Endemism and reemergence potential of the ipomovirus *Sweet potato mild mottle virus* (family Potyviridae) in Eastern Africa: half a century of mystery

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ABSTRACT

Viruses have the ability to frequently colonize new hosts and ecological niches because of their inherently high genetic and evolutionary plasticity. However, a virus may emerge and remain of no or less economic importance until changes in viral and/or environmental factors dictate its epidemiological status. An example is sweet potato mild mottle virus (SPMMV), which was first reported in the 1970s on sweetpotatoes in eastern Africa, has remained endemic in the region and poorly understood, yet accounting for 60-95% losses especially in mixed infections. Unlike other sweetpotato viruses which have a global incidence, SPMMV has never been confirmed outside eastern Africa. This implicates the region as its center of origin, but does not fully account for SPMMV's exclusive geographic delimitation to eastern Africa. Despite its importance, several mysteries and research gaps surround SPMMV, which decelerate efforts for effective virus disease management in sweetpotato. The aim of this review is to articulate research gaps, propose pivotal scientific directions and stimulate knowledge generation for better management of virus diseases in sweetpotato. Vector-mediated transmission of SPMMV remains enigmatic. Here we postulate testable hypotheses to explain SPMMV transmission. Comparisons between SPMMV and cassava brown streak ipomoviruses demonstrate epidemiological "hallmarks" for monitoring SPMMV. Evolutionary forces on SPMMV coupled with the virus' broad host range imply a 'silent build up' of better fit variants in a changing climate, and this could explode into a worse disease conundrum. These information gaps need urgent filling to ease future management of virus disease emergences in sweetpotato.

Key words: Co-infection, Eastern Africa, Helper component protease, Helper virus, *Ipomovirus*, Mixed infections, Phylogeography, Sweetpotato (*Ipomoea batatas*), Sweetpotato reversion, Vector-

mediated transmission, Virus disease epidemiology, Virus emergence, Virus evolution, Virus tropism, Virus-vector relationships

INTRODUCTION

Viruses are not constrained to perpetually occupy a single ecological niche (Malmstrom et al., 2011; Roossinck, 2013; 2015; Lefeuvre et al., 2019). This is because viruses have inherent genetic and evolutionary plasticity which enables acquisition of better fitness with potential to continuously expand their host and geographical ranges (González et al., 2020). A plant virus may "emerge" and remain of no or less 'extraordinary' economic importance until viral and/or environmental factors influence its reemergence and exacerbated virus disease effects (Gibbs et al., 2010). Accordingly, plant virus emergence is a complex process driven by an interaction of genetic and ecological variables. First, the virus acquires ability to infect a new host, followed by adaptation by ensuring successful transmission between hosts, and finally gaining ability to spread epidemically (Rojas & Gilbertson, 2008; Elena et al., 2014; Lefeuvre et al., 2019).

Apparently, plant virus emergence is an "eco-evolutionary" process, because, while the first two steps entail genetic changes in the virus, the third step may require vector or host population changes or other ecological or environmental shifts (Roossinck & García-Arenal, 2015; Geoghegan & Holmes, 2017; Lefeuvre et al., 2019; French & Holmes, 2020). The classical disease triangle accounting for the roles of a virulent pathogen, susceptible host, and favorable environmental conditions for the outbreak of an epidemic is therefore constrained until all the necessary parameters have been effectively optimized (Scholthof, 2007; Jeger, 2020; Islam et al., 2020). Thus, the impact of climate variability on agro-ecosystems makes plant virus emergence a significant factor for 21st

71 century agriculture (Anderson et al., 2004; Jones, 2009; Canto et al., 2009; Rodríguez-Nevado et al.,
72 2018; Jones & Naidu, 2019; Lal et al., 2020; Jones, 2021; Ristaino et al., 2021).
73
74 Sweet potato mild mottle virus (SPMMV; genus *Ipomovirus*, family *Potyviridae*) infects
75 sweetpotatoes (*Ipomoea batatas* Lam) and related wild plants in eastern Africa where it was first
76 reported on sweetpotato in the 1970s (Hollings et al., 1976; Tugume et al., 2010a; Clark et al., 2012).
77 Virus-specific methods frequently used for virus detection in sweetpotato may cause variants of
78 certain viruses to remain undetected and consequently unstudied using genomic sequence analysis.
79 Unconfirmed reports show that SPMMV has also been detected serologically in New Zealand,
80 Indonesia, Peru, Malawi (Carey et al., 1997; Fletcher et al., 2000; Tairo et al., 2005; Mbewe et al.,
81 2021), and by RT-PCR in South Africa (Sivparsad & Gubba, 2013). However, these reports are not
82 backed by genetic sequence data. Therefore, the most reliable detection and sequence data indicate
83 SPMMV is to-date geographically restricted in eastern Africa (Kashif et al., 2012; Clark et al., 2012;
84 Gu et al., 2014; Nhlapo et al., 2018; Jo et al., 2020; Ibaba & Gubba, 2020; Nakasu et al., 2022;
85 Orfanidou et al., 2022).
86
87 In contrast from SPMMV, other viruses infecting sweetpotato within the same eastern Africa region
88 that are also frequently detected elsewhere globally. For example, incidences of the “East African”
89 (EA) strain of sweet potato feathery mottle virus (SPFMV; genus *Potyvirus*, family *Potyviridae*) and
90 sweet potato chlorotic stunt virus (SPCSV; genus *Crinivirus*, family *Closteroviridae*) that were
91 originally thought to be restricted to eastern Africa (Kreuze et al., 2000; Tairo et al., 2005) have been
92 confirmed elsewhere in the world (Clark et al., 2012; Qin et al., 2013a; Qin et al., 2013c; Maina et
93 al., 2018; Kwak et al., 2018; Kreuze et al., 2020). Because sweetpotato was introduced to eastern
94 Africa from Latin America only ca. 400 years ago (Zhang et al., 1999; Zhang et al., 2004),

geographical restriction of SPMMV to eastern Africa has led to conclusions that this region is the center of origin of SPMMV (Mukasa et al., 2003b; Tairo et al., 2005; Tugume et al., 2010a). However, originating from eastern Africa does not fully account for SPMMV's exclusive geographic affinity to this region, nor minimize the virus' reemergence potential in the same region.

In eastern Africa, SPMMV is a component of virus disease complexes that account for 60-95% yield loss and this may reach 100% with increased multiple virus infections in otherwise high-yielding sweetpotato cultivars (Clark et al., 2012). Despite its economic importance, various aspects of SPMMV biology, epidemiology and evolution are unknown, which decelerates efforts to develop appropriate strategies for effective management of disease complexes in which it occurs (Tugume et al., 2016b). Effective management of emerging plant virus diseases is strongly coupled to a good understanding of the virus-vector, virus-host and virus-virus relationships at the community level because they have a strong bearing on host/geographic range and spread of plant viruses (Power, 2008; Borer et al., 2010; Power et al., 2011; Mauck et al., 2012; Shates et al., 2019; Donnelly & Gilligan, 2022). Moreover, SPMMV is characterized by numerous biological contradictions and/or knowledge gaps making it atypical of genus *Ipomovirus*, which itself has features unusual to family *Potyviridae* (Dombrovsky et al., 2014). For example, ever since the first report of SPMMV whitefly-transmission (Hollings et al., 1976), vector-mediated transmission of SPMMV in sweetpotatoes has remained enigmatic, because to-date, no efforts have successfully reproduced SPMMV whitefly-transmission (Misango, 2011; Tugume et al., 2016b). Whiteflies are the vector for other known ipomoviruses in the region such as cassava brown streak ipomoviruses (Maruthi et al., 2005; Maruthi et al., 2017). SPMMV transmission success and efficiency could depend on the virus' tissue-localization in infected plants and feeding habits of vectors but this is unknown. Correlations between the vector populations and SPMMV incidence/prevalence under field conditions is not

119 determined. Until when tomato mild mottle virus (ToMMoV, previously called eggplant mild leaf
120 mottle virus, EMLMV) was characterized (Dombrovsky et al., 2012), SPMMV was the only known
121 ipomovirus encoding Helper component protease (HCPro), a multifunctional protein encoded by
122 viruses of genus *Potyvirus* (Colinet et al., 1996; 1998; Valli et al., 2018).
123
124 The aim of this review is to provide a sharp focus, analysis of current progress and research gaps
125 surrounding SPMMV with the goal of stimulating further scholarship and re-thinking research
126 investments for virus disease management in sweetpotato. We highlight the reemergence potential of
127 SPMMV by drawing comparisons between three taxonomically and phylogeographically related
128 viruses: SPMMV, and the two cassava brown streak disease (CBSD)-causing ipomoviruses, namely,
129 cassava brown streak virus (CBSV) and Ugandan cassava brown streak virus (UCBSV), collectively
130 reffered to as cassava brown streak ipomoviruses (CBSIs). All the three viruses are pathogens of
131 vegetatively propagated crops commonly grown in eastern Africa. CBSIs are the causative agents of
132 CBSD; first reported in the 1930's in eastern Africa, remained of little economic significance until
133 their re-emergence about half a century later in the 1990's to mid-2000's (Storey, 1936; Monger et
134 al., 2001a; Monger et al., 2001b; Hillocks & Jennings, 2003; Alicai et al., 2007). To-date, CBSD
135 continues to severely constrain cassava farming systems across eastern, central and southern Africa.
136 In less than a decade after reemergence of CBSIs, CBSD caused huge economic losses of more than
137 USD100 million per year (Pennisi, 2010; Rey & Vanderschuren, 2017; Mero et al., 2021). Currently,
138 CBSD is estimated to cause annual losses in excess of USD 750 million to the affected communities
139 (Hillocks & Maruthi, 2015; Mero et al., 2021). From the early 1930's to early 1990's, the CBSIs, like
140 SPMMV, remained restricted to coastal eastern Africa but thereafter rapidly spread to mid and high
141 altitude areas in Uganda, Burundi and Zambia (Alicai et al., 2007; Patil et al., 2015; Tomlinson et al.,
142 2018; Mulenga et al., 2018).

Evolutionary analyses show that intra-specific recombination, positive selection and on-going molecular adaptation account for the emergence of CBSIs in eastern Africa (Mbanzibwa et al., 2011a; Ndunguru et al., 2015; Alicai et al., 2016; Tomlinson et al., 2018). Similarly, recombination and positive selective events were detected in SPMMV genome, implying that the virus is undergoing fast evolutionary adaptation under natural conditions in eastern Africa (Tugume et al., 2010a). The availability of additional genomic sequence data is critical for extended analysis of forces imposed on the genome and hence driving the evolution of SPMMV. The evolutionary plasticity observed in the SPMMV isolates characterized to-date shows that point mutations, recombination and selection coupled with the virus' natural infectivity of diverse wild host species may drive a 'silent build up' of better fit genotypes in a changing climate and could explode into a worse disease conundrum, similar to CBSIs. We should urgently fill the existing information gaps in advance to contain possible new disease emergences associated with SPMMV.

Sweetpotato (*Ipomoea batatas* Lam) is grown in all tropical climates and is globally ranked among the 10 most important food crops (Low et al., 2017; Low et al., 2020). The diversity and significance of this crop in food, nutrition and income security in eastern Africa has been reviewed (Woolfe, 1992; Gichuki et al., 2003; Loebenstein, 2009; Yada et al., 2010; Tumwegamire et al., 2011; Low et al., 2017; Low et al., 2020). The importance of sweetpotato, is however challenged by numerous diseases especially viral diseases that hampers productivity (Clark et al., 2012; Loebenstein, 2015; Gibson & Kreuze, 2015; Kreuze et al., 2021; Kreuze et al., 2022). The need to control virus diseases in sweetpotato is recognized as the most urgent activity for increasing productivity in developing countries (Fuglie, 2007; Kleinwechter, 2012; Pemsl et al., 2022; Hambly et al., 2022); yet, viruses still remain the main constraint in sweetpotato production in eastern Africa. Limited research

investments in sweetpotato by developing countries including those in eastern Africa hampers in generating epidemiological information that would otherwise leverage virus disease control.

VIRUSES AND VIRUS DISEASES OF SWEETPOTATO IN EASTERN AFRICA

The first record of suspected viral infection in sweetpotatoes within eastern Africa was during 1939 in Ituri province of DRC (boarders Uganda to the west), and then in 1944 from Uganda (Hansford, 1944). Follow-up studies on plant-infectivity in eastern Africa indicated the occurrence of two viruses, virus "A" and virus "B", that were aphid- and whitefly-transmitted, respectively (Sheffield, 1957; 1958). The descriptions made for virus "A" and virus "B" are very similar to those of SPFMV and SPMMV, respectively (Hollings et al., 1976; Schaefers & Terry, 1976; Colinet et al., 1996). Because sweetpotato was a neglected and less important crop in eastern Africa at the time (Woolfe, 1992; Bermejo & León, 1994; Qaim, 1999), not much research progress into sweetpotato virology was attainable. Subsequently, the interest to study viruses and virus diseases in sweetpotato crops in eastern Africa was largely incited by the devastating effects of cassava mosaic disease epidemic (Martin, 1928; Otim-Nape et al., 1996; Thresh et al., 1997; Zhou et al., 1997) which destroyed cassava crops and re-directed more interest to the use of sweetpotato as an alternative crop for subsistence food security (Minde et al., 1997; Scott et al., 1997; Karyeija et al., 1998). Since then, several viruses and viral diseases of sweetpotato have been reported from many parts of the world including eastern Africa (Tairo et al., 2005; Clark et al., 2012). Currently, at least 17 viruses have been found infecting sweetpotato in eastern Africa have been found out of about 30 viruses that have been reported on the crop globally (Clark et al., 2012; Kreuze et al., 2021). All the 17 viruses currently detected in sweetpotatoes in eastern Africa belong to six families of *Potyviridae*, *Closteroviridae, Geminiviridae*, *Betaflexiviridae, Caulimoviridae* and *Bromoviridae* (Gibson &

Aritua, 2002; Mukasa et al., 2003a; Tairo et al., 2004; Tairo et al., 2005; Miano et al., 2006; Aritua et al., 2007; Wasswa et al., 2011; Clark et al., 2012; Mbanzibwa et al., 2014; Mwaipopo et al., 2021).

Most viruses currently known to infect sweetpotatoes in eastern Africa have (+)ssRNA genomes (Clark et al., 2012; Kreuze et al., 2020). Exceptions are viruses in family *Caulimoviridae* (dsDNA reverse-transcribing viruses or pararetroviruses) and the monopartite begomoviruses (genus *Begomovirus*, family *Geminiviridae*), known as sweepoviruses (Trenado et al., 2011). Transmission of DNA viruses of sweetpotato in eastern Africa is not studied, although it is presumed that sweepoviruses are transmitted through vegetative propagation and semi-persistently by whiteflies. Sweepoviruses have been isolated from sweetpotato plants in different parts of the world, including eastern Africa (Miano et al., 2006; Wasswa et al., 2011; Mbanzibwa et al., 2014) and their impact on sweetpotato root yield is now apparent (Clark & Hoy, 2006; Ling et al., 2010; Wanjala et al., 2020). All viruses currently known to infect sweetpotato (with exception of cucumber mosaic virus, CMV) are unique to sweetpotato. These viruses have been found to infect only a few other host plant species, and sweetpotatoes (or related *Ipomoea* spp.) are not affected by viruses infecting other crops, suggesting that the plant provides some unique tissue or cellular environment in which only specialized viruses can propagate (Kreuze et al., 2021). Whereas several different viruses infect sweetpotato in eastern Africa (Loebenstein et al., 2009; Clark et al., 2012; Loebenstein, 2012; Gibson & Kreuze, 2015; Kreuze et al., 2021), the greatest negative impact on root yield is incited mostly by complex diseases arising from interactions involving SPCSV and other viruses, most especially SPFMV and SPMMV.

Sweet potato chlorotic stunt virus

213 Most times, symptoms of SPCSV are persistent even when infecting alone. Therefore, SPCSV is
214 often considered the most damaging virus affecting individual sweetpotato plants. Symptoms caused
215 by SPCSV in eastern Africa vary with cultivar but generally include deep red to purple or yellow
216 mottle of the lower and middle leaves, and stunting of sweetpotato plants (Gibson et al., 1998;
217 Mukasa et al., 2006). Two serologically-distinguishable strains of SPCSV first isolated from West
218 Africa ($SPCSV_{WA}$) and East Africa ($SPCSV_{EA}$) are known in Africa (Schaefers & Terry, 1976;
219 Gibson et al., 1998). SPCSV has flexuous, filamentous particles ranging from 850 to 950 nm,
220 positive-strand single-stranded RNA genome which consists of two genomic RNAs, RNA1 and
221 RNA2, with complete nucleotide sequences of about 9.4 kb and 8.2 kb, respectively (Kreuze et al.,
222 2002). RNA1 contains overlapping open reading frames (ORFs) that encode replication with five
223 functional domains. RNA2 consists of seven ORFs including the coat protein and heat shock protein
224 homologue (Hsp70h) domains. There are also several subgenomic RNAs (sgRNAs) that have been
225 detected in SPCSV-infected plants. SPCSV is phloem-limited, transmitted in a semi-persistent
226 manner by the whitefly species *Bemisia tabaci*, *B. afer* and *Trialeurodes abutilonea* (Loebenstein,
227 2015). Host range of SPCSV is apparently restricted to species in the family *Convolvulaceae*, with 12
228 wild species reported as natural hosts (Tugume et al., 2013).
229
230 **Sweet potato feathery mottle virus**
231 Sweet potato feathery mottle virus (SPFMV) is the most widespread and characterized virus of
232 sweetpotato, found wherever the crop is grown (Moyer & Salazar, 1989; Clark et al., 2012). The
233 virus is highly variable, with three major strains identified; EA (East Africa), RC (russet crack), and
234 O (ordinary) (Campbell et al., 1974; Karyeija et al., 2000b; Tairo et al., 2005). A previous strain C of
235 SPFMV was deemed as a standalone independent virus species named sweet potato virus C
236 (Untiveros et al., 2010). Previously, synonyms of these strains included russet crack virus, internal

cork virus, russet crack virus, sweetpotato virus A, sweetpotato ringspot virus and sweetpotato leafspot virus. The SPFMV virions are elongated flexuous rods, 810 to 865 nm long, with a monopartite, single-stranded, positive sense RNA genome which is approximately 10.8 to 11 kb (Moyer & Cali, 1985; Yamasaki et al., 2010). The genome contains 5' non-translatable region (NTR), one open reading frame for a single polyprotein giving rise to 10 mature proteins, a 3'NTR and and poly(A) tail (Moyer & Cali, 1985; Yamasaki et al., 2010). In eastern Africa, SPFMV causes transient or no symptoms. Most common symptoms on sweetpotato plants when infected by SPFMV alone include chlorotic coloration along leaf midribs (feathering), faint to distinct chlorotic spots, with or without purple pigmented borders. Both spots and feathering may have purplish margins (Moyer & Salazar, 1989). SPFMV is transmitted in a non-persistent manner by aphids, including *Myzus persicae* and *Aphis gossypi*, *A. craccivora* and *Lipaphis erysimi*, the first being the principal vector (Schaefers & Terry, 1976). Host range of SPFMV is wide, but has been mainly limited to the families Convolvulaceae (*Ipomoea* spp.) and Chenopodiaceae (*Chenopodium* spp.). Recently, SPFMV was found naturally infecting *Chrysanthemum morifolium* (family Asteraceae) and *Amaranthus blitum* (family Amaranthacea) in China (Yan et al., 2020; Zhao et al., 2020). In addition to transmission by aphid vectors, SPFMV is perpetuated through use of infected vine cuttings as planting material. It has been suggested that eastern Africa is a hotspot for evolution and diversification of SPFMV, and isolates from the region cluster distinctly from others (Kreuze et al., 2000; Tugume et al., 2010b; Wokorach et al., 2020).

Sweet potato mild mottle virus

SPMMV is the third most prevalent virus infecting sweetpotatoes in eastern Africa, after SPFMV and SPCSV (Mukasa et al., 2003a; Tairo et al., 2005). Symptoms of SPMMV reported include mild leaf mottling, chlorosis, distorting, streaks of different colors on leaves and plant stunting (Moyer &

Salazar, 1989). SPMMV is the type member of genus *Ipomovirus* in family *Potyviridae*, and has flexous rod-shaped virions 800 - 950 nm in length, (+)ssRNA genome which consists of about 9 kb to 10.8 kb and encoding nine functional domains (Colinet et al., 1996; 1998). Although originally reported as a whitefly transmitted virus (Hollings et al., 1976), the vector(s) transmitting SPMMV has since become controversial and forms an important topic of discussion under this review. The virus, like all other sweetpotato viruses is also perpetuated through use of infected vines as planting material. Symptoms of SPMMV are often not easy to diagnose in the field and the virus can remain latent (Hollings et al., 1976; Skoglund & Smit, 1994). The host range of SPMMV have been reported to be the largest compared to other sweetpotato infecting virus, including over 20 plant species in at least 14 families (Moyer & Salazar, 1989; Brunt et al., 1996; Tugume et al., 2010a). The reasons accounting for SPMMV's broad host range are unknown. Generally, plant viruses are "promiscuous" with their virus-host but not virus-vector relations (Power & Flecker, 2003; McLeish et al., 2019) and the wide host range of plant viruses usually tightly linked to their likelihood of emergence (Anderson et al., 2004; Jones, 2009; Moury et al., 2017). The relatively low incidence of SPMMV in eastern Africa has been attributed to high rate of sweetpotato reversion from infection by the virus compared to low reversion from SPFMV and SPCSV (Ssamula et al., 2019). Effects of SPMMV on yield of sweetpotato are unknown, but it certainly reduces the quality of vines for use as planting material. Although SPMMV commonly occurs as co-infections with SPFMV and SPCSV, interactions with either or both viruses and vector relations are less characterized, e.g., Mukasa et al. (2006). Beyond the clear understanding on geographical restriction of SPMMV largely to eastern Africa where it was first isolated, no detailed epidemiological knowledge exists.

Virus disease complexes in Sweetpotato

Single infections of SPFMV, SPMMV or SPCSV in sweetpotato cause little or no noticeable impact on yield (Gibson et al., 1997; Clark et al., 2012). However, disease complexes arising from multiple infections cause the most destructive effects (Clark et al., 2012). Consequently, co-infections of SPFMV and SPCSV result in up to 1000-fold increase in the titers of SPFMV because the antiviral defense in sweetpotato is suppressed by RNase III protein of SPCSV (Karyeija et al., 2000a; Kreuze et al., 2005; Mukasa et al., 2006; Cuellar et al., 2008; Cuellar et al., 2009; Wang et al., 2021b). The many-fold increase in SPFMV titers in plants results into Sweet potato virus disease (SPVD), the most devastating disease of the crop in eastern Africa (Gibson et al., 1998; Karyeija et al., 2000a; Untiveros et al., 2007; Cuellar et al., 2008). Characteristic symptoms of SPVD include severe stunting, leaf distortion, narrowing, wrinkling, purpling, bronzing of older leaves, vein clearing or chlorotic mottle associated with the midrib, and the disease results in yields less than half that of symptomless plants (Karyeija et al., 1998; Mukasa et al., 2003a). Observations of SPVD and its impact on sweetpotato crops was first reported in the region during the early 1940s near western Uganda (Hansford, 1944) and soon after, viral diseases affecting sweetpotato were reported in Kenya, Tanzania, Rwanda, Burundi, Malawi (Sheffield, 1957). Sweet potato chlorotic dwarf disease (CD) complex (syn. Sweet potato severe mosaic disease, SPSMD) the second most destructive disease complex arises from a synergistic interaction between SPMMV and SPCSV (Mukasa et al., 2006). In the CD, SPMMV titers increase up to 600-fold (Mukasa et al., 2006; Untiveros et al., 2007). Although the original description of SPVD referred to an outcome of synergism between SPCSV and SPFMV (Karyeija et al., 2000a), later studies found many other different viruses of sweetpotato can be synergized by SPCSV (Mukasa et al., 2006; Untiveros et al., 2007; Cuellar et al., 2011b; Cuellar et al., 2015). The division of SPCSV-induced synergisms into SPVD and CD may therefore be artificial with CD considered a case of SPVD used here to illustrate the role of SPMMV in disease complexes. The disease complexes account for 60-95% yield loss and this may reach

100% with increased multiple virus infections of the otherwise high-yielding sweetpotato cultivars in eastern Africa (Clark et al., 2012). Because many landrace sweetpotato cultivars in eastern Africa express high levels of resistance to SPFMV and SPMMV allowing only very low accumulation of the virus, many plants singly infected with these viruses remain undetected in routine serological tests (Gibson et al., 1997; Karyeija et al., 2000a; Mukasa et al., 2006).

Virus disease complexes of sweetpotato in eastern Africa are exacerbated by six characteristic features of sweetpotato cropping systems there. These include: (a) year-round abundance of insect vectors transmitting the viruses, (b) high susceptibility and lack of resistance in sweetpotato cultivars to the disease complexes, although resistance is apparent in single virus infections, (c) lack of an established formal sweetpotato seed system to ensure clean planting material, (d) perennial nature of vegetatively propagated planting material that continuously accumulate virus infections and overlapping planting seasons (Fig. 1), (e) presence of evergreen alternative wild hosts in close proximity to sweetpotato fields (Fig. 1), and (f) high frequency of mixed virus infections (Bashaasha et al., 1995; Tugume et al., 2008; Gibson, 2009; Tugume et al., 2010a; Tugume et al., 2010b; Namanda et al., 2011; Clark et al., 2012; Tugume et al., 2013; Tugume et al., 2016a; 2016b; Ngailo et al., 2016; Echodu et al., 2019; Low et al., 2020; Donnelly & Gilligan, 2022). These characteristics do not only present suitable conditions for a perpetual virus disease burden, but are also a perfect recipe for the emergence of viruses (Fargette et al., 2006; Jones, 2009; Canto et al., 2009; Tugume et al., 2010a; Tugume et al., 2010b; Clark et al., 2012; Alexander et al., 2014; Tugume et al., 2016a; 2016b; French & Holmes, 2020).

VECTOR-MEDIATED TRANSMISSION OF VIRUSES TO SWEETPOTATO IN EASTERN AFRICA

Most of the viruses known to infect sweetpotato in eastern Africa are transmissible by two insect vectors, namely, aphids and whiteflies. The whitefly *B. tabaci* (Gennadius), commonly called as tobacco or sweetpotato whitefly was formerly considered as a single species with a worldwide distribution in tropical and semi-tropical regions. However, molecular studies and reciprocal crossing experiments conducted in the past decade have revealed that *B. tabaci* is a cryptic species complex comprising of more than 40 morphologically indistinguishable species (Maruthi et al., 2001a; Colvin et al., 2004; Liu et al., 2007; Boykin et al., 2007; Xu et al., 2010; Dinsdale et al., 2010; De Barro et al., 2011; Barbosa et al., 2014; Boykin & De Barro, 2014; Tay et al., 2017; Vyskočilová et al., 2018). In sub-Saharan Africa, species of *B. tabaci* are a serious pest on many crops including sweetpotato, cassava, tomato, cotton and beans (Berry et al., 2004; Sseruwagi et al., 2005; Boykin et al., 2007; Ghosh et al., 2015; Mugerwa et al., 2018). Much of the research on whitefly diversity in eastern Africa has been done on cassava whiteflies while six species known to infest other plants; namely Indian Ocean (IO), Mediterranean (MED) (Gueguen et al., 2010), East Africa 1 group (EA1), Middle East-Asia Minor 1 (MEAM1), MEAM2, and Uganda sweetpotato (UgSp) (Sseruwagi et al., 2005). Cassava is known to be infested by 13 putative whiteflies named sub-Saharan Africa (SSA1 to SSA13) (Legg et al., 2002; Mugerwa et al., 2018). Although many of these species were found on sweetpotato, only UgSp is believed to truly colonize the crop (Maruthi et al., 2001b) and thus may potentially transmit viruses in the crop. The nature of the two whitefly populations originally used to transmit SPMMV in sweetpotato plants (Hollings et al., 1976) is unknown except for their origin of host range from cucurbit and tobacco plants. Their genetic relationships to the current day species therefore cannot be established.

Aphids are another group of insects transmitting viruses in sweetpotato of which two species (*M. persicae* and *A. gossypii* have been implicated in non-persistent transmission of SPFMV

(Byamukama et al., 2004; Ndunguru et al., 2009). In Uganda the species *M. persicae* has experimentally, successfully transmitted SPFMV with a single aphid simultaneously transmitting two serotypes of SPFMV (Karyeija et al., 2000b). In Kenya, the species has commonly been trapped in sweetpotato fields (Wambugu, 1991), which presumably could indicate its role in the spread of SPFMV as it visits the sweetpotato fields. Despite the glaring evidence of the ability of aphids to transmit SPFMV, in-depth studies to unravel its vector capacity to transmit the common sweetpotato viruses found in the eastern African region have not been done.

THE ENIGMA OF VECTOR-MEDIATED TRANSMISSION OF SPMMV

Successful transmission within the host community is a good indicator of the virus' adaptive optimization of virus-host and virus-vector relations because these are part of the eco-evolutionary processes driving virus emergence (Rojas & Gilbertson, 2008; Fereres & Moreno, 2009; Elena et al., 2014; Lefeuvre et al., 2019; Chisholm et al., 2019). Indeed, the incidence of SPMMV ranging between 9.0% and 25.0% of sweetpotato samples tested in different countries of eastern Africa (Mukasa et al., 2003a; Tairo et al., 2004; Ateka et al., 2004b; Njeru et al., 2008; Wokorach et al., 2019) may imply such virus-host and virus-vector optimization. SPMMV was reported to be persistently transmitted by *B. tabaci*, Gennadius biotype B (Hollings et al., 1976). SPCSV is also transmitted by whiteflies, *B. tabaci*, *B. afer* and *Trialeurodes abutilonea*, in a semi-persistent non-circulative manner (Cohen et al., 1992; Sim et al., 2000; Gamarra et al., 2010). Epidemiologically, it is reasonable that viruses infecting a common host and transmitted by the same vector should exhibit a high frequency of co-infection (Seabloom et al., 2015; Allen et al., 2019; Moreno & López-Moya, 2020). For this reason, frequent association or co-infection is expected between SPCSV and SPMMV under natural conditions. Such correlation has been observed between the aphid-transmitted viruses SPFMV and CMV in sweetpotatoes (Opiyo et al., 2010), SPFMV and the possibly aphid-

transmissible sweet potato chlorotic fleck virus (SPCFV) (Ateka et al., 2004a; Aritua et al., 2009), and between zucchini yellow mosaic virus (ZYMV) and watermelon mosaic virus (WMV) in cucurbits (Lecoq & Desbiez, 2012; Salvaudon et al., 2013). However, many studies in eastern Africa have consistently shown lack of significant association between SPCSV and SPMMV (Mukasa et al., 2003a; Tairo et al., 2004; Ateka et al., 2004b; Aritua et al., 2007; Njeru et al., 2008; Opiyo et al., 2010). Moreover, studies carried out after those of Hollings et al. (1976) have failed to confirm whitefly transmissibility of SPMMV (Tairo et al., 2005; Misango, 2011), while aphid transmissibility of SPMMV has not been tested. These observations led us to postulate different hypotheses that can be tested to account for vector-mediated transmission of SPMMV.

Hypothesis #1: SPMMV is opportunistically aphid-transmitted with potyvirus SPFMV as a helper virus.

We hinge this hypothesis on the finding that dual-infections of SPMMV and SPFMV are more common than single infections of SPMMV in sweetpotatoes in Kenya (Ateka et al., 2004b) and up to 3-fold more likely than dual-infections of SPMMV and SPCSV in sweetpotatoes and wild plants in Uganda (Mukasa et al., 2003a). These observations are interesting because the common co-occurrence of SPMMV and SPFMV cannot be due to the latter's suppression of the natural resistance to the former in sweetpotato since both viruses do not exhibit synergism to each other (Mukasa et al., 2006; Untiveros et al., 2007). Also, sweetpotato cultivars in eastern Africa are less resistant to SPMMV than SPFMV. In earlier studies of plants co-infected with two viruses, an aphid-transmissible potato virus Y (PVY; originally named potato virus C) was shown to facilitate aphid-transmissibility of a non-aphid transmissible heterologous virus, potato aucuba mosaic virus (Kassanis, 1961; Kassanis & Govier, 1971a; 1971b; Govier & Kassanis, 1974a; 1974b). Moreover,

403 reciprocal assistance mechanisms mediated simultaneous aphid transmission of two aphid non-
404 transmissible strains of zucchini yellow mosaic potyvirus (Desbiez et al., 1999).
405
406 In viruses of genus *Potyvirus*, the CP and HC-Pro proteins function together in aphid transmission
407 (Pirone & Blanc, 1996; Ng & Falk, 2006; Valli et al., 2018). The HCPro of SPMMV contains the
408 highly conserved motif PTK (Tugume et al., 2010a) that is critical in bridging the virion to the aphid
409 stylet during transmission of potyviruses (Blanc et al., 1998). The KITC, KLSC or RITC motif of
410 potyviruses critical for retention of potyviral virions in aphid stylets (Blanc et al., 1998) are also
411 present in the HCPro protein of SPMMV but in a mutated form as KTCC, KACC, or RTCC
412 (Tugume et al., 2010a). In contrast, the CP N-terminus in SPMMV lacks the DAG motif that is
413 essential for aphid-transmission of potyviruses (Atreya et al., 1990; Atreya et al., 1991; Atreya et al.,
414 1992) although DAG motif is present in an ipomovirus CBSV (Ateka et al., 2017) that is whitefly-
415 transmissible (Maruthi et al., 2005; Maruthi et al., 2017). SPMMV was historically the only known
416 HCPro-encoding ipomovirus until when this protein was also found encoded by the genome of a
417 newly characterized tomato mild mottle virus (ToMMoV) and its distant strain designated as
418 ToMMoV-IL (Abraham et al., 2012; Dombrovsky et al., 2012; Dombrovsky et al., 2013; Rabenstein
419 et al., 2013; Dombrovsky et al., 2014). The presence of a highly conserved PTK motif in SPMMV
420 and TomMMoV HCPro may be biologically meaningless due to lack of DAG motif in these viruses
421 (Dombrovsky et al., 2014). Similarly, the presence of DAG motif at amino acid positions 52-54 from
422 the N-terminus of the CP of ipomoviruses CBSV, squash vein yellowing virus (SqVYV), coccinia
423 mottle virus (CocMoV) (Ateka et al., 2017) may be nonsense in absence of the HCPro within the
424 same genome to facilitate aphid transmission. By analogy, whitefly transmission of an ipomovirus
425 cucumber vein yellowing virus (CVYV) was abolished in CVYV mutants lacking amino acids (aa) in
426 the N-terminal region of the CP at 93-105 position (Lindenau et al., 2021), yet a homologous CP aa

sequence is also present in SPMMV CP whose whitefly transmission remains enigmatic. Therefore, although the presence of a DAG motif does not guarantee aphid transmissibility in potyviruses (Johansen et al., 1996; Flasinski & Cassidy, 1998; López-Moya et al., 1999), possibilities to employ a DAG-containing CP from a co-infecting heterologous or homologous co-infecting virus(es) in co-transmission of SPMMV is reasonable.

The hypothesis of "opportunistic aphid transmission of SPMMV" using potyvirus SPFMV as a helper virus presupposes that the two viruses are simultaneously present in tissues of infected plants from which aphids acquire them in a single acquisition access event. However, the alternative scenario is that aphids may first acquire SPFMV before SPMMV. Previously, the recombinant strains, $PVY^{N:O}$ and PVY^{NTN}, were more efficiently transmitted than PVY^{O} when they were sequentially acquired regardless of the order acquired (Mondal et al., 2017; Mondal & Gray, 2017). Hence, the recombinant strains appear to preferentially bind to the aphid stylet over PVY^{O} or they may be preferentially released during inoculation which may preferentially increase incidence of the recombinant strains over PVY^{O} in fields (Mondal et al., 2017; Mondal & Gray, 2017; Mondal et al., 2021). In the case of sweetpotato, this hypothesis can be tested by sequential inoculation access events between single SPFMV (or its encoded protein), SPMMV or dual SPFMV + SPMMV or triply SPFMV + SPMMV + SPCSV infected plants. Indeed, in potyviruses, the HCPro of one virus may allow the transmission of unrelated potyviruses when the HCPro is provided before or concomitantly to virus acquisition by aphids (Granier et al., 1993). For example, a non-aphid-transmissible ZYMV isolate (ZYMV-NAT), which had a transmission-deficient CP, was easily transmitted by aphids from plants infected concomitantly by ZYMV-NAT and a transmissible isolate of PRSV (Bourdin & Lecoq, 1991). This occurred through hetero-encapsidation, in which the RNA of ZYMV-NAT is completely or partially encapsidated by the functional papaya ringspot virus

(PRSV) CP. Moreover, an aphid non-transmissible isolate deficient for the HC-Pro could be transmitted by aphids in mixed infection with an isolate that has a functional HC-Pro through the mechanism of heteroassistance (Lecoq et al., 1991).

Under "opportunistic aphid-transmission" of SPMMV, both heteroencapsidation and heteroassistance may enforce natural maintenance of SPMMV or its variants which may have lost their vector transmissibility, although Desbiez et al. (1999) showed that heteroassistance is more efficient than heteroencapsidation. As noted above, the CP of SPMMV lacks the DAG aphid transmissibility motif compared to the virus' HCPro that harbors some or mutated versions of (and probably ineffective forms of) aphid transmissibility motifs (Tugume et al., 2010a) indicating similarity to the CP and HCPro of another ipomovirus ToMMoV (Abraham et al., 2012). Our hypothesis is further supported by a finding that an Israeli isolate of ToMMoV is whitefly-transmitted (Dombrovsky et al., 2013), yet this contradicts aphid-transmission of the Yemenite isolate of ToMMoV (Walkey et al., 1994). The discrepancy between these two reports could be explained by opportunistic aphid transmission in which ToMMoV is present in a mixed infection with PVY (Dombrovsky et al., 2014). Indeed, Hiskias et al. (1999) detected ToMMoV and PVY in mixed infections, which is reminiscent of abundant SPMMV and SPFMV co-infections in sweetpotato and wild plants in eastern Africa.

Hypothesis #2: Vector-mediated transmission of SPMMV is modulated by synergism with SPCSV.

In plant virology, synergism refers to a simultaneous infection by two distinct viruses where the infection of one or both viruses is enhanced (Smith, 1945; Close, 1964; Malyshenko et al., 1989; Atabekov & Taliansky, 1990; Falk et al., 1995; Froissart et al., 2002; Latham & Wilson, 2008; Aguilar et al., 2015; Zhang et al., 2019). Synergisms may be unidirectional (in which the two viruses

475 are often referred to as the ‘helper’ and the ‘dependent’ virus) or mutualistic, implying that protein(s)
476 from one virus can enhance infection by another (Malyshenko et al., 1989; Latham & Wilson, 2008;
477 Syller, 2012; Syller & Grupa, 2016). A recent study in wheat-infecting viruses revealed that
478 interactions among different unrelated viruses in a co-infected plant host can be more complex, and
479 that synergistic interactions do not always necessarily cause increase in virus titers (Tatineni et al.,
480 2022). The hypothesis that “vector-mediated transmission of SPMMV is modulated by synergism
481 with SPCSV” is hinged on data showing that concentrations of SPMMV are up to 600-fold higher in
482 dual infections with SPCSV than in single infections (Mukasa et al., 2006; Untiveros et al., 2007).
483 For example, earlier observations in beans and potato singly infected with bean common mosaic
484 virus and PVY, respectively, showed that plants containing higher virus titres were better sources of
485 the virus for aphid transmission (Bagnall & Bradley, 1958; Zettler, 1969). Virus transmission rates
486 are expressed as the percentage of plants that become infected following inoculation of viral particles
487 by vectors that have fed previously on infected plants (Maruthi et al., 2005; Maruthi et al., 2017).
488 Therefore, increased titres of one or both viruses in dual infections may result in increased chances of
489 vector transmission which is positively correlated with virus accumulation (Froissart et al., 2010).
490
491 The effect of virus accumulation on transmission was demonstrated for aphid-transmitted viruses,
492 irrespective of the transmission mode (nonpersistent or persistent), as previously demonstrated in
493 several studies (De Bokx et al., 1978; Pereira et al., 1989; Gray et al., 1991; Barker & Woodford,
494 1992). Also, the transmission efficiency of the criniviruses, tomato chlorosis virus and tomato
495 infectious chlorosis virus by whiteflies corresponded to virus concentration in the host in both single
496 and double infections (Wintermantel et al., 2008). Moreover, possibilities of increased infection rate
497 by mite-mediated transmissions from plants doubly infected by wheat streak mosaic virus (genus
498 *Tritimovirus*) and triticum mosaic virus (genus *Poacevirus*) (both members of family *Potyviridae*)

than from singly infected plants have been shown (Tatineni et al., 2010). These studies imply that the possibility of enhancing vector transmission as a result of increasing virus titers in co-infections is not dependent on the mode of transmission of the individual viruses, nor how related the co-infecting viruses are. For SPMMV, possibilities of whitefly transmission or opportunistic transmission by aphids (hypothesis #1) are both plausible since SPMMV titers increase significantly also in the triple infection by SPMMV, SPCSV and SPFMV (Mukasa et al., 2006).

Relatedly, in double infections, both or at least one of the viruses may not only accumulate to a largely increased level, but may also broaden virus distribution within the host, thereby increasing virus availability for feeding vectors (Mascia et al., 2010). Also, it is known that mixed viral infections can affect the biology and preference of virus vectors. The fecundity of *M. persicae* and *Macrosiphum euphorbiae* (Homoptera: Aphididae), the efficient vectors of potato leaf roll virus (PLRV) and PVY, was significantly higher on plants doubly infected with these viruses than on plants singly infected with PVY, but not PLRV (Srinivasan & Alvarez, 2007). Such an outcome could be the result of inhibited phloem transport and increased accumulation of sugars and amino acids in the phloem in the mixed and PLRV-infected plants compared with PVY-infected plants and non-infected plants. Furthermore, both aphid species preferentially settled on doubly infected plants.

It is probable that the visual and/or olfactory stimuli emitted by mixed infected plants were more attractive to aphids than were the stimuli emitted by singly or non-infected plants (Srinivasan & Alvarez, 2007). Plant-mediated interactions between PVY/PLRV and aphid vectors may have significant and far-reaching implications for disease epidemiology, as the two viruses often occur in mixed infections e.g., Srinivasan and Alvarez (2007) and Chatzivassiliou et al. (2008). In sweetpotato, scenarios of host preferences of potential insect vectors with respect to SPMMV

incidence in sweetpotato are not well-known, although previous observations indicated that whiteflies tend to prefer settling on SPMMV-infected than healthy sweetpotato plants (Kisekka, 2016). It is expected that enhancement of SPMMV titers by synergism with SPCSV promotes vector transmissibility of SPMMV, irrespective of the vectors since high titers promote chances of SPMMV acquisition during the access phase. The dynamics of selective virus acquisition and transmission from mixed infections requires detailed investigations because the two viruses (SPCSV and SPFMV) that commonly co-infect plants with SPMMV are transmitted by different vectors.

Hypothesis #3: SPMMV tropism and histo-localization changes upon co-infection with SPCSV.

The specificity of a plant virus for a particular host tissue(s) (tropism) results from a successful tripartite virus-host-vector interaction, which determines the fate of viral infection (Naidu et al., 2015; Mauck & Chesnais, 2020). Plant viruses colonize fewer tissue types than animal viruses because plants have only three basic tissue types: epidermal tissue, vasculature (xylem and phloem), and ground tissue which includes photosynthetic parenchyma, supporting collenchyma, and structural sclerenchyma cells (Spence, 2001; Guillemin et al., 2004). The kind of host plant tissue to which SPMMV shows preference is not well known, yet this has bearing on the virus' vector transmission dynamics. For example, viruses that are found in all tissues of their hosts are transmitted in a non-persistent manner during short intracellular punctures by vectors in the epidermal cells (Esau, 1960; Nault, 1997; Brault et al., 2010; Jiménez et al., 2018; Jiménez et al., 2021a). Virus transmission by vectors in this manner can therefore be optimized with short probing events that are restricted to the dermal tissues (Dietzgen et al., 2016; Jiménez et al., 2018).

In contrast, most semi-persistent and persistently transmitted viruses are phloem-limited where they may also replicate and circulate (Ng & Zhou, 2015; Dietzgen et al., 2016; Jiménez et al., 2018),

indicating that only vector stylet penetrations into phloem tissues can optimize transmission of these viruses (Prado & Tjallingii, 1994; Jiménez et al., 2018; Kappagantu et al., 2020; Jiménez et al., 2021b). Thus, phloem-limited viruses often require longer periods of vector feeding and longer persistence of the virus particles in the vector for successful transmission (Prado & Tjallingii, 1994; Jiménez et al., 2018; Kappagantu et al., 2020; Jiménez et al., 2021a; Jiménez et al., 2021b). Virus diagnostics in sweetpotato routinely use leaf tissues as the starting sample material. The general assumption is that viruses may homogeneously colonize all tissues in which they may get easily detected, yet this may not necessarily be so.

Virus infections within a plant can be structured as metapopulations, indicating that the host plant is not necessarily a "bag" containing a homogeneous or unstructured swarm of viral genomes e.g., (Jridi et al., 2006; Dunham et al., 2014; Tamukong et al., 2020). The prevailing climatic conditions also influence this population structure at individual plant level also influencing virus transmissibility and other plant-vector-pathogen relations (Trębicki et al., 2017; Cunniffe et al., 2021). This is especially so for systemic plant virus infections where the host plants like sweetpotato infected for long cycles in a local cropping system. Like many sweetpotato viruses, SPMMV is easily transmissible mechanically and by grafting upon successful graft union. For example, eight isolates of SPMMV from wild plants that had been graft-transmitted to sweetpotato were also easily sap-transmitted by rubbing sweetpotato infested sap onto carborundum-dusted leaves of *Nicotiana tabaccum*, *N. rustica*, and *N. bethamiana* (Tugume et al., 2010a). SPMMV is also easily graft-transmissible in sweetpotato and the indicator host, *Ipomoea setosa* (Brunt et al., 1996; Tugume et al., 2010a; Ssamula et al., 2019). The ease of sap/mechanical transmission of SPMMV onto susceptible plants may indicate that it is abundant in various tissues of infected plants, just like SPFMV (Karyeija et al., 2000a). This would contrast SPMMV from SPCSV that is phloem-limited

(Karyeija et al., 2000a; Mukasa et al., 2006; Nome et al., 2007) a scenario that is compatible with the phloem-feeding habits of whitefly vectors transmitting SPCSV. However, SPCSV is itself also easily transmissible by grafting and direct rubbing or mechanical friction on sweetpotato stems (Tugume et al., 2013; Zhang et al., 2020).

Single infections of SPMMV (and also SPFMV) in sweetpotato are not easy to detect by ELISA and RT-PCR (Mukasa et al., 2006; Clark et al., 2012). However, both SPFMV and SPMMV become easily to detectable in dual infections with SPCSV. This ease of detection is attributed to enhancement of SPMMV and SPFMV titers due to increased viral multiplication because SPCSV suppresses host anti-viral defense (Mukasa et al., 2006; Untiveros et al., 2007; Cuellar et al., 2008; Cuellar, 2008; Cuellar et al., 2009; Wang et al., 2021c). It is also possible that virus tropism and tissue preference of SPMMV gets altered as a result of a mixed virus infection as shown for other viruses e.g., Alves-Júnior et al. (2009) and Moreno and López-Moya (2020). In various host plants, the infecting begomoviruses all of which are whitefly-transmitted are reported to be phloem-limited e.g., Roy et al. (2021), although in some other cases, begomovirus particles have also been detected outside the phloem in the mesophyll, palisade, parenchyma and epidermal cells (Sudarshana et al., 1998; Wege et al., 2001; Levy & Czosnek, 2003). Both single and mixed infections of tomato yellow leaf curl virus (TYLCV) and tomato yellow leaf curl sardinia virus (TYLCSV) in tomato and *N. benthamiana* were found confined to the phloem (Morilla et al., 2004). In citrus, infection by citrus tristeza virus (CTV) of a resistant or susceptible genotype in which a phloem-limited virus got offloaded from the phloem into other tissues was shown (Dawson et al., 2013). Consequently, tropism of CTV is not simply phloem-limited but tissue specific: virus infection in resistant citrus genotypes was not prevented but mostly restricted to the roots than in shoots (Harper et al., 2014). Pea enation virus 2 (PEMV-2) complements potato leaf roll virus (PLRV) mechanical transmission

595 and facilitates its systemic infection (cell-to-cell movement and inside the phloem movement)
596 (Ryabov et al., 2001).
597
598 In sweetpotato, most cultivars in eastern Africa are naturally resistant to SPMMV where single virus
599 infection may not incite major symptoms, sometimes to undetectable levels (Clark et al., 2012). This
600 resistance is probably comparable to ipomovirus CBSV resistance observed in two elite South
601 American cassava genotypes, DSC167 (highly resistant and immune) and DSC260 (that restricts the
602 virus replication to stems and roots only) (Sheat et al., 2019). The resistance in these cassava lines is
603 not a restriction of long-distance movement but due to preventing virus unloading from the phloem
604 into parenchyma cells for replication, thus restricting the CBSV to the phloem cells only (Sheat et al.,
605 2021). Only a low CBSV signal was found in phloem tissue of DSC 167, indicating that there is no
606 replication in this genotype, while the intense CBSV signals in the phloem of DSC 260 provided
607 evidence for CBSV replication in companion cells. None of the two genotypes showed evidence of
608 virus replication outside the phloem tissues, indicating that in resistant cassava genotypes, CBSV is
609 confined to the phloem tissues only, in which virus replication can still take place or is arrested
610 (Sheat et al., 2021).
611
612 Relatedly, one hypothesis is that SPMMV in resistant sweetpotato clones is restricted within the
613 phloem in a mechanism similar to CBSV in resistant cassava genotypes, but that a co-infection with
614 SPCSV breaks this restriction allowing wide spread and enhanced replication in other non-vascular
615 tissues. For example, the begomovirus abutilon mosaic virus (AbMV) is phloem-limited in single
616 infections; however, co-infection with CMV changes tropism of AbMV to be no longer phloem
617 limited (Wege & Siegmund, 2007). Infections of tomato yellow spot virus (ToYSV) in *N.*
618 *benthamiana* show presence of the virus in mesophyll cells, whereas the related tomato rugose

mosaic virus (ToRMV) does not. However, in dual infections, ToRMV is no longer confined to the phloem and can be found in mesophyll cells similar to ToYSV (Alves-Júnior et al., 2009), suggesting that ToYSV may facilitate the "escape" of ToRMV from the phloem and towards mesophyll tissues. These examples are of DNA viruses, however, in a co-infection of SPFMV and SPCSV both of which are RNA viruses, SPCSV enhances the multiplication and increases titer of SPFMV in non-phloem tissues and abundant in leaves, causing severe SPVD symptoms (Karyeija et al., 2000a). Whether enhanced SPMMV titres and ease of detection in the sweetpotato co-infected with SPCSV is coupled to altered tissue colonization and tropism is unknown and requires extensive investigation.

VIRUS TROPISM AND SWEETPOTATO REVERSION MAY CONSTRAIN SPMMV

Virus distribution in sweetpotato vegetative organs

The presence and distribution of viruses in different sweetpotato plant organs or tissues is not studied. Earlier experiments showed that although infecting viruses may be detected in all tissues of sweetpotato, and that no definite pattern was observed, there was restricted movement of viruses from infected roots (Green et al., 1988). Also, sweetpotato samples taken from leaf petioles gave more reliable results than leaf laminae for the detection of SPFMV (Gibb & Padovan, 1993). Recently, it was shown that sweetpotato stems may be more susceptible than other organs to SPFMV and SPCSV infection (Zhang et al., 2020), however, SPMMV was not part of this study and no quantitative or qualitative assessment of any of studied viral distribution across the different organs were made.

The new sweetpotato crop is normally initiated by planting vines of 15-30 cm in length but in rare circumstances, the storage roots may be used. For example, sweetpotato storage roots were

previously used to generate sprouts for virus testing (Green et al., 1988; Kashif et al., 2012), indicating that the storage roots can function as a reservoir of viruses. Indeed, once sweetpotato vines get infected with single or in mixed virus infections, viruses are capable of infecting storage roots of Ugandan sweetpotato cultivars, and if the storage root is used as seed root, will produce infected sprouts, leading to virus spread (Adikini et al., 2019). Also, infection by a single virus, storage root sprouts may produce mild or no symptoms, and the sprout has the ability to revert from virus infection in the case of SPCSV, or the ability to revert to normal in the case of SPFMV (Adikini et al., 2019). Moreover, SPVD was shown to be latent in sweetpotato storage root; accordingly, using virus-free storage roots and cuttings, purposeful monitoring for SPVD and immediate rouging of infected plants could control and prevent SPVD in sweetpotato (Zhang et al., 2020). However, none of these studies included SPMMV; hence, questions remain whether this virus follows the similar or different scenarios with respect to translocation between roots and shoots. In sweetpotatos, and probably other plants that regenerate vegetatively, a common assumption is that infected cuttings (or tubers) will produce virus-infested plants via systemic translocation of the virus during growth. However, this is not necessarily the case. For example, using potato potexvirus X (PVX), potato andean mottle comovirus (APMoV), PVY (jointly with PVX) or PLRV-infected potato tubers for sowing in Peru, incomplete autoinfection was found in all cases (Bertschinger et al., 2017). Moreover, changing the growing site to higher altitudes decreased autoinfection for all viruses, indicating environmentally dependent incomplete autoinfection (Bertschinger et al., 2017). Such scenarios have not been investigated in sweetpotato.

Vector-mediated transmission and virus distribution in host plants

It is essential to profile virus distribution across the different tissue types or organs because this may also have a bearing in the virus' vector-mediated transmission. The anatomical and spatial

differentiation between aerial and underground tissues may lead to virus population structuring in the same host plant. For example, preference of PVY to underground storage roots constrains its aerial-borne vector transmission while promoting tuber-mediated transmission (da Silva et al., 2020). Accordingly, differences in nucleotide diversities of PVY populations between potato leaves and tubers were transmission mode-dependent with higher diversities in tubers than in leaves for aphid and mechanically transmitted lineages. In sweetpotato, despite the enigmatic nature of SPMMV vector transmission and organ tropism in sweetpotato plants, isolates of SPMMV show high genetic diversity in comparison to other sweetpotato viruses (Mukasa et al., 2003b; Tairo et al., 2005; Tugume et al., 2010a) and other members of genus ipomovirus (Adams et al., 2005; Adams et al., 2011; Webster & Adkins, 2012). The mode of virus transmission is a key determinant of virus population genetic structure both within and between hosts which determines meta-populations at a community level (Power, 2000; Chare & Holmes, 2004; Forrester et al., 2012; Mondal & Gray, 2017; Mauck et al., 2019; da Silva et al., 2020).

It is reasonable that tuber mediated transmission of influences viral diversity of SPMMV as demonstrated for PVY in potato, although the genetic admixture in tubers prevents an efficient fixation of new alleles (da Silva et al., 2020). Such observations could make sense to SPMMV if sweetpotato storage roots accumulate the virus and if the roots are frequently used in propagation. Virus infected shoot sprouts from abandoned sweetpotato tuberous roots are frequently observed in eastern Africa (Tugume et al., 2016b). Also, Kashif et al. (2012) successfully used storage tubers for virus testing of sweetpotato samples from Central America but SPMMV was not detected, presumably because it is not found in Central America. Similarly, SPFMV and SPCSV have been retrieved from root tubers of plants previously infected with these viruses in Uganda (Adikini et al., 2019). Although not being part of this study, it is plausible that SPMMV, too, can be retrieved from

691 the storage roots of SPMMV-infected sweetpotato. As such, SPMMV-infected but symptomless
692 sprouts arising from tuberous roots may occasionally get included in the farmer-selected planting
693 materials together with the sweetpotato vines that have reverted from SPMMV. In this context
694 therefore, it may be speculated that tubers and their sprouts generate diverse virus populations,
695 providing new alleles to the SPMMV metapopulation and the diversity can be constrained by host
696 reversion and random subsampling when SPMMV is spreading in field through vector transmission.
697 Due to high non-synonymous versus synonymous variability, diversifying selection was detected in
698 the P1 protein of SPMMV whereas purifying selection was implicated for the HC-Pro-, P3-, 6K1-
699 and CP-encoding regions (Tugume et al., 2010a). If SPMMV HC-Pro mediates vector transmission
700 as hypothesized earlier under this review, the purifying selection in this protein would make sense.
701 An extensive analysis of evolutionary signatures in SPMMV should be possible when additional
702 genomic data are available. Nonetheless, the high genetic diversity observed in these isolates coupled
703 with recombination and adaptive evolution (Mukasa et al., 2003b; Tugume et al., 2010a) and
704 infectivity of a broad host range (Hollings et al., 1976; Brunt et al., 1996) may imply an on-going
705 fixation of better fit genotypes in the SPMMV population.
706
707 **East African sweetpotato genotypes most frequently revert from SPMMV infection**
708 Absence of viral infection in plants that were previously infected (reversion) was reported in some
709 eastern African sweetpotato varieties that had been infected with SPFMV (Mwanga et al., 2013;
710 Gibson et al., 2014; Ssamula et al., 2019). Similar reports were made in cassava infected with
711 UCBSV and CBSV (Mohammed et al., 2016). Furthermore, reversion from virus infection was
712 observed on storage root sprouts infected singly with SPFMV, whereas those infected with SPCSV
713 alone showed reversion, and none of the storage root sprouts infected by both viruses showed
714 reversion (Adikini et al., 2019). In cassava, UCBSV-infected plants had a higher rate of reversion

715 when compared to plants infected with CBSV (Mohammed et al., 2016), supporting another line of
716 evidence of devastating nature of CBSV (Alicai et al., 2016). Ssamula et al. (2019) provided the first
717 evidence of sweetpotato reversion from SPMMV. Moreover, highest reversion rates were observed
718 from SPMMV and SPLCV both from sweetpotato genotypes from east Africa and USA and this was
719 postulated to explain the comparatively low field prevalence of SPMMV observed in east Africa
720 (Aritua et al., 1999; Mukasa et al., 2003a; Tairo et al., 2004; Ateka et al., 2004b; Njeru et al., 2008)
721 and SPLCV in the United States (Clark et al., 2012). Observations by Ssamula et al. (2019) show that
722 reversion from viral infection in sweetpotato was virus-specific and also affected by environmental
723 factors, as well as on whether or not the virus is in single or mixed infections. Furthermore, there was
724 host genotypic predisposition to reversion in both sweetpotato cultivars from both east Africa and
725 USA. Higher rates of reversion from SPMMV are certainly beyond the host and environment alone
726 because these are uniform variables with other viruses in the sweetpotato pathosystem of eastern
727 Africa. It is likely that higher reversion rates from SPMMV have to do with unknown virus-specific
728 variables, which require extended studies.
729
730 **GENETIC VARIATION AND EVOLUTIONARY TRAJECTORY OF SPMMV**
731
732 **Genetic variability in isolates of SPMMV**
733 Only 32 isolates of SPMMV have been sequence-characterized to-date (Tairo et al., 2005; Tugume et
734 al., 2010a). Most of the genetic sequence information is available for virus' 3'end of the genome
735 (except for 16 isolates also characterized for their 5' genomic ends) compared to 3 complete (size
736 10818-, 10832-, and 10864-nt) and 13 nearly complete (7679-7688 nt) nucleotide sequences (Colinet
737 et al., 1998; Mukasa et al., 2003b; Tairo et al., 2005; Tugume et al., 2010a). Eight of the 13 nearly
738 complete sequences of SPMMV are of isolates from wild plants belonging to five species (Tugume et

al., 2010a). Availability of these nucleotide sequences has enabled phylogenetic analysis, genetic diversity studies and detection of recombination signatures in sequences of SPMMV (Colinet et al., 1998; Mukasa et al., 2003b; Tairo et al., 2005; Tugume et al., 2010a). The nucleotide sequences of the 3' end of SPMMV isolates from sweetpotato and wild plants share identities of >85% (Mukasa et al., 2003b; Tugume et al., 2010a). In absence of enough complete genomes, the coat protein gene is used for demarcation of *Potyviridae* virus species (Shukla et al., 1994), and as such suggests occurrence of a single species of SPMMV (Tugume et al., 2010a). Phylogenetic analysis of a limited number isolates from wild plants formed a separate cluster, suggesting host-driven evolution of SPMMV isolates (Tugume et al., 2010a). However, this is difficult to ascertain with greater precision due to limited sequence information as phylogentic clusters of isolates from both wild and cultivated species of *Ipomoea* are weakly supported (Fig. 2). It is possible that SPMMV isolates in different wild plants have fixed mutations (diversifying selection) that allow them to colonize those plants but have retained ability to be transmitted by vector(s) to sweetpotato plants.

Selection pressure and recombination are some of the key drivers of plant virus diversity and evolution (García-Arenal et al., 2001; 2003; Elena et al., 2014; Stobbe & Roossinck, 2016; Pagán, 2018). Therefore, studies showed majority of amino acid sites in the characterized protein-encoding genomic regions of SPMMV were under strong purifying selection although a few amino acid sites in which genes were found to be under adaptive evolution (Tugume et al., 2010a). Similarly, strong purifying selection was also reported in the CBSIs (Mbanzibwa et al., 2009a; Mbanzibwa et al., 2011a) but a different study indicated CBSV was evolving faster than UCBSV and had many sites under positive selection as compared to UCBSV (Alicai et al., 2016). Occurrence of positively selected sites in genomes of viruses allows adaptation to new functions. On the other hand, strong purifying selection, which is common to most sites in genomes of both SPMMV and CBSIs, is in line

763 with what would be expected of viruses with small genomes, which is the case with ipomoviruses
764 and other viruses in the family *Potyviridae*.
765
766 Recombination events were detected in the 5'-proximal genomic end of 14 isolates (from wild plants
767 and sweetpotato) and for the 3'end of genomes of 29 isolates (Tugume et al., 2010a). The lack of
768 evidence for major and minor parent like sequences in that study was in favor of the argument that
769 SPMMV originated in eastern Africa and has been evolving there (Tugume et al., 2010a). No
770 SPMMV isolates have been detected or characterized outside this region. Recombination events have
771 been detected in other ipomoviruses, the CBSIs with eastern Africa geographical restrictions
772 (Mbanzibwa et al., 2011a). Analyzing complete genomes of CBSV and UCBSV, respectively,
773 revealed recombination events in the 3'proximal region of CBSV and in P1, CI, VPg, NIb, HAM1h,
774 CP encoding regions and the 3'UTR (Mbanzibwa et al., 2011a; Ndunguru et al., 2015). Therefore,
775 recombination appears to be the main driving force for the evolutionary diversification of SPMMV
776 and CBSIs. These three ipomoviruses, while embracing strong purifying selection, they both allow
777 exchange of genetic material through homologous recombination as a means of maintaining genetic
778 variability.
779
780 **Genomic differences and similarities between SPMMV and other ipomoviruses**
781 SPMMV and CBSIs are geographically and taxonomically related because they are both important
782 ipomoviruses of economically important vegetatively cultivated crops in eastern Africa. The typical
783 genome of SPMMV is structurally similar to that of potyviruses and translates into a polyprotein that
784 autocatalytically cleaves into ten mature proteins (Colinet et al., 1998; Adams et al., 2005; Valli et
785 al., 2006; Valli et al., 2007; Cui & Wang, 2019). However, SPMMV differs from its cogners in the
786 genus *Ipomovirus* in many aspects and here we specifically consider CBSIs which like SPMMV

emerged in eastern Africa, although the former have now fast expanded their initial geographic niches to neighboring areas including parts of south, central and western Africa (Rey & Vanderschuren, 2017; Mulenga et al., 2018; Casinga et al., 2021). Firstly, SPMMV has an HC-Pro, which is not found in the genomes of CBSIs. Absence of HC-Pro is observed in all ipomoviruses except SPMMV and recently assigned ipomoviral species ToMMoV (Abraham et al., 2012; Walker et al., 2020). Secondly, the size of SPMMV P1 (83kDa) is nearly twice as large as P1s of CBSIs (42kDa) (Colinet et al., 1998; Adams et al., 2005; Valli et al., 2007; Mbanzibwa et al., 2009b). Thirdly, the CBSIs have encoded a Maf/HAM1-like sequence, which is recombined between the replicase and coat protein domains in the 3'- proximal part of their genomes. Only two other viruses, the potyvirus Euphorbia ringspot virus (Mbanzibwa et al., 2009b; Knierim et al., 2017) and the torradovirus Cassava torrado-like virus (Leiva et al., 2022) infecting euphorbiaceous plants have encoded this class of sequence in their genomes. Fourthly, whereas all proteolytic cleavage sites in sequences of CBSIs are easy to predict based on conserved amino acids around cleavage sites (Adams et al., 2005), the cleavage site for NIb/CP in the sequence of SPMMV has not been completely resolved (Colinet et al., 1998; Mukasa et al., 2003b; Tugume et al., 2010a). Different sites for cleavage of SPMMV CP from NIb have been proposed and it could be that there is more than one site as the sizes of CP reported differ. Whereas Hollings et al. (1976) reported the size of the SPMMV CP to be 37.7kDa, Tugume et al. (2010a) demonstrated that SPMMV CP size was 35kDa. Understanding cleavage sites in the sequences of SPMMV is important; for instance, the NIb/CP cleavage site is normally used for insertion of foreign sequences, which enable use of viruses as vectors and allow studies of gene functions (Kelloniemi et al., 2008). Moreover, this cleavage site has become interesting to virologists following the finding that in some viruses, including CBSIs, foreign sequences are naturally recombined between CP and NIb (Mbanzibwa et al., 2009b; Tomlinson et al., 2019a; Tomlinson et al., 2019b; Goh & Hahn, 2021; Palani et al., 2021).

The P1 protein of CBSIs, ToMMV and SPMMV are not duplicated compared to those of other known ipomoviruses (Valli et al., 2006; Valli et al., 2007; Cui & Wang, 2019). The much larger P1 protein of SPMMV compared to other viruses in the family *Potyviridae* makes it difficult to achieve accurate multiple sequence alignment of P1 sequences when SPMMV sequences are included in the alignment. Thus, one may argue that it is likely that P1 of SPMMV is also duplicated but that determination of a cleavage site will require laboratory experiments. Comparison of nucleotide sequences of P1s of ipomoviruses shows that P1 of SPMMV is distantly related (<25%) to P1 of CBSV, UCBSV and coccinia mottle virus (CocMoV) and to P1a and P1b of CVYV, SqVYV and CocMoV (Table 1). However, CI (50.9-55%) and NIb (51.6-57%) have the highest nucleotide sequence similarities between SPMMV and other ipomoviruses including CBSIs. A phylogenetic analysis using complete genomic sequences of all seven known species in the genus *Ipomovirus* places SPMMV into a separate group distinct from that of CVYV, SqVYV, CBSV and UCBSV (Fig. 3). The other virus with HCPro, ToMMV forms a third and distinct group from other groups (Fig. 3). Amino acid similarities with SPMMV are highest with ToMMV (Table 1). The close sequence identities (Table 1) and phylogenetic clustering of ToMMV with SPMMV separate from other ipomoviruses (Fig. 3) and these being HCPro-encoding members suggests a likelihood that SPMMV and ToMMV may belong to a genus that is different from that for CBSV and UCBSV and other ipomoviruses.

Homologous proteins shared between SPMMV and CBSIs may contain conserved amino acid motifs performing different functions. For instance, their P1 proteins contain the basic LxRA and zinc finger motifs, which are associated with RNA silencing suppression in plants (Mbanzibwa et al., 2009b; Giner et al., 2010; Valli et al., 2018). The DAG motif in the CP is associated with transmission of

potyviruses by aphids (Atreya et al., 1995). However, the DAG motif is missing in SPMMV and UCBSV (Colinet et al., 1998; Mukasa et al., 2003b; Tugume et al., 2010a; Ateka et al., 2017) yet it is present in CBSV, CocMoV and SqVYV (Ateka et al., 2017). Unlike for potyviruses, the HCPro of SPMMV does not participate in RNA silencing suppression and occurrence of PTK motif in the SPMMV HCPro has not been evaluated for SPMMV transmission by aphids. Recently, Lindenau et al. (2021) demonstrated that the N-terminal region of CVYV CP (between 93-105 aa positions) functions in whitefly transmission. This region shows conserved aa residues between CVYV, CBSIs, CocMOV, SqVYV, ToMMoV and SPMMV (Lindenau et al., 2021). The *HAM1* gene in CBSIs is speculated to encode proteins which reduce mutagenesis by intercepting and preventing incorporation of non-canonical nucleoside triphosphates into DNA and RNA (Galperin et al., 2006; Mbanzibwa et al., 2009b) as determined previously (Noskov et al., 1996; Takayama et al., 2007). However, no experimental evidence of CBSV *HAM1* gene to protect CBSV's genome from mutations was found (Tomlinson et al., 2019b). These discrepancies in presence and/or absence of genes or motifs underpin the necessity of complementing bioinformatics predictions with experimentations because presence of homologous genes or motifs may not necessarily imply similar homologous functions. Similar motifs and genes present in SPMMV and potyviruses may be an indication of evolutionary relics of common ancestry between SPMMV (or other ipomoviruses) and potyviruses that got fixed with enhanced functions in the latter while having lost those functions in the former.

COMPARABLE EMERGENCE OF SPMMV AND CBSIs IN EASTERN AFRICA

The emergence of SPMMV is comparable to that of CBSIs, the causative agents of cassava brown streak disease (CBSD). Initially, CBSIs were endemic and limited only to the eastern Africa coastal areas and had not been detected in West Africa and other countries far from the great lakes region of Africa. However, recent reports indicate a wide spread of CBSIs into southern, central and western

parts of Africa (Bigirimana et al., 2011; Mulimbi et al., 2012; Hillocks & Maruthi, 2015; Chipeta et al., 2016; Rey & Vanderschuren, 2017; Koima et al., 2018; Munganyinka et al., 2018; Ano et al., 2021; Casinga et al., 2021). The rapid westwards and southward spread of CBSIs in Africa projects a heavy presence of CBSIs and CBSD by 2030 (Jarvis et al., 2012; Rey & Vanderschuren, 2017; Mero et al., 2021; Ano et al., 2021).

Albeit limited knowledge on alternative hosts of CBSIs, it seems that, unlike SPMMV, they have a narrow natural host range and it is believed that, like SPMMV, they originated from eastern Africa from a yet to be identified natural host. CBSIs have been detected in *Manihot glaziovii* in cassava in Tanzania and Mozambique (Mbanzibwa et al., 2011a; Mbanzibwa et al., 2011b; Amisse et al., 2019). Only CBSV has been detected in non-cassava relatives, *Zanha africana* (Radlk.) Exell. and *Trichodesma zeylanicum* (Burm.f.) R.Br., in Mozambique (Amisse et al., 2019). However, the CBSIs were not detected in plants of 60 alternative hosts in Uganda (Legg et al., 2011) suggesting a narrow natural host range or a recent introduction of these viruses through planting material.

It is likely that the natural hosts, from which the SPMMV and CBSIs jumped to sweetpotato and cassava plants, respectively, remain unknown to-date. This has implications on our understanding of the ecologies of these viruses and their potential of reemergence. For instance, during colonial rule, CBSD was eradicated in Uganda only to reemerge in the late 1990's (Alicai et al., 2007). Reemergence of CBSD in Uganda was attributed to the introduction of the viruses into an area with high whitefly populations and susceptible cassava varieties (Alicai et al., 2007). Indeed, a recent study has shown CBSD incidence increases with increased whitefly populations (Shirima et al., 2020). This could also be due to occurrence of unknown alternate host(s) of the virus and arrival of new cassava genotypes generated during the efforts to eradicate cassava mosaic disease (CMD). This

883 argument is supported by the fact that the new cassava genotypes, with resistance to CMD, are
884 susceptible to CBSD (Alicai et al., 2007; Beyene et al., 2016). While today SPMMV is less of a
885 problem especially in single infections, it can in the future through some circumstances become a
886 threat to sweetpotato production just like CBSV and UCBSV to cassava production in the region.
887
888 **EMERGENCE OF SPMMV VIA "NEW ENCOUNTER SCENARIO" IN EASTERN AFRICA**
889
890 **Endemism versus exoticism of SPMMV in eastern Africa**
891 The endemism of SPMMV in eastern Africa is a hypothesis arising from lack of evidence of the virus
892 outside the region, postulating that SPMMV invaded the newly introduced sweetpotato in eastern
893 Africa via a new "encounter scenario" about 400 years ago (Tairo et al., 2005; Tugume et al., 2010a;
894 Clark et al., 2012; Lindenau et al., 2021). "New encounter scenarios" describe situations where plants
895 (in this case sweetpotato) introduced into new areas allows them to come into contact with viruses
896 with which they have not interacted before and to which they express no resistance (Jones, 2009;
897 Jones & Coutts, 2015; Jones, 2020; Jones, 2021). It also refers to situations where plant viruses are
898 transferred from their indigenous hosts to cultivated hosts or are transported to other areas as new
899 disease agents. In this sense, plant viruses and their principle hosts should have common centres of
900 origin, unless if the viruses were derived from new encounter scenarios (Lovisolo et al., 2003; Jones,
901 2009; Jones & Coutts, 2015; Jones, 2020; Jones, 2021). Sweetpotato originated in tropical America
902 (Austin, 1975; 1988; Roullier et al., 2013) and dispersed around the world mainly via human-
903 mediated migration towards the start of the 16th century (Lebot, 2010). An exception is Australasia
904 and South Pacific where there is evidence for prehistoric sweetpotato cultivation (Yen, 1963;
905 O'Brian, 1972; Huang & Sun, 2000; Zhang et al., 2004; Switek, 2013). Indeed, evidence shows that
906 the introduction of sweetpotato into Polynesia pre-dates human colonization of the region by

907 thousands of years, probably via long-distance mediated buoyant seed dispersal by ocean currents
908 (Muñoz-Rodríguez et al., 2018). The crop was introduced to Africa by the Portuguese later in the 16th
909 century (Zhang et al., 1999; Zhang et al., 2004), probably first into eastern Africa in Tanzania, then
910 from east to west Africa (Lebot, 2010). Accordingly, west Africa sweetpotato germplasm was
911 derived from east African sweetpotato and would imply co-dispersal with SPMMV from eastern
912 Africa to western Africa. However, SPMMV is absent in western Africa (Clark et al., 2012; Gutierrez
913 et al., 2012; Tibiri et al., 2020). In addition, the lower diversity of sweetpotato in east Africa than in
914 west Africa suggests that west African sweetpotato is not simply a sub-sample from eastern Africa
915 but might have been independently introduced into west Africa later on (Glato et al., 2017).
916
917 In contrast, the hypothesis of "exoticism of SPMMV in eastern Africa" is derived from the second
918 definition of new encounter scenarios; that is, transport of the viruses in plants (in this case
919 sweetpotato) to distant areas as new disease agents there (Jones, 2009; Jones, 2020). Under this
920 hypothesis, SPMMV or its progenitors existed in the wild plants in tropical America prior to their
921 domestication as sweetpotato in their centre of origin and were later dispersed across the world
922 including introduction to eastern Africa. It is believed that prior to plant domestication, plant viruses
923 co-evolved or co-existed with their natural wild host plants in the plants' centers of origin (Lovisolo
924 et al., 2003; Jones, 2009; Jones, 2020; Jones, 2021), but this co-evolutionary 'balance' was
925 drastically altered following the domestication of wild plants and agricultural intensification
926 (Diamond, 2002; Jones, 2009; Jones & Coutts, 2015; Purugganan, 2019; Jones, 2020). This
927 disruption was further exacerbated by the gradual dispersal of crops away from their original centers
928 of domestication to other regions (Harlan, 1965; 1971; Lovisolo et al., 2003) which created
929 opportunities for "new encounter scenarios" between host plants and viruses (Jones, 2009; Jones,
930 2020). However, absence of SPMMV outside eastern Africa even when globally most or all

sweetpotatoes originate from one centre of origin in tropical America makes the hypothesis of "exoticism of SPMMV in eastern Africa" quite unlikely. The only possibility becomes if east Africa provided a "unique conducive environment" for the initial perpetuation and persistence of SPMMV something that did not happen elsewhere including the centre of origin in tropical America e.g.,Stobbe et al. (2012) and Lefeuvre et al. (2019). Wild relatives of sweetpotato from Uganda in eastern Africa are the only alternative hosts that have been analysed for SPMMV infectivity indicating close genetic identities with isolates from cultivated plants (Tugume et al., 2010a); and whether SPMMV occurs in wild plants in tropical America is unknown. Together, these observations support the hypothesis of SPMMV endemism in eastern Africa and indicate incompatibilities between origins of sweetpotato host and SPMMV. SPMMV is probably not a "sweetpotato virus": it existed in east African wild *Ipomoea* species and/or other closely related taxa (Verdcourt, 1963; Blundell, 1992; Agnew & Agnew, 1994; Tugume et al., 2008) as primary hosts and invaded sweetpotato when introduced from tropical America. To-date, there are only few reports, mostly in Australia, of plant viruses still restricted to wild plants and natural ecosystems (Jones, 2009; Vincent et al., 2014; Jones & Coutts, 2015; Jones, 2020) which is a contrast from SPMMV in east Africa. A coalescent and more articulate phylogeographic analysis and evolutionary trajectories of SPMMV should be done when sufficient genetic, genomic, and biological information is available.

Eastern Africa as a hotspot of plant virus emergence and reemergence

The Great Lakes region of eastern Africa is known for supporting the emergence and diversification of unique genotypes of plant viruses. In the sweetpotato cropping system, the expectation is that fairly identical viruses, viromes and/or their progenies occur worldwide in sweetpotato, if virus dispersal happened along with their host. This seems to be the case for some viruses such as SPFMV, SPCSV, SPCFV, SPVC, and various sweepoviruses. However, some specific genotypes or strains of

these viruses were also previously reported as restricted to eastern Africa. For example, the "EA strain" of SPFMV was initially known to be restricted to eastern Africa where it shows greatest prevalence over other SPFMV strains (Kreuze et al., 2000; Mukasa et al., 2003c; Tairo et al., 2005; Tugume et al., 2010b; Tugume et al., 2013; Wokorach et al., 2020), although subsequent reports show the presence of this strain elsewhere. Similar observations are made for SPCSV that has only two contrasting strains, of which the "EA strain" is the only strain prevalent in eastern Africa (Tairo et al., 2005; Tugume et al., 2013; Qin et al., 2013b; Qin et al., 2013c) and only lately detected elsewhere. Isolates of the "EA strain" of SPCSV from wild plants and sweetpotato eastern Africa may encode or not encode an RNA silencing suppressor p22-encoding sequence at the 3'-proximal region of RNA1. In contrast, the p22 gene is consistently absent in isolates from outside eastern Africa (Cuellar, 2008; Cuellar et al., 2011a; Tugume et al., 2013; Qin et al., 2013c; Wang et al., 2021a), indicating unique variability of isolates of "EA strain" of SPCSV from eastern Africa. One new viral species related to SPCSV and encoding an RNase3-like RNA-silencing suppressor protein has also been detected in Uganda (Tugume et al., 2013) and Tanzania, although this virus seems to be currently rare in cultivated sweetpotato. Isolates of the carlavirus SPCFV from eastern Africa also shown to cluster alone or with those from Peru and separate from those originating from Asia (Tugume et al., 2016a). These data show that specific viral genotypes of SPCFV or trains are more common than others in eastern Africa. The geographical range of plant viruses and successful perpetuation within an agro-ecosystem is constrained more by virus-vector than by virus-host plant relations (Power, 2000; Power & Flecker, 2003; Power, 2008; Power & Flecker, 2008) and this may influence dominance of one or more strains over the others. The optimization of virus-host and virus-vector may therefore be an ongoing process in unique viral genotypes in eastern Africa with an on-going adaptive evolution generating better fit genotypes.

The significance of east Africa and its native wild flora on the evolution and diversification of SPMMV parallels that of viruses in other important crops in the region, besides sweetpotato. Besides the CBSIs already mentioned above in cassava, the virulent recombinant strain of cassava mosaic begomoviruses (CMBs) exhibited a gradient of decreasing prevalence east-to-south of Africa (Legg, 1999; Ndunguru et al., 2005; Bull et al., 2006; Patil & Fauquet, 2009). Rice yellow mottle virus (RYMV, genus *Sobemovirus*) showed phylogenetic congruence with geographical origin of isolates on east-to-west transect across Africa and decreased nucleotide diversity westward across Africa (Fargette et al., 2004; Traore et al., 2005; Fargette et al., 2006; Traoré et al., 2009; Ochola et al., 2015; Jones, 2020; Ramathani et al., 2021). Most of the strains of RYMV, including the most divergent ones, were found in the eastern Arc Mountains of east Africa (Fargette et al., 2004; Traore et al., 2005; Traoré et al., 2009; Trovão et al., 2015; Pinel-Galzi et al., 2015). The Eastern Arc mountains occupy the eastern coast of Tanzania and parts of offshore islands of Pemba and Zanzibar and constitute the main biodiversity 'hotspot' in Africa containing several endemic vascular plants, herbs and grasses (Myers et al., 2000; Küper et al., 2004; Burgess et al., 2007; Skarbek, 2008; Dimitrov et al., 2012). The climate in these areas is modulated by the Indian Ocean promoting variabilities of micro-climatic gradients away from east Africa (Marchant et al., 2007; Nicholson, 2017; Blau & Ha, 2020) which may promote emergence of different genotypes of plant virus populations. Indeed, unique historical climate changes in eastern Africa account for the increase in abundance of whiteflies and contributing to crop disease pandemics (Kriticos et al., 2020). The south-west Indian Ocean islands off the coast of east Africa, for example, are home to an emerging begomovirus species complex that is associated with serious disease outbreaks in bean, tobacco and tomato plants (Delatte et al., 2005; Lefeuvre et al., 2007; Scussel et al., 2018). CBSIs also postulated to originate in eastern Africa show distinct species of these viruses in mainland eastern Africa (the Lake Victoria basin) and Indian Ocean coastal areas are found (Mbanzibwa et al., 2009a; Mbanzibwa

et al., 2011a; Mbanzibwa, 2011; Ndunguru et al., 2015). The main difference from SPMMV is that the phylogeographic scenarios exhibited by RYMV, CMBs and CBSIs in east Africa are seemingly absent in SPMMV, possibly due to lack of sufficient genomic sequence data available for extended analysis. Nevertheless, these data further demonstrate the wealth of eastern African region with respect to plant virus emergence and evolution.

CONCLUSIONS AND FUTURE RESEARCH DIRECTIONS

The emergence and reemergence of plant viruses remains one of the most pressing challenges to the 21st century agriculture (Jones, 2009; Jones, 2020; Amari et al., 2021; Jones, 2021). Because virus emergence is a fundamental result for climate change effects on both agro-ecosystems and natural ecosystems (Jeger, 2020; Jeger, 2022), it is likely that agriculture will experience more frequent episodes of plant virus emergencies and probably more destructive virus disease epidemics. The climatic patterns in eastern Africa as influenced by the Indian Ocean dipole creates a series of irregular microclimatic gradients away from the Indian Ocean coastal areas (Marchant et al., 2007; Blau & Ha, 2020) may drive emergence and perpetuation of different virus genotypes. In cases where the virus genotypes have not caused economic impact on the host crop plants, scenarios like that of CBSIs reemergence on cassava in eastern Africa may arise. The CBSIs incite highly damaging CBSD on cassava but this disease remained of no economic importance since the 1930's when it was first reported in eastern Africa until 1990's to mid-2000's when the disease "exploded" (Storey, 1936; Monger et al., 2001a; Monger et al., 2001b; Hillocks & Jennings, 2003; Alicai et al., 2007). CBSD remains the single most destructive threat to cassava farming in Africa (Pennisi, 2010; Ano et al., 2021). Whereas efforts to contain CBSD in eastern Africa were at first successful, the disease has already extended its borders and is fast spreading westwards with projections to affect western Africa by 2030 (Jarvis et al., 2012; Ano et al., 2021). Using this analogy, SPMMV was first reported on

sweetpotato in eastern Africa in the 1970's (Hollings et al., 1976; Tairo et al., 2005; Tugume et al., 2010a). Since then, SPMMV has remained restricted in the region where it is the third most prevalent virus on sweetpotato (Clark et al., 2012). Like CBSIs prior to the 1990's, SPMMV is currently considered to be of less economic significance, except when the virus occurs in mixed infections with SPCSV, hence becoming a component of virus disease complexes of sweetpotato (Mukasa et al., 2006; Tugume et al., 2010a). However, as demonstrated for CBSIs in eastern Africa, and numerous other viruses elsewhere (Jones & Naidu, 2019; Jones, 2020; Jones, 2021), it is expected that SPMMV is not constrained to perpetually occupy a single ecological niche in eastern Africa because the virus has genetic and evolutionary potential for enhancement of its fitness advantages (Tugume et al., 2010a). Due this high likelihood SPMMV reemergence, it is critical that we build essential basic scientific information in advance that has remained vague or absent for now half a century on this virus. Lack of accurate information on a virus and the associated disease(s) limits effectiveness of measures used in disease management: hence, the following information is most needed in the effective management of any disease outbreak(s) associated with SPMMV.

Firstly, vector transmission dynamics including determining the actual vector(s) transmitting SPMMV and the factors that may enhance or limit this process require urgent profiling. This review has articulated different hypotheses for evaluation through rigorous experimentation. The hypothesis on "opportunistic transmission of SPMMV by aphids using SPFMV as a helper virus" may be tested through sequential aphid feeding (or inoculation) access events between single SPFMV (or its encoded protein), SPMMV or dual SPFMV + SPMMV or triply SPFMV + SPMMV + SPCSV infected plants. The second hypothesis of "SPMMV transmission being modulated by synergism with SPCSV" may be tested by transmission experiments where source plants are singly or co-infected with SPCSV, irrespective of the candidate vector. It is noteworthy that vector

transmissibility is often lost or compromised during serial mechanical passages of plant viruses (Legavre et al., 1996; Gray & Banerjee, 1999; Garcia et al., 2019). It is therefore essential that the transmission experiments use SPMMV isolates directly from nature or those that have not stayed for long outside the natural environment.

Secondly, the transmission dynamics of SPMMV need to be evaluated under field conditions in association with other sweetpotato viruses. Most surveys conducted in the eastern Africa have provided only a sketchy evidence of association of virus incidence and, in some cases, severity of virus disease complexes with the abundance of whiteflies and aphids. For example, Ndunguru et al. (2009) reported a high positive correlation of whitefly abundance in the Lake Victoria basin with SPVD incidence but not severity. The same study also reported clear correlation of low SPVD incidence with low whitefly and aphid population in southern Tanzania. A positive correlation of whitefly abundance with SPMMV severity in Uganda was previously observed (Kisekka, 2016), although correlation of the disease or virus with abundance of a potential insect vector does not necessarily confirm the vectoring ability. Similarly, Maruthi et al. (2005) reported the inability of *B. afer* to transmit the CBSV despite the apparent observed association of the CBSD with the whitefly species in the field being evident. Therefore, comprehensive field associations between viruses and virus diseases with vectoring agents require extended studies especially in contrasting microclimates of coastal east Africa lowlands and mainland areas of high altitude closet the Lake Victoria basin. Sweet potato leaf curl virus (SPLCV) is an emerging virus of sweetpotato in east Africa. It is also important that virus associations with SPMMV be extended beyond sweetpotato RNA viruses in east Africa (SPCSV and SPFMV). For example, Wanjala et al. (2020) demonstrated increased symptoms severity and reduced yields when there is coinfection of SPLCV with SPCSV and SPFMV, but

whether or not a coinfection of SPMMV with SPLCV results into reduced yields and enhanced disease symptoms is not known.

Thirdly, histo-localization of SPMMV, and whether or not this localization is variable according to the multiplicity of infection should be profiled. This may be collaborated with the feeding behavior of the vector(s) transmitting SPMMV for which optimization may be either by short probing events in the epidermal tissues or by stylet penetrations into the phloem tissues under field conditions. Moreover, plant virus populations in an individual host plant may show population structuration as was recently shown for PVY in potato in which tuber-mediated transmission generated higher diversity of viral populations (da Silva et al., 2020). Similarly, potato, which has similar growth habit and propagation means as sweetpotato shows organ-based accumulation of PVY (Kogovšek et al., 2011). The exception is that sweetpotatoes are propagated from cuttings as opposed to stem tubers in potato, although sweetpotato tubers may be used in propagation. A finding that sweetpotato stems are more susceptible to SPFMV and SPCSV infection (Zhang et al., 2020) may further imply possibilities of organ- or tissue-dependent structuration of SPMMV although SPMMV was not part of this study. Furthermore, sweetpotato shows reversion most frequently from SPMMV infection (Ssamula et al., 2019): this phenomenon require extended analysis at the individual host plant level prior to meaningful extrapolation on plant community level.

Last, but not least, it is necessary to improve the extremely limited sequence data for SPMMV, especially for whole genomes to enable thorough testing of genetic and evolutionary hypotheses. Additional sequence information covering both complete SPMMV genomic space as well as comprehensive geographical coverage is urgently needed. This is useful in extending analysis of driving forces in reemergence of the virus and to model possible evolutionary trajectories in the

sweetpotato cropping system of eastern Africa and allow empirical comparisons with existing scenarios. Virus diseases still rank highest in importance and urgency for research on sweetpotato (Fuglie, 2007; Clark et al., 2012; Zhang et al., 2020). Therefore, all these information gaps should be filled in advance before reports of reemergence, if they are to be applied in the containment of emerging disease epidemics.

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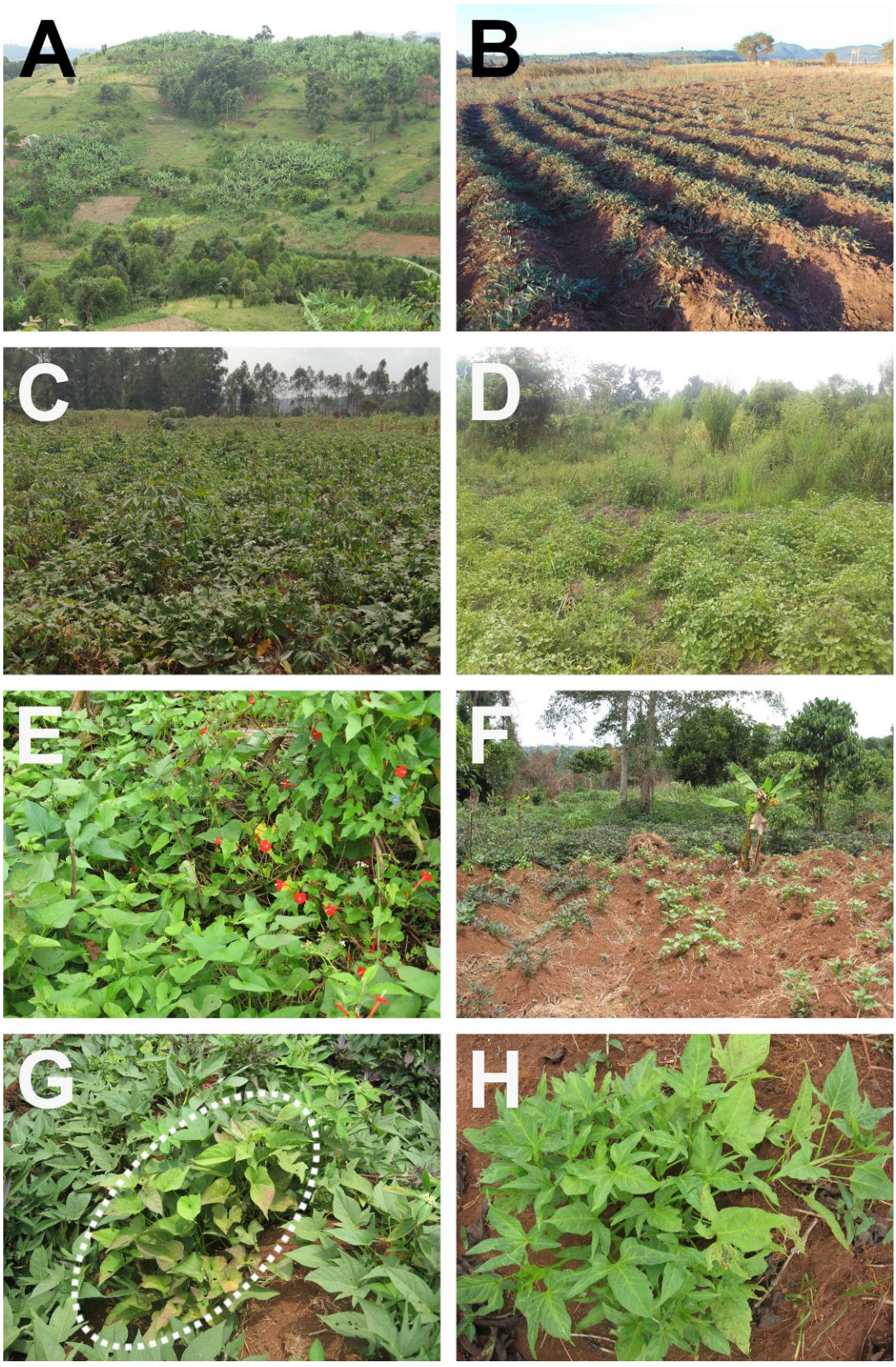
Table 1. Percent nucleotide sequence identities of protein encoding sequences of SPMMV (accession NC_003797) with homologous proteins of other known ipomoviruses. Comparisons were made using ClustalW method implemented in MEGA7 (Kumar et al., 2016).

Protein	CBSV	UCBSV	ToMMV	SqVYV	CVYV	CocMoV
P1	20.6	21.1	17.8	-	-	-
	-	-	-	20.6[a]	22.5[a]	20.6[a]
	-	-	-	16.9[b]	15.7[b]	17.6[b]
HC-Pro	-	-	44.5	-	-	-
P3	45.7	47.6	37.5	43.6	39.1	43.7
6K1	49.3	48.7	44.5	47.2	48.1	50.0
CI	54.8	53.6	50.9	52.8	55.0	53.7
6K2	42.2	45.3	44.2	43.9	41.4	43.9
VPg	45.1	46.8	42.3	45.2	43.4	44.3
NIa	42.6	40.7	39.8	40.7	40.0	39.5
NIb	54.7	53.7	51.6	53.7	54.4	57.0
CP	37.4	39.6	42.5	38.8	39.2	38.5

SqVYV, CVYV and CocMoV have duplicated P1 proteins in form of P1a and P1b instead of a single P1 protein. The numbers marked with [a] and [b] refer to percent identities of P1a and P1b nt sequences of these viruses, respectively, with the P1 of SPMMV. Similarly, other viruses except SPMMV and ToMMV lack the HCPro. The dashes (-) imply absence of protein in given virus genome.

1123



1124

Fig. 1. Some of the characteristic features of sweetpotato cropping systems in eastern Africa that promote the perpetuation and persistence of viruses and their vectors on the crop as well as ease of vector-mediated transmission. **(A)** Sweetpotato is one of the many crops in a locality with a mixture of crop husbandry and agro-forestry in Kanungu, southwestern Uganda, creating a heterogeneous community of crop stands of a diversity of alternative hosts of viruses and vectors; **(B)** Medium-sized sweetpotato farm in Arusha, Tanzania with a single variety that could allow easy perpetuation of virus diseases; **(C)** A mixed crop stand of sweetpotato and cassava in Mukono, central Uganda which allows continuity of vectors and facilitating repeated transmission; **(D)** A sweetpotato field adjacent to numerous species of wild plants which favors ease of virus transmission between wild plants and sweetpotato in Kabarole, western Uganda; **(E)** A sweetpotato garden with some vines at the hedge growing intertwined with plants of *Ipomoea hederifolia* (with red petalled flowers), a natural reservoir of SPMMV, SPFMV and SPCSV in Rukungiri, southwestern Uganda; **(F)** Sweetpotato gardens in Mpigi, central Uganda at different stages of growth and adjacent to each other: vines from an old garden in the background (4-months' old) were used to initiate the garden on the left foreground (1-month old), and right foreground (3-weeks' old) allowing simultaneous transmission and perpetuation of viruses in the crop; **(G)** Virus-infected plant with symptoms of SPVD (dotted circle) most likely from a vine planted with the infection and surrounded by healthy-looking allowing vines allowing ease of virus transmission to nearly plants in Kakamega, western Kenya; **(H)** Symptoms of sweet potato virus disease on a plant that emerged as a sprout from an abandoned storage tuber from a previous garden of sweetpotato in Mbale, eastern Uganda.

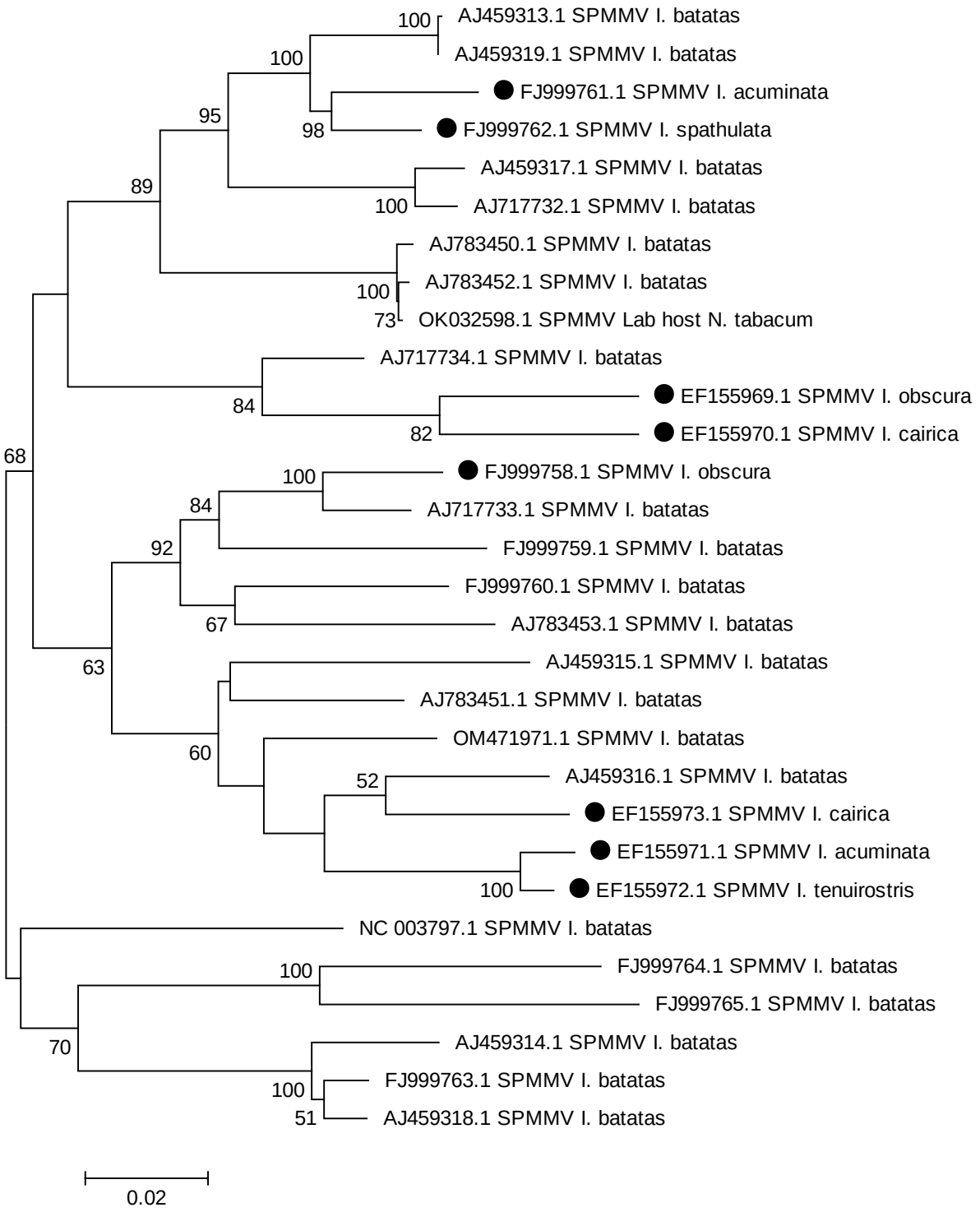


Fig. 2. Molecular phylogenetic tree generated using 30 nucleotide sequences (ca. 1,800nts) encoding the 3' end of SPMMV genome containing partial NIb, complete CP and 3'UTR. Only isolates with this genomic region sequenced were included in the analysis. The isolates whose sequences are used were either isolated from sweetpotato (*Ipomea batatas*) or alternative wild plants (*I. acuminata, I. spathulata, I obscura, I. cairica,* and *I. tenuirostris* with a symbol ● preceding sequence accession

number). Phylogenetic clustering was inferred by using the Maximum Likelihood method based on the Tamura-Nei model (Tamura & Nei, 1993). Values shown at the branch nodes represent bootstrap values of 1000 replicates; only values greater than 50% are shown. The tree is drawn to scale, with branch lengths measured in the number of substitutions per site. All positions containing gaps and missing data were eliminated and therefore there were a total of 1775 positions in the final dataset. Phylogenetic analysis was conducted in MEGA7 (Kumar et al., 2016).

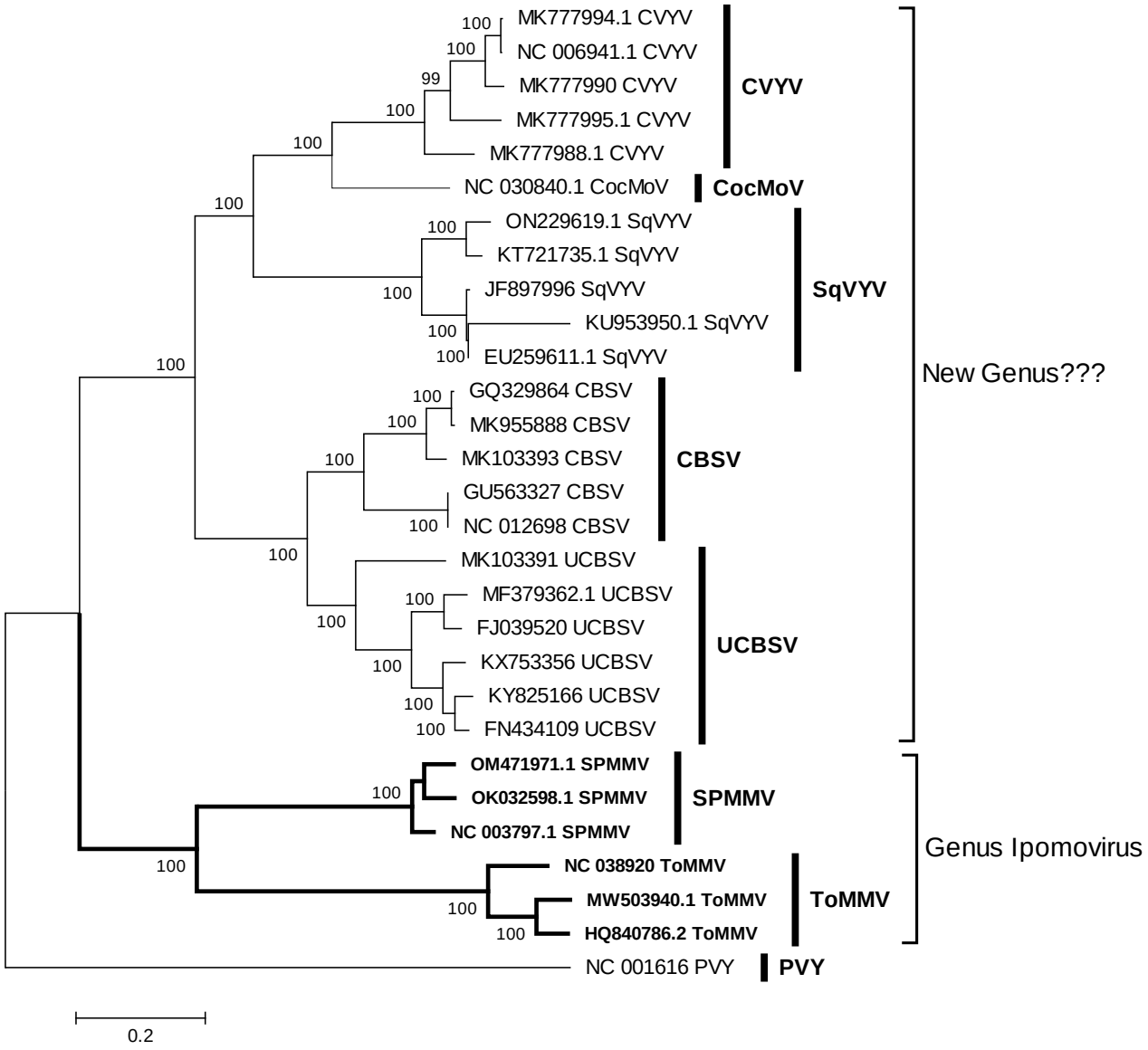


Fig. 3. Molecular phylogenetic tree generated using 29 representative complete genome sequences of ipomoviruses. Currently, the International Committee on Taxonomy of Viruses (ICTV) recognizes only seven virus species. In the analysis, each species was represented by at least one viral sequence: UCBSV, CBSV, SqVYV, CocMoV, CVYV, SPMMV and ToMMV. Accession numbers are shown for each sequence before the abbreviations of virus names. Potato virus Y (PVY; *Potyvirus*) was used as an out-group. Names and branches of HC-Pro encoding members are shown in bold. Phylogenetic tree was inferred by using the Maximum Likelihood method based on the Tamura-Nei model (Tamura & Nei, 1993). Values shown at the branches represent bootstrap values of 1000 replicates; only values greater than 50% are shown. The tree is drawn to scale, with branch lengths measured in

the number of substitutions per site. Although the analysis involved complete nucleotide sequences, all positions containing gaps and missing data were eliminated and therefore, there were a total of 7554 positions in the final dataset. Phylogenetic analysis were conducted in MEGA7 (Kumar et al., 2016).

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