HERITABILITY ESTIMATES OF RESISTANCE TO MAIZE STREAK VIRUS AND TURCICUM LEAF BLIGHT IN TWO MAIZE POPULATIONS.

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CHAPTER ONE

1.0 INTRODUCTION

1.1 Back ground

Maize (*Zea mays* L) is the most popular cereal crop widely grown in many sub-Saharan countries and the worldover (CIMMYT, 2005; Gupta *et al.*, 2009), with 57% of Ugandan farm households growing the crop (UNHS, 2005). It is believed that maize was introduced from Americas to Europe by Christopher Columbus through his various voyages (Purseglove, 1988) and then introduced to the African coasts by the Portuguese in the 17th century although much less information has been available as to when it spread to the rest of the interior (Miracle, 1965).

However in Uganda, maize is said to have been introduced in 1861(Sprague, 1987) but was first recorded as a minor crop in 1882 (Purseglove, 1988) and by 1900 it had been an established crop grown widely in many parts of Uganda (Balirwa, 1992). Maize was until 1930, the government promoted crop but this policy was later reversed due to maize crop's heavy up take of soil nutrients and competition with cotton, the major export crop at the time (Jameson, 1970). It is currently the third important crop in Uganda after plantains and cassava (Steve *et al.*, 2010). It is estimated that the total area under maize production by 2008 was 862,000 hectares with an estimated production of 1,266,000 metric tones and an average yield production of 1434 Kg/Ha (FAO, 2009).

Maize became an important food crop for both humans and live stock in 1970s to meet increased consumption demand from urban consumers and institutions (Anon, 2006). By 1999/2000, maize contributed 25% of the caloric intake at the national level, only second to root tubers which contributed 37% (Kasenge *et al;* 2001). It is used as food in form of roasted maize, 'posho' and at times taken as

porriedge. It is also used as feed for livestock and raw material for making local brew that has made it become one of the major export and foreign exchange earner in Uganda (BOU, 2007). The ability of maize to provide vital sources of nutrients in form of carbohydrates , protein, vitamin B, and minerals has made it compare favourably with root tubers to satisfy human and animal needs (http://www.faqs.org/nutrition/Ca-De/Corn-or-Maize-Based-Diets.html).

Inspite of its importance, maize production in Uganda is constrained by a number of factors that include largely pests (maize storage weevils and Lepidopterous stem borers) and diseases (Maize streak Virus, Turcicum leaf blight, gray leaf spot) low soil fertility paticulary deficiency of N and P, water stress and lack of implementation of recommended agronomic practices (timely planting, weeding) and post harvest technologies that include proper drying and storage (IITA, 1993, Sseruukuuma *et al* 2001, Bekunda *et al.*, 1997).

Among the major diseases of economic importance are maize streak virus disease (MSD) and Turcicum leaf blight (*Exserohilum turcicum)*-TLB (Bigirwa *et al.*, 1993, NRI, 2005). The MSD causes ~~is an important disease~~ in sub-saharan Africa ~~with~~ an estimated yield loss of up to 100% (Dionne *et al.*, 2007, Lagat *et al.*, 2008, Kyetere *et al.*,1999) while TLB is a widespread disease of maize which can cause yield losses of up to 70% (Yeshitila, 2003) especially in cool humid locations (Juliana *et al.*, 2005). Significant yield loss in the lowland tropical and highland agroecologies of Uganda is attributed to these two diseases (Kyetere *et al.*, 1998, Lagat *et al.*, 2008).

In many economically important plant pathosystems, disease is controlled by breeding for genetic resistance in the host plant (Fakorede *et al.*, 2003; Pratt *et al.*, 2003) and the control of TLB and MSD has been directed towards this (Slavica *et al.*, 2007, Olaoye, 2009). Breeding for resistance begins with generation of breeding populations from which selection of better parents is made (Kevin *et al.*2006). The starting genetic material of a breeding program in development of resistant lines to these diseases

is very important, since it determines the potential improvement under selection. Modern maize breeding is mainly based on F_2 (elite x elite inbred crosses), backcross and synthetic populations; with the F_2 generation being unique in plant breeding since it is usually the first generation in which selection is practiced due to few population numbers, making selection to have the greatest impact (Jensen, 1998). During selection of the traits, heritability is an important statistic that measures the phenotypic variance, which is attributable to genetic cause. It indicates the structure of population and the extent to which a given character can be transmitted to the next generation there by helping the plant breeder to predict the behaviour of the succeeding generation and making desirable selections. The higher the heritability, simpler will be the selection process and greater will be the response to selection.

1.2 Problem statement.

The risk of crop loss due to diseases occurs in Uganda because maize is produced by small-scale farmers who have no access to means of crop protection like use of pesticides against MSD and TLB. Due to a narrow genetic base background (Dionne, 2007, Magenya *et al,* 2008), efforts to breed for resistant varieties/~~hybrids~~ against infections like MSD and TLB has been hard as the developed resistance usually breaks down probably due to a single gene control ~~combined with changes in environment~~. Two new F_2 maize populations bearing resistant traits for MSD and TLB have been introduced into Uganda from CIMMYT, Nairobi but their heritability estimates and nature of genetic variation ~~for these diseases~~ is not known to predict the ~~direction~~ of the selection of resistance ~~lines/families for future hybrid development.~~

1.3 Justification

Where as average maize yield in USA and other high-income countries is 8.3 tons per hectare, most of Sub Saharan Africa and other developing countries have achieved yield below 2.0 tons per hectare

(Kevin *et al*, 2006). ~~Most of this high yield reduction results from~~ losses ~~due to~~ pests and diseases, poor soil fertility, and water shortage (AFY, 2009) as well as low genetic potential (Luciano *et al*.,2000). Of the many diseases, MSD and TLB are one of the most common maize diseases that cause greatest damage in Uganda (Asea., 2005, Bigirwa *et al*., 2001). Gardner *et al;* 2007 reported that breeding of resistant genotypes against diseases is one of the most desirable ways to reduce their impact in maize production. This begins with introduction of new populations which can be made from one region to the other easily and is used for further manipulation to develop breeding lines (Jamal *et al*., 2009) which are resistant to the two diseases. Selection provides an opportunity for carrying out any breeding program effectively (Abdus *et al*; 2003) in these populations. Hence the broader the range of heritable variation to MSD and TLB resistance, the more effective will be the selection of resistant parents for the next generation and vice versa. Therefore the effectiveness of the selection depends on heritability. The knowledge of heritability will help in predicting the behaviour of the succeeding generation and making desirable selections (Shoukat *et al*; 2007) for resistance. ~~Heritability estimates presented in this research work will enable plant breeders to make predictions about the possible progress that can be achieved by making the selection~~ more effective.

1.4 Research objectives

- To ~~estimate~~ the heritability for resistance to MSD and TLB ~~using WL-429-40.E.21 and WL-429-40.E.66 populations.~~
- To ~~predict~~ the nature of gene action conditioning resistance for TLB and MSD in WL-429.E.21 and WL-429-40.E.66.

1.5 Hypotheses

- The heritability estimates for resistance to MSD and TLB is high in both WL-429-40.E.21 and WL-429-40.E.66 populations.

The nature of

- Resistance for MSD and TLB in WL-429-40.E.21 and WL-429-40.E.66 populations is ~~conditioned by~~ quantitative ~~genes~~.

CHAPTER TWO

2.0 LITERATURE REVIEW

Review under each objective

2.1 Heritability for resistance to MSD and TLB

Heritability is a measure of the phenotypic variance attributable to genetic causes, with a predictive function of breeding crops (Songsri *et al.*, 2008) and the higher the heritability estimates, the simpler are the selection procedures (Khan *et al.*, 2008).

Heritability has been useful in breeding especially in defining the direction of any breeding programme. Hakiza *et al* (2004) used heritability estimates for resistance to TLB to predict gain from selection. The $F_{2:4}$ families were found to have heritability estimates consistently higher than those from the $F_{2:3}$ families. Hakiza *et al* (2004) suggested that heritability estimates greater than 0.5 indicated that resistance to TLB was achievable through phenotypic selection in replicated plots. Gordon *et al.*, (2006) used heritability estimates of incubation and disease severity for breeding purposes and reported that heritability estimates of incubation period were lower and more variable than those based on disease severity. Onyimbo (2007) used heritability estimates for gray leaf spot (GLS) and Phaeosphaeria Leaf Spot resistance and suggested that SRS method based on phenotypic variance in the field without progeny testing could be used to improve GLS.

Asea, (2005), observed highly variable estimates of heritability for resistance among genotypes developed and attributed it to variable reaction of progenies due to extreme drought during the first season in 2003 that may have affected virus multiplication, plant growth, and resultant symptom expression.

Nick (1978) in his evaluation of F_1 and F_2 generation on five maize populations found out that narrow sense heritability estimates for MSD resistance ranged between 0.34 to 0.52; populations UK-

198X7721, UK-330 X7721, TZY-SR X7721, "Revolution" x 7721 and Tan-Lita x 7721 gave estimates of 0.39, 0.49, 0.34, 0.52 and 0.39 respectively.

Sibiya., (2009) in his study for narrow sense heritability for resistance to Phaesphaerial leaf spot (PLS) found out that the estimates ranged from 40 to 74% and were slightly lower than 70-85% as earlier reported by Carson (2001) and concluded that the presence of dominance and epistasis could have affected the estimates of heritability downwards. Falconer and Mackay (1996) asserted that such differences in heritability estimates amongst different studies could be a result of differences in either the environment and or the genotypes used as well as different levels of additive variance versus dominance and epistatic variances.

Walter R. Fehr, (1993), describes that heritability estimates can be got where selection is based on single plants data in a population that is not sub-divided into plots, although the estimates were said to be slightly lower if compared with the estimates on entry mean basis.

2.2 Resistance of MSD and TLB

TLB resistance is controlled by both quantitative and qualitative genes (Welz *et al* 2008). Quantitative resistance to *E. turcicum*, causal agent of TLB was found to be inherited polygenically (Pataky *et al.*, 1986) and five qualitative dominant or partially dominant genes, *Ht*, *Ht2*, *Ht3*, *HtM* and *HtN* control resistance to specific races of *E.turcicum* (Lipps *et al.*, 1997), but may individually be overcome by virulent genes present in such specific races of the pathogen (Juliana *et al.*, 2005). Pratt and Gordon (2006) found out that combinations of qualitative and quantitative resistance genes are generally employed in breeding for resistance, with the emphasis now on quantitative genes, due to their higher phenotypic stability. Resistance conferred by Ht genes is characterized by chlorotic and necrotic lesions without spore formation thereby limiting the spread of the disease (Singh *et al.*, 2004) while resistance

conferred by minor genes is not absolute as in qualitative resistance but chances of new biotypes overcoming quantitative resistance are considered to be minimal (Ojulong *et al.*, 1995). Quantitative resistance to TLB was found to be highly heritable in general, although it ranged from low to high estimates of between 0.26 to 0.89 in these trials (Welz *et al.*, 1998). High levels of Quantitative resistance have been demonstrated in inbred lines such as H99 (Hakiza *et al* 2004). However, despite the availability of these resistant sources, lately TLB incidences and severity had increased in Africa probably most of the resistance that was available in some regional hybrids could have been qualitative in nature which is not stable (Vivek *et al.* 2009).

Although a significant progress has been made in the breeding of MSD-resistant maize (Efron *et al.* 1989), in practice commercially available maize hybrids are at best moderately tolerant to the disease. (Darren *et al*; 2009) said that even the ultra-short season hybrids popular amongst commercial farmers for their high yields and very quick growth, have no MSD tolerance/resistance, and that farmers growing these are at particular risk of losing their entire crops to MSD . The resistance to MSD is controlled by qualitative and quantitative genes, with the latter being controlled by 2-3 genes (Efron *et al.*, 1989). Redinbaugh *et al* (2004) found that qualitative resistance in MSD control was not reliable for it broke down under field conditions and they argued that use of quantitative genetic resistance provides a long lasting resistance for the disease. Use of tester CML 202 was found to have got a positive impact in influencing resistance in development of resistant inbred lines for it lowered the disease expression where it was used as a parent (Pernet *et al.*, 1999).

While there are several ways to differentiate various forms of genetic mechanisms (**Simmonds, 1982**), one of the criteria is to monitor the distribution or variation pattern of a specific character in a given population to determine whether the trait is qualitatively or quantitatively controlled (Paul de Souza *et al.*, 1999) hence frequency histogram distribution of disease mean scores for TLB and MSD are useful

in this prediction.

Based on visual assessment of the frequency distributions of TLB ratings of F_2 (Jenkins and Robert, 1952) and F_3 progenies (Jenkins *et al.* 1952) derived from crosses of resistant with susceptible inbred lines, Jenkins and co-workers concluded that TLB resistance was polygenically inherited in those populations as they assumed that most of the genes had minor and a few major effects.

2.4 Reaction of Maize populations to MSD and TLB which objective??

Various genotypes differ in their resistance to MSD and TLB with many susceptible to the diseases, this is attributed to environmental factors, the virulence of the strains of the pathogen and the genotype of the host plant (Campbell *et al.*, 1974). Selection of resistant genotypes has been done either by exposing plants to natural infection in the field or by artificial infestation of the viruliferous leafhoppers (IITA, 1977) and use of artificial inoculums on sorghum kernels for MSD and TLB respectively. Selection of genotypes/families resistant to diseases is based on low symptom manifestation of such plants when exposed to intense disease pressure (IITA, 1977, Mariote, 2007) and using a visual severity rating scale. Clark *et al.*, (1989) in their study on MSD concluded that symptom manifestation is influenced by cultivar susceptibility, degree of stress, growth stage or virulence of the isolate; as many isolates/strains were known to occur in different localities with some being more virulent than others. Also in his study to MSD, Kyetere *et al.*, (1996, 1998), found that some resistant parents previously reported to have complete resistance by Kim *et al.*,(1989) had mean score higher in their experiments and the difference in scores was attributed to change in environments, difference in isolates/strains, disease factors and heterogeneity among different Tzi4 accession used.

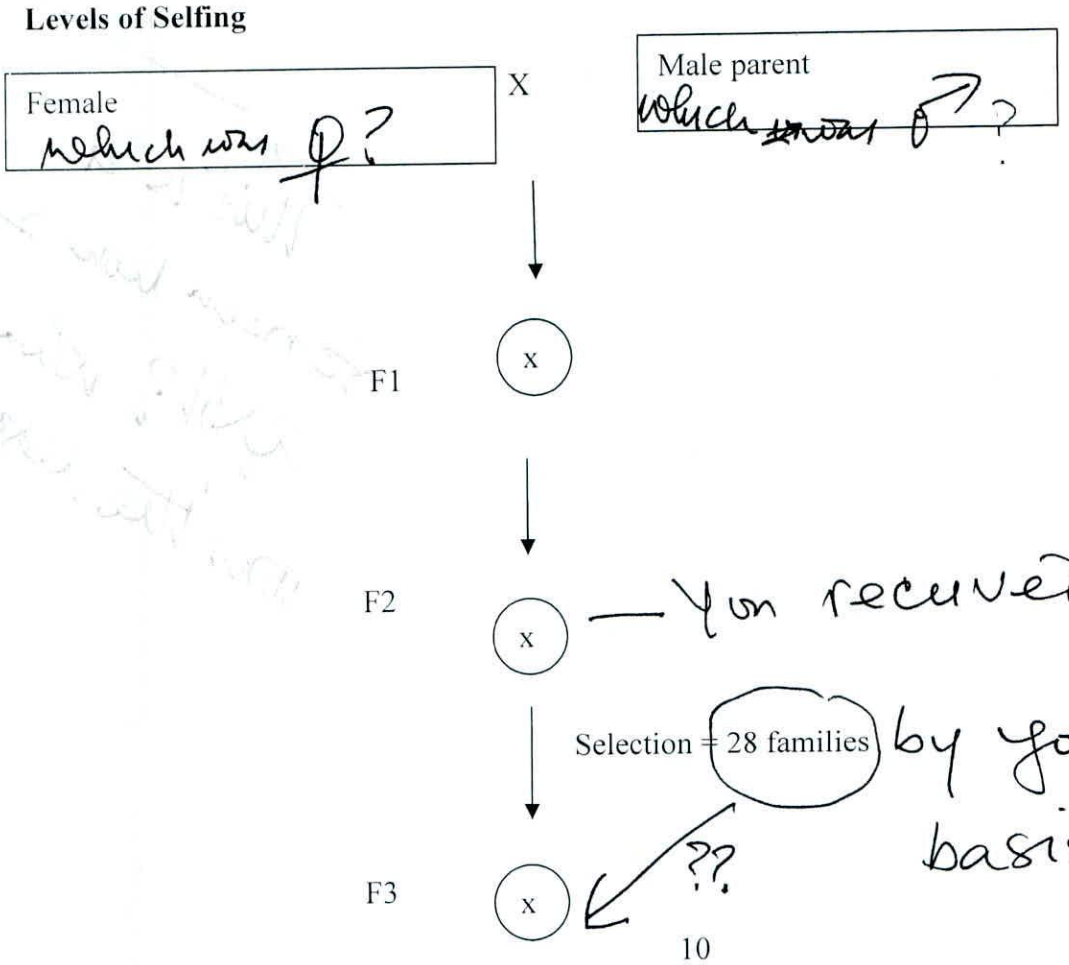
CHAPTER THREE

3.0 MATERIALS AND METHODS

3.1 Germplasm to be used

Two F_2 maize populations, WL-429-40.E.21 (Stock: D376-21) and WL-429-40.E.66 (Stock: D376-66), referred to respectively by their pedigree (WL-429-40/[CML442/CML197//[UXPSEQ]CYF2/P49-SR]F2) and WL429-40[CML442/CML197//[TUXPSEQ]CTF2//P49-SR]F2, respectively developed at CIMMYT Nairobi were used in this research. WL-429-40.E.21 and WL-429-40.E.66 were developed by making a three way cross followed by self-pollinating single F_1's to produce F_2 populations. F_3 populations consisting of families were developed by self pollinating F_2 plants in each population to form F_3 lines in a pedigree fashion.

Levels of Selfing



3.2 Experimental lay out and selection

F_2 populations (90 plants per population) were planted each on a single plot, assessed for disease resistance, 28 families showing resistance trait were selected (representing a 30% selection intensity) based on a single plant selection following pedigree selection method. The selected plants were selfed to produce F_3 family lines from January 2011-April 2011 at National crops resources research institute (NaCRRI). The selected 28 F_3 family lines in each population were planted such that each family represented a plot of 10 plants and their reaction to TLB assessed between December 2011 – April 2012 while MSD assessment was done separately between March –July 2012 because leaf hopper populations were still low in cages during December 2011 planting to enable a successful inoculation. Assessment was on the basis of visual scores (Percent Leaf Area Affected-PLAA) with no replication since Presello, 2001 said that plants grown under pedigree, selection can be performed among and within families which are generally grown on a single plot with no replication. At F_3 five representative maize plants selected randomly per family were tagged and disease scores taken and an average score computed per family/plot in each as described by Walter, 1991. The best families showing resistance to MSD and TLB were advanced to F_4 generation for experimental continuity. Selection of F_2 plants for advancement to the next generation was conducted in such a way that plants with "zero" (0 for TLB and 1for MSD) severities were not selected and only those that showed symptoms with severity scores of 3 or less were selected (Mariote, 2007), since selection of plants with zero scores as "resistant" plants were previously found to be mere escapes (Fajemisin *et al.*, 1982). Standard management practices for maize production like timely weeding, fertilizer application was done.

3.3 Infecting plants with maize streak virus

Leaf hoppers (*Cicadulina mbila*), already captured and reared in cages at NACCRI, were fed on potted maize plants showing severe MSD symptoms and raised in screen house. They were placed in the

acquisition cage for a period of 2 days (Kyetere *et al.*, 1998), there after the cages containing leafhoppers were covered using a dark sheet leaving an opening on one side for light penetration where the hoppers would move. This enabled the removal of MSD infested plants and their replacement with health potted maize seedlings ~~populations.~~ This was done at V3 stage without making hoppers escape, after which the cages ~~would be~~ were closed and the sheet removed. To ensure uniform viral infection, the insects were disturbed every six hours. After two days of leaf hopper feeding, the seedlings were then transplanted ~~to~~ into the field (Bua, *et al.*, 2010, Opio *et al.*, 2010). in a randomised design? what design??

3.4 ~~Preparation of~~ TLB inoculum

Inoculum was prepared following the method ~~as~~ used by Hakiza *et al.*, (2004). Leaf tissues bearing lesions of the disease were collected from the field at NaCRRI, portions of leaf tissues were surface sterilized in 1% sodium hypochlorite for 30 s, rinsed in sterile distilled water (Okori, 1997) and then placed under high humidity under fluorescent light for 3 days to initiate sporulation (Lipps *et al.*, 1997). Single conidia were then picked from conidiophores with a glass needle and placed onto potato dextrose agar (PDA) in petridish and after two weeks, one petridish culture was placed in ~~each~~ a plastic bag containing autoclaved sorghum kernels. The bags were maintained on the lab bench for 10-14 days and shaken every other day to distribute the fungus evenly over the kernels (Ngwira *et al.*, 2005). Infested kernels were air dried on laboratory benches for 3 to 4 days and thereafter kept dry until they were used in inoculating maize plants in the field (Lipps., *et al.*, 1997). ~~Plants were inoculated~~ by placing approximately 50 infested sorghum kernels into the whorl of each plant at V6 stage just before sun set to enable conditions for initiation of spore germination during the night (Hakiza *et al.*, 2004).

3.5 Determination of Disease severity.

TLB disease severity (as percentage area leaf affected) were determined using a modified scale from

whose method as by who??

that used by Elliot and Jenkins, (1946) quantitative scale of 0-5 as the method was used by (Muiru *et al*; 2007); where: 0 = No symptoms, 0.5=very slight infection one or two lesions on lower leaves, 1 = slight infection, a few scattered lesions (3-8) on lower leaves, 2 = light infection, moderate number of lesions (9-15) on lower leaves, 3 = moderate infection, abundant lesions(>16) on lower leaves and a few on lower laves, 4 = Heavy infection, lesions abundant on lower and middle leaves and extending to the upper leaves, 5 = Very heavy infection, lesions abundant on all leaves and plants may be killed prematurely. The first disease score was made at the R1 growth stage (Ritchie *et al.,* 1989), when 50% of plants have visible silk exertion. Visual estimates for F3 were made on whole plot rather than individual plant assessments because each plot constituted of a family and reaction of the plants within a family to infection was assumed to be similar (Asea, 2005). A total of two assessments will be made at one-week intervals.

at this level – you still have segregation!!!

MSD severity ~~shall be~~ was determined using a quantitative scale of 1-5 with an increment of 0.5 as used by Kyetere *et al.*, (1998) and Welz *et al:* (1998) where 1 = no symptoms on leaves, 1.5 = very few streaks on leaves, 2 = light streaking on old leaves, gradually decreasing on young leaves, 2.5 = light streaking on old and young leaves, 3 = moderate streaks on old and young leaves, 3.5 = moderate streaks on old and young leaves and slight stunting, 4 = severe streaking on 60% of leaf area, plants stunted, 4.5 = severe streaking on 75% of leaf area, plants severely stunted, 5 = severe streaking on 75% or more of the leaf area, plants severely stunted, dying or dead (usually within 4 weeks after infestation).In this case 1=resistant while 5=highly susceptible. MSD symptoms ~~will be~~ were assessed and rated at 52 days after infestation. MSD symptom severity was rated about 7 and 8 wk (two scores) after infestation as described by Kevin *et al.*, (2006).

?? – 52 Days = 7 & 8 wk ??

~~CHAPTER FOUR~~

~~4.0 DATA ANALYSIS.~~

3.6 Data analysis

3.6.1 ~~4.1~~ Estimation of heritability

Heritability estimates for resistance to MSD and TLB were estimated using parent-offspring regression ~~since it has been advocated as a means of estimating heritability~~ because of the likelihood that few assumptions are violated, such as freedom of all genetic loci affecting the trait from linkage (Nyquist, 1991; Foolad and Jones, 1992). It is also the only simple method for estimating heritability that is not biased by selection on the parents (Nyquist, 1991; Lynch and Walsh, 1998). ~~The method was also used by Hanson *et al.*, 2002 by regressing of F_3 family means on the values of the F_2 parents in tomatoes.~~

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3.6.2 ~~4.2~~ Analysis for the Genetic variation

The genetic variation among the populations was analyzed basing on the type of distribution of the disease severity scores as used by Jenkins *et al.*, (1952) and Paul de Souza *et al.*, (1999) in their ~~investigations on genetics of anthracnose panel canker disease resistance in *Hevea brasiliensis* and~~ inheritance studies on TLB respectively. The character which show continuous variation is often referred to as a ~~likely~~ candidate for polygenic inheritance while that where the trait is clearly cut is qualitatively inherited (Paul de Souza *et al.*, 1999; Mayada *et al.*, 2010).

cite under appropriate lit review

Here just state method used with authority —

CHAPTER FIVE

5.0 RESULTS AND DISCUSSION

Narrow-sense heritability estimates based on the regression coefficients from parent-offspring regression at final disease ratings for MSD were found to be 0.492 and 0.563 for WL-429.E.21 and WL-429.E.66 respectively. While narrow sense heritability estimates for TLB were 0.458, 0.58 for WL-429.E.21 and WL-429.E.66 respectively as indicated in Table1 below.

Table1: **Regression coefficients of Population WL-429.E.21 and WL-429.E.66 F2 family means on to F3 family means based on Percent Leaf Area Affected (PLAA) used to estimate narrow sense heritability for resistance to TLB and MSD at NaCRRI, Uganda between 2011-2012.**

Population WL-429.E.66			**Population WL-429-21**	
	TLB	**MSD**	**TLB**	**MSD**
$h^2 \pm se$	0.580± 0.136	0.563±0.192	0.458 ±0.075	0.492±0.213
R^2	0.412491	0.249	0.59	0.17
p-value (1%)	<.001	0.007	<.001	0.029

In all characters studied, the regression model showed a significant relationship between F_2 and F_3 in the two populations. However the R^2 values for MSD in the two populations appeared low (R^2 =0.249 and R^2 =0.170 for WL-429.E.66 & WL-429.E.21 respectively) and this could have been due to the drought that was experienced soon after transplanting inoculated plants to the field, which could have made some families weakened and show slightly higher disease severities than their parental F_2's as indicated in Fig.1

The narrow sense heritability estimates for MSD in the two populations agree with those reported by Nick, (1981) Kim *et al* (1989). Heritability estimates for MSD resistance have been low to high, e.g., 22% (Asea, 2005), 34%-54% (Nick, 1981), 83% (Kim *et al.,* 1989), 62 and 93% (Welz *et al.*, 1998), 73 to 98% (Pernet *et al.*, 1999) with one major QTL (Kyetere *et al.,* 1998) and two, three, or "few" total genes implicated (Efron *et al.*, 1989). Heritability estimates for TLB also agree with those reported by Hakiza *et al.*, (2004). Heritability estimates have been also low to high, for example Hakiza *et al.*, (2004) found narrow sense heritability estimates of between 27-80% in the populations they investigated ~~on~~ for resistance to TLB. The heritability estimates of the two populations for resistance indicated that both MSD and TLB were moderately heritable, although with population WL-429.E.21 having estimates slightly lower than those of population WL-429.E66.

The relatively medium estimates of heritability observed in this study indicate that substantial portion of genetic variation is heritable in nature (Sibiya, 2009), therefore improvement of MSD and TLB resistance in these populations can be realized through breeding as suggested by Carson (2001). ~~in these populations~~. The moderate narrow sense heritability estimates indicate that conventional pedigree and early generation selection could be effective for initial improvement (Sibiya. 2009) of MSD and TLB in these two maize populations. Asea (2005) ~~stated~~ that moderate estimates of heritability based on parent-offspring coefficients indicate that resistance to pathogens ~~was~~ heritable and early generation selection could result in improved germplasm under high disease pressure evaluations. Analysis of frequency distribution ~~histogram~~ for mean population severities for TLB and MSD in Fig. 2-8, below indicated that the MSD and TLB severity scores at F_3 were fairly following a normal distribution when compared to their parents, F_2's where the disease severity scores had a clear cut. Such behavior of variation pattern is an indicative of a quantitative character that is polygenically controlled as described by Jenkins, (1952) and Paul de Souza., *et al.*, (1999). However the frequency distribution of MSD severity in WL-429.E.66 was sharper than that of WL-429.E.21, an indication that the former

population is better to transfer the MSD quantitative resistance to the next generation.

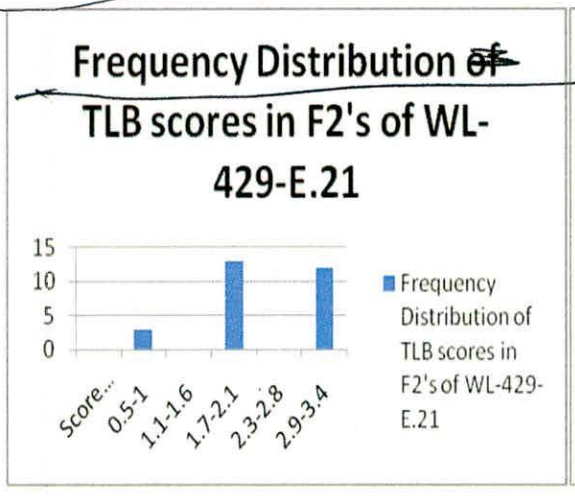
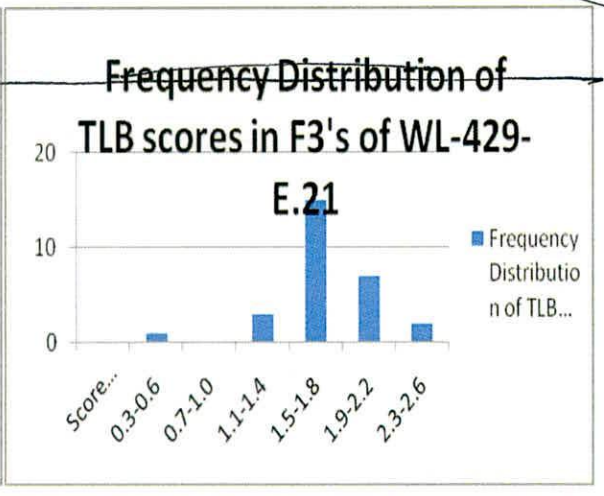


Fig.1



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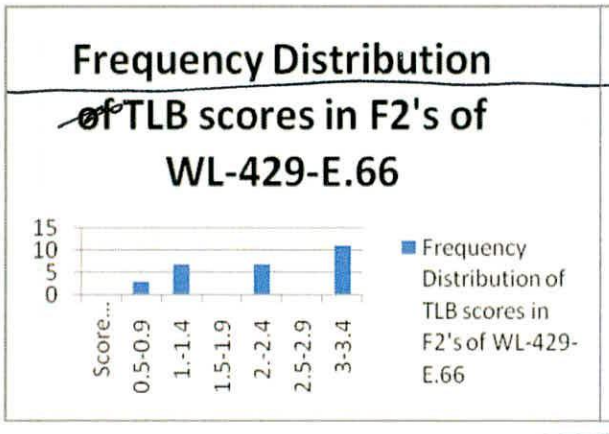

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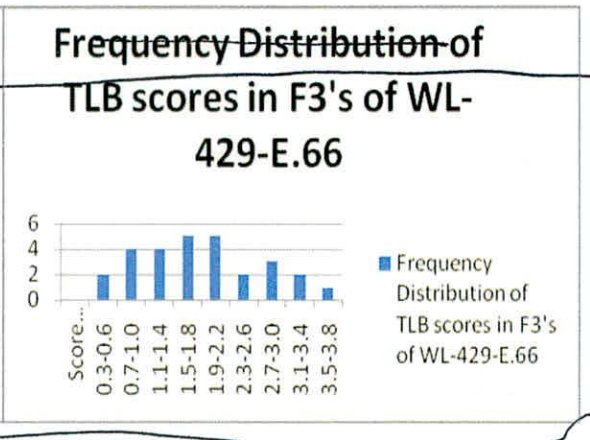

Fig.4

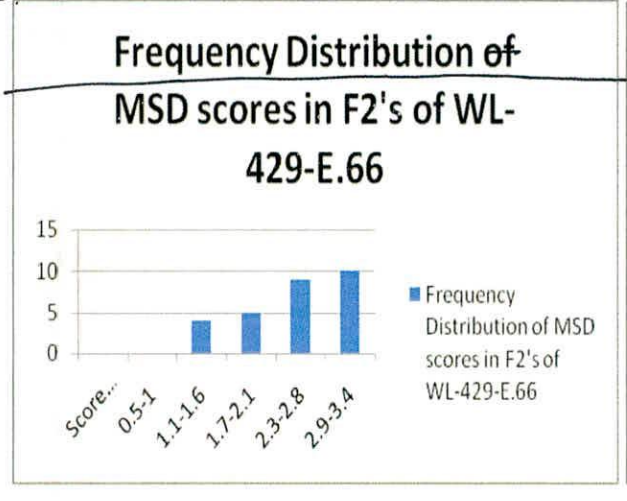

Fig.5

Fig 3

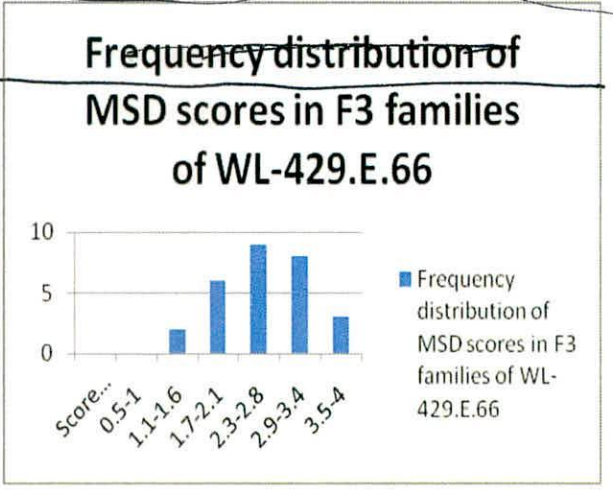

Fig.6

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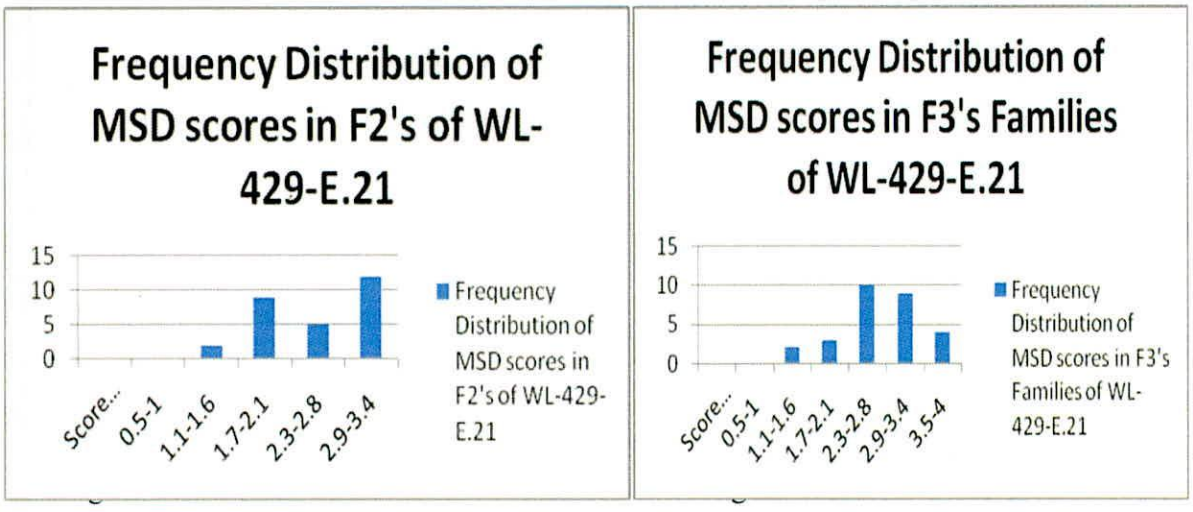
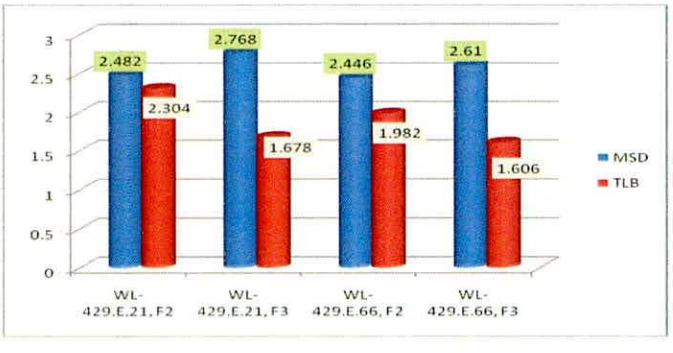


Figure 4: Showing the F_2 and F_3 mean Disease severity scores for population WL-429.E.21 and WL-429.E.66. when? which season??



5.1 Conclusion and Recommendations

must be based on your hypotheses

Moderate heritability estimates for MSD and TLB resistance observed, coupled with the characteristic of a normal distribution of severity mean scores in the two populations is an indication of a good positive step in breeding for durable resistance. The results show that the resistance is under the control of poly genes which can be relied on and does not break down easily compared to qualitative. Moderate heritability estimates means that selection can therefore fairly be based on these characters and their phenotypic expression would be a good indicator of their genotypic potentiality in early generations.

Moderate heritability estimates showed that selection for resistance to MSD and TLB at F_2 in the two

populations can be effective and would permit early elimination of susceptible lines and allow breeders to focus resources on the promising genotypes (Nyquist, 1991).

However parents and off-springs (F_2&F_3) were evaluated in different seasons and at the same location (Namulonge), so heritability estimates could have been probably higher if it was not biased by a genotype-by-location component as indicated by Nyquist, (1991) and Hanson *et al.*, (2002). So this experiment needs to be repeated under multi-location trial under the same season in Uganda to probably come out with conclusive results which are not biased by the environment. Irrespective of this, this research, has laid ground for quantitative resistance presence, its ability to be inherited and for selection to be made in early generation in the two populations.

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Appendix

a) Regression analysis for TLB on WL.429.E.66

Response variate: F3_Mean_TLB_score
Fitted terms: Constant, F2_TLB_Mean_SCORE

Summary of analysis

Source	d.f.	s.s.	m.s.	v.r.	F pr.
Regression	1	8.32	8.3173	18.25	<.001
Residual	26	11.85	0.4556		
Total	27	20.16	0.7468		

Estimates of parameters

Parameter	estimate	s.e.	t(26)	t pr.
Constant	0.456	0.298	1.53	0.137
h^2	0.580	0.136	4.27	<.001

b) Regression analysis for TLB means on WL-429.E.21

Response variate: Mean_TLB_score_F3
Fitted terms: Constant, TLB_score_F2

Summary of analysis

Source	d.f.	s.s.	m.s.	v.r.	F pr.
Regression	1	2.872	2.87249	37.47	<.001
Residual	26	1.993	0.07666		
Total	27	4.866	0.18021		

Estimates of parameters

Parameter	estimate	s.e.	t(26)	t pr.
Constant	0.622	0.180	3.45	0.002
h^2	0.4584	0.0749	6.12	<.001

c) Regression analysis for MSD means on WL-429.E.66

Response variate: F3
Fitted terms: Constant, F2

Summary of analysis

Source	d.f.	s.s.	m.s.	v.r.	F pr.
Regression	1	2.429	2.4289	8.56	0.007
Residual	26	7.378	0.2838		
Total	27	9.807	0.3632		

Estimates of parameters

Parameter	estimate	s.e.	t(26)	t pr.
Constant	1.234	0.481	2.56	0.016
h^2	0.563	0.192	2.93	0.007

d) Regression analysis for MSD on WL-429.E.21

Response variate: F3
Fitted terms: Constant, F2

Summary of analysis

Source	d.f.	s.s.	m.s.	v.r.	F pr.
Regression	1	1.752	1.7517	5.33	0.029
Residual	26	8.552	0.3289		
Total	27	10.303	0.3816		

Estimates of parameters

Parameter	estimate	s.e.	t(26)	t pr.
Constant	1.547	0.540	2.87	0.008
h^2	0.492	0.213	2.31	0.029