Theoretical and Applied Genetics (2021) 134:3785–3803
https://doi.org/10.1007/s00122-021-03914-y

REVIEW

Maize streak virus research in Africa: an end or a crossroad

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Received: 10 January 2021 / Accepted: 10 July 2021 / Published online: 26 July 2021

Abstract

The economic importance of the maize streak virus disease to the African maize production dynamic is to be appreciated now more than ever due to the preponderant influence of a changing climate. Continued dependence on a single major-effect quantitative trait locus (QTL) called *Msv1* on Chromosome 1 of Maize (*Zea mays* L.) is not guaranteed to ensure durable resistance to the causal pathogen. With over ten decades of research on the disease and its associated host plant resistance mechanisms, it is pertinent to consider future approaches to attaining durability by looking to the synergistic roles of moderate- and minor-effect QTLs located on other chromosomes so as to facilitate a secure farming system for sub-Saharan Africa. For this review, more than 40 publications relating to maize streak disease research were methodically analysed with about 30% making specific reference to conventional, molecular and transgenic approaches employed in introgressing, maintaining and improving streak resistance in maize. A meta-analysis of mapped QTLs conferring streak resistance was conducted in a bid to reveal any inter-dependence or co-localization of resistant loci and to aid decision-making for marker-assisted breeding. With the changing climatic conditions around the globe, man's preparedness in the event of an epidemic following any evolutionary process in the streak viral genome was determined as insufficient. Modern breeding approaches including gene pyramiding that could be considered in maize breeding programmes to ensure durability for streak resistance were proposed while improving maize for other abiotic stress tolerance, particularly drought.

Introduction

Maize (*Zea mays* L.) is a food crop of choice for millions of people residing on the continent of Africa (Olawale and Tontsa 2015; Mahoussi et al. 2017), where it acts as an energy source making for a common component of meals or as a source of animal calories in compounded feeds and also used for fuel, fibre and raw materials production. The rapid adoption of the crop by farmers in sub-Saharan Africa (SSA) since its introduction stemmed from its desirable attributes including being a short-season and easy-to-propagate food crop (Mahoussi et al. 2017). It is also suitable for intercropping purposes and could be planted during the major and minor cropping seasons that characterizes rain-fed agriculture in the region. All-year round production of maize in SSA is achievable and should be a target for national programmes on the African continent.

Of the more than 197.20 million hectares (ha) of land area harvested in the world in 2019, 20.64% was under maize cultivation in Africa (FAOSTAT 2020). On this continent, Eastern Africa has maintained the lead on maize area harvested with an average of 41.39% (16,675,227.75 ha) between 2015 and 2018. Between 2018 (17,097,810 ha) and 2019, a decrease of 7.73% was recorded, most likely attributable to the devastating effect of the locust outbreak in the subregion as well as climate change (Devi 2020; Salih et al. 2020). Over the same period, Western Africa accounted for an average of 32.41% of Africa's harvested land area with a net increase of 1.98% in 2019. Maize was cultivated on roughly 28% of harvestable land area by the rest of Africa with Southern Africa occupying an average

Communicated by Rajeev K. Varshney.

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of 6.46% between 2015 and 2019. In terms of production volume, Africa produced 7.13% (81,891,311 tonnes) of the world's maize grains in 2019, a figure that depicts a loss of 7.41 million tonnes from its 2017 estimate (FAOSTAT 2020). Although a similar trend played out with production volumes in the subregions as that of harvestable land area in the period under review, a reduction of 2.81% (2,708,529 tonnes) in Eastern Africa between 2018 (33,463,604 tonnes) and 2019 alludes to the threats to food security through crop loss due to obvious factors including pest infestation. Meanwhile, production volumes of other regions between 2015 and 2019 exhibited no significant improvement or worse still, a further drop in volume.

Efforts to boost maize yields in Africa through enhanced agronomic practices including the use of more efficient water–weed–pest management strategies and pragmatic development of elite cultivars and hybrid varieties that possess desirable adaptive features have seen some success (Badu-Apraku et al. 2013; Abate et al. 2015; Nzioki et al. 2016; Jovanovic et al. 2020; Nafi et al. 2021). For instance, in a commodity intelligence report, the Foreign Agricultural Service of the United States Department of Agriculture (USDA) indicated that South Africa's 2017/2018 maize yields reached second highest on record with 5.09 t/ha, following those of the previous year at 5.89 t/ha. This record was attributed to abundant rainfall during the critical pollination and grain-filling stages occurring towards the end of January through the end of April as well as to the use of improved maize seed varieties that perform well under drier conditions and higher plant populations, an evidence of yield gains through adoption of innovative technology (Reynolds 2018).

However, from a global perspective, these yield estimates connote a continent lagging very far behind others who grow the crop on a lot less land and with even lesser area harvested (FAOSTAT 2018). Factors causing this trend includes: depleting soil fertility, weed infestation, increasing rates of dry spells due to climate change and newly emerging pests and disease pathogens (Reynolds 2018; Lobulu et al. 2019). Low yields and evitable crop loss due to these constraints will need a pragmatic, well-tailored and contextually adaptive framework in order to secure present and future maize demands, while at the same time boosting yield gains by maximizing transformative advances (Bailey-Serres et al. 2019).

At present, the causal agents of numerous biotic constraints hindering the achievement of Africa's maize yield potentials are to some extent being controlled using several strategies including host plant resistance (Martin and Shepherd 2009). Yet significant damages are still being perpetuated, especially following regular recombination in the genome of pathogens resulting often in direct mutagenic products or selectively favoured mutants (Varsani et al. 2008; Monjane et al. 2012). Whatever the case, proactive crop improvements to withstand the present circumstances and any unforeseen events in the future are hinged on possession of sufficient host plant immunity to either tolerate or outrightly resist emerging pathogens, taking into account the estimated rate of their evolution.

One such disease-causing pathogen is the maize streak virus (MSV) that belongs to the family Geminiviridae and a well-known member of the genus *Mastrevirus* implicated in the maize streak virus disease (MSVD). The virus possesses a single component of circular, single-stranded DNA genome of about 2700 bases encapsulated in germinate particles (Shepherd et al. 2010) and is persistently transmitted by a leafhopper of the Genus *Cicadulina* (Family Cicadellidae, Order Hemiptera), which is a migratory vector.

MSD is a devastating disease of maize endemic only to SSA, and the economic impact of MSV attack in terms of the cost to national economies in the region via yield loss, lost income and higher maize prices has been succinctly approximated (Martin and Shepherd 2009). Some authors reported of up to 100% yield loss in crops infected with MSV (Wambugu and Wafula 2000; Lagat et al. 2008). However, according to Martin and Shepherd (2009), approximately 10–100% yield reductions can result based on timing of infection per infected plant. In actual fact, a 100% yield loss would occur only in a scenario where susceptible maize seedlings planted to individual fields were infected with a virulent strain of MSV before they reached the 2nd leaf stage as no seed will be yielded. Furthermore, between US$120 million and US$480 million per year is lost in terms of lost income and higher maize prices in SSA (Martin and Shepherd 2009).

For over ten decades, in-depth studies have been carried out on MSV as the most significant virus affecting maize production in sub-Saharan Africa (Mesfin et al. 1992; Bosque-Pérez 2000). Aspects relating to the geographical distribution of MSV (Alegbejo et al. 2002), its diversity at the molecular, genomic and strain levels as well as its host plant range, the virus/vector ecology and epidemiology in key regions of Africa (Bosque-Pérez 2000; Magenya et al. 2009; Martin and Shepherd 2009), the biology of its vector and its relationship with MSV and efforts at breeding for resistance against the virus [whether conventional (Welz et al. 1998; Danson et al. 2006; Lagat et al. 2008; Ladejobi et al. 2018) or transgenic (Shepherd et al. 2007a, b, 2014; Owor et al. 2011) have all been documented in the literature.

Following such magnitude of inquiry into this important pathogen, it is now common knowledge to identify its characteristic manifestation in diseased maize plants. Features including an initial appearance of small, pale, spherical, chlorotic spots can be seen on the lowest exposed parts of the youngest leaves which subsequently progresses to prominent long streaks as the leaves expand while mature leaves remain

uninfected. Also, a general reduction in plant growth, leaf size and yield as well as a severe stunting in plants infected early are observed (Rojas et al. 2018). MSV's 11 strains can infect more than 80 plant species in the *Poaceae* family with MSV-A being the most virulent and itself having five sub-types. MSV-A_1 is documented as the prominent strain recognized to cause crop losses in highly cultivated subregions and countries within SSA.

Ten years after an extensive review on the pathogen profile of the infamously old and complex "emerging" MSV was done in addition to the earlier eight decades of MSVD research (Bosque-Pérez 2000; Shepherd et al. 2010), this review seeks to explore whether a lasting solution to combat the streak disease in maize now exists and if not, to highlight possible research goals towards the host plant improvement for MSVD resistance. While several strategies including conventional and transgenic approaches for developing crops resistant to Geminiviruses have been proposed (Shepherd et al. 2009, 2014), these are threatened by the high rate of strand-specific mutations and recombination associated with Mastrevirus (Monjane et al. 2012).

Notwithstanding, to tackle this disease in an environmentally friendly and a cost-effective manner through host plant resistance, the meticulous mapping of a major locus on chromosome 1 of maize known as *Msv1* has been documented. This accounts for 40–76% phenotypic variation as reported in diverse MSV-resistant lines where it has been consistent (Welz et al. 1998; Kyetere et al. 1999; Pernet et al. 1999a, b). A further fine mapping of this particular locus has delimited the region to 0.87 cM bearing a candidate gene named GRMZM2G046848 which is a U-box domain containing tyrosine kinase family protein (Nair et al. 2015). In addition, using 948 DArT markers, new loci for MSV recovery resistance in maize has been identified by researchers at the International Institute of Tropical Agriculture (IITA) in Nigeria using $F_{2:3}$ population of a cross between KU1414×9450)×9450)-15-2-1-BBB-1-B*11 and GT-MAS:GkxKU1414SRxGT-MAS:Gk)-8-1-2-4-B*12 (Garcia-Oliveira et al. 2020). They mapped 18 QTLs with moderate to minor genetic effect for MSV resistance on chromosomes 1, 2, 3, 4, 5 and 7 accounting for 3.1–21.4% of the phenotypic variance. Earlier, four QTLs, two bearing significant effects on chromosome 3 and accounting for 47–51% of the total phenotypic variance and two QTLs with lesser effects on chromosome 7 and 9 accounting for 28–32% of the total variation was identified by genotyping 250 S1 maize lines with 269 SNP markers performed using kompetitive allelic-specific PCR (KASP) method on Kbiosciences' KASPar assay platform from LGC Genomics (Ladejobi et al. 2018).

On a local scale, a number of moderately resistant maize varieties and germplasm are available to maize producers in Africa facing streak infestation on their farmlands. For central Uganda and in the Southern Highlands of Tanzania, *Longe 1* and *Longe 6H* are MSV-resistant maize varieties recommended for adoption since they possess a strong form of resistance (Gibson et al. 2005; Bua and Chelimo 2010). In Western Kenya, an early maturing, high-yielding and MSV/Striga weed-tolerant hybrid known as WH 502 is promoted over local varieties for the upper and lower midland zones (Salasya et al. 2007). In Ghana, Tigli and Dorke SR are recommended streak-resistant varieties for cost-effective and practical control of MSV (Adu et al. 2014). In Nigeria, improved OPVs, namely TZSR-W/Y and TZESR-Y, and a few improved hybrid varieties including 8505-2 and 8505-3 are available for production in areas prone to the disease (Olaniyan 2015).

Even with the above feat, reports of a resurgence of MSV in a few countries in SSA may suggest a breakdown of *Msv1* resistance as cautioned in time past (Welz et al. 1998; Nair et al. 2015; Garcia-Oliveira et al. 2020). There is a graphical representation of this recent development where countries previously known to experience widespread MSV infestation are shown (Thottappilly et al. 1993) along with countries presently reporting MSV incidence such as in Ghana (Oppong et al. 2015), Benin (Personal communication), Ethiopia (Guadie et al. 2019), Uganda and Kenya (Pande et al. 2017) (Fig. 1).

Maintaining durable resistance by the maize plant is a time-old discussion which goes beyond the possession of a single major resistance locus and bearing in mind that MSD resistance is of a quantitative nature (Pernet et al. 1999b). More so, the general perception of the use of genetic engineering to proffer solutions to agricultural challenges and the subsequent adoption of the biotechnological products in the form of improved transgenic seeds for maize production by various national governments in Africa is somewhat negative or completely misunderstood. This notion may be based upon an insufficient evidence about the exact procedures or methods used in developing genetically modified seeds for planting, the associated health risks in the long term, the cost–benefit ratio derived from using such methods or even the extent of durability provided by them (Ayele 2007; Takeshima and Gruère 2011; Adenle et al. 2013). These gaps in knowledge necessitate steps towards providing clarity on key subjects such as the benefit of modern genetic and genomic tools for maize improvements, especially in light of the changes in the climatic conditions being experienced since the turn of the decade (Rani and Usha 2013).

Important as well are key concerns regarding the spontaneous, yet-to-be quantified evolutionary processes of MSV, its unpredictable vector population dynamics, predisposing drought conditions and an overdependence on rain-fed agriculture in areas highly prone to its epidemic; this situation gives credence for a thorough dissection of the subject around durable MSV resistance. The review proposes a

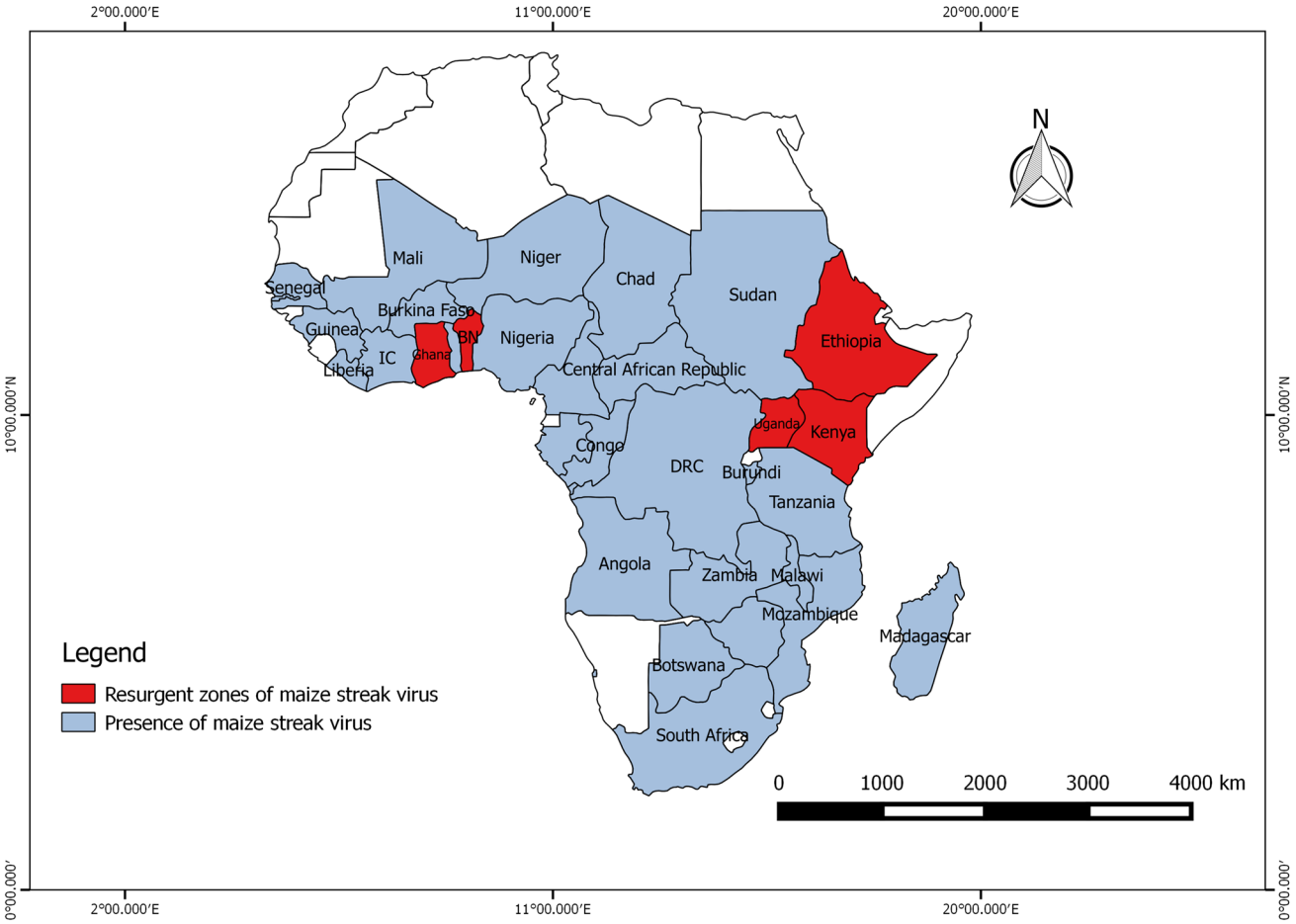

Fig. 1 Graphical Representation of MSV Resurgence in SSA

complementary approach aimed at efficiently incorporating useful minor to moderate streak resistance genes to enhance resilience in maize varieties currently being developed for stress tolerance in the tropics.

Meanwhile, a number of questions have arisen on the subject of durable MSVD resistance that are hoped to be addressed through the objectives stated below including: (a) what proportion of cultivars and hybrid varieties under cultivation in Africa possess durable streak resistance and therefore are equipped for any future evolutionary event?; (b) what is the fate of newly discovered sources of streak resistance and if there is a consensus at the molecular level that translates to future usefulness in improvement programmes?; (c) what pathways can be charted for the leveraging of new sources of resistance to ensure durability?; and lastly (d) are current maize improvement programmes sufficiently dynamic in coping with any eventual streak viral evolution?

The objectives of this review are to (i) bring to light the current streak resistance status of varieties and cultivars already developed and distributed in Africa for use in field production of maize; (ii) dissect the output of a meta-analysis carried out on all mapped quantitative trait loci (QTL) bearing MSV resistance and discuss its implication for future breeding programmes; (iii) propose modern breeding approaches that can be inculcated into maize breeding and seed production programmes to ensure enhanced streak resistance particularly, during improvements for emerging abiotic stresses; and (iv) determine man's preparedness in the event of an outbreak following any evolutionary process in the viral genome of MSV.

Retrospective account of the genetics and genomics of host plant resistance to MSV and the path forward

The search for a source of resistance to MSV has gone on for as long as the disease had first been noticed by Claude Fuller, the Government Entomologist of Natal in South Africa, in his report in 1901. He described it as a disease of "mealie variegation" and reported several symptoms as is known today but perhaps wrongly attributed its causal agent

to either a soil nutrient deficiency or a chemical enzyme acquired from the soil (Shepherd et al. 2010).

Over time, resistance has been discovered in several maize germplasms: firstly in the variety known as "Peruvian Yellow" in South Africa as far back as 1931, and has been used to develop hybrid populations through a series of selections, inbreeding, hybridization, backcrossing, population formulation and improvements (Alegbejo et al. 2002). Initial efforts to breed against MSV attacks were based on thirteen genotypes (Table 1) representing a few sources of streak resistance that were leveraged upon.

In all, it is well established that a major QTL on chromosome 1 of maize known as the *Msv1* is responsible for resistance to the disease and has a large effect in a majority of resistant germplasm. It is stable in all germplasms where it is present and is expressed irrespective of the environment it is established in. This QTL has been mapped and fine-mapped to an interval of 0.87 cM on chromosome 1 with the aid of molecular tools particularly restriction fragment length polymorphism (Welz et al. 1998; Kyetere et al. 1999), simple sequence repeats (Lagat et al. 2008) and single-nucleotide polymorphism (Nair et al. 2015; Ladejobi et al. 2018; Garcia-Oliveira et al. 2020) genetic markers. It is also documented that there are involvements of a few modifying genes and/or minor-effect QTLs that act synergistically with the major-effect *Msv*1 to boost resistance in various genotypes in which they have been found (Efron et al. 1989; Pernet et al. 1999a, b; Nair et al. 2015).

The recent but very elaborate work done by a team of researchers at the International Centre for Maize and Wheat Improvement (CIMMYT) on the fine mapping of this major-effect QTL through QTL isogenic recombinant (QIR) strategy delimited the *Msv1* to a 7.62 Mb interval, flanked by two SNPs, namely PZE-101090728 (GCTGAGACGATG TTCTTGAACCAAGCTCCCTGGAAACTAGGGCTGCCT CT[A/G]TTTTGATTGTTACCCGGAGACTCAGGTGAG GCTTGATTTTTGGAAGTCAG) and PZA00944.1 (CAA ATGAGGTGCCACTTCGGGGGAAAAATATGCTTGTAT AGTGGATCGACACGGTTGTC[A/G]TCCACGGTGATA GTGCTGTCATCTCCAAGGAATGGAAAGTCATACAGG TAGCTAGGCAAC) with a genetic distance of 0.87 cM in a large F_2 population of CML206 × CML 312 based on two-point linkage analysis (Nair et al. 2015). This team went ahead to develop three kompetitive allele-specific PCR (KASP) assays from three SNPs, namely PZE-101093951 (TAACTCTCTGCTGTTGCTTGTCTTCAGGTTGTCATG AGAGATCCTCACAT[A/G].

GCAGCAGATGGCTTCACCTACGAAGCTGACGCT CTTAGATACTGGCTCGA), PZE-0186065237 (ACATCT CCAGTAACAAACAGAAGTCTTTCGAATCGTGATACC ATCCCCAA[T/C]

CACGCACTGCGKTCGGCCATCCAAGAATACCTC CGGCAGAACGAGCTGCA) and PZE-0186365075 (AGA AGAAAATGGCCTGCCATATATATATCCCGGTTAATC GCTARTGCATT[A/C].

TCAGGAATCATTCTCATAGGTCATAAGACGAGC AAGGGATACTCTTCTAC) that co-segregated with PZE-101090728, one of the flanking markers delimited in the *Msv1* interval where they showed significant association with response to MSV based on a haplotype trend regression. This KASP assays have been validated and currently in use for classifying phenotypes based on their response to MSV to a fairly high degree of precision. A recent study was carried with the aim of validating the diagnostic ability of the three SNPs that co-segregated with PZE-101090728 above using the high-throughput KASP assay technology in 151 early generation inbred lines with diverse genetic backgrounds together with nine MSV-resistant elite lines and a susceptible check known as cv. Pool-16 (Sime et al. 2021). Upon categorization of

Table 1 Sources of MSV resistance used in earlier conversions of elite maize germplasm

Source of resistance	Origin of germplasm	Types of germplasm	Type of QTL or gene present	Selected reference
Peruvian yellow	South Africa	Cultivar	Single, incompletely dominant gene	Efron et al. (1989)
Arkells hickory	South Africa	Cultivar	Single, incompletely dominant gene	Efron et al. (1989)
CIRAD390	Réunion island	–	1 major-effect gene and 7 minor-effect genes	Pernet et al. (1999a)
TZ-Y	IITA Nigeria	Population	–	Efron et al. (1989)
IB32	IITA Nigeria	Inbred line	Major genes Kim et al., 1989	Kim et al. (1981)
Tzi4	IITA Nigeria		Major gene on Chromosome 1	Kyetere et al. (1999)
CML 202	CIMMYT	Inbred line	Major gene on Chromosome 1	Welz et al., 1998
CML 204	CIMMYT	Inbred line	Major gene *Msv1*, 2–3 minor genes	Welz et al. (1998)
CML 206	CIMMYT	Inbred line	Major QTL *Msv1*, 2 minor-effect QTLs	Nair et al. (2015)
TZIL07A01005	IITA Nigeria	Inbred line	Recovery QTLs on Chromosome 3, 7, 9	Ladejobi et al. (2018)
TZIL07A01322	IITA Nigeria	Inbred line	Recovery QTLs on Chromosome 3, 7, 9	Ladejobi et al. (2018)
La revolution	La reunion island	Cultivar	–	Efron et al., (1989)
D211	Réunion island	–	1 major-effect gene and 4 minor-effect genes	Pernet et al., (1999b)

the phenotypic responses of the maize lines by artificial inoculation of MSV using viruliferous leafhoppers under screenhouse conditions into resistant, moderately resistant, susceptible and highly susceptible, the three SNPs associated with MSV resistance were detected in 131 of these lines, classifying them into resistant (54), moderately resistant (76) and susceptible (1), thereby confirming strong association of SNPs with MSV resistance.

This feat described above has great advantages for ease of use and is cost-efficient, especially in routine screening during a variety development programme aimed at other emerging targets for stress tolerance. Meanwhile, caution would need to be taken by guarding against an overdependence on the *Msv1* (Garcia-Oliveira et al. 2020) as the only QTL for declaring resistance to this pathogen, firstly because of the unpredictable nature of its evolutionary process in the face of a changing climate and also because this resistance is of a quantitative type and as such, a need to optimize the contribution of other QTLs of importance in this context. This may have been a motivating factor leading to the recently discovered and mapped MSV recovery resistance by a team of scientists at the International Institute for Tropical Agriculture, Nigeria (Ladejobi et al. 2018). They mapped four SNP loci conferring recovery resistance to the streak disease in already resistant elite lines of the TZL category, i.e. Tropical Zea Inbred lines, especially the TZ-Y (Tropical Zea Yellow). The TZ-Y resistance sources express very few streak symptoms (on < 5–30% of the leaf area compared to susceptible lines with streak symptoms > 75%) or the resistant plants initially produce severe symptoms (streaks on > 75% of the leaf lamina) but leaves emerging post-infection show symptom remission, termed as recovery resistance. The IB32 is a classic example of a resistant line derived from Tropical Zea Yellow material bearing quantitative inheritance of resistance to MSV conditioned by 2–3 genes with additive type of gene action and has been used in the development of streak-resistant varieties due to its stability in time and space (Efron et al. 1989; Kim et al. 1989).

In Ladejobi et al. (2018)'s study, lines fell susceptible in the first few weeks of infection but afterwards "recovered" from such symptoms and emerge with healthy upper leaves in time for accumulation of assimilates required for seed development. Garcia-Oliveira et al. (2020) mapped eighteen QTLs for different components of MSD resistance including traits associated with MSD severity, per cent MSD recovery and area under the disease curve (Table 2). Three novel genomic regions (two on chromosome 4 and one on chromosome 7) are narrowed down in their work as effective targets for breeding purposes going forward as they consist of unique sources of phenotypic variation for the reduction in MSV symptom severity. A downside in these most recent works appears to bother on the small size of the F_2 and $F_{2:3}$ segregating populations assessed which raises pointers to the role of large epistatic interactions in explaining the total variance.

The mechanism behind recovery resistance to MSV is attributed to a decline in severity of streak symptoms from the lower (older) leaves to the upper (newer) leaves (Sime et al. 2021). Host plant-related genetic factors at the molecular level implicated in recovery resistance remains to be identified. However, the quantitative nature of this type of resistance suggests the involvement of multiple loci which are associated with different levels of resistance to MSV in maize (Garcia-Oliveira et al. 2020). The partial protection contributed by this type of resistance may suggest a synergistic association with the *Msv1* QTL for durable effects. To develop recovery resistance in already adopted germplasm, gene pyramiding (Andersen et al. 2018) through multiple cycles of marker-assisted backcrossing may be sought.

Validation of these new sets of QTLs in large diverse genetic backgrounds including accurate estimations of genotype by environment effects, fine mapping to improve marker quality, development of their KASP assays and finally a rapid deployment in future maize varieties are key recommendations to enable durability of resistance going forward. The contribution of these other kinds of alleles in addition to the set of KASP assays for *Msv1* already deployed as routine markers will go a long way in ensuring effective field protection against MSV.

Epidemiology and evolutionary potentials of MSV

Several incidences regarding the occurrence of MSV in many countries in sub-Saharan Africa dating back to more than eleven decades have been sufficiently recorded (Marchand et al. 1995; Bosque-Pérez 2000; Alegbejo et al. 2002; Magenya et al. 2009; Shepherd et al. 2010; Monjane et al. 2012; Karavina et al. 2014). A reoccurrence of the disease every 3–10 years is stated as being inevitable, primarily due to environmental influences on the virus' migratory vector species causing an estimated yield loss of between 17 and 100% in a typical epidemic year (Martin and Shepherd 2009; Nair et al. 2015). These estimated losses are detrimental to the future of maize production in Africa and in meeting increasing demands on the continent. For these reasons, quite some attention has been and continues to be garnered in MSV research towards mapping new sources of durable and sustainable host plant resistance (Ladejobi et al. 2018; Garcia-Oliveira et al. 2020). Measuring the impact of this vector-borne virus in order to predict its level of devastation in a single year is done by evaluating the age-old three-pronged factors known to be present for a successful disease development (Martin and Shepherd 2009). These authors reported strong statistical associations between the climatic conditions that favour a proliferation of

Table 2 Genetic Information for performing a meta-analysis for MSV Resistance

Trait	QTL name	Flanking marker	Chromosome	Linkage group	LOD	R^2 (PVE)	Position (cM)	Lower limit of the confidence interval (cM)	Upper limit of the confidence interval (cM)	Author
Major resistance	*Msv1*	PZA00944.1 (L), csu1138.4 (R)	1	1	11.76	0.6736	131.5	131.021772	131.978228	Nair et al. (2015)
Major resistance	*Msv1*	PZA01396.1 (L), PHM17210.5 (R)	3	3	3.46	0.086	225.7	221.954251	229.445749	Nair et al. (2015)
Major resistance	*Msv1*	PZA01919.2 (L), PHM13687.14 (R)	10	10	4.87	0.104	21.2	18.102554	24.297446	Nair et al. (2015)
Recovery resistance	PHM5502_31	PZA00508_2, PZA00667_2	3	3	3.04	0.47	37	34.7446808	39.2553192	Ladejobi et al. (2018)
Recovery resistance	PZA02616_1	PZA000084_2, PHM4135_15	3	3	3.08	0.51	112.2	110.121586	114.278414	Ladejobi et al. (2018)
Recovery resistance	PZA02872_1	PHM2776_11, PZA01154_15	7	7	2.4	0.37	97.9	95.0351351	100.764865	Ladejobi et al. (2018)
Recovery resistance	PHM1766_1	PHM1911_173, sh1_12	9	9	2.3	0.29	33	29.3448276	36.6551724	Ladejobi et al. (2018)
MSVD severity 4WAI	*qMS4wai_4a*	4771778 (L), 2400658 (R)	4	4	5.1	0.133	464	445.024	482.976	Garcia-Oliveira et al. (2020)
MSVD severity 4WAI	*qMS4wai_4b*	2487440 (L), 4776609 (R)	4	4	8.2	0.214	556	544.206	567.794	Garcia-Oliveira et al. (2020)
MSVD severity 4WAI	*qMS4wai_5*	4582804 (L), 4593399 (R)	5	5	3.2	0.078	298	265.643	330.357	Garcia-Oliveira et al. (2020)
MSVD severity 5WAI	*qMS5wai_2*	4771758 (L), 5584933 (R)	2	2	2.5	0.138	414	395.711	432.289	Garcia-Oliveira et al. (2020)
MSVD severity 6WAI	*qMS6wai_2*	2466894 (L), 5583192 (R)	2	2	2.5	0.072	211	175.947	246.053	Garcia-Oliveira et al. (2020)
MSVD severity 6WAI	*qMS6wai_3*	25946752 (L), 4771193 (R)	3	3	3.7	0.113	237	214.665	259.335	Garcia-Oliveira et al. (2020)
MSVD severity 6WAI	*qMS6wai_7*	4580643 (L), 4771917 (R)	7	7	3.0	0.031	68	13.413	149.413	Garcia-Oliveira et al. (2020)
MSVD severity mean	*qMMS_1*	2426379 (L), 24026807 (R)	1	1	2.6	0.073	694	659.427	728.573	Garcia-Oliveira et al. (2020)
MSVD severity mean	*qMMS_3*	21696188 (L), 4584669 (R)	3	3	2.5	0.182	385	371.133	398.867	Garcia-Oliveira et al. (2020)
MSVD severity mean	*qMMS_4a*	4771778 (L), 2400658 (R)	4	4	3.7	0.111	465	442.263	487.737	Garcia-Oliveira et al. (2020)
MSVD severity mean	*qMMS_4b*	2487440 (L), 4776609 (R)	4	4	5.0	0.147	557	539.831	574.169	Garcia-Oliveira et al. (2020)
MSVD severity mean	*qMMS_5*	2432489 (L), 2472175 (R)	5	5	2.9	0.08	386	354.452	417.548	Garcia-Oliveira et al. (2020)

Table 2 (continued)

Trait	QTL name	Flanking marker	Chromosome	Linkage group	LOD	R^2 (PVE)	Position (cM)	Lower limit of the confidence interval (cM)	Upper limit of the confidence interval (cM)	Author
Per cent MSVD recovery	*qPMR_7*	24027065 (L), 4580643 (R)	7	7	3.0	0.122	62	41.313	82.687	Garcia-Oliveira et al. (2020)
AUDPC	*qAUDPC_1*	2426379 (L), 24026807 (R)	1	1	2.5	0.072	692	656.947	727.053	Garcia-Oliveira et al. (2020)
AUDPC	*qAUDPC_4a*	4771778 (L), 2400658 (R)	4	4	3.7	0.114	462	439.861	484.139	Garcia-Oliveira et al. (2020)
AUDPC	*qAUDPC_4b*	2487440 (L), 4776609 (R)	4	4	5.3	0.178	557	542.821	571.179	Garcia-Oliveira et al. (2020)
AUDPC	*qAUDPC_5a*	2432489 (L), 2472175 (R)	5	5	3.0	0.078	387	354.643	419.357	Garcia-Oliveira et al. (2020)
AUDPC	*qAUDPC_5b*	2506202 (L), 4583014 (R)	5	5	2.7	0.07	436	399.945	472.055	Garcia-Oliveira et al. (2020)

WAI, weeks after inoculation, *AUDPC* area under the disease progress curve, *MSVD* Maize streak virus disease *LOD* Logarithm of Odd, R^2 = Phenotypic variance explained

leafhopper populations, the variety of choice grass populations available as alternative hosts during the insect's mating season (e.g. maize and wild grass hosts like *Digiteria sanguinalis* and *Bracharia* spp.) and the composition of strains of MSV present within the leafhopper vectors and on plant host species.

It is imperative to state that up-to-date information on vector population dynamics and MSV strain composition within the guts of plant leafhopper vectors at multiple locations in SSA is lacking in the literature and is cautioned in this current review to be crucial if accurate pest and disease management protocols are to be designed. A knowledge of the variation in strain composition present on farmers' fields using appropriate molecular and modern virus tracing tools will improve prediction and estimation of recombination rates and as such will present options for effective genetic strategies to tackle, prevent or alleviate huge crop losses in a given year or cropping season.

With MSV-A being the dominant strain and present in a significant number of countries in sub-Saharan Africa, studies relating to its co-occurrence with the other ten known strains of the virus and the role of such associations in increasing virulence or enhancing evolution is limited (Martin and Shepherd 2009; Monjane et al. 2012). In addition, it is stated that mixed infections of MSV-A and MSV-B are quite naturally occurring and as such validates genetic recombination as potential mechanisms for evolutionary adaptation on the part of the virus.

It is also evidently documented that a few strain variants of the MSV-A (for instance, the MSV-A_4 in South Africa) and even the first known MSV-A itself evolved as a result of inter-strain or interspecies recombination event within the eleven known MSV strains (Varsani et al. 2008; Martin and Shepherd 2009). This serves as a pointer to the obvious risk of evolution of other crop-adapted MSV virulent strains through any such process. With this in mind, prevention of future epidemics must follow proactive and modern steps.

Preceding an outline of these proactive steps, a close look at some genomic resources that are presently available through a consensus of QTLs as extracted from a meta-analysis of all mapped streak resistance loci using SNP molecular markers is detailed. This piece of knowledge can be wired into the fabrics of future maize improvement programmes for onward routine breeding. Breeding strategies that encourage the incorporation of moderate- to minor-effect QTLs located in newer germplasms are discussed posteriorly.

A query into current genomic resources for future improvement for streak resistance

With decades of elaborate studies on resistance to MSV, many scientists have dissected various mapping populations developed from diverse sources of streak resistance to

understand the genetic architecture of this trait. Projecting the information generated over the years onto a consensus map and subjecting such to a meta-analysis is crucial so as to greatly simplify the inventory of candidate genes and sufficiently unravel all genetic factors underlying MSV disease resistance (Sosnowski et al. 2012). A successful implementation of a meta-analysis for MSV will serve to identify consensus genomic regions controlling the trait of interest and, hence, facilitate the development of efficient markers for use in marker-assisted breeding (Ayenan et al. 2018, 2019).

Rossi et al. (2019) did a meta-analysis of QTLs for resistance to fungi and viruses in maize using a total of 110 previous studies to identify genomic regions carrying primarily major-effect QTLs for resistance. The method used focused on accounting for the positions of QTL with relatively large effect for the phenotypic variation, depending heavily on an odds ratio approach rather than the use of a reference map. The perspective of the current review is that the latter approach is equally as important since it is effective in identifying clusters of both major- and minor-effect loci rather than highlighting only genomic regions (bins) bearing major-effect QTLs. Rossi et al (2019) claim that such meta-analysis done with a reference map is "traditional" citing inadequacy of reference genome and an inability to locate the physical position of some genes or QTLs on the chosen version of a reference genome. They, thereafter, admitted to the possibility of doing a meta-analysis using other approaches apart from theirs which could further "increase the power of molecular breeding."

Moreover, for the particular case of the MSVD and its associated resistance QTLs, we combined all articles that utilized diverse genetic markers including the restriction fragment length polymorphism (RFLP) together with those that used high-density markers like SNPs in mapping *Msv1* QTL (Welz et al. 1998; Kyetere et al. 1999; Pernet et al. 1999a, b; Lagat et al. 2008; Nair et al. 2015). Updated information including the recent mapping of minor-effect QTLs (recovery resistance) for MSV has come to light (Ladejobi et al. 2018; Garcia-Oliveira et al. 2020) and was not included in Rossi et al. (2019)'s study. These newly mapped QTLs ought to be reflected in a discourse aimed at promoting durable levels of MSV disease resistance in future released varieties for sustainable maize production.

Since not much literature is available on the fine mapping and validation of the minor-effect QTLs related to this adaptive trait, their exact delimitations in the maize genome with well-defined confidence intervals and their subsequent inclusion into routine screening prior to varietal release by both national and international programmes have been limited if not completely excluded. It has been emphasized (Nair et al. 2015; Garcia-Oliveira et al. 2020) of the need to avoid an overreliance on a singular source of major-effect resistance as this could result in a possible breakdown of the *Msv1*-mediated resistance in the long term since multiple strains of MSV-A are reported to sporadically co-occur with the most virulent MSV-A_1 strain in different parts of SSA. Expediting genetic gain in breeding for stress tolerance can be seamlessly achieved with the use of more markers linked to important adaptive traits such as MSV resistance, especially when selection for other stress-related traits such as heat and drought tolerance is major considerations (Nair et al. 2015).

For the present enquiry, a meta-analysis for MSV disease resistance was performed and a consensus map was constructed by leveraging on the BioMercator software version 4.2.3. The aim of undertaking this task was to detect co-localized QTLs also known as meta-QTLs (MQTLs), if any, associated with both major and recovery resistance to MSV by following described procedures (Veyrieras et al. 2007). To do this, three journal articles out of all other mapping studies were selected for obtaining QTL information principally because of the use of single-nucleotide polymorphism (SNP) molecular markers in ascertaining the intervals of interest in their studies (Nair et al. 2015; Ladejobi et al. 2018; Garcia-Oliveira et al. 2020). Necessary data including logarithm of odd (LOD) values, R^2, confidence interval (CI) and position in cM required in the generation of a QTL map input were extracted from these studies as shown in Table 2. A 95% CI for each QTL was computed using the formulae $CI = 530/(R^2 \times N)$ for F_2 populations (Darvasi and Soller 1997), with N representing the size of each mapping population and R^2 as the proportion of variance explained by each QTL. The start and end positions of each CI were derived as Lower limit = (QTL's position − CI/2) and upper limit = (QTL's position + CI/2).

A maize genetic map, IBM SNP50 corresponding to the genome of maize germplasm B73, was downloaded in a text format from the maize database (www.maizegdb.org 2020) and was used as the reference map. The information was transferred to an excel spread sheet and formatted to fit the BioMercator file format before being converted back to a text format and called the Map file for onward upload into the package. With the compiled information, a consensus map was constructed using the QTL input file and the IBM SNP50 map file. The formatted Map files and QTL file are made available upon request.

In all, a total of twenty-five QTLs from three most recent SNP-based mapping experiments were identified and uploaded into the package. Among the 25 QTLs are loci identified for MSV resistance that are found on chromosomes other than chromosome 1 where *Msv1* has been mapped. Details regarding these QTLs, their phenotypic variance explained as well on which chromosomes they are located are documented in Table 2. Two of the recent SNP-based mapping studies reported varying degrees of tolerance to MSVD if the QTLs mapped in their study are utilized. For 18 identified

QTLs associated with different components of MSD resistance, 3.1–21.4% of the phenotypic variance was accounted for (Garcia-Oliveira et al. 2020). Of four QTLs identified in the other study, two putative QTLs with significant effect on chromosome 3 together accounted for 47–51% of the total phenotypic variance while the other two QTLs with reduced effect on chromosomes 7 and 9 accounted for 28 to 32% of the total variation (Ladejobi et al. 2018). However, the study recorded no detection of co-localized QTLs when the option "Meta-Trait" was chosen to detect Meta-QTLs. A meta-QTL can be identified where there is a cluster of QTLs (co-localizations) such that the genomic region can be targeted for candidate gene identification. Therefore, no meta-QTLs were projected onto the B73 maize reference genome; rather, only 5 individual QTLs were visualized on the consensus map out of the 25 QTLs initially imputed (Fig. 2) with two of such found on chromosome 3 and the rest on chromosome 7.

A close look at the 5 QTLs projected onto the consensus map showed the mean of the difference between the lower and upper limits of the confidence intervals of each of the two QTLs on chromosome 3 (qLT1 and qLT2) to be 4.33 cM with high R^2 values above 40 (Fig. 2). On chromosome 7, qLT3 spanned an interval limit of 5.73 cM with an R^2 value of 37; qGT7 spanned the entire chromosome with a low R^2 value of 3.1, whereas qGT13 spanned an interval of 41.374 cM with an R^2 value of 12.2. Although no meta-QTLs were detected in the present meta-analysis, the QTLs with high R^2 values (qLT1, qLT2, qLT3 and qGT13) individually accounted for significant phenotypic variation for MSV recovery resistance. Each of these can be targeted for conferring durable streak resistance through marker development for marker-assisted selection or varietal conversions in future maize breeding efforts.

Whereas a very large confidence interval would be of limited importance in the identification of genes and their onward application in plant breeding, overlapping regions could be of interest in the subject of fine mapping. This is seen in the genomic regions where qLT3 and qGT13 overlapped on qGT7 (Fig. 2). It is worthy of reiteration that the aforementioned QTLs were mapped in two separate sources of recovery resistance and would inform future hybridization efforts towards streak resistance improvements. These five unique QTLs all fall within chromosomes separate from chromosome 1, which is known to bear the major-effect QTL, *Msv1*.

Resistance status of released cultivars in Africa

Consistent release of maize hybrid varieties on a yearly or biannual basis is a key target of national and international maize breeding programmes all around the globe, especially seed companies, who are at the top of their games in terms of utilizing favourable alleles for product enhancements and recombination. Improvement of breeding materials with adaptive traits like MSVD and maize lethal necrosis (MLN) resistance along with possession of other desirable agronomic, nutritional, culinary and organoleptic features are key areas where advocacy is highest even as attention is shifted towards stress-tolerant, locally adapted and adoptable varieties that can brace up to the vagaries of a changing climate (Abate et al. 2017; Bailey-Serres et al. 2019).

A recent survey of maize cultivars grown in Africa was done during the 2013/2014 main cropping season in 13 countries and it brought to light the overdue replacement of the entire maize seed system operational from southern Africa to the eastern and western Africa, with newer and more climate-resilient maize hybrids and varieties (Abate et al. 2017). It was noted that the average age of maize cultivars grown on the continent varied significantly. The average age of hybrid maize cultivars for SSA was 13 years, whereas OPVs were more than 18 years old, with the overall weighted average of all cultivars being 15 years. Pockets of literature suggest cases of general improvement of cultivars for resistance to MSV using the major quantitative resistance, *Msv1*, as the only check for resistance, but this should not be the case going forward (Ladejobi et al. 2018).

Hybrid varieties are widely adopted in Southern Africa and used by more than one country including PAN53 (popularly grown in Malawi and Zambia), SC513 (in Zimbabwe and Zambia) with a slight difference for PAN67 (popularly grown in both the East African country of Tanzania and Mozambique). All these hybrids have been in existence for not less than 10 years and were developed by ensuring at least significant tolerance to streak virus. PAN53 is medium-maturing, flint-grain, white hybrid maize variety of a three-way cross that is tolerant to MSV and with a grain yield of 13.87 tonnes/ha (Tripp and Ragasa 2015; Mubanga et al. 2018). SC513, a three-way hybrid under co-infection with turcicum leaf blight, had a disease score of 2.433 while scoring 2.0 in the streak disease rating with a grain yield of 10.35 tonnes/ha (Karavina et al. 2014). The reactions of PAN67 (also white, low to medium-maturing flint type grain hybrid) to MSV under chemical and non-chemical fertilization in community plots in Gandajika, DR-Congo were rated 1.5 and 1.2, respectively (Lyimo et al. 2014; Mbuya et al. 2010).

In West Africa, special cases of varieties such as EV8443SR, DMRESRW, EV8430SR developed and released for southern Benin and TZBSR and TZPBSR for the northern parts of Benin Republic over the last 15–20 years as well as drought-tolerant open pollinated varieties including TZECOMP3DT, MVDC2SYNF2, DTSRWC2 and EV97DTSTRW released over a decade ago (Abadassi 2013) by the joint efforts of CIMMYT, IITA and the national programme housed at the Institut National

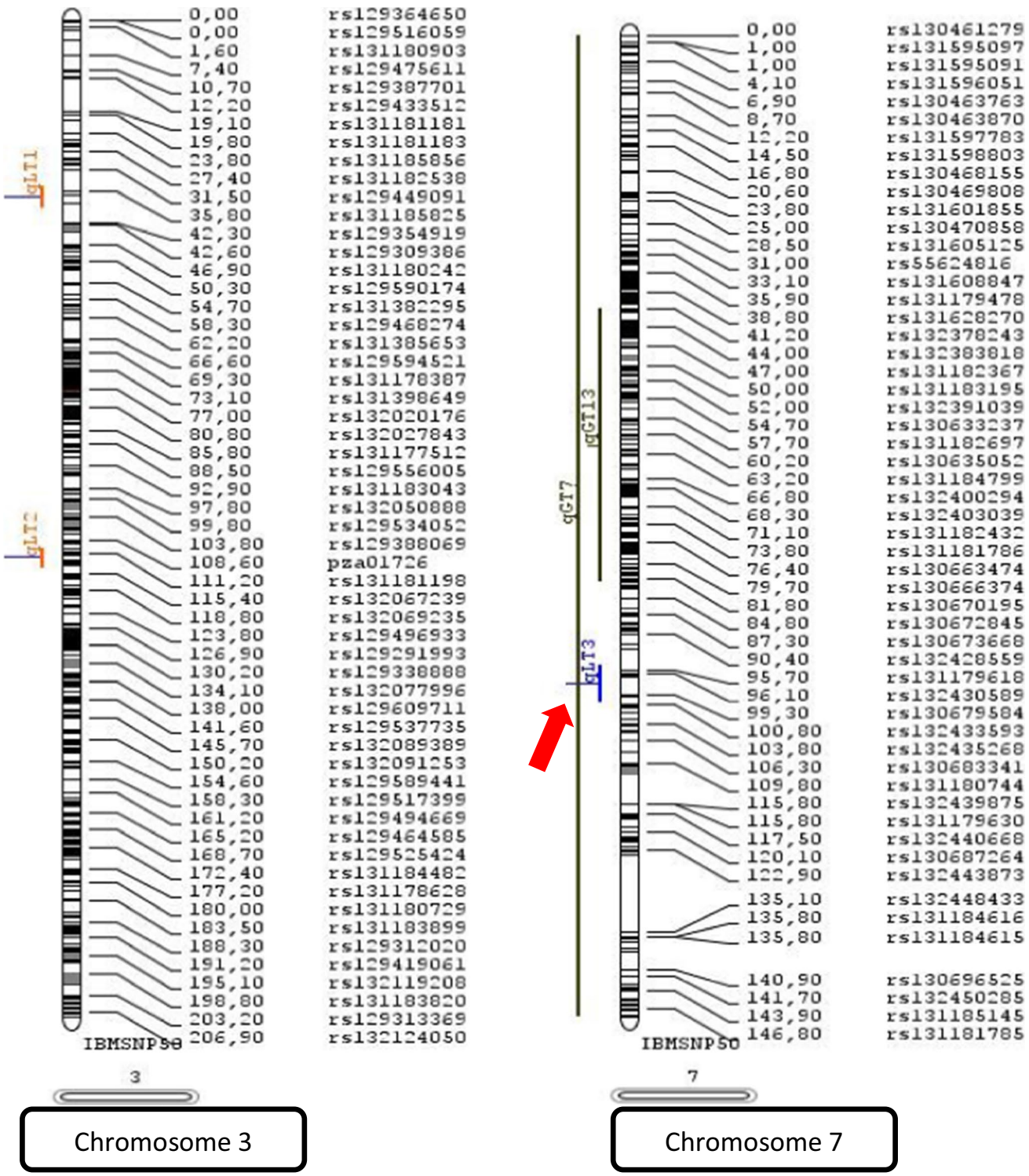

Fig. 2 Mapped QTLs for MSV Resistance aligned to the B73 maize reference genome

des Recherches Agricoles du Bénin (INRAB) are a few examples of areas for continuous improvements for MSV resistance. It is most probable that the varieties mentioned had been previously improved with *Msv1* since they were released after the 1980s (Efron et al. 1989; Bosque-Pérez 2000).

This status for varieties grown in Benin and other countries in Western Africa is not far from those cultivated in

Southern, Central or Eastern Africa as the source of resistance deployed has been relegated to the singular QTL present on chromosome 1 in Maize and used for screening of seeds for phytosanitary certifications or for all kinds of enhancements towards MSV when improvements for other traits are ongoing.

Transgenic enhancements by employing replication proteins engineered into maize germplasms that inhibit the gene expression on the part of the virus (Shepherd et al. 2007b; Reddy et al. 2009; Owor et al. 2011; Rani and Usha 2013) has barely seen the light of day in terms of being used in the conversion of cultivated maize varieties for enhanced resistance to MSV. It is of course not a debatable topic since there hovers a societal as well as regulatory circumscription on the use of such biotechnological outputs even though no sufficient evidence has supported any harmful claims conclusively.

Modern approaches to improve maize for enhanced streak disease resistance

To appreciate the need for continued and sustainable exploitation of MSV resistance sources for enhancing maize varieties towards durable streak resistance, a short analysis of the work that has been done so far and what more has to be done is imperative. This will help in shaping the pathway for current maize breeding programmes in keeping a great pace ahead of any unforeseen viral evolution and thereby maintain durable resistance to MSV in future released varieties. By the end of the 1980s, Efron et al.(1989) reviewed the extensive work done through the cooperation between CIMMYT and IITA scientists in partnership with several of Africa's national research programmes in finding and developing an array of MSV resistance sources and using such in the enhancement of diverse kinds of maize populations that suited different markets and ecologies. A common understanding about the genetics of the resistance to MSV in such sources was that the resistance was quantitative and so followed a non-mendelian inheritance. More than two gene pairs were revealed to be responsible for the phenotypic variation observed (Kim et al. 1981, 1989) and coupled with the evidence of unique and different streak symptoms observed on different MSV-resistant inbred lines, and the involvement of minor genes was strongly suggested, although the location of the genes on the maize genome was not discussed extensively.

By then, three basic types of breeding methodologies with slight modifications had already been proposed and elaborately used for achieving what was termed a flexible breeding strategy to meet the various requirements of social and ecological demands. They were the following: population improvement through recurrent selection, modified backcross conversion programme, and inbreeding and hybridization. The resulting improved germplasm gotten from each rigorous methodology in combination with other breeding goals like downy mildew resistance, which was also an economically important disease at the time, produced a diverse range of improved populations with higher grain yields and stronger MSV resistance and also inbred lines that could be used for the production of open pollinated synthetics or to develop hybrids and/or top crosses and so on, which were sent to the national research programmes for continued improvements for other traits, yield stability analysis and onward release.

From the review, it was documented that the modified backcross conversion approach gave better steak-resistant varieties at the second backcross generation and even greater resistance, yields and stability at the fourth generation if open pollinated varieties were used as the initial recurrent parents. Works on MSVD resistance after 1990 bordered partly on the discovery that there were more than one isolate of the MSV causing the disease (Mesfin et al. 1992; Martin and Rybicki 1998). Other scientists continued on to improving for complete or partial resistance having found that the resistance for MSV was under a genetic control involving loci with major dominant genes and other loci with minor genes that confer partial resistance (Rodier et al. 1995). A great deal of resources went into the mapping of genes conferring resistance to the streak virus, especially with the advent of biotechnological tools like genetic markers that can help in detecting the targeted loci or gene in a population under improvement.

Restriction fragment length polymorphism (RFLP) genetic markers were extensively used to study and/or map both major and minor genes/QTLs found to be located on several chromosomes of maize (Welz et al. 1998; Kyetere et al. 1999; Pernet et al. 1999a, b) while simple sequence repeat (SSR) markers were explored (Lagat et al. 2008) in mapping the major MSV-resistant genes or QTLs on chromosome 1 and was subsequently used in marker-assisted selections by the end of the first decade in the new millennium (Abalo et al. 2009; Asea et al. 2012). More recently as already stated in this review, single-nucleotide polymorphism markers have been employed in mapping and fine mapping the major-effect QTL on chromosome 1 (Nair et al. 2015). Even the techniques and technologies employed over the years towards specifically, rapidly and more efficiently tracking the target gene(s) or loci of interest have evolved from the use of a fragmentized portions of DNA (as in RFLP) to a more allele-specific and/or nucleotide-specific array (as in SNP). To this end, technologies including the kompetitive allele-specific PCR (KASP) assay have now been employed in the production of high-throughput production markers for the *Msv1* QTL

on chromosome 1 and are now used for routine screening of breeding pipelines (Nair et al. 2015).

This singular feat towards a particular locus on just one of the chromosomes where resistance is found raises a number of questions, especially since a meta-analysis carried out in the present study confirmed the other minor loci to be non-congruent, and as such can be exploited independently. The following questions are put forward: (i) How can the synergistic roles of minor-effect QTLs found on other chromosomes of maize apart from chromosome 1 be optimized and successfully brought into elite genetic backgrounds so as to boost resistance? (ii) Where, precisely, are they located on each chromosome so as to facilitate marker-assisted selection? (iii) What additional phenotypic variation do they account for if utilized for population improvement and/or in hybrid development as well as their stability in sub-optimal production environments like drought-prone areas? (iv) What breeding approaches/methodologies as well as technological advancements can be utilized to fast-track selection in a forward breeding programme? And finally, (v) Are there available plant genetic resources that bear these minor-effect loci and are sufficiently genetically broad-based bringing other desirable agronomic traits? To answer these questions, several considerations are put forth under different themes as outlined below.

Marker-assisted breeding

Since plant breeding takes advantage of the dynamics of population genetics, improving streak resistance in highly cross-pollinated crops like maize will require a higher frequency of minor-effect alleles, if they are to be represented in a breeding population. This is irrespective of the breeding method adopted or mating design implemented. Boosting the frequencies of minor-effect alleles in segregating populations of a breeding programme should always inform the choice and quantity of germplasms being used as parents in the initial crosses. This has to be a conscious effort to enhance durability to MSV resistance. Deliberately broadening the genetic base of an actively mating population by incorporating more than one donor parent bearing minor-effect streak resistance relative to that harbouring major-effect resistance will amount to a potential doubling or tripling of the proportion of the targeted allele frequency and result in a greater probability of the minor-effect loci being successfully incorporated and discovered in developed progenies. A higher success in selections using markers can be achieved in order to rapidly advance progenies bearing all desirable gene combinations for further recombination. As a hypothetical example, consider a marker-assisted backcrossing (MABC) integrated in a Pyramiding scheme put forward below in order to enhance local broad-based maize cultivars towards enhanced resistance (Fig. 3).

Briefly, the recurrent parent (RP) can be a susceptible cultivar or a germplasm possessing some level of resistance, which could be major or minor effect or in combinations. The donor parents are listed as follow: DP A (mmAACCbb—bearing alleles for at least two minor-effect QTLs, A and C) and DP B (mmaaccBB—bearing alleles for one minor-effect QTL, B) as sources of minor-effect QTLs while DP is a MSV-resistant inbred line (MMaaccbb—bearing the major-effect QTL known as the *Msv1*). Crossing each donor separately at the first season with the RP (mmaaccbb—susceptible at all target loci) to produce the F_1 (mmAaCcbb, mmaaccBb and Mmaaccbb) and then backcrossing to the RP gives a population of BC_1F_1 lines with diverse recombination during the second season.

At Season 3, all BC_1F_1 lines will be genotyped and only progenies that bear appreciable recombination from each set of backcrosses (i.e. mmAaCcbb, mmaaccBb and mmaaccbb) will be selected for selfing to produce BC_1F_2 and for pyramiding among any two sets of backcrosses, i.e. mmAaCcbb × mmaaccBb; mmaaccBb × Mmaaccbb and mmAaCcbb × Mmaaccbb. This will yield mmAaCcBb (for mmAaCcbb × mmaaccBb), MmaaccBb (for mmaaccBb × Mmaaccbb) and MmAaCcbb (for mmAaCcbb × Mmaaccbb). The resulting genotypes are selfed separately at Season 4, and also a pyramided cross is made at the same time between mmAaCcBb and MMaaccbb, which is a progeny of Mmaaccbb from BC_1F_1 to give MmAaCcBb. By Season 5, it is only the selfing of MmAaCcBb from Season 4 that will result in MMAACCBB (Fig. 4).

In the hypothetical example, the probability of obtaining the genotypes bearing the target alleles at each season displayed above depends on number of plants genotyped in the population. A 95% chance of obtaining 1 desired genotype was gotten by the formula: LN(1-0.95)/LN(1-Probability Value). Getting two plants then means multiplying the total number of plants required to obtain one plant by two and so forth (Table 3).

Due to recombination during segregation, probabilities of finding the alleles of the minor-effect QTLs in a population are low, and hence, more plants have to be genotyped in order to achieve successful outcomes. At a probability of 0.0039 for finding MMAACCBB, a complete representation of all target alleles, it will take not less than 765 plants to be genotyped in order to find 1 desired genotype and 1531 for 2 such genotypes and then 2295 plants to obtain 3 plants with complete dominance at all loci. All the while, appropriate markers will have to be employed in distinguishing desired genotypes and advancing them to the next generation.

The genetic gain from selection for each season can only be appreciated when increased phenotypic variation can be accounted for following marker-assisted selection and field evaluation under well-monitored artificial infestation but an even greater gain will suffice with a boost in yields and

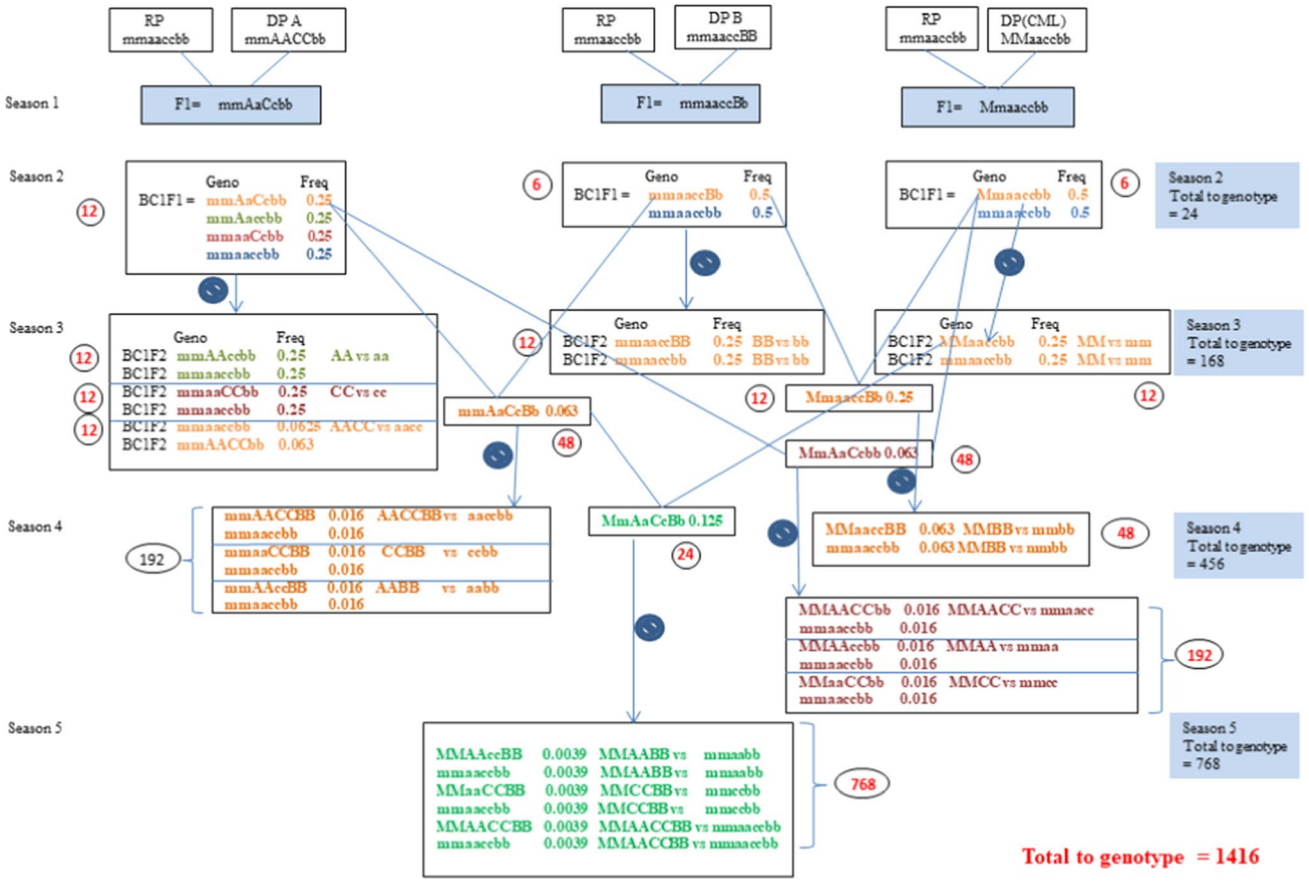

Fig. 3 Marker-assisted backcrossing scheme for germplasm improvement against MSV

other desirable traits as required during overall population improvement and/or hybrid development for sub-optimal production environments like drought-prone areas.

Genomic selection (GS)

Improvements employing MAS or MABC involving minor-effect QTLs require deep knowledge of trait inheritance and resolute mapping in order to efficiently call markers that influence the phenotypic variation being sought. These requirements are not fulfilled when GS is implemented. Rather, a reliance on statistical models built on the inclusion of all functional marker information acquired through the extensive use of molecular markers is sufficient to estimate genomic breeding values. This approach takes into consideration most of the variation arising from the minor-effect QTLs as captured in the prediction model (Ayenan et al. 2019). Hence, it is assumed that enhancing maize germplasm for recovery resistance to streak disease could be significant as well as time saving with a more precise estimation of genetic gain when a large number of QTLs are being utilized.

Plant genetic resource availability

Answering the call for enhanced resistance to the streak virus actually begins with obtaining and/or discovering appropriate germplasms bearing the target loci of interest and that also have synchronized and desirable agronomic traits as well as good combining ability with locally adapted but susceptible genetic materials. Minor-effect QTLs conferring MSV resistance have been reported to be in play in germplasms like CML 202 (Welz et al. 1998), CIRAD390 (Pernet et al. 1999a), D211 (Pernet et al. 1999b) and those conferring recovery resistance have been found in TZIL07A01005 and TZIL07A01322 (Ladejobi et al. 2018), and in (KU1414 × 9450) × 9450)-15-2-1-BBB-1-B*11 (Garcia-Oliveira et al. 2020) to name a few. A majority of the above-mentioned lines are inbred lines obtainable from CIMMYT, IITA and from CIRAD in France. Some germplasms like CML 202, D211 and CIRAD390 are reported to carry the major-effect loci, *Msv1* on chromosome 1 of maize as well as modifying or minor-effect genes found on chromosomes 2, 3, 4, 5, 6, 8 and 10 while *Msv1* was reported to be absent in TZIL07A01005 and TZIL07A01322 but instead possess recovery resistance effects contributed by QTLs

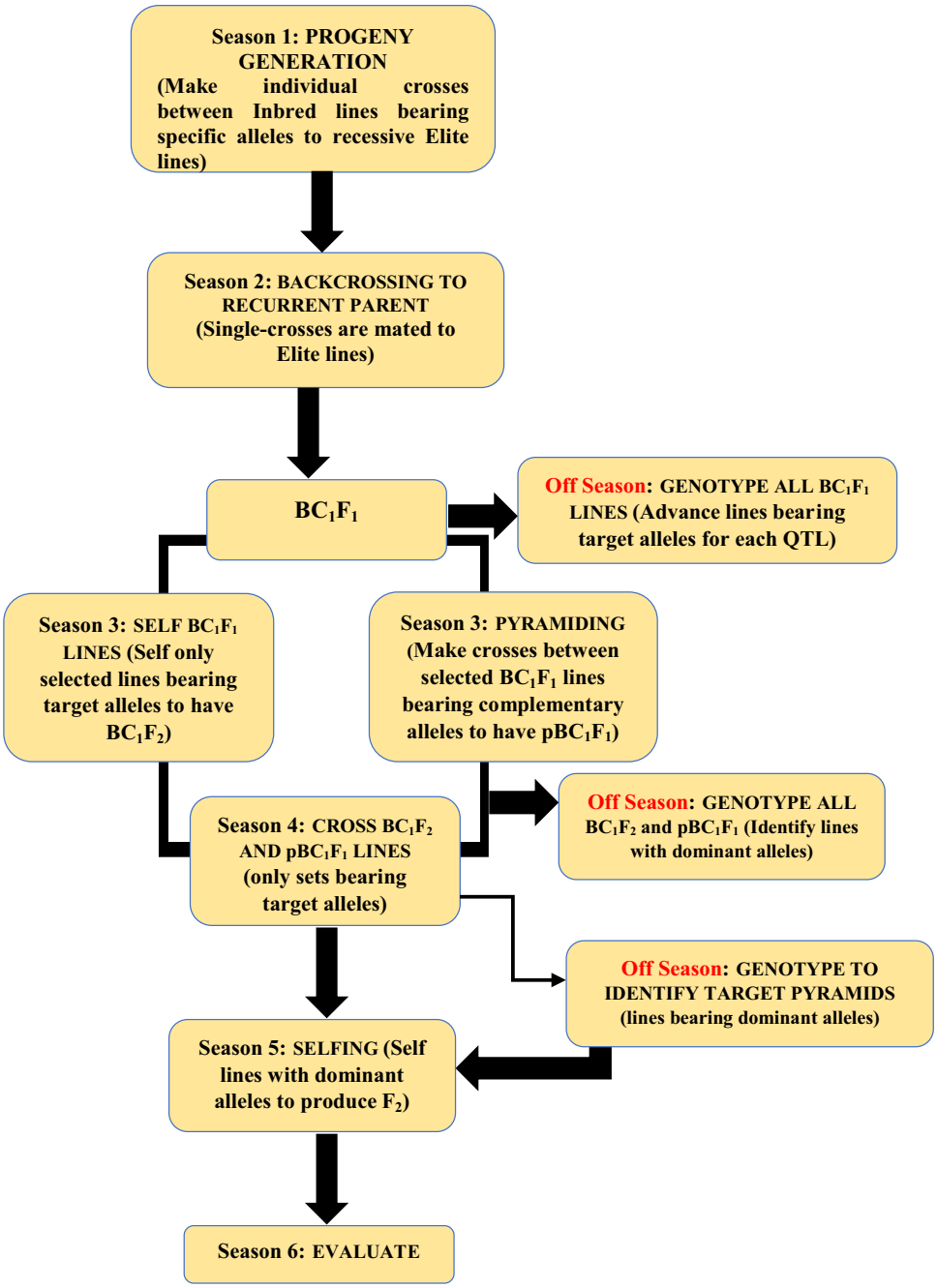

Fig. 4 Pathway for the enhancement of elite lines through the stacking of favourable alleles of major- and minor-effect QTLs conferring streak resistance in Maize (*Zea mays* L.) pBC1F1: pyramided backcross progenies; BC1F1: first progenies of a backcross; F2: progenies developed by selfing a previous generation)

Table 3 Number of plants to genotype to be 95% certain of 1, 2 or 3 genotypes with target alleles

Probability of genotype	No of plants to genotype to be 95% certain of 1 plant with desired genotype	No of plants to genotype to be 95% certain of 2 plants with desired genotype	No of plants to genotype to be 95% certain of 3 plants with desired genotype
0.5	4	9	13
0.25	10	21	31
0.125	22	45	67
0.0625	46	93	139
0.015625	190	380	571
0.0039	765	1531	2296

present o chromosome 3, 7 and 9. Utilizing these lines in the conversion of current maize cultivars and elite varieties will speed up the goal of durability for streak resistance, especially when appropriate breeding methodologies are applied and allele-specific marker technologies are leveraged upon.

Creating new germplasm through inter-mating resistance sources

Due to the nascent state of maize hybrid production in many parts of SSA, synthetic cultivars could be a viable option for future improvement of maize germplasm where seasonal production of hybrids is not feasible. This could be achieved by inter-mating six or more inbred lines bearing all known and recently developed sources of streak resistance and planting the resulting seeds as cultivars. This takes advantage of or exploits some amount of hybrid vigour while minimizing the inbreeding depression that results from open pollination in hybrid development (Bernardo 2002). These can be released directly to farmers or plugged in as breeding materials in new cycles of marker-assisted recurrent selections targeting other important traits.

Technological/laboratory facilities

Efficiency of selections in plant breeding has been countlessly proved to be enhanced by the use of molecular markers. The presence or absence of whole gene segments or quantitative trait locus or even a variation in a single-nucleotide base, which accounts for the manifestation of a phenotype that is progressive or deleterious, has been distinctly observed through the use of the several kinds of genetic markers that act as signposts to the location of the observed differences. Data generated from the sole use of these technological advancements or when combined with actual measured phenotypic traits have aided in drawing sufficient inferences and making informed, rapid and accurate selections leading to genetic gains. The current availability and wider accessibility to high-throughput sequencing facilities and steadily reducing costs of genotyping continues to facilitate a cut down in time and resources required to achieve marker-assisted breeding and hence quicker release of improved varieties to farmers.

The question regarding where the minor-effect loci are located on each chromosome and the methods and facilities necessary to efficiently select genotypes bearing them in a population is important to aid the development of effective markers for marker-assisted selection. Recent advances in genotyping and marker development technologies include those that are able to distinguish between alleles at even a single nucleotide. They include: kompetitive allele-specific PCR technology (KASP), tetra-primer amplification refractory mutation system–polymerase chain reaction (Tetra-ARMS PCR), among others. Both technologies mentioned are used to genotype single-nucleotide polymorphisms (SNPs) although the number of primers used differ. The Tetra-ARMS PCR is a simple and economical method to genotype SNPs using four primers in a single PCR and is followed just by gel electrophoresis while KASP genotyping assays are based on competitive allele-specific PCR and enable bi-allelic scoring of SNPs and insertions and deletions at specific loci. The key point being that such kinds of technology can be proactively explored for use in routine screening of major- and minor-effect resistance loci in forward breeding for other target stresses, especially drought, which is known to be influential to MSV epidemics.

Human resources

Handling selections involving minor-effect loci, especially in large populations, are as much a technical endeavour as it is subjective. Skills necessary for on-field visual scoring as well as genotyping using the modern biotechnological tools are required for drawing informed inferences and making accurate selections, especially during routine screening in preliminary and advanced trials. National and International maize breeding programmes must be sufficiently equipped and upgraded to handle modern equipment as well as newer protocols and statistical analyses required for critical adaptive traits such as this.

Broadening MSV resistance in maize germplasm requires a clear breeding programme outline that strategically utilizes modern breeding tools in introgressing this all-important adaptive trait that is most relevant to the sustainability of the maize production system of SSA. It is proposed that the maize breeding strategy for SSA going forward should be such that constantly incorporates inbred lines that possess new sources of recovery resistance into MAGIC (multi-parent advanced generation intercross) populations for the purpose of broadening the genetic base. A continuous effort is to be expended in fine mapping of mapped genomic regions and validation of genetic markers linked to streak recovery resistance and then in utilizing these markers for selections following high-throughput phenotyping. The outcome of this strategy will result in the development of functional markers for routine selections by breeders and production of climate-resilient recombinants and cultivars that can be released for adoption in the region.

Conclusion

This review has sought to bring to light the insufficiency of the control measures presently being deployed towards a virus as much a threat today as it was potent at first discovery over 11 decades ago. Even as host plant resistance

remains the best course of action for its inestimable evolutionary regimen, more need to be done towards incorporating the minor-effect QTLs that have been mapped for recovery to streak disease in maize plants. Along with all other constraints to the optimum potentials of maize production in SSA, the imminent change in climatic conditions has brought the realities of water stress to bear and with this, a need to tighten the belt on the resilience of maize, especially as this is a predisposing factor to an MSV epidemic.

Private and public maize breeding programmes targeting future releases of hybrid and synthetic varieties for SSA must embrace newer genomic tools in selections for key adaptive traits including streak resistance. A widening of the gene pool at the preliminary stages of hybridization to accommodate inbred lines with several forms of minor streak resistance while maximizing the benefits of hybrid vigour contributed during the interplay and yet applying genomic selection as the trials advance would more than triple the genetic gain over cycles and dollar spent.

None of these propositions would hold if ardent attention is not given to efficient validation of the newest sets of minor-effect QTLs in large diverse genetic backgrounds including accurate estimations of their genotype by environment effects, and a fine mapping to improve marker quality, with the development of a chip of KASP assays for future routine screening against MSV.

Acknowledgements The authors wish to express their gratitude to Mr. Mathieu T. Ayenan for his immense guidance in the use of BioMercator software for meta-analysis of QTLs conferring MSV resistance. The assistance of Prof. Clay Sneller of Ohio State University, USA, towards the meticulous construction of a marker-assisted backcross scheme for incorporating minor-effect QTL into a breeding programme is highly appreciated. Also, Dr. Alcade C. Segnon and Mr. Felicien Akohoue were profoundly helpful with general literature search using the web search engines, Scopus and Web of Science. We appreciate the Intra-Africa Academic Mobility Program (GENES) of the European Union as well as the International Foundation for Science (IFS), Sweden, for funding the first author.

Author Contribution EGAD and ME designed and outlined the manuscript. ME wrote the first draft of the manuscript as part of the literature review for her PhD dissertation and EGAD critically revised the work while all authors commented on previous versions of the manuscript. All authors read and approved the final manuscript.

Funding The first author is a PhD candidate under the scholarship awarded by the Intra-Africa Academic Mobility Program (GENES) of the European Union as well as a grantee of the International Foundation for Science (IFS), Sweden.

Availability of data and material Files used for meta-analysis are freely available on request and the B73 maize reference genome was downloaded from www.maizegdb.org.

Declarations

Conflicts of Interest The authors declare no conflict of interest.

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