (iii)}$$

Indirect ear rot selection

Data were also recorded for Natural ear rot infection (ER), Husk cover tightness (HC), and grain texture (GT) as described by Betr'an *et al*. (2002); where 1 = flint; round crown kernel with vitreous appearance, to 5 = dent: kernel dented and having a floury endosperm. Husk cover was determined

using 1 - 5 scale described by Tembo *et al.* (2016); where 1 = good, tight husk extending beyond the tip of the cob to 5 = poor, loose short husk with exposed cob tip.

Estimation of heritability

The genotype responses were subjected to analysis of variance (ANOVA) and restricted maximum likelihood (REML) approaches. The relationship between the three disease scores was evaluated by performing a correlation analysis of the disease score means and the associated traits of interest. The relative contribution of General Combing Ability (GCA) and Specific Combining Ability (SCA) was estimated using Baker's ratio (BR) (Baker 1978), computed as:

$$BR = \frac{(\sigma^2 GCA1 + \sigma^2 GCA2)}{(\sigma^2 GCA1 + \sigma^2 GCA2 + \sigma^2 SCA)} \quad \text{(iv)}$$

Where: GCA_1 represents the male parent; and GCA_2 represents the female parent; σ^2GCA and σ^2SCA are the variance components of GCA and SCA, respectively.

The effects of general combining ability (GCA) and specific combining ability (SCA) for the crosses hybridised in a North Carolina 2 (NC II) design, were determined. An inbred line's General combining ability (GCA) was determined through progeny testing by crossing it with other inbred lines using North Carolina 2 (NC II) design and comparing their resultant single cross hybrids' overall performance for the the traits of interest. Specific combing ability describes those cases in which certain hybrid combinations do relatively better or worse than what would be expected based on the average performance of the parent (inbred lines). (All the GCA and SCA data analyses were performed using GenStat computer package,(VSN International (2022). Genstat for Windows 22th Edition VSN International, Hemel Hempstead , UK). Two tailed t-tests were used to determine the level of significance of GCA effects.

Since the parents used in the experiment were fixed, variance ratios were used to obtain the broad-sense coefficient of genetic determination (BSCGD) and narrow-sense coefficient of genetic determination (NSCGD). Estimates of broad-sense coefficient of genetic determination (BSCGD) were based on the formula suggested by Dabholkar (1992); described below:

$$H^2 = \frac{\sigma^2 GCAi + \sigma^2 GCAj + \sigma^2 SCAij)}{(\sigma^2 GCAi + \sigma^2 GCAj + \sigma^2 SCAij + \sigma^2 e)} \quad \text{(v)}$$

Where: H^2 = Estimated broad sense heritability; σ^2GCA = Variance due to additive effects; σ^2SCA = Variance due to non-additive effects; and σ^2e = Environmental error variance component.

GCA effects were calculated and assessed for significant difference from zero, using t-test, *viz*:

$$t_{gca} = \frac{GCA - 0}{SEMgca} \quad \text{(vi)}$$

Where: GCA = General combining ability value; and SEM = Standard Error of Means.

Narrow sense heritability of the various traits of interest in the study, was derived from estimates of narrow sense coefficient of genetic determination of a derivative from the expression of the ratio of

additive genetic variance to the phenotypic variance, from a formular suggested by Dabholkar (20—.) (as cited by Simpasa *et al*., 2018):

$$h^2 = = \frac{\sigma^2 GCAi + \sigma^2 GCAj}{(\sigma^2 GCAi + \sigma^2 GCAj + \sigma^2 SCAij + \sigma^2 e)} \quad \text{..... (vii)}$$

Where: h^2= Estimated narrow sense heritability; ó^{2}GCA= Variance due to additive effects; ó^{2}SCA=Variance due to dominance effects; and ó^2_e= Environmental error variance component.

Results

Disease development in parental inbred lines

The highest level of susceptibility to *S. maydis,* among the inbred lines, was recorded at Bulindi where the average disease severity score was 5.0 (Table 1). The highest disease scores for *A. flavus* infection were recorded for maize grown at NaCRRI (2.6). Notably *S. maydis* had the highest severity among the three ear rot pathogens, with *F. graminearum* having the least severity.

Table 1. Fungal disease severity scores across genotypes for inbred lines evaluated in this study

Location	Asp Lab (1-5 scale)	PKI(%)	PSIK(%)	ISIK(%)	*F. graminearum* (1-6 scale)	*S. maydis* (1-5 scale)
NaCRRI	2.6	90.4	29.5	28.9	1.8	4.9
Bulindi	2.3	6.7	19.5	18.7	1.7	5
Ngetta	2	60.5	23.5	12.8	1.1	4.3
Means	2.6	33.6	24.2	20.1	1.5	4.7

Severity ratings scores, Asp Lab (*A. flavus*): 1 = 0 - 25 %; 2 = 26- 50 %, 3 = 5 - 75 %, 4 = 76 - 99 % and 5 = 100, *F. graminearum*: 1 = 1 – 3%, 2 = 3 – 10%, 3 = 11 – 25%, 4 = 26 – 50%, 5 = 51 – 75%, and 6 = 76 – 100% , *S. maydis*: 1 = 0 - 25 %; 2 = 2 6 - 50 %, 3 = 5 - 75 %, 4 = 76 - 99 % and 5 = 100.

Asp lab = *A. flavus* kernel sample colonisation score (laboratory evaluation)
PKI = Percent-Kernel-infection calculated as (number of infected kernels x 100/total number of incubated kernels)
PSIK = Percent-severely-infected kernels, calculated as (number of severely infected kernels x 100/ number of infected kernels)
ISIK = Incidence of severely-infected Kernels calculated as (number of severely infected kernels x 100/ total number of incubated kernels)

Specific combining ability (SCA) for ear rot fungal disease infection, maize husk cover, grain texture and yield among single crosses in single and multi-location evaluation

Table 2 shows SCA effects of a number of crosses and their reaction to A. *flavus*, *F. graminearum, S. maydis* infections, husk cover and grain texture and yield. SCA analysis revealed that many crosses showed significant SCAs towards resistance to individual and multiple ear rot diseases whereas some showed large SCA effects towards susceptibility. Some of the crosses showing multiple ear rot resistance also combined well for good husk cover, grain texture and high yields. Single cross hybrids that

Table 2. Specific combining ability (SCA) effects of selected maize single crosses (multi location) for *S. maydis, F. graminearum*, husk cover and grain texture

Single crosses (Hybrids)	Kernel infection rate (Laboratory)				Field traits				
	Asp Lab	PKI	PSIK	ISIK	Fus	Steno	HC	Tex	Yield
CML 384/CML 506	-0.17	-15.37	0.72	-1.47	0.33	0.05	-0.34	-0.2	-0.22
CML 506/D264-14	-0.23	-9.63	-1.19	-13.2	0.05	-0.02	0.03	-0.03	0.44
CML 506/D264-15	-0.05	-5.31	7.63	-2.24	-0.12	-0.06	0.54	0.22	0.01
CML 506/D264-2	-0.02	3.58	1.65	-0.04	-0.1	0.04	0	-0.03	-0.34
CML 506/D264-3	-0.7	-4.31	2.39	-4.53	-0.29	-0.08	-0.09	0.16	0.09
CML 506/D264-5	0.07	-3.32	-0.32	6.93	0.08	-0.09	0.03	0.08	0.81
CML 506/D264-8	-0.12	-6.59	2.07	-2.31	-0.22	-0.15	0.25	-0.01	-0.77
NML 141/WL 118-10	0.24	7.88	4.57	2.89	0.13	0.23	-0.2	0.12	0.64
CML 506/TZAR 102	0.1	-0.18	7.61	2.86	-0.12	0.05	-0.08	-0.21	0.34
CML 506/TZAR 104	-0.06	0.43	0.66	-3.01	0.05	-0.17	-0.25	-0.42	-0.26
CML 506/TZAR 106	0.17	1.4	0.86	5.16	0.15	0.07	-0.05	-0.06	0.85*
CML 506/WL118-15	-0.01	0.22	-5.04	-6.38	0.05	0.05	-0.03	-0.12	0.54
D264-13/WL 429-35	0.13	2.41	12.76	8.03	0.03	-0.03	0.18	-0.11	0.95
D264-14/CML506	-0.23	-9.63	-1.19	-13.2	0.05	-0.02	0.03	-0.03	-0.95*
D264-14/WL 118-10	0	7.62	22.64***	8.19	-0.05	-0.03	-0.04	0.02	0.27
D264-15/WL 118-10	0.03	2.11	8.39	1.55	0.11	0	-0.08	-0.08	0
D264-2/CML 506	-0.02	3.58	1.65	-0.04	-0.1	0.04	0	-0.03	-0.14
D264-2/WL 118-10	0.06	-0.61	8.73	3.41	0.06	0.13	0.09	0.1	-0.52
D264-6/CML 506	0	0	5.23	0	0	0	0	0	-0.91*
D264-7/CML 506	0.11	1.77	2.85	0.22	-0.09	0.06	0.26	0.08	0.25
D264-7/WL 118-10	-0.05	-3.12	5.08	2.09	-0.02	-0.05	-0.08	-0.01	-0.77
D264-9/CML 506	-0.05	5.35	0.81	0.35	0.16	0.06	-0.51	0.19	0.01
D264-9/WL 118-10	0.06	6.59	3.35	0.39	-0.15	-0.03	0.08	-0.07	0.46
D264-9/WL 429-35	-0.04	-9.26	4.48	-0.56	0.08	0	0.18	-0.03	-0.26
NML 141/WL 118-10	-0.11	-7.08	1.00	-2.15	-0.02	-0.16	0.07	-0.17	-0.29
NML 141/WL 429-35	-0.01	3.14	5.58	0.70	-0.05	0.04	0.01	0.11	0.54
TZAR 106/CML 506	0.17	1.40	0.86	5.16	0.15	0.07	-0.05	-0.06	-0.95*
TZAR 106/WL118-10	-0.16	-11.67	0.57	-5.71	-0.02	-0.08	-0.12	-0.01	0.91
WL 118-10/D264-14	0	7.62	22.64***	8.19	-0.05	-0.03	-0.04	0.02	0.12
WL 118-10/D264-8	0.12	6.59	10.32	2.31	0.22	0.15	-0.25	0.01	0.34
WL 429-35/D264-14	0.47	4.02	27.97**	10.05	0.01	0.10	0.01	0.02	0.24
WL 429-35/TZAR 101	0	0	1.74	0	-0.02	-0.04	-0.12	-0.02	0.72
WL 429-35/TZAR 106	-0.17	8.88	0.85	-4.60	-0.28	-0.05	0.35	0.13	0.90

Asp lab = *A. flavus* kernel sample colonisation score (laboratory evaluation), PKI = Percent-Kernel-infection, PSIK = Percent-severely-infected kernels, ISIK = Incidence of severely-infected Kernels. Fus = *Fusarium graminearum* ear rot, Steno = *Stenocarpella maydis* ear rot, HC = Husk cover tightness, GT = Grain texture. *** = Significant at $p \leq 0.001$, ** = Significant at $p \leq 0.01$,* = Significant at $p \leq 0.05$

possessed multiple resistance to the three ear rot diseases as revealed by their commonly low and negative SCA effects included; CML 506/D264-15, CML 506/D264-3 and CML 506/D264-8; these also had positive SCA effects for yield (Table 2). Crosses; CML506/D264-8 and NML 141/WL 118-10 possessed multiple ear rot resistance but yielded poorly (Table 2). However some single cross hybrids generally possessed high and positive SCA effects for all the three ear rot diseases despite having significant high SCA effects for yield. These hybrids were generally susceptible to the three ear rot diseases, and included; NML 141/WL 118-10, CML 506/TZAR 106 and D264-15/WL 118-10 (Table 2).

Correlation of disease scores against yield of the parental inbred lines

A positive but non-significant correlation existed between scores of kernel sample colonisation by *A. flavus* (Asp lab) and scores of *F. graminearum* (r = 0.11), and *S. maydis* (Table 3). However, a significant ($p \leq 0.05$) moderately strong correlation (r = 0.53) among the genotypes was observed between PKI Asp and *S. maydis* scores. There was a moderate positive relationship (r = 0.43) between *S. maydis* scores and *F. graminearum* scores, among the inbred lines. Highly significant but moderately strong (r = 0.62, $p \leq 0.01$) positive correlation values were recorded for *A. flavus* kernel incidence scores of PSIK, ISIK and husk cover scores. This was, however, not the same for *F. graminearum* and *S. maydis* scores; which correlated positively but very weakly with husk cover and grain texture scores (r = 0.04 and 0.08, respectively). Notably, *S. maydis* correlated very weakly (r = -0.05), but negatively with the grain texture scores.

Table 3. Correlation of disease scores against maize grain yield among the parental inbred lines evaluated

	Asp Lab	PKI	PSIK	ISIK	Fus	Steno	ER	HC	GT
Asp Lab	-								
PKI	0.14	-							
PSIK	0.38	-0.4	-						
ISIK	0.57*	0.07	0.82***	-					
Fus	0.11	0.19	0.09	0.26	-				
Steno	0.35	0.53*	-0.14	0.13	0.43	-			
ER	-0.17	-0.06	-0.03	-0.06	0.16	0.37	-		
HC	0.37	-0.01	0.62**	0.62**	0.04	0.09	-0.08	-	
GT	-0.41	0.09	-0.25	-0.15	0.08	-0.05	-0.23	-0.2	-

Asp lab = *A. flavus* kernel sample colonisation score (laboratory evaluation); PKI = Percent-Kernel-infection; PSIK = Percent-severely-infected kernels; and ISIK = Incidence of severely-infected Kernels. Fus = *Fusarium graminearum* ear rot; Steno = *Stenocarpella maydis* ear rot; ER = Natural ear rot infection; and HC = Husk cover tightness, GT = Grain texture

*** = Significant at $p \leq 0.001$, ** = Significant at $p \leq 0.01$,* = Significant at $p \leq 0.05$

Correlation of disease scores with yield and other agronomic traits

Highly significant ($p \leq 0.001$), but moderate positive correlation values (Table 4), were observed for lab kernel infection measurements (Asp Lab and PSIK); with the exception Percent-Kernel-Infection (PKI) and Incidence of severely-infected Kernels (ISIK), which had negative but weak correlation with yield (r = -0.23 and r = -0.28, respectively). Similarly, weak but negative non-significant (r = -0.16) correlations existed between yield and *F. graminearum* disease scores. These results showed that yield amounts among the test crosses, to a small extent, decreased with increase in resistance to colonisation by *A.*

Table 4. Correlation of disease scores against husk cover and grain texture among the test crosses

	Asp Lab	PKI	PSIK	ISIK	Fus	Steno	ER	HC	GT	Yield
Asp Lab	-									
PKI	0.64***	-								
PSIK	0.59***	0.38*	-							
ISIK	0.79***	0.58***	0.73***	-						
Fus	0.05	0.02	0.11	0.11	-					
Steno	0.11	0.22	0.18	0.03	-0.12	-				
ER	0.32	0.21	0.16	0.20	0.17	-0.03	-			
HC	0.05	0.02	0.11	0.11	1.00***	-0.12	0.17	-		
GT	0.18	0.04	-0.16	-0.10	-0.08	-0.01	-0.21	-0.08	-	
Yield	-0.33*	-0.23	-0.31*	-0.28	-0.16	0.03	-0.57***	-0.16	0.30	-

Asp lab = *A. flavus* kernel sample colonisation score (laboratory evaluation); PKI = Percent-Kernel-infection; PSIK = Percent-severely-infected kernels; and ISIK = Incidence of severely-infected Kernels. Fus = *Fusarium graminearum* ear rot; Steno = *Stenocarpella maydis* ear rot; ER = Natural ear rot infection; HC = Husk cover tightness; and GT = Grain texture

*** = Significant at $p \leq 0.001$, ** = Significant at $p \leq 0.01$,* = Significant at $p \leq 0.05$

flavus and *F. graminearum*. With a weak and non-significant correlation (r = 0.03), yield was more or less affected by *S. maydis* infestation. A moderately strong but highly significant negative correlation (r = -0.57, $p \leq 0.001$) was observed between yield and PKI.

Correlation of ear rot against maize yield and other agronomic traits among the single crosses

The four parameters measured for colonization and incidence of *A. flavus,* were highly correlated ($p \leq 0.001$) (Table 5). *F. graminearum* and *S. maydis* correlated weakly for all the four *A. flavus* infection

Table 5. Correlation of disease scores and natural ear rot infection, against husk cover and grain texture among the single crosses

	Asp Lab	PKI	PSIK	ISIK	Fus	Steno	ER	HC	GT	Yield
Asp Lab	-									
PKI	0.50***	-								
PSIK	0.59***	0.38***	-							
ISIK	0.66***	0.34**	0.74***	-						
Fus	0.13	0.06	0	0.05	-					
Steno	0.1	-0.02	0.1	0.14	0.19	-				
ER	0.03	-0.07	-0.15	-0.05	0.02	0.02	-			
HC	0.07	0.02	0.02	-0.04	-0.03	-0.08	0.12	-		
GT	-0.08	0.05	-0.04	-0.09	0.05	0.23	0.08	0.33**	-	
Yield	-0.1	-0.12	-0.24*	-0.02	-0.01	0.03	-0.05	0.01	-0.17	-

Asp lab = *A. flavus* kernel sample colonisation score (laboratory evaluation), PKI = Percent-Kernel-infection, PSIK = Percent-severely-infected kernels, ISIK = Incidence of severely-infected Kernels. Fus = *Fusarium graminearum* ear rot, Steno = *Stenocarpella maydis* ear rot, ER = Natural ear rot infection, HC = Husk cover tightness, GT = Grain texture

*** = Significant at $p \leq 0.001$, ** = Significant at $p \leq 0.01$,* = Significant at $p \leq 0.05$

measurement parameters. *Fusarium graminearum* and *S. maydis* also correlated weakly. Yield correlated weakly, but negatively with *F. graminearum,* as well as with all the *A. flavus* incidence measurements. On the other hand, as with the test cross hybrids, yield among the single crosses correlated positively, but weakly with *S. maydis* scores (r = 0.03). Likewise, among the test crosses; there was a significant ($p \leq 0.05$), but negative correlation between the hybrid yield means and PSIK.

Test cross variance component ratios (Baker's ratios) and heritability

Multi location analyses of the test cross results revealed that with the exception *S. maydis*, Baker's ratios for all the other two ear rot pathogen measures, yield and traits; husk cover and grain texture was greater than 0.6 (Table 6). Broad sense heritability for *A. flavus* infection measurements were moderate to high, ranging from 0.5 to 0.61; whereas the narrow sense heritability measurements were between 0.35 and 0.78. Broad sense and narrow heritabilities for *F. graminearum* and *S. maydis,* were low both below 0.1. Yield, grain texture and husk cover all had very high Baker's ratios. Broad and narrow sense heritabilities were moderate to high for yield, moderate for grain texture and very high for husk cover tightness.

Table 6. Baker's ratios and heritability estimates (single crosses) for the laboratory and field ear rot colonisation for disease colonisation and traits closely associated with ear rot resistance

	NaCRRI			Bulindi			Ngetta			Multi location		
	BR	BSH	NSH	BR	BSH	NSH	BR	BSH	NSH	BR	BSH	NSH
Asp	0	0	0	1	0.48	0.48	0	0	0	0.69	0.5	0.35
PKI	0	0	0	1	0.43	0.43	0.29	0.15	0.04	0.90	0.52	0.47
PSIK	1	0.03	0.03	1	0.28	0.28	0.49	0.30	0.15	0.89	0.5	0.45
ISIK	1	0.03	0.03	1	0.24	0.24	0.53	0.13	0.07	0.96	0.61	0.58
Fus	0.05	0.47	0.02	0	0.15	0	0	0.37	0	1	0.10	0.10
Steno	1	0	0	0	0.10	0	0.18	0.61	0.11	0	0	0
HC	0.68	0.80	0.55	0.73	0.86	0.63	0.06	0.49	0.03	0.94	0.83	0.78
Tex	1	0.26	0.26	0.95	0.74	0.70	0.22	0.33	0.07	1.00	0.43	0.43
Yield	0.43	0.59	0.25	0.32	0.70	0.22	0.85	0.44	0.37	0.91	0.57	0.52

BR = Baker's ratio; BSH = Broad sense heritability; NSH = Narrow sense heritability; Asp lab = *A. flavus* kernel sample colonisation score (laboratory evaluation); and PKI = Percent-Kernel-infection
PSIK = Percent-severely-infected kernels; ISIK = Incidence of severely-infected Kernels; Fus = *Fusarium graminearum* ear rot; Steno = *Stenocarpella maydis* ear rot; HC = Husk cover tightness; and GT = Grain texture
*** = Significant at $p \leq 00.001$, ** = Significant at $p \leq 00.01$,* = Significant at $p \leq 00.05$

Baker's ratios and heritability values among the ear rot diseases varied from site to site, with PSIK and ISIK for *A. flavus* having very high and consistent Bakers ratios in all the sites. Broad and narrow sense heritabilities for *F.graminearum* and *S. maydis,* were generally low in single and multi-location analyses. Baker's ratio value for *S. maydis* at NaCRRI was very high (BR=1), but analysis gave very low Baker's ratios at the other two sites and in the pooled data analysis.

Single cross variance component ratios and heritability

Multi-location analysis of Bakers ratios resulted in very high ratios for all *A. flavus* infection and incidence measures ranging from 0.87 to 1 (Table 7). Baker's ratio for *F. graminearum* was high at

Table 7. Baker's ratios and heritability estimates (single crosses) for *A. flavus* kernel infection rate, *S. maydis, F. graminearum* colonisation and traits closely associated with ear rot resistance

	NaCRRI			Bulindi			Ngetta			Multi location		
	BR	BSH	NSH	BR	BSH	NSH	BR	BSH	NSH	BR	BSH	NSH
Asp	0.05	0.93	0.05	0.38	0.6	0.23	0.25	0.56	0.14	0.87	0.03	0.03
PKI	1	0.26	0.26	1	0.07	0.07	0.24	0.15	0.04	1.00	0.06	0.06
PSIK	1	0.32	0.32	0	0	0	0.13	0.69	0.09	1.00	0.04	0.04
ISIK	0.78	0.58	0.45	0.45	1	0.45	0.04	0.56	0.02	1.00	0.03	0.03
Fus	1	0.07	0.07	1	0.14	0.14	0	0	0	0.66	0.17	0.11
Steno	1	0.04	0.04	1	0.11	0.11	1	0.2	0.2	1.00	0.00	0.00
HC	1	0.33	0.33	0.68	0.75	0.51	0.47	0.29	0.14	1.00	0.02	0.02
Tex	0.79	0.84	0.66	0.60	0.84	0.50	1	0.19	0.19	0.93	0.12	0.12
Yield	0.64	0.52	0.33	0.37	0.63	0.24	0.96	0.35	0.34	0.75	0.17	0.13

BR = Baker's ratio, BSH = Broad sense heritability; and NSH = Narrow sense heritability
Asp lab = *A. flavus* kernel sample colonisation score (laboratory evaluation); PKI = Percent-Kernel-infection; PSIK = Percent-severely-infected kernels,; and ISIK = Incidence of severely-infected Kernels. Fus = *Fusarium graminearum* ear rot; Steno = *Stenocarpella maydis* ear rot; HC = Husk cover tightness; and GT = Grain texture
*** = Significant at p≤00.001, ** = Significant at p≤00.01,* = Significant at p≤00.05

0.66, whereas and that of *S. maydis* was very high at 1. Likewise, Bakers ratios for husk cover, yield and grain texture were also very high, ranging from 0.75 to 1. Narrow sense and broad sense heritability estimates for resistance to all the three ear rot pathogens and the ear rot associated traits including yield, were low. The single location Baker's ratios, broad sense and narrow sense heritability (BSH) estimates, varied across locations. Baker's ratios for the three ear rot pathogens were generally high; whereas broad sense and narrow sense heritabilities were generally low.

Discussion

Inbred locational variations for colonization by the dieases

Analysis of the inbred disease data in the three locations data revealed that the most aggressive ear rot pathogen was *S .maydis* (1-5 score scale, Table 1). This generally showed that the maize germplasm studied generally had a high resistance to *F. graminearum* and to *A. flavus* colonisation, but were very susceptible to *S. maydis*. Evaluation of the disease severity in inbreds was based on a study by Hung and Holland (2012); for which they reported ear rot resistance differentiation due to hybrid vigour contribution. They further reported that hybrids had 27% less ear rot and 30% less mycotoxin content, compared to their inbred parents; thus demonstrating the importance of hybrid vigour to disease resistance and mycotoxin contamination. They suggested that the most efficient way to improve ear rot and mycotoxin contamination resistance in hybrids is to evaluate and select among inbred lines before using resources to create and evaluate hybrids.

Correlation among the fungal disease scores against husk cover and grain texture among the parental inbreds, test crosses and single crosses

Among the test cross and single cross trials, correlation analyses among these four *A. flavus* kernel infection and incidence measures generally gave strong and highly significant positive responses (Tables 4 and 5). These results show that among the hybrids, the four measurements are directly correlated; implying that each of them can provide a reliable accurate measure for *A. flavus* infection.

For *A. flavus*, the same trend applies for the parental inbreds, but with less reliability as shown in the parental inbred trial (Table 3). On the hand, among the inbred lines; only Incidence of severely-Infected Kernels (ISIK) correlated moderately strongly with the overall kernel sample *A. flavus* infection score (Asp lab)(r=0.57*). ISIK also correlated positively and strongly with PSIK (R = 0.82**). This indicates that in *A. flavus* resistance, results from any of the two *A. flavus* colonization measures can reliably be used to predict their corresponding scores. This may help save resources and time in conducting multiple measurements for *A. flavus* infection.

In general, the *A .flavus* kernel infection and incidence measures correlated weakly, but positively with the scores of *F. graminearum* and *S. maydis* among the parental inbreds, test crosses and single crosses (Tables 3, 4 and 5). The exception was with Percent-Kernel-infection (PKI) among the inbreds that had a moderately positive, but significant (r = 0.53*) correlation with *S. maydis* scores. Correlations between *F. graminearum* and *S. maydis* varied among the inbred lines, test cross and hybrid trials.

Fusarium graminearum correlated moderately, but positively with *S. maydis* (r=0.43) among the inbred lines (Table 2), weakly but positive (r = 0.19) among the test crosses (Table 4) and weakly, but negatively among the single crosses (r = -0.12) (Table 4). This signifies that among the inbred materials, selection for resistance for one of these pathogens (*S. maydis* and *F. graminearum*), fairly or moderately favours indirect selection for resistance to the other pathogen. This indirect selection strategy among the inbred lines, can also be applied to select for resistance to *A. flavus;* but with less reliability.

This can be attributed to the low positive correlation values between the *A. flavus* scores and the other two ear rot pathogen scores, especially *F. graminearum*. A highly significant and strong positive correlation (r = 0.62, p<0.01) was recorded for the two *A. flavus* kernel incidence measurements of PSIK, ISIK and husk cover among the parental inbred lines.

Among the test cross and single cross hybrids, yield correlated weakly, but negatively with each of three ear rot diseases (Tables 4 and 5). This suggests that there is a general independent relation between the three fungal ear rot infections and maize yield. Natural ear rot infestation generally correlated weakly, sometimes negatively with artificial infection by the three pathogens. This points to an independent relationship between natural and artificial ear rot infections in both parental inbreds and hybrids. This suggests that genotypes selected under artificial infection may not necessarily be superiorly resistant under natural infection.

Mesterházy *et al*. (2012) in a review of studies involving *Fusarium* spp., emphasizes that both artificial and natural infections are of great significance. They stress that genotypes selected under artificial infection pressure must demonstrate superior resistance under natural infection pressures. They further state that for this reason, research that clarifies the relationships between artificial and natural infection results is of high priority.

In *Fusarium* spp. studies, Mesterházy *et al*. (2012) observed a close correlation between the results of the silk channel inoculation method and natural infection (r = 0.75–0.96). In related *Fusarium* spp. studies, Palaversi¡ic´ *et al.* (2010) found medium to close (r = 0.66, 0.61, 0.84) correlations between the silk channel and natural infection severity data. Based on these studies, Mesterházy *et al.* (2012) alleges that the data support the view that artificial and natural infection data tend to be closely correlated. Results from our study point out that this hypothesis may not hold true as natural infection generally weakly correlated with artificial inoculation. This might have been due to natural fungal

inoculum pressure being very low in all the three environments. Additionally this hypothesis may not hold for the other fungal ear rots, other than *F. graminearum*. Further, a toothpick inoculation method pierced through the ear husk, other than silk channel inoculation, was used in this study and may not give the same effects to support the hypothesis. With regard to this hypothesis, Mesterházy *et al.* (2012) states that resistance to the two main modes of fungal access into the ear, *via* the silk or through kernel wounds, is not correlated in all genotypes. Mesterházy *et al.* (2012) noted that the reason for this lack of correlation was not clear. They further reiterated that when silk channel resistance was assessed, data from natural and artificial inoculation trials correlated well. They concluded that comparable data relating to kernel resistance have not been published. In the light of this, more studies based on these findings need to be done to test this hypothesis.

Among all the three trials (inbred line, test cross and hybrid trials), correlation among the three ear rot pathogens and three associated ear rot resistance traits i.e., husk cover and grain texture were mainly weak and negative (Tables 3, 4 and 5). The exception was among the parental inbred lines, where significant correlation values (r = 0.62**) were observed between husk cover and both PSIK and ISIK. The weak and negative correlation between husk cover and *S. maydis* scores, had earlier been recorded by Mukanga (2010); who noted a negative relationship between husk cover and *S. maydis* scores. Mukanga (2010) reported that the intensity of *F. graminearum* infestation increased with the tightness of the husk cover. The husk cover ceased being a barrier to ear rot pathogen infection, after being broken by artificial tooth pick inoculation. This, therefore, suggested that grain texture and husk cover had little or less contributions to reducing the progress of ear rot colonization once the resistance barrier had been broken by the pathogens.

The mainly weak and non-significant correlation values among the three ear rot mean disease scores, as indicated by the inbred, test cross and single cross trial correlation results (Tables 3, 4 and 5) suggests that resistance to the three pathogens is inherited independently. Citation has also been made by other scientists arguing that inheritance of resistances to Stenocarpella ear rot and other infections by Stenocarpella fungal species such as Diplodia leaf spot and Diplodia stalk rot, are likely to be independent of each other (Mesterházy *et al.*, 2012).

Heritability

Based on Specific combining ability (SCA) analyses, single cross hybrids constituted in the study, such as CML 216/D264-3, had low and negative SCAs for all the three ear rot diseases; in addition to having high yields, good husk cover and grain texture (Table 2). This designates their superiority in terms of multiple ear rot resistance and agronomic performance. This, therefore, supports the need to test for combining ability when developing hybrids to come up with the best crosses in similar studies. Multiple location heritability analysis for ear rot, husk cover and grain texture measurements generally gave high Baker's ratio estimates and low narrow sense heritability (NSH) estimates. The high Baker's ratio estimates for the ear rot disease and associated trait measurements give a stronger backing for the fact that additive gene effects were the most important in the inheritance of resistance to the three ear rot pathogens among the study inbred lines. The high heritability estimates also suggest that, though inheritance is quantitative, it is only controlled by a few genes and transmissivity of resistance is low. Across location broad sense heritability estimates for resistance to the three ear rot, pathogens were generally found to be low. Mukanga (2010) reported that there is no confirmation for complete resistance to Aspergillus ear rot, Fusarium ear rot and Stenocarpella ear rot, or for cross resistance to two or more ear rots. He further concluded that although the reported heritability values are low for Aspergillus and Fusarium ear rots, in principle, progress to selection would still be made since the gene effect is largely additive. The results from the present study also indicate no complete resistance among the germplasm used in the study to any of the three fungal diseases.

This low broad sense heritability (BSH) estimate indicates that the proportion of heritability for ear rot resistance, good husk cover and good grain texture controlled by genetic effects is low. The low values of NSH estimates for all the ear rot pathogen resistance estimates indicates that transmissivity of these traits across generations is low. High Baker's ratios were generally obtained for the three ear rot diseases, yield and the associated traits; husk cover and grain texture. This indicates that the variation obtained among the crosses for these traits can easily be fixed and selected for, through breeding especially in early generations (Robertson et al., 2006). It should also be possible to implement backcrossing methods to infer multiple ear rot resistance. The high heritability also confirms that the visual scoring system for ear rots and the associated traits is effective and progress can be made by the breeders for multiple ear rot resistance breeding. Furthermore, the fungal ear rots can be screened visually and so is less expensive and less time consuming to evaluate than laboratory assays for mycotoxins.

This further indicated that heritability for multiple ear rot resistance was generally high and selection could be done in early generations though the proportion of resistance due to genetic factors as shown by the low BSH was generally low and its transmission (NSH) was low. Environmentally induced resistance can, therefore, be enhanced by breeding for materials with a good husk cover and more flinted maize to encourage more environment induced resistance against the ear rots. These heritability results, therefore, suggest that resistance to *S. maydis* and *F. graminearum* cob rots is quantitative and determined wholly or partly by additive gene action and non-additive effects, as earlier reported in separate ear rot studies by Rossouw *et al*. (2013). They further reported that resistance to ear rots was controlled by additive gene effects, with low dominance and interaction effects. Similar studies indicate that ear rot resistance in maize, in a related pathosystem, when singly infected by *A. flavus,* is conditioned by epistasis and additive gene action (Bello *et al*., 2017) Results from the present study, therefore, conform to similar earlier studies that reported that overall, resistance to cob and kernel infection is conditioned by quantitative gene action (Zila *et al.*, 2014) Resistance to Fusarium ear rot has been reported to be polygenic with relatively low heritability (Lanubile et al., 2017). Robertson *et al*., (2006) reported low to moderate heritability values in two well-known North American Populations that comprised of 213 $BC_1F_{1:2}$ of GE440 to FRI1064 (GEFR) and 143 recombinant inbred lines from the cross of NC300 to B104 (NCB)

Conclusions

Both general combining ability of parents and specific combining ability of hybrids are critical to obtain superior cultivars in resistance to ear rots in maize. Therefore, hybrids involving inbred lines with favorable alleles to ear rot resistance and good specific combining ability to decrease the severity of the fungal diseases are a promising strategy to improve the grain quality in maize breeding. Since the sources of multiple ear rot resistance used in this study did not confer complete resistance to any or a combination of the three ear rot diseases, it is recommended that breeding efforts continue so as to find sources that can confer complete multiple resistances. Such sources are usually controlled by single genes showing dominance. When those sources are found, it is recommended that they should be bred into commercial varieties that already possess the quantitative genes from sources used in this study. This would be most ideal because in the event of breakdown of the dominant gene, additive genes are available to avert disaster.

Given the moderate correlation between some of the three ear rot pathogen mean scores, indirect selection is possible for ear rot resistance but with less reliability. Therefore, direct selection against specific ear rot pathogens should be prioritized over indirect selection by researchers in future resistance

studies when using single pathogen inoculation. However, indirect selections are less time and less money consuming than direct single pathogen selections for multiple pathogen resistance introgression.

Both additive and non-additive genes contributed to resistance to all the three fungal ear rot diseases. However, the major contribution of the inheritance of resistance to the three ear rot pathogens was additive gene action. It was further revealed that, resistance to the three pathogens is inherited independently as the three pathogen infection scores did not correlate strongly and significantly. Yield performance among hybrid maize was found to be independent from infection scores of each of the three fungal infections. Results from this study therefore do not indicate that similar resistance genes in a given genotype (hybrid or parental inbred) are responsible for yield performance and resistance to any of the three specific ear rot pathogens.

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