CHAPTER 1

INTRODUCTION AND JUSTIFICATION

Maize (*Zea mays* L.) is the most important staple crop in most Southern African countries. According to Byerlee and Eicher (1997), maize accounts for more than 50 % of all the calories consumed in Southern Africa and its demand is increasing. In Zimbabwe, most of the maize is produced by smallholder or communal farmers who cultivate about 70 % of the national arable area under maize (Eicher and Kupfuma, 1997). However, maize yield levels in these areas is very low (an average of 1.2 t/ha) (Jonga, Mariga, Chivinge, Munguri and Rupende, 1997). The rather low yield can be attributed to various constraints including water stress, poor accessibility to inputs and also low and unattractive producer prices. Of these, water stress has been singled out as the most important factor affecting maize production (Pixley, Harrington and Ransom, 1997). Production is also affected by biotic factors such as diseases, weeds and insect pests. Maize streak virus (MSV) is one of the major diseases of importance in Africa as it can cause a total crop failure on susceptible varieties during severe epidemics and is reported to cover an estimated 60 % of the total arable area in Africa (DeVries and Toenniessen, 2001).

The disease is endemic throughout the maize producing areas in Africa. It is transmitted by *Cicadulina* leaf hoppers in a non-persistent manner after a latent period of five to twelve hours (Williams, Mbiele and Nkouka, 1988). Epidemics have been reported in many parts of Africa. However, these occur irregularly both in time and location and these depend on similar transmission by vectors. In other words, it is impossible to

transmit MSV from one plant to another without the involvement of vectors. The incidence of MSV is therefore dependant on a vector population which is also influenced by rainfall, temperature and the availability of alternate hosts (Rose, 1978). Therefore, the erratic nature of MSV occurrence from year to year and also across regions in the same season is caused by this interplay of these various factors. Maize streak virus is usually severe in late planted crops and in crops planted under irrigation as these conditions favor the multiplication of vectors.

Symptoms of MSV are small cream to white flecks or spots on young leaves which then elongate and coalesce forming long chlorotic streaks along the length of the leaf. The result will be a leaf with many white stripes with green stripes in between (Ngwira and Pixley, 2000). Plants affected at an early stage become severely stunted and may die before producing grain. Yield loss due to MSV is affected by two factors that is, the level of susceptibility of the maize variety and the growth stage of the plant during the time of infection (Williams *et al*., 1988). Resistant varieties show very little or no symptoms of MSV and therefore the disease has little effect on yield in these varieties (Ngwira and Pixley, 2000). On the other hand if susceptible varieties are infected within the first week of emergence, the entire yield will be lost but if infection occurs eight weeks after emergence, yield reduction due to the disease will be negligible (Williams *et al*., 1988).

Different methods can be used to control MSV but the most effective, economically viable and environmentally friendly method of controlling the disease is through the use of resistant varieties. In Zimbabwe most varieties are tolerant to MSV. Examples of high

yielding tolerant varieties produced by the company Seed Co. (Pvt) Ltd. include SC 403, SC 407 and SC 621 (Seed Co. Production Manual, 2003). Some varieties from other organizations like Pioneer Hybrid International and CIMMYT also have tolerance. However, there is need for the production of more maize varieties that are tolerant to MSV, especially dwarf maize since all the released dwarf maize varieties currently have limited resistance to the disease.

Dwarf maize varieties were recently developed and released by the African Centre for Fertilizer Development (ACFD). Dwarf maize varieties are an important technology especially in the resource poor and drought prone rural communities of Zimbabwe. Dwarf maize is reportedly more efficient in its use of water and fertilizer as it does not produce unnecessary biomass. Thus dwarf maize varieties have a comparative advantage especially in marginalized areas of Zimbabwe. Mwale (1999) reported that dwarf maize from Zimbabwe yielded more than some Zambian tall varieties in experiments carried out in Zambia. Dwarf maize is also more tolerant to high populations as compared to tall varieties and is able to resist lodging when grown in populations that are higher than the recommended populations especially when all inputs are supplied to the optimum. Commercial varieties of dwarf maize produced by ACFD include AC 71 and AC 31. Another registered dwarf hybrid, AC 133, is on the verge of being marketed and distributed in Zimbabwe. Though these hybrids are high yielding, they all have limited resistance to MSV. Thus the challenge now is to incorporate more dependable MSV resistance into the inbred parents of these varieties.

In order to develop effective breeding plans for the incorporation of MSV resistance in dwarf maize varieties, it is imperative to know the mode of gene action for the inheritance of resistance. Various reports concerning the inheritance of MSV resistance have been written and they seem to indicate different modes of gene action. Gorter (1959) reported that resistance to maize streak virus in Peruvian Yellow x Arkill's Hickory was quantitative. However, Storey and Howland (1967) reported that mainly a single and incompletely dominant gene control resistance in Peruvian Yellow x Arkill's Hickory. The genetic study of a maize population, IB32, indicated that resistance to MSV was quantitatively inherited and that resistance was controlled by two to three gene pairs (Kim, Efron, Fajemisin and Buddenhagen, 1989). Later, after studying 500 S_1 and 93 S_2 maize lines, Rodier, Marchand and Herve (1995) suggested that resistance was controlled by major genes, controlling high to complete resistance, associated with minor genes controlling partial resistance. More recently, the results from a 10 x 10 diallel experiment between streak resistant lines from CIMMYT and streak susceptible varieties from various sources carried out at CIMMYT (Zimbabwe) showed that additive gene action was important in the inheritance of resistance to maize streak virus disease. The results showed that MSV resistance is controlled by polygenic genes and is inherited quantitatively (Nhlane and Caligari, 1997). On the contrary, Kyetere (1997) when using molecular markers, reported that MSV resistance, in progenies derived from the cross TZi4 x Hi34, was controlled by a single gene which had some level of dominance.

However, there has been no research carried out to look into the inheritance of maize streak virus resistance in dwarf maize plants. It does not necessarily follow that dwarf

maize plants will show a similar model to that exhibited by normal height plants. Therefore, there is need to analyze the inheritance of maize streak virus resistance in dwarf maize *per se*. This is crucial in breeding for streak resistance in dwarf maize as it will help breeders develop appropriate breeding plans for incorporating MSV resistance in dwarf maize. Therefore, this study seeks to provide information on the inheritance of MSV resistance in dwarf maize and its implication in breeding MSV resistant dwarf maize cultivars.

1.1 Aim

The aim of this study was to determine the mode of inheritance of resistance to MSV in dwarf maize inbred lines.

1.2 Specific Objectives

i) To determine the general combining ability (GCA) and specific combining ability (SCA) and their relative importance in the inheritance of MSV resistance in dwarf maize using progeny from a six parent half-diallel cross.

ii) To determine the heritability estimates of MSV resistance

1.3 Hypotheses

i) It was hypothesized that maize streak virus resistance is faithfully transmitted from parents to progeny in dwarf maize. That is to say, additive genetic effects are more important in the inheritance of MSV resistance in dwarf maize.

ii) MSV resistance is highly heritable in the dwarf maize.

CHAPTER 2

LITERATURE REVIEW

2.1 Maize Production

Maize (*Zea mays* L.) is the most important staple crop in most Southern African countries including Zimbabwe, Mozambique, Malawi, Zambia, South Africa and Tanzania. According to Byerlee and Eicher (1997), maize accounts for more than 50 % of all the calories consumed in Southern Africa and its demand is increasing. Maize is so important that most of the land in Southern Africa especially in the communal areas is grown to maize. For example, in Malawi, maize occupied about 80 % of the total area cultivated in the 1995 season (Byerlee and Eicher, 1997). On a global basis, maize is the third most important cereal after rice and wheat. However, in the developing countries, maize seems to be more important as the area to which it was grown increased more than that of rice and wheat in recent years. According to Hess (1997), the area grown to maize in developing nations has increased by 41 % between 1961 and 1996 as compared to that grown to rice and wheat, the later of which increased by 29 % and 36 %, respectively. Maize is also one of the most adapted cereals as it grows on a wider geographical range and variety of environments than other cereal crops (Hess, 1997).

2.1.1 Maize Production Constraints

In Zimbabwe, most of the maize is produced by smallholder or communal farmers who cultivate about 70 % of the national area under maize (Eicher and Kupfuma, 1997). Maize is important both as food crop and cash crop to these farmers. However, maize yield level in these areas is very low (an average of 1.2 t/ha). This can be attributed to

various constraints. These include water stress which is due to unreliable and poorly distributed rainfall, shortage of inputs and their unaffordability, poor soil fertility and also low and unattractive prices. Water stress has arguably been singled out as the most important constraint affecting maize production (Pixley, Harrington and Ransom, 1997). Perhaps equally important is the availability and accessibility of plant nutrients. In Zimbabwe bumper harvests are realized when adequate rainfall is coupled with access to inputs such as fertilizers by farmers.

Maize production is also affected by biotic stresses such as pests, diseases and weeds. Many diseases have been found to affect maize production in southern Africa. This is so mainly because of the mild winters which allow pests and inoculum to overwinter readily on crop debris or on alternative host species. These conditions result in disease and pest outbreaks. Major maize pests in Southern Africa include storage pests and stalk borers while major maize diseases include leaf blight caused by (*Exserohilum turcicum* Pass.), common rust (*Puccinia sorghi* Schw.), maize streak virus, grey leaf spot (*Cercospora Zea maydis* Tehon. and Daniels.), ear rots (*Fusarium spp. and Diploidia spp.*) and head smut (*Sphacelotheca reiliana* (Kühn) Clint.) (Vivek, Pixley, Odongo, Ojuguna, Imanywoha, Bigirwa and Diallo, 2004). Of these diseases, maize streak virus is the most important as it can cause a total crop failure if not dealt with especially in severe epidemics.

2.2 Maize Streak Virus (MSV)

Maize streak virus was first described as mealie-variegation in the Natal Province of South Africa at the turn of the twentieth century where it naturally occurred in maize *(Zea mays* L.) and oats *(Avena sativa*) and wild grasses (Fuller, 1901). The disease occurs only in Africa where it is widely distributed and in the adjacent Indian Ocean Islands (Ngwira and Pixley, 2000). Maize streak virus is one of the most ecologically versatile diseases of maize as it occurs in both savannah and forest ecologies and also from sea level to an elevation of 1800m (Williams *et al*., 1988). In addition to maize and oats, the disease affects other cereals including wheat (*Triticum aestivum* L.), barley (*Hordeum vulgare* L.), rye (*Secale cereale* L.), rice (*Oryza sativa* L.), sugarcane (*Saccharum officinarum* L.), teosinte, finger millet (*Eleusine coracana* L.) and many wild grasses (Shurtleff, 1986). In a study carried out in Kenya, eleven wild grass species were found to be naturally infected by maize streak virus (Njuguna, Gordon and Luoie, 1997). However, MSV is only of economic importance in maize.

2.2.1 Description of the Virus

The disease was initially thought to be a physiological disorder (Fuller, 1901) but was later found to be caused by a Gemini virus. The virions are isometric with a diameter of approximately 20 nm and they frequently occur in pairs with a size of about 20 x 30 nm (Shurtleff, 1986). The virus has a single stranded circular deoxyribonucleic acid (DNA) of about 2.7 kilobases (Rybicki, 1994 and Briddon and Markham, 1995). Various strains of the virus are known and each strain is particular to a specific host (Rose, 1978; Shurtleff, 1986).

2.2.2 Symptoms of MSV

Maize streak virus manifests initially as small, cream to white spots or flecks on young leaves. These spots appear on the lowest exposed portion of the leaf. Only new leaves develop the symptoms of maize streak virus while those leaves below the point of infection remain healthy. These spots elongate and coalesce to form chlorotic streaks running several millimeters in length along leaf veins. These then fuse laterally to give narrow broken chlorotic stripes which may extend over the entire length of the affected leaf. Eventually, the affected leaf will consist of many white stripes with green stripes in between (Ngwira and Pixley, 2000) and (Shurtleff, 1986). Chlorosis is thought to be due to failure of chloroplasts to develop in the tissue surrounding the vascular bundles and this chlorosis results in reduced photosynthesis and high respiration. As a result, plants affected by this disease at an early stage become severely stunted producing undersized and misshaped cobs, poorly filled cobs or no yield at all (Shurtleff, 1986). Symptoms may however differ depending on the isolate infecting the host plant. Some strains cause severe symptoms while others cause mild symptoms. Differences also occur in the type of streaks induced by the different strains and these streaks may have white, yellow or red pigmentation. Besides, the same strain may give different symptoms in different hosts (Shurtleff, 1986).

2.2.3 Yield Losses due to MSV

Yield loss caused by maize streak virus is directly related to the time of infection and the level of inherent resistance. Plants infected at the seedling stage without a high level of resistance will give no yield at all or are killed and various levels of yield reductions

occur if infection occurs at a later stage in the growth of the plant depending on when the infection would have occurred. According to Williams *et al*. (1988), the effect of the age of the plant at the time of infection on yield is such that when the plant is infected less than one week after emergence, all the yield is lost, when infection occurs in the third week of emergence, 50 % of the yield is lost and nearly full yield is obtained if infection occurs after the eighth week of emergence. The nutrition level of the soil also contributes to the degree of yield loss. The higher the level of plant nutrition, the less the damage caused by streak and vice versa (Williams *et al*. (1988). Though maize streak virus can cause total crop failure in cases of severe attack, losses of 1 -5 % are usually experienced in the Eastern and Southern African region (Ngwira and Pixley, 2000).

2.2.4 Transmission of MSV

Maize streak virus is vector-transmitted by five *Cicadulina* species with *Cicadulina mbila* Naude. being the most important (Shurtleff, 1980). The leafhoppers transmit the virus as they feed on maize plants. Since MSV has an obligate association with the vector, virus incidence is therefore influenced by vector population which is also influenced by climatic conditions of rainfall and temperature and also by the availability of alternative hosts. Thus the erratic nature of the occurrence of the disease from year to year, between seasons and within a particular field in any season derives from this complexity and interplay of various factors (Rose, 1978). Warm weather accelerates the development of nymphs into adults but in cool weather, oviposition and nymphal development is slow. Leafhoppers transmit the virus persistently after a latent period of about 5 – 12 hours at 30 °C. The latent period for transmission is dependant on

temperature such that it increases as temperatures get lower. The latent period represents the time required for the virus to pass from the lumen to haemocoel and then salivary glands for infection (Storey, 1928). The disease is usually severe on maize or cereal plants grown under irrigation or towards the end of the rainy season and on susceptible varieties. Spread of the virus between crops can be attributed to successive cropping of susceptible plants and also by the presence of wild grasses which facilitates the retention of both the virus and the vectors (Williams *et al*., 1988). In the warm and wet season, the fecund, that is, the long bodied form of *C. mbila* is produced. This fecund can only fly for about 10 m or less. Therefore, there are only isolated patches of infection. However, towards the end of the season when the crops mature or when there is a drought and the food supply for the insects is drying out, the stronger – flying short bodied form of *Cicadulina mbila* is produced. These then migrate to irrigated crops spreading the disease over great distances causing widespread crop failure (Rose, 1978). A major limiting factor in the epidemiology of MSV is that it is not seed transmitted and this makes it easier for its control (Jonker and Flett, 1997).

2.2.5 Available MSV Control Methods

Several methods can be used to control MSV. The virus can be controlled by the method of avoidance whereby plantings are adjusted to avoid migrating leaf hoppers from landing on young maize plants (Rose, 1978). For example, in Zimbabwe, *C. mbila* is known to be in flight from around April to September (Zitsanza[1], 2006: *Personal communication*). Therefore maize planting, especially if it is under irrigation, should not be done until after October. The vector can also be controlled using systemic insecticides

as seed dressing or applied to the planting furrow at planting. However, the most economically viable means of controlling streak epidemics is through the use of tolerant varieties. These can allow infestation but are able to perform well despite the infection or they may exhibit lower disease incidence (tolremicity) particularly due to insect resistance (Bosque-Perez and Buddenhagen, 1997). In Zimbabwe, through conventional breeding methods, most varieties are now tolerant to maize streak virus. Examples of high yielding tolerant varieties produced by Seed Co. (Pvt) Ltd. include SC 403, SC 407, and SC 621 (Seed Co. Production Manual, 2003). More recently, the first transgenic MSV tolerant maize have been produced in South Africa using the mutated MSV replication associated gene (Shephered, Mangwende, Martin, Bezuidenhout, Kloppers, Carolissen, Manjane, Rybicki and Thompson, 2007). Biotechnology has also been exploited to produce transgenic maize that is resistant to MSV. The transgenic maize was reported to delay symptom development, decrease symptom severity and increase survival rates after infestation by MSV. However, there is need for the production of more maize varieties tolerant to maize streak virus especially for dwarf maize varieties, which presently have limited resistance to the disease.

2.3 Dwarf Maize

2.3.1 Brief Morphology of Dwarf Maize

Dwarf maize is a type of maize that is much shorter than normal varieties because of genetic factors. Dwarf maize plants have shortened internodes and their leaves are brought close together to form a rosette. Three types of dwarfness have been reported in maize depending on the presence or absence of certain modifier genes. There is what can

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be called severe dwarfness, medium dwarfness and mild dwarfness. Severe dwarfness is whereby the plants have an extremely short internode length. This is not very desirable as the cobs are also very small, thus the yield is usually subdued. In medium dwarfness, the height of the plant is the average of the severe dwarfs and mild dwarfs. Mild dwarfs are almost as short as normal maize varieties. Medium and mild dwarfs have good vigor and produce the higher yields when compared to the severe dwarfs. Dwarfing is also sometimes observed to occur below the cob, above the cob or both below and above the cob. (Mutengwa[2], 2006: *Personal communication*). In Zimbabwe, dwarf maize varieties were first released into the market by the African Centre for Fertilizer Development (ACFD) in 1998.

2.3.2 How Zimbabwean Dwarf Maize was developed

Dwarf maize is homozygous recessive for the *brachytic-2* gene which causes dwarfness. It is reported that the trait for dwarfness is simply inherited and several dwarfing genes have been discovered including *d3*, *b-2* and *di* (Sangoi and Salvador, 1997). In Zimbabwe, the *brachytic-2* gene is used by ACFD to dwarf their materials (Mutengwa, 2006: *Personal Communication*). The source of the dwarfing gene used in the ACFD breeding programme was the super dwarf hybrid developed in Mexico at the University of Saltillo using the concept of crop ideotypes. This super dwarf was reported to have an optimum density of 130 000 plants per hectare and a yield of 21 t / ha (Muchena[3]: *Personal Communication, 2007*). In the programme, inbreds developed from the super dwarf hybrid were backcrossed to selected locally adapted inbreds and other exotic

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[3] Maize Breeder and Managing Director, ACFD

germplasm to produce a series of dwarfed inbreds from which a series of dwarf maize hybrids were produced.

2.3.3 Advantages of Dwarf Maize

2.3.3.1 Water and Fertiliser Use Efficiency

Of all the genes deployed in plant breeding programs, those for dwarf stature in cereals are among those that had a significant impact on world food production (Rasmusson, 1984). Dwarf maize is reportedly more efficient in its use of water and fertilizer as it does not produce unnecessary biomass. By reducing the stem height, assimilate is saved and used in reproductive parts therefore there is greater ear development and also increased grain setting. This is important especially in marginalized areas (Agro-ecological regions III and IV) of Zimbabwe. These areas usually receive inadequate and poorly distributed rainfall. In addition, even the areas of the country that receive good rainfall are often visited by mid-season droughts. Besides the rainfall problem, the soils in marginalised areas are usually quite infertile and most of the people in these areas cannot afford to buy inputs like fertilizers which may be in very short supply. Dwarf maize has proved to be better adapted to these conditions and often produce superior yield (Muchena, 2006: *Personal Communication*). Yamaguchi (1974) reported that there is a significant positive correlation between grain yield of maize and either reduced plant height or ear height. Moshi (1982) also reported that shorter selections had higher yields under conditions of low nitrogen supply and poor weed control. These nutrient and water use efficiencies have been attributed to the reduced plant biomass in the dwarf maize plant whilst retaining the size of the harvested part. Hurd (1974) have reported that the widely grown

semi-dwarf winter wheat have longer and more extensive root systems than older tall varieties. Hence, this difference in the root system has been thought to contribute to the water and nutrient use efficiencies. However, Tongoona, Muchena and Hendrikz (1984), working with dwarfing genes in pearl millet, reported that while dwarfing genes induced a relatively large rooting system in some backgrounds, it also induced a reduction in the rooting systems in other backgrounds. Chinhema (1997) also reported that the dwarfing gene had no effects on root depth but pleitropically induced a finer and more fibrous root system in maize.

2.3.3.2 Ability to withstand High Population Densities

Besides its water and nutrient use efficiencies, dwarf maize has a better standability and can withstand high plant populations under favorable edaphic and climatic conditions (Sangoi and Salvador, 1997). Thus dwarf maize can be used by commercial farmers to increase plant populations above those recommended since they can provide the optimum conditions for the maize. Much of the improvement in the US Corn Belt has been as a result of the ability of improved genotypes to respond to higher plant densities without lodging while maintaining or reducing the level of barrenness (Russell, 1974). This is likely to give a better yield for the same land size. Dwarf maize has also been reported to be better yielding than normal maize varieties (Mwale, 1999).

Commercial dwarf maize hybrid varieties released by ACFD include AC 71 and AC 31. These hybrids are good and high yielding but they all have limited resistance to maize

streak virus. Thus the challenge is to incorporate maize streak virus resistance into these hybrids. This will greatly enhance their performance.

2.4 Mechanisms of Resistance to Viruses

Two major categories of resistance to plant viruses have been alluded to; non-host resistance and host resistance (Fraser, 1990). Non-host resistance which can also be referred to as immunity, involves situations where all genotypes within a plant species fail to be infected by a particular virus, that is to say, there is no genetic polymorphism for susceptibility to the virus in that taxon. However, the mechanisms of such a resistance are largely unknown but likely to be diverse. On the other hand, host resistance occurs where genetic polymorphism for susceptibility is observed in the plant taxon, that is to say, some genotypes show heritable resistance to a particular virus while some genotypes in the gene pool are susceptible. Genotypes that exhibit this type of resistance are said to be either resistant or tolerant. Resistant genotypes are those that are resistant to the virus in such a way that the multiplication and spread of the virus through the plant is demonstrably restricted relative to susceptible hosts. In such plants, even under disease pressure, disease symptoms are highly localized or are not evident at all (Kang, Yeam and Jahn, 2005). Tolerant varieties are those that are resistant to disease symptoms but may be fully susceptible to the virus. In this case, the virus may move through the host in a manner that cannot be distinguished from that in the susceptible host but disease symptoms are not observed or if they are observed, do not cause economic loss in yield (Kang *et al.,* 2005). Where MSV is concerned, maize mostly exhibit tolerance to the

disease. In this paper, the general term resistant was used to refer to those genotypes that strictly exhibited high levels of tolerance.

2.5 Sources of Resistance to Maize Streak Virus

Various sources of tolerance to maize streak virus are locally available at CIMMYT. In the Eastern Africa regional maize nursery, more than 50 lines with a high level of tolerance to maize streak virus had been identified by 1998 (Ngwira and Pixley, 2000). The African Centre for Fertiliser Development has also identified one source of streak tolerance in a family of inbreds of Mexican origin (Muchena, 2006: *Personal Communication*). Completely resistant lines have also been reported in Reunion for example DZ 11. However, such sources of resistance are few in the world (Dintinger, Pernet, Rodier, Reynaud and Marchand, 1997).

2.6 Inheritance of Maize Streak Virus Resistance.

Various reports concerning the inheritance of maize streak virus have been given. Gorter (1959) reported that resistance to maize streak virus in Peruvian Yellow x Arkill's Hickory was quantitative. However, Storey and Howland (1967) reported that resistance in progeny derived from the same cross, that is Peruvian Yellow x Arkill's Hickory was found to be controlled mainly by a single and incompletely dominant gene. The genetic study of a maize population, IB32, indicated that resistance to MSV was quantitatively inherited (mainly additively) and that resistance was controlled by two to three gene pairs (Kim *et al*., 1989). Later, after studying 500 S_1 and 93 S_2 maize lines, Rodier *et al*. (1995) suggested that there were two different systems controlling resistance; one involving loci

with major genes controlling complete resistance, the other with minor genes controlling partial resistance. The results from a 10 x 10 diallel experiment between streak resistant lines from CIMMYT and streak susceptible varieties from various sources carried out at CIMMYT (Zimbabwe) showed that additive gene action was important in the inheritance of maize streak virus disease. The results showed that maize streak virus is controlled by polygenic genes and is inherited quantitatively (Nhlane and Caligari, 1997). These results suggest that the resistance can be effective and potentially durable by serving as a buffer to a sudden shift in pathogens. Breeders should also be able to select genotypes with a high level of resistance and by simple recurrent selection or backcrossing; they should be able to concentrate the frequency of desirable genes (Nhlane and Caligari, 1997).

These conflicting reports in terms of the mode of gene action for the inheritance of MSV resistance may have been caused by different factors including use of different leafhopper infestation methods (natural versus controlled), use of different MSV isolates, different rating scales to measure host reaction, use of different statistical analyses and also illusions in visual assessment that may occur when measuring host response (Kyetere, 1997). When Kyetere (1997) analysed 87 recombinant inbred lines (RILs) from two parents having divergent origins that is Hi34 (susceptible) and TZi4 using restriction fragment length polymorphisms (RFLPs), he reported that resistance was controlled by a single gene showing some level of dominance. However, there has been relatively little or no research carried out to look into the inheritance of maize streak virus resistance in dwarf maize plants. It does not necessarily follow that dwarf maize plants will show a similar model to that exhibited by normal plants since dwarf maize plants are quite

different to maize plants with normal height. Therefore, there is need to analyze the inheritance of maize streak virus in dwarf maize *per se*. This may prove to be quite useful in facilitating breeding for streak resistance in dwarf maize.

2.7 Mating Designs Used in Genetic Analysis

Different mating designs can be used for genetic analysis of maize populations. For most of these analyses, the following assumptions have to be made (Hallauer and Miranda, 1981):

i) There is no epistasis.

ii) The population is "Mendelian" and is in linkage equilibrium.

iii) Individuals are selected at random from the population that is for random models.

iv) Individuals form diploids during meiosis.

v) There are no maternal effects.

Mating designs most frequently used include; the biparental mating design, North Carolina design 1 (NC 1), North Carolina design 11 (NC 11), North Carolina design 111 (NC 111) and the diallel design.

2.7.1 Biparental Mating Design

This is one of the simplest mating designs and it was introduced by Mather (1949). It is a random model which involves crossing pairs of plants taken at random from the population. That is to say from n parents, $n_{/2}$ crosses are made. This design allows the estimation of variance components. It provides information needed to determine if

significant genetic variation is present in a population but does not provide information to determine the type of genetic variance.

2.7.2 North Carolina Design 1 (NC 1)

This method was first introduced by Comstock and Robinson (1948). It is the second most commonly used mating design after the diallel (Hallauer and Miranda, 1981). This design is appropriate for estimating genetic components of variance for a reference population. Parents used for this design are selected randomly from the reference population. Plants are selected randomly from the population and these are designated male. Each of these is mated to a set of females which are also selected randomly from the population. The male plants are different from the female plants and are each mated to different set of females. This design is also called the nested design because the females are nested within the males. This design also allows for the estimation of the different components responsible for genetic variation and whether it is important in a population or not.

2.7.3 North Carolina Design 11 (NC 11)

This design was also introduced by Comstock and Robinson (1948). It is also called the factorial design. In this design, the parents are divided into males and females. Each male is crossed to every female. Parents are divided due to certain characteristics that can separate them for example disease susceptibility and resistance. This design gives similar information to that given by the diallel. It allows for the estimation of genetic components of variance of a reference population. It also gives the GCA and SCA

estimates for both males and females. This design is ideal when wanting to include a large number of parents from the population while using limited resources.

2.7.4 Diallel Analysis

Most inheritance analysis is done using diallel analysis. A diallel system is a system that involves all possible crosses among a group of parents (Viana, Cruz and Cardoso, 1999). The crosses are usually between two parental groups for example, inbred lines susceptible to maize streak virus and inbred lines resistant to maize streak virus (fixed model). Diallels can also be used to study genetic components of variance for a reference population that is if the parental lines were selected at random from the reference population (random model). However, there is limited use of the random model since the number of crosses increases rapidly as the number or parents increase. In such cases NC 11 is more appropriate. A diallel analysis is used to study polygenic systems that control quantitative traits. They allow the estimation of genetic and non-genetic component of variation. Diallel analysis can also show us the relative importance of additive and non-additive variation in determining the inheritance of a trait. It also allows the estimation of both the general and specific combining abilities of lines involved in the diallel. A diallel analysis can use data from the F_1 and the parents or from the F_1, F_2 and the parents. Diallel analysis can be done using methods set out by Griffings (1956). The diallel design can be performed using the following four methods:

i) A full diallel whereby every other cross is made,

ii) Without reciprocals but with parents included,

iii) Without parents but with reciprocals, and

iv) Without parents and reciprocals, using F_1 only.

The diallel is very useful especially regarding a fixed model whereby the parents are the population of inference. This is because unlike the other designs, it allows crosses between all parents to be made. This allows the calculations of the GCA and SCA effects to be more accurate. Since this study only considered six parents, a fixed model diallel analysis was used.

2.8 Evaluation of Resistance to Diseases

Many diseases cause visible symptoms on maize plants. These usually border on the reduction in yield quality or quantity. In evaluating disease resistance, the objective is to find affordable techniques that maximize expression of genetic differences and enable scientists to make accurate decisions when selecting lines, hybrids or varieties to be used for research or by farmers. Various methods can be used for disease inoculation. These include natural inoculation and artificial inoculation. For disease scoring, visual assessments can be used. However, because this method seems to be subjective, a more accurate method which involves the use of molecular markers can also be employed.

The following are the guidelines for disease evaluation that was used by the collaborators of the maize Regional Nursery Program (REGNUR) of CIMMYT (Ngwira and Pixley, 2000).

2.8.1 Disease Inoculation

- Has to be artificial to allow for uniform disease pressure and thus reliable evaluation of resistance.

- Inoculation has to be as early as possible in maize growth for the methodology in use.
- Growing young leaf and plant tissues are most appropriate for inoculation.
- Inoculation should be repeated at least once

2.8.2 How to Score Disease Symptoms.

- The purpose of scoring is to adequately estimate relative resistance of the plants.
- A scale of 1 – 5 or 1 – 9 is used. Where;
    - 5 (or 9 if using a scale of 1 – 9) is the best score, and it includes plants with no or very few symptoms.
    - 1 is the worst score and it includes heavily diseased plants.
- A method of scoring disease symptoms which is based on that adopted by the collaborators at REGNUR is shown in Table 2.1.

Table 2.1: Disease symptom scoring system

% Diseased tissue	Score	
	(1 to 5)	**(1 to 9)**
0 - 2	5	9
3 – 5	4.5	8
6 – 10	4	7
11 – 25	3.5	6
26 – 44	3	5
45 – 60	2.5	4
61 – 75	2	3
76 – 80	1.5	2
81 – 100	1	1

(From Ngwira and Pixley, 2000).

2.8.3 When to Score for Disease Symptoms

- Disease symptoms should be scored at least twice. This should be done two weeks after infestation by the pathogen and during flowering in maize. Scoring two weeks after infestation allows one to assess whether the infestation was successful or not. Scoring at the flowering period allows one to assess the full impact of the pathogen since at this time the plant will have attained full vegetative growth.
- Disease symptom scores are only recorded if there are differences among entries

These guidelines are quite useful and inexpensive and thus were made use of in this study. However, there are limitations to these guidelines especially because of the fact that disease scoring is based on visual assessment. Kyetere (1997) used molecular markers linked to MSV resistance when evaluating resistance of maize to MSV. Though this method is more accurate, it is quite expensive and hence the visual assessments tend to be more commonly used.

CHAPTER 3

MATERIALS AND METHODS

3.1 Materials used

Seeds from a six parent diallel cross (F_1s only without parents and reciprocals) that was conducted at ACFD in the 2004/05 summer season were planted at the University of Zimbabwe farm in Harare in the 2006/07 summer season. Table 3.1 shows the six parents' code names and their disease reaction.

Table 3.1: Description of the dwarf maize inbred lines used in the experiment.

Inbred line	Disease Reaction
21	Susceptible
32	Susceptible
51	Susceptible
72	Susceptible
CML1	Resistant
CML 7	Resistant

3.2 Experimental Design

The seeds from the 15 crosses (F_1s only) were grown in a randomized complete block design with three replications or blocks. Each plot consisted of two three-metre rows spaced 0.75 m between rows and 0.3 m between planting hills. A distance of 0.5 m was allowed between plots while the blocks were separated by a distance of 1.0 m. Five-metre border rows planted to a commercial hybrid (AC 71) with some resistance to streak were

allowed for at each side to keep the MSV from spreading. This gave a plot size of 3 m^2 and a block size of 70 m^2. The total size of the land required for the experiment was 820 m^2.

3.3 General Agronomy

Three seeds were planted per station and these were thinned to one plant per station after two weeks after emergence. A basal compound fertilizer (7% N: 14 % P_2O_5: 7 % K_2O) was applied at planting at a rate of 300 kg/ha. A topdressing ammonium nitrate (34.5 % N) was also applied at a rate of 250 kg/ha five to six weeks after emergence. Weeds were controlled by hand hoeing when necessary.

3.4 Infestation and Scoring for MSV

Plants were infested artificially with viliferous *C. mbila* nymphs from CIMMYT 10 to 14 days after emergence. Due to shortage of insects, one out of every two plants was infested instead of the usual two insects per plant. Disease rating was done two weeks after infestation and also at flowering, that is, 8 to 10 weeks after emergence. Disease rating was based on an individual whole plant basis using a modification of the disease scoring system employed by REGNUR collaborators (Ngwira and Pixley, 2000). This means that each plant was rated after which the mean score for each plot was obtained. The analysis was done using the mean plot score. Genotypes scored for maize streak virus with a score of 1 to 3.4 were regarded as susceptible, 3.5 to 6.4 as intermediate and 6.5 to 9 as resistant (Nhlane and Caligari, 1997).

3.5 Other Agronomic Traits Recorded

Apart from MSV ratings, other agronomic traits that were recorded include;

i) Plant height (cm)
ii) Ear height (cm)
iii) Ear position
iv) Ear length (cm)
v) Ear diameter (cm)
vi) Shelling percentage
vii) Number of kernel rows per ear
viii) Number of kernels per row
ix) 100 seed grain weight (g)

3.6 Statistical and Genetic Analysis

The data for yield and the other agronomic traits were analyzed using by the REML method using Field book statistical package (Banziger and Barreto, 1999). The preliminary analysis of variance for MSV scores at flowering was done manually using statistical tables (Table 3.2). The same was also done for the combining ability analysis (Table 3.3).

Table 3.2: Form of analysis of variance (ANOVA) for crosses and replications

SOURCE	DF	MS
Replication	r -1= 2	
Crosses	v -1= 14	
Error	[1/2n (n-1)-1] r-1= 28	
Total		

Where r = number of replicates; n = number of parental lines; v = number of crosses

Table 3.3: The partitioning of mean squares for combining ability analysis

Source	**DF**	**MS**	E.M.S (variance components)
GCA	n-1 = 5	M1	(M1 – M3)/ n-2 = σ^2 GCA
SCA	1/2n(n-3) = 9	M2	M2 –M3 = σ^2 SCA
Error	[1/2n (n-1)-1] r-1= 28	M3	M3 = σ^2 error

Where r = number of replicates; n = number of parental lines;
M1 = mean square due to GCA; M2 = mean square due to SCA; M3 = error mean square.

The formulae used to calculate the sums of squares due to GCA and SCA and the error mean square were as described by Singh and Chaudhary (1985). This was based on Model 1 (GCA and SCA fixed), Method 4 (F_1 only) (Griffings, 1956). General and specific combining ability effects were estimated using Model 1 (GCA and SCA fixed), Method 4 (F_1 only) (Griffings, 1956). The formulas used to calculate the general and specific combining abilities were as described by Singh and Chaudhary (1985) (Appendix 1). The basic model to be used in the analysis of variance was;

$$Y_{ijk} = \mu + rep_k + g_i + g_j + s_{ij} + e_{ijk}$$

Where Y_{ijk} = mean value of the k^{th} plot of the cross between the i^{th} and j^{th} parent;

μ = overall mean

rep_k = replication or block effects

g_i and g_j = gca effects of the i^{th} and j^{th} parent respectively

s_{ij} = sca effects of the cross between the i^{th} and j^{th} parent and

e_{ijk} = error peculiar to the ijk^{th} observation

The narrow sense heritability was calculated as $h^2 = \frac{4\,\sigma^2\,GCA}{4\,\sigma^2\,GCA + 4\,\sigma^2\,SCA + \sigma^2\,error}$

The relative importance of additive and non-additive gene effects was determined using Baker's (1978) formula -: $\frac{2\,\sigma^2\,GCA}{2\,\sigma^2\,GCA + \sigma^2\,SCA}$

This formula can be applied to both fixed and random effects models.

CHAPTER 4

RESULTS

4.1 MSV Scores

The mean MSV ratings at flowering time are presented in Table 4.1.

Table 4.1: Mean MSV Scores of the fifteen hybrids in a six parent dwarf maize diallel.

Line	21	32	51	72	CML1	CML7
21		1.0	1.0	1.8	7.0	6.7
32			2.0	1.7	5.2	5.0
51				1.0	5.2	4.8
72					6.2	5.0
CML1						6.5
CML7						

1 = susceptible; 9 = resistant p<0.05 LSD (0.05) = 1.3

The mean MSV ratings for F_1 hybrids ranged from 1.0 for hybrids 32 x 21, 21 x 51 and 51 x 72 to 7.0 for hybrid 21 x CML 1 where high values indicate resistance.

The results showed that there were generally three main hybrid groups when classified according to their response to MSV infestation that is susceptible hybrids, hybrids with intermediate resistance and resistant hybrids. Examples of plants taken from experimental plots showing these different responses to MSV are illustrated Figure 4.1. The first picture (a) was taken from a plot containing hybrid 21 x 51 and as can be seen, the plants were susceptible and severely devastated by the disease.

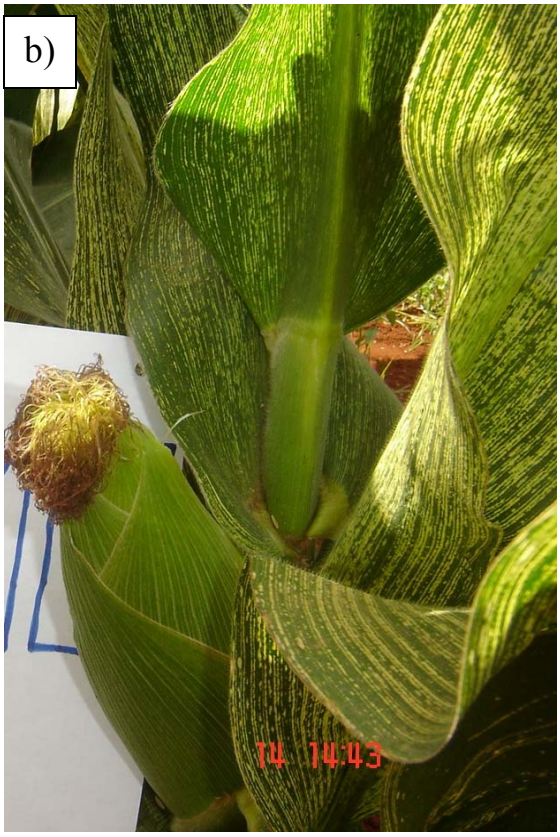

Figure 4.1: Illustration of the three hybrid classes according to their response to MSV.

a) **susceptible,** score of 1 to 3.4
b) **intermediate resistance**, score of 3.5 to 6.4
c) **resistant,** score of 6.5 to 9.0

The second picture (b), was taken from a plot grown to hybrid 32 x CML7, the plants were severely streaked but were still quite vigorous. These were classified as having intermediate resistance. The last picture (c) was taken from a plot planted to 21 x CML7. The plants only had mild streaks and these were classified as resistant.

There were six susceptible hybrids; six hybrids that had intermediate resistance and three resistant hybrids (score of 6.5 – 9.0). These are shown in Table 4.2

Table 4.2: Reaction of the 15 F_1 Hybrids to MSV.

Line	21	32	51	72	CML1	CML7
21		**S**	**S**	**S**	**R**	**R**
32			**S**	**S**	**I**	**I**
51				**S**	**I**	**I**
72					**I**	**I**
CML1						**R**
CML7						

1.0 - 3.4 = susceptible **(S)**
3.5 – 6.4 = intermediate **(I)**
6.5 – 9.0 = resistant **(R)**

All hybrids generated from crosses between susceptible lines were susceptible. These are hybrids 21 x 72, 32 x 21, 21 x 51, 51 x 72, 32 x 72 and 32 x 51. The mean MSV score for susceptible hybrids was 1.4. Resistant hybrids were generated from the cross between the two resistant lines and those of the resistant lines with susceptible parent 21. The mean MSV score for the resistant hybrids was 6.7. All the hybrids with intermediate resistance

were generated from crosses between resistant lines and susceptible lines except line 21. The mean MSV score for hybrids with intermediate resistance was 5.2 (Figure 4.2).

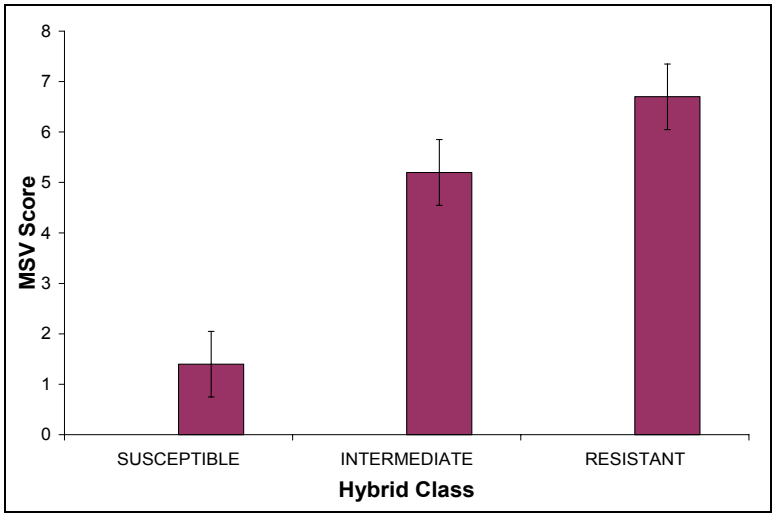

Figure 4.2: Mean MSV scores for the three hybrid classes based on their response to MSV

4.2 Combining Ability Analysis

Table 4.3 shows the mean squares for replications and crosses. There was significant variation ($P< 0.05$) among crosses for MSV scores.

Table 4.3: Mean squares from analysis of variance (ANOVA) for MSV Scores at flowering based on diallel analysis model 1, method 4

Source	DF	MS
Replications	2	0.017 ns
Crosses	14	15.774*
Error	28	0.201

*: Significant at 5 % level of probability
ns: Not significant at 5 % level of probability

These differences were due to both GCA and SCA effects since these were both significant ($p< 0.05$) (Table 4.4).

Table 4.4: Mean squares and variance components from combining ability analysis based on diallel analysis model 1, method 4.

Source	DF	MS	E.(M.S)
GCA	5	12.64*	2.86
SCA	9	1.21*	1.14
Error	28	0.07	

*: Significant at 5 % level of probability.

However, the total variation for the crosses was attributed more to differences in the GCAs among parents than SCA effects. The GCA variance when compared to the SCA variance gave a ratio of 2.51. The GCA variance was about three times larger than the SCA variance. Also the relative importance of GCA and SCA, calculated according to Baker (1978), gave a value of 0.83 for MSV.

4.2.1 GCAs of the Six Parents and SCAs of the Fifteen Crosses

Estimates of GCA effects are presented in (Table 4.5) on the diagonals. These were compared to determine the relative contribution of individual parents to MSV resistance as measured by the MSV scores. A negative GCA indicates a contribution towards greater MSV susceptibility while a positive GCA indicates a contribution towards greater resistance.

Table 4.5: Estimates of General Combining Ability (GCA) (diagonal) for the six parents and Specific Combining Ability (SCA) (below diagonal) for the 15 hybrids in a six parent dwarf maize diallel

Line	21	32	51	72	CML1	CML7
21	**-0.63**					
32	1.61	**-1.28**				
51	1.76	3.19	**-1.51**			
72	2.28	2.61	2.06	**-1.09**		
CML1	5.08	4.06	3.86	4.57	**2.52**	
CML7	5.13	5.86	3.81	3.73	2.83	**1.99**

The most tolerant plant or parent was CML1 with a GCA effect of 2.52 while the most susceptible parent was parent 51 with a GCA effect of -1.51. Generally, resistant parents had positive GCA effects while susceptible parents had negative GCA effects.

Specific Combining Ability effects are also presented in Table 4.6 (below the diagonal). All the SCA effects were in a positive direction for these particular crosses. The crosses 21 x CML1, 21 x CML7 and 32 x CML7 had the highest SCAs of 5.08, 5.13 and 5.86 respectively.

4.3 Heritability estimates

The narrow sense heritability estimate for MSV at flowering was 71 % and was relatively high. This high heritability estimate further supports the additivity which was obtained from the diallel analysis.

4.4 Grain yield

The mean grain yield data for all the hybrids are shown in Table 4.6.

Table 4.6: Grain yield (t / ha) for the 15 hybrids

Line	21	32	51	72	CML1	CML7
21						
32	0.56					
51	0.51	0.21				
72	1.56	0.95	0.38			
CML1	7.01	7.07	9.01	7.12		
CML7	8.34	7.70	5.79	9.25	9.00	

LSD (0.05) = 2.7

There was a significant difference ($P<0.05$) among the crosses for yield. The yields ranged from 0.21 t / ha for hybrid 32 x 51 to 9.25 t / ha for hybrid 72 x CML7.
Hybrid 32 x 51, which had the lowest yield, was also generated from the two parents with the lowest GCAs for MSV resistance (Table 4.6).

The grain yield means of the three classes of hybrids based on their reaction to MSV is shown in Figure 4.3. The resistant hybrids had the highest grain yield of 8.17 t / ha followed by those that had intermediate resistance with a grain yield of 7.66 t /ha and the susceptible hybrids had the least yield at an average of 0.69 t / ha.

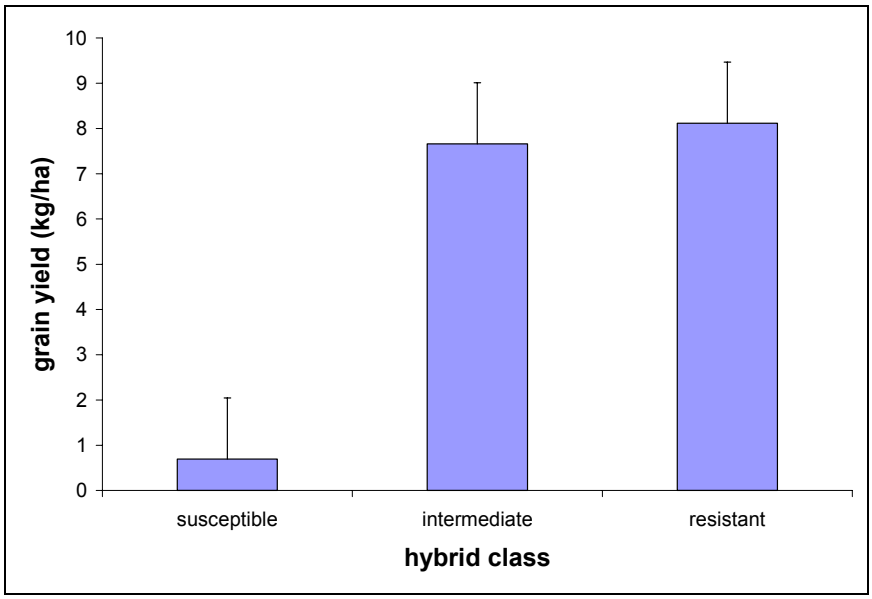

Figure 4.3: Mean grain yields (t/ha) for the three hybrid classes based on their reaction to MSV

The mean yield for hybrids that were classified as resistant to MSV was not significantly different ($P<0.05$) from that of the hybrids that were classified as having intermediate resistance. However, the means of both classes (intermediate and resistant) were significantly different from that of the susceptible hybrids.

4.5 **Other Agronomic Traits**

Results on data for other agronomic traits were not included in this discussion because these are of general interest but were beyond the scope of this study. These are however presented in Appendix 2.

CHAPTER 5
DISCUSSION

5.1 Introduction

The MSV scores are explained in terms of their relationship to the mode of gene action responsible for MSV resistance. The same is also done for the GCA and SCA effects, variance ratio and heritability. The mode of inheritance of resistance to MSV is also discussed in terms of its implications on breeding systems that can be used in breeding for MSV resistance. The GCAs of the inbred lines are discussed in such a way that the best parent for MSV resistance is identified and the reasons for it being so are explained. Yield of the fifteen hybrids is related to their response to MSV and the deviations from what is expected are explained. Results from this study are also compared to those of studies previously done. It is important to note that, since a fixed effects model was used, the findings of this study are interpreted with reference only to this particular group of experimental hybrids and should not apply to dwarf maize in general.

5.2 MSV Scores

All the ranges of MSV scores were represented in the hybrids. Six hybrids were susceptible, six of intermediate resistance and three were tolerant. This suggests that mainly additive gene action was responsible for the inheritance of resistance to MSV in the dwarf maize lines used. Significant differences ($P < 0.05$) that were observed among crosses for MSV scores suggested that there was genetic variation present in the hybrids that were evaluated. This therefore necessitated the combining ability analysis to determine the nature of the genetic variation.

5.3 Combining Ability Analysis

The analysis of variance for GCA and SCA effects showed that the significant variation ($P<0.05$) found among crosses for MSV scores can be attributed both to GCA and SCA effects. The significant GCA and SCA effects suggest that both additive and non additive gene effects are involved in the transmission of MSV resistance in the dwarf maize lines. Nhlane and Caligari (1997) also reported significant GCA and SCA effects among F_1 crosses in a diallel analysis involving ten tall maize inbred lines of which five were resistant and the other five were susceptible to MSV.

However, variation due to GCA effects was shown to be more important than that caused by SCA effects as shown by the value of the relative importance of GCA to SCA calculated according to the formula given by Baker (1978). The importance of the GCA effects over the SCA effects was also confirmed by the variance ratio of 2.83. Similar results were reported by Nhlane and Caligari (1997) where the GCA effects were found to be more important than SCA effects in a similar study focusing on tall maize inbred lines. This suggests that additive portion of genetic variance is more important than dominance or epistatic variances in the inheritance of resistance to MSV in the dwarf maize inbred lines used. This means that MSV resistance is controlled quantitatively by several genes each having a small effect. The quantitative nature of the inheritance of MSV resistance also suggests that the resistance is most likely to be effective, stable and will be able to withstand sudden shifts in the composition of the pathogen. The stability of the resistance to MSV in resistant tall maize cultivars was demonstrated by Mawere, Vincent and Pixley (2006) when they evaluated the resistance of four MSV resistant

maize inbred lines to twenty MSV isolates collected from the wild over a period of two years from locations across Zimbabwe. They reported that, though there were significant differences in the final maize streak symptom scores induced by the isolates, they only differed by not more 20 %. The additivity also suggests that the variation obtained for the crosses can be fixed and selected for by breeding.

On the other hand, the fact that SCA effects were significant, even though they were less important as compared to GCA effects means that some non additive gene effects like dominance and epistasis were involved in the control of the trait in these inbred lines. Therefore it is important to do tests for hybrid combinations when selecting a hybrid for MSV resistance.

A comparison of the GCAs of the six parents showed that line CML1 had the highest GCA of 2.52. This means that the mean performance of the crosses involving CML1 was the best when compared to the mean performance of the crosses involving the other inbred lines. In other words CML1 seemed to transmit the greatest MSV resistance to its progeny when compared to the other inbred lines. Therefore, CML1 can be used as a parent where MSV resistance is required, for example in backcrossing schemes to convert MSV susceptible lines to MSV resistant lines. This is, however, not withstanding other agronomic traits. On the other hand, line 51 had the lowest GCA meaning that it produced progenies with the greatest susceptibility to MSV. This also suggests that it had the least genetic factors that contribute to MSV resistance as compared to the other lines

used in the experiment. In general terms, the GCAs for susceptible lines (21, 32, 51and 72) were negative while those for resistant parents (CML1 and CML7) were positive.

The SCAs of all the crosses were in a positive direction for MSV tolerance. This was because the crossing resulted in more efficient MSV tolerance as compared to the mean performance of the single parents in all crosses. The hybrids seemed to show a level of heterosis for MSV resistance. Hybrids 21 x CML1, 21 x CML7 and 32 x CML7 had the highest SCAs indicating that these experimental hybrids were superior crosses in terms of MSV resistance. This suggests that there is need to test for combining ability when developing hybrids resistant to MSV in order to come up with the best crosses.

The narrow sense heritability estimate for MSV tolerance at flowering of 71 % was quite high. This high heritability estimate further support the fact that additive gene effects were the most important in the inheritance of MSV tolerance in these inbred lines. The high heritability estimate also suggests that, though the inheritance is quantitative, it is only controlled by a few genes. Therefore, the variation obtained for crosses can be easily fixed and selected for through breeding. It should also be easy to implement backcrossing methods to confer resistance to susceptible lines. The high heritability also confirms that the visual scoring system for MSV is effective and will enable breeders to register some genetic progress.

These results agree with those of Nhlane and Caligari (1997) and Kim *et al.* (1989) who reported high heritability estimates for MSV tolerance. They also reported that resistance

to MSV was controlled quantitatively by a small number of genes. This, however, differs from the findings of Kyetere, Ming, McMullen, Pratt, Brewbaker and Musket (1999) and Storey and Howard (1967) who reported the presence of a single major gene controlling resistance. Rodier *et al* (1995) however, reported that MSV resistance in population CVR3-C3 was controlled by two different genetic systems; one involving loci with major genes controlling complete resistance, the other with minor genes controlling partial resistance.

5.4 Grain Yield

The significant variation ($P<0.05$) among crosses for yield suggest that MSV has a significant effect on the yielding ability of dwarf maize. However, the effect of MSV on yield was also confounded by the differences in the combining abilities of these inbred lines for yield. There also was a significant variation among the mean yields of the three classes of hybrids according to their response to MSV. As expected, the mean yield of susceptible hybrids had the lowest yield and this yield was significantly different from that of the intermediate resistance hybrids and tolerant hybrids. However, the mean yield of the intermediate resistant hybrids was not significantly different from that of the resistant hybrids. This could be because dwarf maize hybrids with intermediate resistance to MSV are able to tolerate the disease. That is to say, though the symptoms of MSV are present, the yield is negligibly affected. Several hybrids (21 x CML 7, 51 x CML 1, 72 x CML 7 and CML 1 x CML 7) had high yields under high levels of MSV infestation, therefore, these hybrids can be considered where high yielding MSV tolerant varieties are

required. The results for the other characteristics that were measured (Appendix 2) were not included in the report since they were beyond the scope of this study.

CHAPTER 6

CONCLUSIONS AND RECOMMENDATIONS

6.1 Conclusions

1. Results from this study showed that though non-additive gene action played a role, the major contribution of the inheritance of MSV resistance in the dwarf maize inbred lines that were studied was additive gene action. Thus genetic control of MSV resistance in the inbred lines studied is polygenic and quantitative. This suggests that the resistance is likely to be effective, stable and should be able to withstand sudden changes in the races of the pathogen.

2. The high heritability obtained suggests that, though the inheritance of resistance to MSV is controlled quantitatively, only several genes as compared to many genes are involved. Therefore it should be easy to implement breeding strategies that fix the trait, like backcrossing.

3. The high heritability for MSV resistance in the dwarf maize inbred lines also suggest that response to selection based on visual scoring will be quite effective. Therefore, there is no need to resort to other more sophisticated and expensive breeding methods like marker assisted selection when breeding for MSV resistance in dwarf maize.

4. The results also showed that the hybrids which are classified as having intermediate resistance to MSV (MSV score of 3.5 to 6.4) performs as well as those that are resistant (MSV score of 6.5 to 9) in terms of yield.

6.2 Recommendations

1. When breeding for a hybrid with high resistance to MSV, one should use selection schemes such as recurrent selection in order to select cultivars that are highly resistant to MSV. These improved populations can then become sources of MSV tolerant inbred lines. Other important agronomic traits should however, not be ignored.

2. Since the sources of MSV resistance used in this study did not confer complete resistance to the disease, it is recommended that breeding efforts continue so as to find sources that can confer complete resistance. Such sources are usually controlled by single genes showing dominance; therefore it is also recommended that should those sources be found they should be bred into dwarf maize varieties that already possess the quantitative genes from sources used in this study. This would be most ideal because in the event of the breakdown of the dominant gene, additive genes are present to prevent any disaster.

3. When breeding dwarf maize for resistance to MSV, one should use the visual scoring method since it is not expensive and does not require a high level of skill but still gives a high level of accuracy.

4. It is recommended that resistant hybrids and those that had intermediate resistance be taken up for further trials. This may lead to the eventual production of some of these single crosses as successful MSV tolerant varieties.

5. It is recommended that this experiment be repeated with a larger number of dwarf maize inbreds in order to come with more conclusive information on the inheritance of MSV resistance in dwarf maize germplasm.

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APPENDICES

Appendix 1: Formulae used for combining ability analysis based on Singh and Chaudhary (1985)

Sums of squares due to GCA = $^{1}/_{n-2}\sum Y^{2}i - {}^{4}/_{n(n-2)}\,Y^{2}..$

Sums of squares due to SCA = $\sum\sum Yij^{2} - {}^{1}/_{n-2}\sum Yi.^{2} + {}^{2}/_{(n+1)(n-2)}Y^{2}..$

Sums of squares due to error = Error sums of squares (for crosses)/number of replications

Variance component due to GCA = $^{1}/_{n-1}\sum gi^{2} = {}^{(Mg-Ms)}/_{(n-2)}$

Variance component due to SCA = $^{2}/_{n(n-3)}\sum\sum_{i<j} sij^{2} = Ms - M'e$

Estimation of GCA effects: $gi = {}^{2}/_{n(n-2)}[nYi - 2Y..]$

Estimation of SCA effects: $sij = Yij - {}^{1}/_{n-2}(Yi. + Yij) + 2/(n-1)(n-2)Y..$

Where: - n = number of parents
Mg = mean square due to GCA effects
Ms = mean square due to SCA effects
M′e = mean square due to error

Appendix 2: Results of the other traits measured

Hybrids	PH	EH	EPO	MOI	NP	NKR	NKPR	EL	ED	100GW	SHELL
32 x 21				18.7	3.0					21.0	67.1
32 x 51				18.6	5.0					16.4	59.7
32 x 72	134.6	26.1	0.31	17.0	8.0	16.1	31.0	12.2	4.7	20.1	53.6
32 x X_2	146.3	44.1	0.27	19.9	18.0	15.5	37.1	15.4	4.7	33.0	77.4
32 x X1	155.9	47.3	0.30	23.1	18.0	17.0	34.8	14.7	4.8	35.3	70.8
21 x 51				20.0	3.0					28.3	77.3
21 x 72	152.6	39.2	0.28	18.4	7.0	14.4	38.3	19.8	3.9	26.1	57.2
21 x X_2	171.6	51.8	0.32	22.0	18.0	15.0	37.0	18.6	4.3	39.0	78.9
21 x X1	186.2	67.8	0.37	22.6	16.0	15.9	37.8	20.9	4.3	36.3	77.8
51 x 72				19.4	2.0					21.5	68.2
72 x X_2	162.0	52.2	0.27	21.4	18.0	14.3	38.7	17.4	4.6	41.5	80.5
72 x X1	164.8	74.2	0.42	23.4	18.0	15.8	39.1	17.5	4.6	41.0	81.6
X_2 x X1	164.2	67.8	0.41	23.7	20.0	17.3	34.5	17.7	5.1	34.9	78.4
51 x X_2	152.2	56.5	0.34	20.0	19.0	14.6	32.1	15.9	4.2	31.3	81.4
51 x X1	156.4	61.8	0.43	21.9	19.0	15.9	35.2	19.5	4.4	34.3	71.9
Mean	**158.8**	**53.5**	**0.34**	**20.7**	**12.8**	**15.6**	**36.0**	**17.2**	**4.5**	**30.7**	**72.1**
LSD (0.05)	**0.0**	**9.8**	**0.07**			**1.8**			**0.0**	**8.4**	
MSe	**280.5**	**134.7**	**0.00**	**1.6**	**16.6**	**1.0**	**4.0**	**2.1**	**0.2**	**24.0**	**146.3**
CV	**10.5**	**21.7**	**16.00**	**6.1**	**31.7**	**6.5**	**5.6**	**8.4**	**10.8**	**16.0**	**16.8**
p	**0.001**	**0.000**	**0.000**			**0.038**			**0.008**	**0.000**	
p	**	***	***	ns	ns	*	ns	ns	**	***	ns
Min	**134.6**	**26.1**	**0.27**	**17.0**	**2.0**	**14.3**	**31.0**	**12.2**	**3.9**	**16.4**	**53.6**
Max	**186.2**	**74.2**	**0.43**	**23.7**	**20.6**	**17.3**	**39.1**	**20.9**	**5.1**	**41.5**	**81.6**

Key
PH = plant height (cm)
EH = ear height (cm)
EPO = ear position
MOI = moisture content (%)
NP = number of plants
NKR = number of kernel rows per ear
NKPR = number of kernels per row
EL = ear length (cm)
ED = ear diameter (cm)
100GW = 100 grain weight (g)
SHELL = shelling percentage (%)
X_1 = CML1
X_2 = CML7