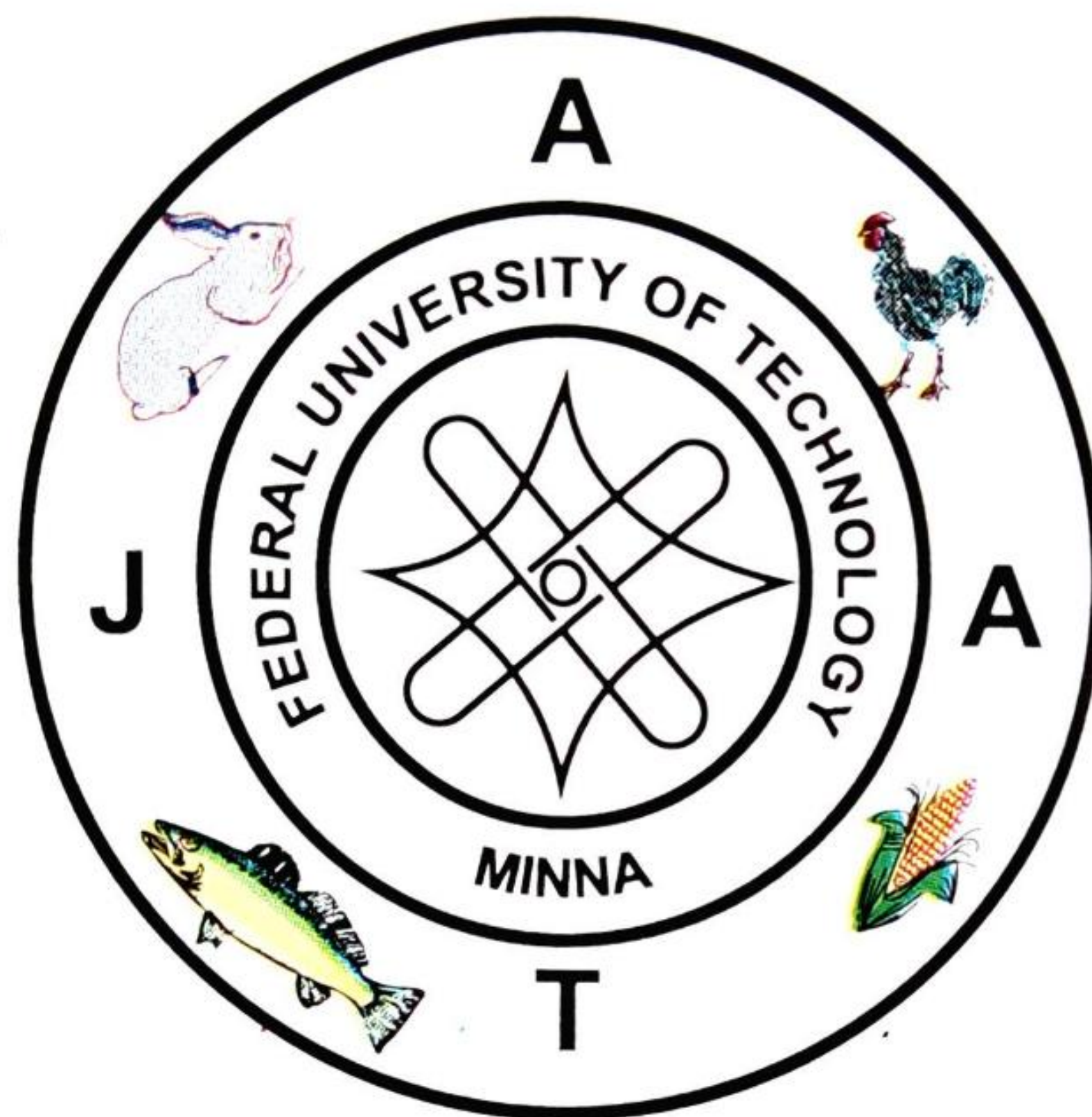


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A Review of Breeding for Resistance to Maize streak virus Disease in Sub-Saharan Africa

Salaudeen, M. T^{1*} and Bashir, M².

¹Department of Crop Production, Federal University of Technology, P. M. B 65, Minna, Niger State Nigeria

²National Biotechnology Development Agency, Lugbe, Abuja, Nigeria

*Corresponding author: mtsalaudeen@futminna.edu.ng

Phone number: +234 8063330183

ABSTRACT

Meeting the food demand for the teeming population is a herculean task in sub-Saharan Africa (SSA), where population growth rate is rapid at the expense of crop productivity. Maize is a staple food for millions of people but yield per unit area is limited by an array of biotic and abiotic factors. Among the several viruses infecting maize, Maize streak virus (MSV; genus *Mastrevirus*, family *Geminiviridae*) is the most economically important in the sub-region. Maize streak virus induces white chlorotic streak on the leaves of infected plants, necrosis, dieback, stunting and sterility. Incidence and yield losses vary from 0 to 100 % depending on the level of tolerance or susceptibility of the maize germplasm. Resistance to MSV infection is conditioned by monogenic or oligogenic genes. Of the various control strategies recommended, adoption of genetically resistant cultivars is the most cost-effective, efficient and sustainable. Breeding for MSV resistance is accomplished using conventional or Marker-Assisted Breeding (MAB). In addition, transgenic MSV tolerant maize plants have been developed using the mutated MSV replication associated gene. This paper reviews the current status of breeding activities for maize streak virus disease resistance in sub-Saharan Africa.

Key words: Breeding; Maize; MSV; genetic resistance; MAB

INTRODUCTION

Maize (*Zea mays* L.) is the third most popular cereal crop worldwide (FAO, 2010) after wheat and rice. It is a major food source in many developing countries of Latin America, Africa and Asia (Sofi *et al.*, 2009) and about 15 % of the global output is consumed annually. At global level it accounts for 15 % of proteins and 20 % of calorie intake (Sofi *et al.*, 2009). Although maize productivity is seriously threatened by over 32 viruses, the most economically important is the *Maize streak virus* (Thottappilly *et al.*, 1993). Maize streak disease (MSD) symptoms are influenced by several factors including the level of resistance or susceptibility of the maize genotype, age at infection, virulence of the strain and environmental factors (Bosque-Pérez *et al.*, 1998; Salaudeen *et al.*, 2010, 2011). In susceptible maize plants streak disease normally manifests as minute, pale, circular spots on the lowest exposed portion of the youngest leaves. As leaves expand, conspicuous chlorotic streaks ranging from broken to almost continuous streaks are evident along the veins on most of the leaf laminae and, since the virus is systemic, symptoms manifest on the inoculated and subsequent leaves (Thottappilly *et al.*, 1993). As infection advances, less streaks are found on the primary veins, compared to the secondary and tertiary veins. Lesion colour is either white or yellow, but some MSV strains induce red pigmentation on maize leaves and abnormal shoot and flower bunching in

grasses (Shepherd *et al.*, 2010). The streak pattern arises from the failure of chloroplasts to develop in tissues surrounding the vascular bundles (Bosque-Pérez, 2000), and this reduces the photosynthetic ability of the plant (Mesfin *et al.*, 1995). Immuno-histochemical studies have demonstrated that the virus is restricted only to vascular tissues and does not invade the apical meristems within the shoot apex. Conversely, in mature tissues which exhibit streak symptoms, MSV is not limited to vascular tissue (Lucy *et al.*, 1996). Infection is more severe in younger than older plants and early infection results in stunting, small sized ears or complete yield loss. Infection of young plants can lead to plant death (Shepherd *et al.*, 2010).

It is somewhat difficult to assess the overall economic importance of MSV in any region owing to variation in disease incidence between seasons and the effect of growth stage at time of infection on the magnitude of yield loss (Fajemisin *et al.*, 1986a). However, it ranges from 0 to 100 % (Alegbejo *et al.*, 2002). Infected seedlings may die or produce no seed and, plants infected at the second, sixth and tenth leaf stages suffer about 55 %, 40 % and 25 % losses in grain weight, respectively (Bock, 1982; Bosque-Pérez and Buddenhagen, 1999; Kaitsha, 2001). Additionally, MSV outbreaks resulting in economic yield losses have been reported in over 20 countries in Africa (Kim *et al.*, 1981). In East

Africa, yield reduction of about 33 – 56 % has been reported (Gutherie, 1977). Also, yield losses of up to 100 % have been confirmed in many countries of West Africa and Zaire (Fajemisin *et al.*, 1986b). Experiments conducted at the International Institute of Tropical Agriculture (IITA) between 1978 and 1981 reported an average yield reduction of about 70 % to MSV infection (Kim *et al.*, 1981).

Management of MSV is quite challenging owing to the variability of the pathogen, susceptibility of locally adapted varieties and highly variable leafhopper vector migratory and survival patterns. Control strategies include crop rotation, timely planting, chemical seed treatment, roguing, insecticidal control of insect vectors and host plant resistance. However, the use of resistant maize genotypes which are cost-effective, safe, sustainable and compatible with integrated disease management (Pratt *et al.*, 2003; Ngwira and Khonje, 2005) is the best option (Danson *et al.*, 2006). Several sources of resistance to MSV have been reported in maize genotypes including Mex. 37-5 and Yellow Bounty (Etienne and Rat, 1973), TZ-Y (Tropical Zea Yellow), and IB32 (Kim *et al.*, 1989). Genetics of resistance has been variously described as being monogenic or oligogenic (Kim *et al.*, 1989; Kyetere *et al.*, 1995; Pernet *et al.*, 1999a, b) depending on the source and sophistication of the technique employed.

Development of resistant varieties makes use of conventional approach which is often characterized by delay and association of undesirable trait. There is a renewed attempt to explore marker-assisted selection (MAS) (Foolad and Sharma, 2005). It is an approach in which selection of individual plants is based on their genotype. Marker-assisted selection involves the use of deoxyribonucleic acid (DNA) markers for determining the presence of gene- (s) of interest such as drought and disease resistance. Molecular markers are tags or signposts indicating the presence of gene of economic importance on the chromosome of a plant. Generally, they help to facilitate the development and availability of desirable varieties (Mungo and Hoisington, 2001). Marker-assisted selection can be used for introgression of major quantitative trait locus (QTL) (Abalo *et al.*, 2009). Therefore, this strategy enhances the possibility of selecting desirable genotypes more effectively. This paper reviews the current status of breeding activities for maize streak virus disease resistance in sub-Saharan Africa.

Properties of *Maize streak virus*

Maize streak virus is a member of the genus *Mastrevirus* (family: *Geminiviridae*) (Willment *et al.*, 2001). *Geminiviruses* are plant viruses having monopartite or bipartite circular single-stranded DNA (ssDNA) genomes. They are 2.5 – 3.0 kb particles of 20 × 30 nm in size (Frischmuth and Stanley, 1993). The *Geminiviridae* comprises four genera (*Mastrevirus*, *Curtovirus*, *Topocuvirus* and *Begomovirus*) based on their genome organizations and biological properties (Fauquet *et al.*, 2003). The particle measures 18 × 30 nm in size (Bosque-Pérez, 2000). Each virion of the MSV contains a single, covalently bound, circular, ssDNA molecule of about 2.7 kb (Francki *et al.*, 1980; Rybicki *et al.*, 2000) which codes for four potential products (Isnard *et al.*, 1998). Replication occurs through double-stranded DNA (dsDNA) intermediates, using a rolling circle replication mechanism (Laufs *et al.*, 1995a, b). *Geminiviruses* do not encode their own DNA polymerases but rely on the nuclear DNA replication machinery of the host (Munoz-Martin *et al.*, 2003). Recent studies have proved that 'recombination-dependent replication' mechanisms are also involved in *Geminivirus* replication (Jovel *et al.*, 2007). In replicative dsDNA molecules, genetic expression arises from both strands, and diverges from an intergenic region containing the virion – sense origin of replication. Rolling circle replication begins by binding of the virus replication – associated protein (*Rep*) to the virion-strand origin of replication, where the protein begins and terminates virion strand DNA synthesis (Stenger *et al.*, 1991; Willment *et al.*, 2007). *Maize streak virus Rep* consists of two complementary sense open frame (ORFs), C1 and C2. Spliced transcript (*Rep*) or unspliced transcript (*Rep A*) is produced from the C1:C2 containing an intron. Although MSV replication is facilitated by *Rep* alone (Liu *et al.*, 1998), *Rep A* plays a variety of important additional functions during its life cycle. Such activities include the modulation of host cell cycle regulation, and probably other development pathways (Gutierrez, 1999). Also present in the MSV genome are the movement protein (MP) and the coat protein (CP). The former enhances the movement of the virus from the site of replication (nucleus) to adjacent cells (Boulton, 2002). Conversely, CP is responsible for the encapsidation of the viral nucleic acid and determines virus – vector interactions (Briddon *et al.*, 1990). Additionally, MSV CP binds non-specifically to both ssDNA and dsDNA; it is also required for cell-to-cell and systemic spread of the virus in plants (Liu *et al.*, 2001); and interacts

specifically with MP to move virus DNA out of the nucleus.

Genetics of MSV disease resistance

Investigations into the genetics of plants resistance to MSV started in South Africa using the cultivars Peruvian yellow (Fielding, 1933) and cv Arkell's Hickory. A hybrid ($P \times H$) of these two cultivars was later used as an MSV-resistance donor (Rose, 1936). In $P \times H$ inbred lines, resistance was found to be mainly controlled by an incompletely dominant gene, deviation from theoretical segregation ratios being attributed to modifying genes (Storey and Howland, 1967). However, other workers ascribed MSV resistance to simple inheritance with apparently strong dominance component (Fourie and Piennar, 1983), and five dominant genes (Engelbrecht, 1975) of quantitative inheritance (Rose, 1938; Gorter, 1959; Lazarowitz, *et al.*, 1989). Soto *et al.* (1982) found that resistance in inbred TZ-Y was simply inherited, but Kim *et al.* (1989) reported that resistance in inbred IB32 was quantitatively inherited through additive action of several genes. Rodier *et al.* (1995) found that resistance in $CVR_3 - C_3$ involved loci with major genes controlling high to complete resistance, and a locus with minor genes controlling partial resistance. The major 'system' was said to be monogenic or oligogenic. Conversely, the 'minor system' was speculated to be polygenic. Similarly, Pernet *et al.* (1999a, b) identified a major QTL in the same genomic location on chromosome 1S, and postulated that MSV resistance was under the control of two genetic systems: one arising from a major gene on the short arm chromosome 1 with dominance effect (*Msv-1*), and with other minor genes on chromosomes 2, 3 and 10 that confer quantitative resistance. Additionally, Kyetere *et al.* (1999) used molecular markers to identify a single, partially dominant gene on the short arm of chromosome 1 (*Msv-1*) in TZ4. Since there was no other genomic region associated with MSV resistance, it was described as being monogenic.

Sources of resistance genes to maize streak virus disease

Following the discovery of MSV resistance in the maize genotype $P \times H$ and SA31 (Fielding, 1933; Rose, 1936), Gorter (1959) reported resistant varieties including 3NA (Rhodesia), 29-29A-5-4, Max 37-5 and Vrg. 54, and 'Yellow Bounty'. Additionally, some maize lines of Colombia and Mexican origin were identified to have combined resistance to rust and MSV infection (Rubaihayo, 1974). These lines were used to develop the

variety "White Star". In Kenya, IRAT and KAF found resistance in 'La Revolution'. This source of resistance has been demonstrated to be generally effective. In West Africa, efforts to identify sources of MSV resistance began in Nigeria in 1970s, following the epidemics of 1970 (Fajemisin *et al.*, 1976). Although some plants were observed to show an appreciable level of tolerance, they could not be fixed as resistance sources due to lack of reliable artificial screening technique. In 1975, IITA scientists selected source of resistance from the population TZ-Y developed from a cross between Planta Baja [from the Centro Internacional de Mejoramiento de Maíz y Trigo (CIMMYT)] and some East African germplasm. This source was maintained through continuous selfing under artificial streak infection and named IB 32. It has since been widely utilized at IITA and in many national and regional programs across Africa to improve the susceptible varieties (Fajemisin *et al.*, 1984).

Breeding methods for resistance to maize streak virus disease

From time immemorial, breeding crop cultivars that are resistant to diseases has been the major goal of the plant breeders. Breeding activities designed to improve grain yield in MSV endemic regions usually require a good knowledge of combining ability of the breeding materials to be used (Gichuru *et al.*, 2011). Breeding for MSV resistance began in tropical Africa in 1960s. In Nigeria, attempts to develop streak resistant maize cultivars started at IITA in 1975 and by 1979 usable resistance had been identified and tested on a large scale (IITA, 2012). The strategy adopted was based on avoidance of virus strain specificity; large scale vector rearing and field infestation to enhance early infection; selection only for tolerance when infected; and not separating virus resistance selection from simultaneous selection for yield; adaptation and resistance to other diseases of importance in each target ecology (Buddenhagen and Bosque-Pérez, 1999). Breeding for MSV resistance is being conducted at conventional and molecular levels. In 1966, breeding for MSV resistance commenced and by 1975 some prominent research institutes including IITA, the National Agricultural Research Systems (NARS), and later CIMMYT (Centro Internacional de Mejoramiento de Maíz y Trigo) were keenly involved. This was aimed to develop maize varieties that combine MSV resistance with other desirable agronomic characters (Fakorede *et al.*, 2001). Currently, marker assisted breeding (MAB) is being explored to speed up the

development of resistant maize genotypes, at reduced costs (Abalo *et al.*, 2009).

Conventional breeding for MSV resistance

Selection criteria: The first task is to select plants with phenotypic resistance to MSV. During the early growth stage seedlings are inoculated with the virus. The infected resistant plant is retained while the uninfected or highly susceptible plant is removed from each stand. Where both plants exhibit MSV symptoms the more tolerant is retained. Also, where both plants are not infected, the more vigorous one is retained (Fajemisin *et al.*, 1984). At tasseling, plants exhibiting resistance to MSV and desirable agronomic characteristics (low ear-and plant-height and resistance to other prevailing diseases) are selected. Recurrent selection scheme is then undertaken.

Recurrent selection scheme: It is a two-phase method involving population formation and improvement. During the population formation susceptible selected germplasm is crossed with the streak resistant variety with minimum of 200 ears. At the IITA, Ibadan, Nigeria, Tropical Zea Streak Resistant-W-1 (TZSR-W-1) and Tropical Zea Streak Resistant-Y-1 (TZSR-Y-1) were the first streak resistant populations with a broad genetic background ever developed (IITA, 1981; Kim *et al.*, 1981), and used as MSV resistance gene donors. In practice, the F_1 is planted as a balanced bulk and selfed to produce also a minimum of 200 ears. These are then grown ear- to row and half-sib recombination done under MSV disease pressure. The product is further subjected to one to three more cycles of half-sib recombination in order to ensure a complete recombination (Efron *et al.*, 1989).

Population improvement

The populations formed are subjected to recurrent selection through multi-location international testing trials. Several MSV resistant populations have been maintained and improved under this technique. The procedure was designed by CIMMYT but later modified by IITA for a number of reasons including high costs and highly variable data generated. It is a two-year scheme comprising formation of reciprocal full-sib families (normally 250 full-sibs) during the first season of the first year, and international testing in six locations using two replications per location, in the second season. Activities in the second year include within - family improvement of selected

families across locations through self-pollination of selected plants in each full-sib family in the first season, and half-sib recombination of selected S_1 families to re-synthesize the population for the next cycle of improvement in the second season. After each international full-sib testing, experimental varieties are obtained for recombining the best 10 families of each testing site, together with one variety based on selected families across all the progeny testing locations (Fajemisin *et al.*, 1984; Efron *et al.*, 1989).

Intra-population improvement

This is usually done where some resistance can be found even at a very low frequency in high-yielding and locally adapted populations or varieties. The resistance level and frequency is then increased gradually without altering the genetic make-up of the population. Intra-population improvement can also be employed even if MSV resistance gene cannot be identified within the population in the variety. This is carried out if only a small proportion of the population is being crossed with an appropriate streak resistance donor. Few resistant progenies of such a cross can be released into the population to initiate the gradual build-up of resistance (Efron *et al.*, 1989).

Backcross

The backcross conversion program began in 1980 at IITA, Ibadan. It basically involves evaluation of maize varieties under MSV disease pressure and several other environments under streak free conditions. The varieties developed after the backcross 2 (BC_2) and backcross 4 (BC_4) generation are compared with the recurrent and donor parents (Efron *et al.*, 1989). It is possible to obtain streak resistant varieties after the second backcross, under streak pressure conditions. Investigations have shown that when an open - pollinated is being upgraded by backcross, four backcross generations are adequate but more backcross generations might be required if better recovery of the recurrent parents gene frequencies is intended (Efron *et al.*, 1989).

Molecular breeding for MSV resistance

The conventional is said to be time consuming and very depended on environmental conditions, the use of MAS is very essential. For instance, in conventional method 8 - 12 years are required to breed a new variety (Abalo *et al.*, 2009). Conventional breeding for MSV resistance is less preferable because more than one gene confers resistance and due to association of undesirable traits with resistance (Shepherd *et al.*, 2007).

Undoubtedly, molecular breeding would contribute significantly to food security in developing countries (Ribaut *et al.*, 2010). However, the success of MAS for complex traits requires an accurate identification of QTL position and the magnitude of their effects (Willcox *et al.*, 2002). The QTL is then mapped at specific regions of the susceptible maize genotypes (Redinbaugh *et al.*, 2004). Marker-assisted selection operates on the principle that it is possible to predict the availability of a gene from the presence of a marker that is tightly linked to the gene. Thus if the marker and the gene are widely located on the chromosome there is less likelihood of being transmitted to the offspring, owing to double crossover recombination (Drinić *et al.*, 2004).

Studies have shown that the presence of several strains or races of a pathogen could impede the application of MAS. There is no consensus regarding the number of genes controlling MSV resistance. Where resistance is conditioned by a single dominant gene MAS is simplified but such resistance may not be durable if case of emergence of a severe strain of the pathogen. On the other hand, where resistance is conferred by several genes, gene pyramiding could be undertaken. Therefore, molecular marker technology offers the tools needed to identify, select and combine favourable alleles via genotypic selection (Sanford *et al.*, 2001). Marker-assisted breeding could be used to maintain virus resistance alleles even in the absence of infection (Redinbaugh *et al.*, 2004). Molecular markers are useful in identifying and pyramiding of unique genes governing disease resistance. Also, they can be used to define the number of genes influencing a trait, the magnitude of their effects, and serve as a strong point for map-based cloning. Abalo *et al.* (2009) reported that MAS method was cheaper than conventional selection by 26 %.

Several marker systems have been employed for genetic analysis including restriction fragment length polymorphisms (RFLPs), random amplified polymorphism DNAs (RAPDs), amplified fragment length polymorphisms (AFLPs), and simple sequence repeats (SSRs). For instance, Mafu *et al.* (2014) used SSRs to genotype some maize lines for MSV resistance genes. Much of the QTL analyses for MSV resistance genes relied on the application of RFLPs (Welz, 1998; Kyetere *et al.*, 1999), SSRs (Danson *et al.*, 2006, Lagat *et al.*, 2008). Single nucleotide polymorphisms (SNPs) have been used

extensively in QTL genotyping for a number of traits (Ribaut *et al.*, 2010) in maize, including resistance to MSV (Nair *et al.*, 2015). Single nucleotide polymorphism refers to the polymorphism occurring between DNA samples with respect to single base (Jehan and Lakhanpaul, 2006). Single nucleotide polymorphisms are quite abundant molecular markers in plant genome. It has been documented that on the average, two randomly sampled maize DNA sequence has one SNP every 104 base pairs (bp) (Tenailon, 2001). Elsewhere, it has been discovered that in maize genome there is one SNP per 20 bp. Therefore, SNPs have become the marker of choice due to their abundance in genome, and ability to provide basis of a superior and highly informative genotyping assay (Jehan and Lakhanpaul, 2006). Furthermore, SNPs are less mutable as compared to other markers particularly microsatellites. Thus, the low rate of genetic mutation make them evolutionarily stable and excellent markers for studying complex genetic evolution. Although SNPs require high quality DNA, they are easy to use, highly automated, reproducible, and cost effective.

Single nucleotide polymorphisms detection and genotyping within a genetic locus can be accomplished using several techniques including Cleavage assay (Huang *et al.*, 2002), reduced representation shotgun (RRS) (Altshuler *et al.*, 2000), electrophoretic assays, temperature modulated heteroduplex assay (TMHA), fluorescence resonance energy transfer, alpha screen, capillary array electrophoresis (CAE), electrochemical detection of mismatches in nucleic acids (EDEMNA), microarrays, genetic bit analysis (GBA), padlock probes, pyrosequencing, and invader assay (Jehan and Lakhanpaul, 2006). Maize streak virus resistant maize genotypes have also been developed using dominant negative mutants of the virus replication-associated protein gene (*Rep*) (Shepherd *et al.*, 2007a, b, 2014). The MSV resistance inherited was effective up to the T₃ generation. Such plants were able to delay expression of streak symptom when inoculated with the virus.

CONCLUSION

Maize is a staple food source for millions of people in sub-Saharan Africa. However, productivity is seriously constrained by Maize streak virus. Although several management strategies are being employed the use of resistant genotypes remains the most viable and sustainable measure. Breeding of maize germplasm for MSV

resistance is of high priority in research institutes including IITA and CIMMYT where resistant donor varieties are used to improve the susceptible genotypes. Such breeding efforts now give preference to MAS in order to save the long time and high costs associated with conventional breeding.

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