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Phytobiomes Journal
A Transdisciplinary Journal of Sustainable Plant Productivity

REVIEW

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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Accepted for publication 29 November 2022.

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 or environmental factors dictate its epidemiological status. An example is sweet potato mild mottle virus (SPMMV), which was first reported in the 1970s on sweetpotato in eastern Africa. SPMMV has remained endemic in the region and poorly understood, yet accounting for 60 to 95% of losses, especially in mixed infections. Unlike other sweetpotato viruses which have global incidences, 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 more 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.

Keywords: coinfection, 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

Viruses are not constrained to perpetually occupy a single ecological niche (Lefeuvre et al. 2019; Malmstrom et al. 2011; Roossinck 2013, 2015). 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 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 the ability to infect a new host, followed by adaptation by ensuring successful transmission between hosts and, finally, gaining ability to spread epidemically (Elena et al. 2014; Lefeuvre et al. 2019: Rojas and Gilbertson 2008).

Apparently, plant virus emergence is an "ecoevolutionary" process, because, whereas 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 (French and Holmes 2020; Geoghegan and Holmes 2017; Lefeuvre et al. 2019; Roossinck and García-Arenal 2015). 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

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The author(s) declare no conflict of interest.

have been effectively optimized (Islam et al. 2020; Jeger 2020; Scholthof 2007). Thus, the impact of climate variability on agroecosystems makes plant virus emergence a significant factor for 21st century agriculture (Anderson et al. 2004; Canto et al. 2009; Jones 2009, 2021; Jones and Naidu 2019; Lal et al. 2020; Ristaino et al. 2021; Rodríguez-Nevado et al. 2018).

Sweet potato mild mottle virus (SPMMV; genus *Ipomovirus*, family *Potyviridae*) infects sweetpotato (*Ipomoea batatas* Lam) and related wild plants in eastern Africa, where it was first reported on sweetpotato in the 1970s (Clark et al. 2012; Hollings et al. 1976; Tugume et al. 2010b). Virus-specific methods frequently used for virus detection in sweetpotato may cause variants of certain viruses to remain undetected and, consequently, unstudied using genomic sequence analysis. Unconfirmed reports show that SPMMV has also been detected serologically in New Zealand, Indonesia, Peru, and Malawi (Carey et al. 1997; Fletcher et al. 2000; Mbewe et al. 2021; Tairo et al. 2005), and by reverse-transcription (RT)-PCR in South Africa (Sivparsad and Gubba 2013). However, these reports are not backed by genetic sequence data. Therefore, the most reliable detection and sequence data to date indicate that SPMMV is geographically restricted in eastern Africa (Clark et al. 2012; Gu et al. 2014; Ibaba and Gubba 2020; Jo et al. 2020; Kashif et al. 2012; Nakasu et al. 2022; Nhlapo et al. 2018; Orfanidou et al. 2022).

In contrast to SPMMV, other viruses infecting sweetpotato within the same eastern Africa region are also frequently detected elsewhere globally. For example, incidences of the East African (EA) strain of sweet potato feathery mottle virus (SPFMV; genus *Potyvirus*, family *Potyviridae*) and sweet potato chlorotic stunt virus (SPCSV; genus *Crinivirus*, family *Closteroviridae*) that were originally thought to be restricted to eastern Africa (Kreuze et al. 2000; Tairo et al. 2005) have been confirmed elsewhere in the world (Clark et al. 2012; Kreuze et al. 2020; Kwak et al. 2018; Maina et al. 2018; Qin et al. 2013b, c). Because sweetpotato was introduced to eastern Africa from Latin America only approximately 400 years ago (Zhang et al. 1999, 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. 2010b). 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 to 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 or geographic range and spread of plant viruses (Borer et al. 2010; Donnelly and Gilligan 2022; Mauck et al. 2012; Power 2008; Power et al. 2011; Shates et al. 2019). Moreover, SPMMV is characterized by numerous biological contradictions 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 sweetpotato 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, 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 or prevalence under field conditions is not determined. Until tomato mild mottle virus (ToMMoV, previously called eggplant mild leaf mottle virus) was characterized (Dombrovsky et al. 2012), SPMMV was the only known ipomovirus encoding helper component protease (HCPro), a multifunctional protein encoded by viruses of genus *Potyvirus* (Colinet et al. 1996, 1998; Valli et al. 2018).

The aim of this review is to provide a sharp focus and analysis of current progress and research gaps surrounding SPMMV, with the goal of stimulating further scholarship and rethinking research investments for virus disease management in sweetpotato. We highlight the reemergence potential of SPMMV by drawing comparisons between three taxonomically and phylogeographically related viruses: SPMMV and the two cassava brown streak disease (CBSD)-causing ipomoviruses; namely, cassava brown streak virus (CBSV) and Ugandan cassava brown streak virus (UCBSV), collectively referred to as cassava brown streak ipomoviruses (CBSIs). All three viruses are pathogens of vegetatively propagated crops commonly grown in eastern Africa. CBSIs are the causative agents of CBSD; first reported in the 1930s in eastern Africa, the viruses remained of little economic significance until their reemergence approximately half a century later in the 1990s to mid-2000s (Alicai et al. 2007; Hillocks and Jennings 2003; Monger et al. 2001a, b; Storey 1936). To date, CBSD continues to severely constrain cassava farming systems across eastern, central, and southern Africa. In less than a decade after reemergence of CBSIs, CBSD caused huge economic losses of more than U.S. $100 million per year (Mero et al. 2021; Pennisi 2010; Rey and Vanderschuren 2017). Currently, CBSD is estimated to cause annual losses in excess of U.S. $750 million to the affected communities (Hillocks and Maruthi 2015; Mero et al. 2021). From the early 1930s to early 1990s, the CBSIs just as SPMMV remained restricted to coastal eastern Africa but, thereafter, rapidly spread to mid- and high-altitude areas in Uganda, Burundi, and Zambia (Alicai et al. 2007; Mulenga et al. 2018; Patil et al. 2015; Tomlinson et al. 2018).

Evolutionary analyses show that intraspecific recombination, positive selection, and ongoing molecular adaptation account for the emergence of CBSIs in eastern Africa (Alicai et al. 2016; Mbanzibwa et al. 2011b; Ndunguru et al. 2015; Tomlinson et al. 2018). Similarly, recombination and positive selective events were detected in the SPMMV genome, implying that the virus is undergoing fast evolutionary adaptation under natural conditions in eastern Africa (Tugume et al. 2010b). 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 (*I. batatas* Lam) is grown in all tropical climates and is globally ranked among the 10 most important food crops (Low et al. 2017, 2020). The diversity and significance of this crop in food, nutrition, and income security in eastern Africa has been reviewed (Gichuki et al. 2003; Loebenstein 2009; Low et al. 2017, 2020; Tumwegamire et al. 2011; Woolfe 1992; Yada et al. 2010). However, the importance of sweetpotato is challenged by numer-

ous diseases, especially viral diseases that hampers productivity (Clark et al. 2012; Gibson and Kreuze 2015; Kreuze et al. 2021, 2022; Loebenstein 2015). The need to control virus diseases in sweetpotato is recognized as the most urgent activity for increasing productivity in developing countries (Fuglie 2007; Hambly et al. 2022; Kleinwechter 2012; Pemsl 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 the generation of 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 sweetpotato within eastern Africa was during 1939 in Ituri Province of the Democratic Republic of the Congo (which borders 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 (Colinet et al. 1996; Hollings et al. 1976; Schaefers and Terry 1976). Because sweetpotato was a neglected and less important crop in eastern Africa at the time (Bermejo and León 1994; Qaim 1999; Woolfe 1992), 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 a cassava mosaic disease epidemic (Martin 1928; Otim-Nape et al. 1996; Thresh et al. 1997; Zhou et al. 1997), which destroyed cassava crops and redirected more interest to the use of sweetpotato as an alternative crop for subsistence food security (Karyeija et al. 1998; Minde et al. 1997; Scott et al. 1997). Since then, several viruses and viral diseases of sweetpotato have been reported from many parts of the world, including eastern Africa (Clark et al. 2012; Tairo et al. 2005). Currently, at least 17 of approximately 30 viruses that have been reported on the crop globally have been found infecting sweetpotato in eastern Africa (Clark et al. 2012; Kreuze et al. 2021). All 17 viruses currently detected in sweetpotato in eastern Africa belong to six families: *Potyviridae*, *Closteroviridae Geminiviridae*, *Betaflexiviridae*, *Caulimoviridae*, and *Bromoviridae* (Aritua et al. 2007; Clark et al. 2012; Gibson and Aritua 2002; Mbanzibwa et al. 2014; Miano et al. 2006; Mukasa et al. 2003a; Mwaipopo et al. 2021; Tairo et al. 2004, 2005; Wasswa et al. 2011).

Most viruses currently known to infect sweetpotato in eastern Africa have positive-sense single-stranded RNA ((+)ssRNA) genomes (Clark et al. 2012; Kreuze et al. 2020). Exceptions are viruses in family *Caulimoviridae* (double-strand DNA 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 semipersistently by whiteflies. Sweepoviruses have been isolated from sweetpotato plants in different parts of the world, including eastern Africa (Mbanzibwa et al. 2014; Miano et al. 2006; Wasswa et al. 2011), and their impact on sweetpotato root yield is now apparent (Clark and Hoy 2006; Ling et al. 2010; Wanjala et al. 2020). All viruses currently known to infect sweetpotato (with the 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 sweetpotato (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 (Clark et al. 2012; Gibson and Kreuze 2015; Kreuze et al. 2021; Loebenstein 2012; Loebenstein et al. 2009), 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.

SPCSV. Most times, symptoms of SPCSV are persistent even when it infects plants alone. Therefore, SPCSV is often considered the most damaging virus affecting individual sweetpotato plants. Symptoms caused by SPCSV in eastern Africa vary with cultivar but generally include deep red to purple or yellow mottle of the lower and middle leaves, and stunting of sweetpotato plants (Gibson et al. 1998; Mukasa et al. 2006). Two serologically distinguishable strains of SPCSV first isolated from West Africa ($SPCSV_{WA}$) and East Africa ($SPCSV_{EA}$) are known in Africa (Gibson et al. 1998; Schaefers and Terry 1976). SPCSV has flexuous, filamentous particles ranging from 850 to 950 nm in length and a (+)ssRNA genome which consists of two genomic RNAs (RNA1 and RNA2), with complete nucleotide sequences of approximately 9.4 and 8.2 kb, respectively (Kreuze et al. 2002). RNA1 contains overlapping open reading frames (ORFs) that encode replication with five functional domains. RNA2 consists of seven ORFs, including the coat protein (CP) and heat shock protein homologue domains. There are also several subgenomic RNAs that have been detected in SPCSV-infected plants. SPCSV is phloem limited, and transmitted in a semipersistent manner by the whitefly species *Bemisia tabaci*, *B. afer*, and *Trialeurodes abutilonea* (Loebenstein 2015). Host range of SPCSV is apparently restricted to species in the family Convolvulaceae, with 12 wild species reported as natural hosts (Tugume et al. 2013).

SPFMV. SPFMV is the most widespread and characterized virus of sweetpotato, found wherever the crop is grown (Clark et al. 2012; Moyer and Salazar 1989). The virus is highly variable, with three major strains identified: EA (East Africa), RC (russet crack), and O (ordinary) (Campbell et al. 1974; Karyeija et al. 2000b; Tairo et al. 2005). A previous strain C of SPFMV was deemed as a standalone independent virus species named sweet potato virus C (SPVC) (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, (+)ssRNA genome which is approximately 10.8 to 11 kb (Moyer and Cali 1985; Yamasaki et al. 2010). The genome contains a 5′ nontranslatable region (NTR), one ORF for a single polyprotein giving rise to 10 mature proteins, a 3′ NTR, and a poly(A) tail (Moyer and 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), and faint to distinct chlorotic spots, with or without purple pigmented borders. Both spots and feathering may have purplish margins (Moyer and Salazar 1989). SPFMV is transmitted in a nonpersistent manner by aphids, including *Myzus persicae* and *Aphis gossypi*, *A. craccivora*, and *Lipaphis erysimi*, the first being the principal vector (Schaefers and 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 perpetu-

ated through the 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. 2010a; Wokorach et al. 2020).

SPMMV. SPMMV is the third most prevalent virus infecting sweetpotato 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 and Salazar 1989). SPMMV is the type member of genus *Ipomovirus* in family *Potyviridae*, and has flexuous, rod-shaped virions 800 to 950 nm in length, and a (+)ssRNA genome which consists of approximately 9 to 10.8 kb and encodes nine functional domains (Colinet et al. 1996, 1998). Although originally reported as a whitefly-transmitted virus (Hollings et al. 1976), the vector 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 and Smit 1994). The host range of SPMMV has been reported to be the largest compared with other sweetpotato-infecting virus, including over 20 plant species in at least 14 families (Brunt et al. 1996; Moyer and Salazar 1989; Tugume et al. 2010b). 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 (McLeish et al. 2019; Power and Flecker 2003) and the wide host ranges of plant viruses are 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 a high rate of sweetpotato reversion from infection by the virus compared with 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 in coinfections with SPFMV and SPCSV, interactions with either or both viruses and vector relations are less characterized (Mukasa et al. (2006). Beyond the clear understanding of 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 (Clark et al. 2012; Gibson et al. 1997). However, disease complexes arising from multiple infections cause the most destructive effects (Clark et al. 2012). Consequently, coinfections of SPFMV and SPCSV result in up to 1,000-fold increase in the titers of SPFMV because the antiviral defense in sweetpotato is suppressed by the RNase III protein of SPCSV (Cuellar et al. 2008, 2009; Karyeija et al. 2000a; Kreuze et al. 2005; Mukasa et al. 2006; L. Wang et al. 2021). The many-fold increase in SPFMV titers in plants results in sweet potato virus disease (SPVD), the most devastating disease of the crop in eastern Africa (Cuellar et al. 2008; Gibson et al. 1998; Karyeija et al. 2000a; Untiveros et al. 2007). 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 were 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, and Malawi (Sheffield 1957). The sweet potato chlorotic dwarf disease (CD) complex (syn. sweet potato severe mosaic disease), 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 that many other different viruses of sweetpotato can be synergized by SPCSV (Cuellar et al. 2011b, 2015; Mukasa et al. 2006; Untiveros et al. 2007). Therefore, the division of SPCSV-induced synergisms into SPVD and CD may 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 to 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 (i) year-round abundance of insect vectors transmitting the viruses; (ii) high susceptibility and lack of resistance in sweetpotato cultivars to the disease complexes, although resistance is apparent in single-virus infections; (iii) lack of an established formal sweetpotato seed system to ensure clean planting material; (iv) the perennial nature of vegetatively propagated planting material that continuously accumulates virus infections, and overlapping planting seasons (Fig. 1); (v) presence of evergreen alternative wild hosts in close proximity to sweetpotato fields (Fig. 1); and (vi) high frequency of mixed virus infections (Bashaasha et al. 1995; Clark et al. 2012; Donnelly and Gilligan 2022; Echodu et al. 2019; Gibson 2009; Low et al. 2020; Namanda et al. 2011; Ngailo et al. 2016; Tugume et al. 2008, 2010a, b, 2013, 2016a, b). These characteristics not only present suitable conditions for a perpetual virus disease burden but also are a perfect recipe for the emergence of viruses (Alexander et al. 2014; Canto et al. 2009; Clark et al. 2012; Fargette et al. 2006; French and Holmes 2020; Jones 2009; Tugume et al. 2010a, b, 2016a, b).

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 tobacco or sweetpotato whitefly, was formerly considered to be a single species with a worldwide distribution in tropical and semitropical regions. However, molecular studies and reciprocal crossing experiments conducted in the past decade have revealed that *B. tabaci* is a cryptic species complex comprising more than 40 morphologically indistinguishable species (Barbosa et al. 2014; Boykin and De Barro 2014; Boykin et al. 2007; Colvin et al. 2004; De Barro et al. 2011; Dinsdale et al. 2010; Liu et al. 2007; Maruthi et al. 2001; Tay et al. 2017; Vyskočilová et al. 2018; Xu et al. 2010). In sub-Saharan Africa, species of *B. tabaci* are a serious pest on many crops, including sweetpotato, cassava, tomato, cotton, and bean (Berry et al. 2004; Boykin et al. 2007; Ghosh et al. 2015; Mugerwa et al. 2018; Sseruwagi et al. 2005). 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. 2001) 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. Therefore, their genetic relationships to present-day species cannot be established.

Aphids are another group of insects transmitting viruses in sweetpotato, of which two species (*M. persicae* and *Aphis gossypii*) have been implicated in nonpersistent 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

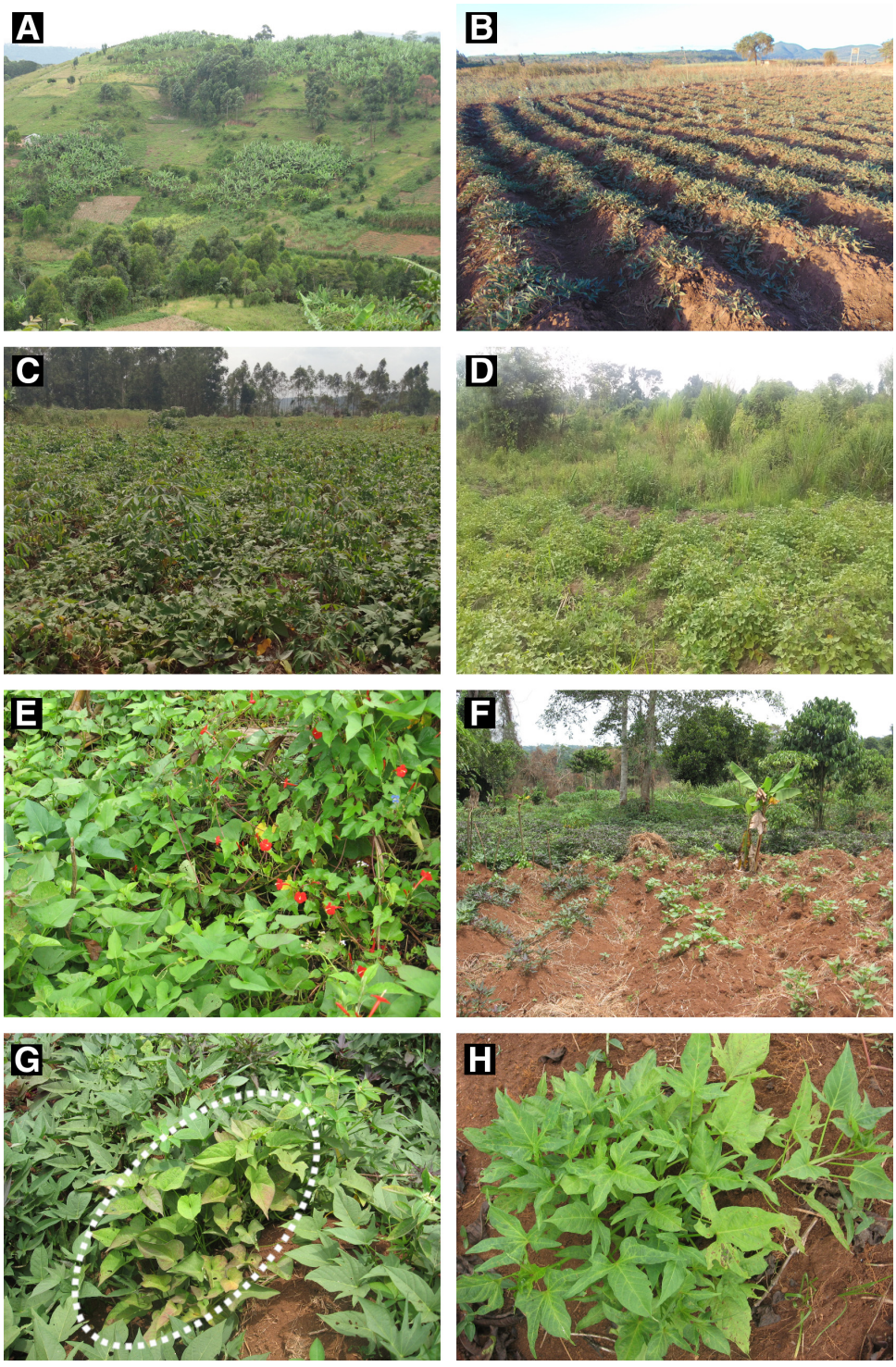

Fig. 1. Some of the characteristic features of sweetpotato cropping systems in eastern Africa that promote the perpetuation and persistence of viruses and their vectors on the crop as well as ease of vector-mediated transmission. **A,** Sweetpotato is one of the many crops in a locality with a mixture of crop husbandry and agroforestry 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,** Mixed-crop stand of sweetpotato and cassava in Mukono, central Uganda, which allows continuity of vectors and facilitating repeated transmission. **D,** 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,** Sweetpotato garden with some vines at the hedge growing intertwined with plants of *Ipomoea hederifolia* (with red-petalled flowers), a natural reservoir of sweet potato mild mottle virus, sweet potato feathery mottle virus, and sweet potato chlorotic stunt virus 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 sweet potato virus disease (dotted circle), most likely from a vine planted with the infection and surrounded by healthy-looking 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.

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 ecoevolutionary processes driving virus emergence (Chisholm et al. 2019; Elena et al. 2014; Fereres and Moreno 2009; Lefeuvre et al. 2019; Rojas and Gilbertson 2008). Indeed, the incidence of SPMMV, ranging between 9.0 and 25.0% of sweetpotato samples tested in different countries of eastern Africa (Ateka et al. 2004b; Mukasa et al. 2003a; Njeru et al. 2008; Tairo et al. 2004; 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 semipersistent noncirculative manner (Cohen et al. 1992; Gamarra et al. 2010; Sim et al. 2000). Epidemiologically, it is reasonable that viruses infecting a common host and transmitted by the same vector should exhibit a high frequency of coinfection (Allen et al. 2019; Moreno and López-Moya 2020; Seabloom et al. 2015). For this reason, frequent association or coinfection is expected between SPCSV and SPMMV under natural conditions. Such correlation has been observed between the aphid-transmitted viruses SPFMV and CMV in sweetpotato (Opiyo et al. 2010), SPFMV and the possibly aphid-transmissible sweet potato chlorotic fleck virus (SPCFV) (Aritua et al. 2009; Ateka et al. 2004a), and zucchini yellow mosaic virus (ZYMV) and watermelon mosaic virus in cucurbits (Lecoq and Desbiez 2012; Salvaudon et al. 2013). However, many studies in eastern Africa have consistently shown lack of significant association between SPCSV and SPMMV (Aritua et al. 2007; Ateka et al. 2004b; Mukasa et al. 2003a; Njeru et al. 2008; Opiyo et al. 2010; Tairo et al. 2004). Moreover, studies carried out after those of Hollings et al. (1976) have failed to confirm whitefly transmissibility of SPMMV (Misango 2011; Tairo et al. 2005), 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 sweetpotato in Kenya (Ateka et al. 2004b) and up to threefold more likely than dual infections of SPMMV and SPCSV in sweetpotato and wild plants in Uganda (Mukasa et al. 2003a). These observations are interesting because the common cooccurrence of SPMMV and SPFMV cannot be due to the latter's suppression of the natural resistance to the former in sweetpotato, because these 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 coinfected 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 (Govier and Kassanis 1974a, b; Kassanis 1961; Kassanis and Govier 1971a, b). Moreover, reciprocal assistance mechanisms mediated simultaneous aphid transmission of two aphid-nontransmissible strains of zucchini yellow mosaic potyvirus (Desbiez et al. 1999).

In viruses of genus *Potyvirus*, the CP and HC-Pro proteins function together in aphid transmission (Ng and Falk 2006; Pirone and Blanc 1996; Valli et al. 2018). The HCPro of SPMMV contains the highly conserved motif PTK (Tugume et al. 2010b) that is critical in bridging the virion to the aphid stylet during transmission of potyviruses (Blanc et al. 1998). The KITC, KLSC, or RITC motifs of potyviruses critical for retention of potyviral virions in aphid stylets (Blanc et al. 1998) are also present in the HCPro protein of SPMMV but in a mutated form as KTCC, KACC, or RTCC (Tugume et al. 2010b). In contrast, the CP N terminus in SPMMV lacks the DAG motif that is essential for aphid transmission of potyviruses (Atreya et al. 1990, 1992; Atreya et al. 1991), although the DAG motif is present in ipomovirus CBSV (Ateka et al. 2017) that is whitefly transmissible (Maruthi et al. 2005, 2017). SPMMV was historically the only known HCPro-encoding ipomovirus. until this protein was also found encoded by the genome of a newly characterized ToMMoV and its distant strain, designated as ToMMoV-IL (Abraham et al. 2012; Dombrovsky et al. 2012, 2013, 2014). The presence of a highly conserved PTK motif in SPMMV and ToMMoV HCPro may be biologically meaningless due to lack of the DAG motif in these viruses (Dombrovsky et al. 2014). Similarly, the presence of the DAG motif at amino acid positions 52 to 54 from the N terminus of the CP of ipomoviruses CBSV, squash vein yellowing virus (SqVYV), and coccinia mottle virus (CocMoV) (Ateka et al. 2017) may be nonsense in the absence of the HCPro within the same genome to facilitate aphid transmission although and both CBSV and UCBSV were recently detected in aphids collected from CBSD-infected cassava plants in Uganda (Nanyiti et al. 2023). By analogy, whitefly transmission of an ipomovirus, cucumber vein yellowing virus (CVYV), was abolished in CVYV mutants lacking amino acids in the N-terminal region of the CP at position 93 to 105 (Lindenau et al. 2021), yet a homologous CP amino acid sequence is also present in the SPMMV CP, whose whitefly transmission remains enigmatic. Therefore, although the presence of a DAG motif does not guarantee aphid transmissibility in potyviruses (Flasinski and Cassidy 1998; Johansen et al. 1996; López-Moya et al. 1999), possibilities to employ a DAG-containing CP from a coinfecting heterologous or homologous coinfecting viruses in cotransmission 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 and Gray 2017; Mondal et al. 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 and Gray 2017; Mondal et al. 2017, 2021). In the case of sweetpotato, this hypothesis can be tested by sequential inoculation access events between plants infected by single SPFMV (or its encoded protein), SPMMV, dual SPFMV + SPMMV, or triple SPFMV + SPMMV + SPCSV. Indeed, in potyviruses, the HCPro of one virus may allow the transmission of unrelated potyviruses when the HCPro is provided before or concomitantly with 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 papaya ringspot virus (PRSV) (Bourdin and Lecoq 1991). This occurred through heteroencapsidation, in which the RNA of ZYMV-NAT is completely or partially encapsidated by the functional PRSV CP. Moreover, an

aphid-nontransmissible 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 with the virus' HCPro, which harbors some or mutated versions of (and probably ineffective forms of) aphid transmissibility motifs (Tugume et al. 2010b), 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 coinfections 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 (Aguilar et al. 2015; Atabekov and Taliansky 1990; Close 1964; Falk et al. 1995; Froissart et al. 2002; Latham and Wilson 2008; Malyshenko et al. 1989; Smith 1945; Zhang et al. 2019). Synergisms may be unidirectional, in which the two viruses are often referred to as the "helper" and the "dependent" virus; or mutualistic, implying that proteins from one virus can enhance infection by another (Latham and Wilson 2008; Malyshenko et al. 1989; Syller 2012; Syller and Grupa 2016). A recent study in wheat-infecting viruses revealed that interactions among different unrelated viruses in a coinfected plant host can be more complex, and that synergistic interactions do not always necessarily cause increase in virus titers (Tatineni et al. 2022). The hypothesis that "vector-mediated transmission of SPMMV is modulated by synergism with SPCSV" is hinged on data showing that concentrations of SPMMV are up to 600-fold higher in dual infections with SPCSV than in single infections (Mukasa et al. 2006; Untiveros et al. 2007). For example, earlier observations in bean and potato singly infected with bean common mosaic virus and PVY, respectively, showed that plants containing higher virus titers were better sources of the virus for aphid transmission (Bagnall and Bradley 1958; Zettler 1969). Virus transmission rates are expressed as the percentage of plants that become infected following inoculation of viral particles by vectors that have fed previously on infected plants (Maruthi et al. 2005, 2017). Therefore, increased titers of one or both viruses in dual infections may result in increased chances of vector transmission, which is positively correlated with virus accumulation (Froissart et al. 2010).

The effect of virus accumulation on transmission was demonstrated for aphid-transmitted viruses, regardless of the transmission mode (nonpersistent or persistent), as previously demonstrated in several studies (Barker and Woodford 1992; De Bokx et al. 1978; Gray et al. 1991; Pereira et al. 1989). Also, the transmission efficiency of the criniviruses tomato chlorosis virus and tomato infectious chlorosis virus by whiteflies corresponded to virus concentration in the host in both single and double infections (Wintermantel et al. 2008). Moreover, possibilities of increased infection rate by mite-mediated transmissions from plants doubly infected by wheat streak mosaic virus (genus *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 coinfections is not dependent on the mode of transmission of the individual viruses, or how related the coinfecting viruses are. For SPMMV, possibilities of whitefly transmission or opportunistic transmission by aphids (hypothesis 1) are both plausible because SPMMV titers also increase significantly 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 also may 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 and 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 noninfected plants. Furthermore, both aphid species preferentially settled on doubly infected plants.

It is probable that the visual or olfactory stimuli emitted by mixed-infected plants were more attractive to aphids than were the stimuli emitted by singly or noninfected plants (Srinivasan and Alvarez 2007). Plant-mediated interactions between PVY or PLRV and aphid vectors may have significant and far-reaching implications for disease epidemiology, because the two viruses often occur in mixed infections (Chatzivassiliou et al. 2008; Srinivasan and Alvarez 2007). 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, regardless of the vectors, because 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 coinfect plants with SPMMV are transmitted by different vectors.

Hypothesis 3: SPMMV tropism and histolocalization changes upon coinfection with SPCSV. The specificity of a plant virus for a particular host tissues (tropism) results from a successful tripartite virus–host–vector interaction, which determines the fate of viral infection (Mauck and Chesnais 2020; Naidu et al. 2015). 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 (Guillemin et al. 2004; Spence 2001). 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 nonpersistent manner during short intracellular punctures by vectors in the epidermal cells (Brault et al. 2010; Esau 1960; Jiménez et al. 2018, 2021a; Nault 1997). Therefore, virus transmission by vectors in this manner can be optimized with short probing events that are restricted to the dermal tissues (Dietzgen et al. 2016; Jiménez et al. 2018).

In contrast, most semipersistent and persistently transmitted viruses are phloem limited, where they may also replicate and circulate (Dietzgen et al. 2016; Jiménez et al. 2018; Ng and Zhou 2015), indicating that only vector stylet penetrations into phloem tissues can optimize transmission of these viruses (Jiménez et al. 2018, 2021b; Kappagantu et al. 2020; Prado and Tjallingii 1994). Thus, phloem-limited viruses often require longer periods of vector feeding and longer persistence of the virus particles in the vector for successful transmission (Jiménez et al. 2018, 2021a, b; Kappagantu et al. 2020; Prado and Tjallingii 1994). 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 (Dunham et al. 2014; Jridi et al. 2006; Tamukong et al. 2020). The prevailing climatic conditions also influence this population structure at the individual plant level, also influencing virus transmissibility and other plant–vector–pathogen relations (Cunniffe et al. 2021; Trębicki et al. 2017). This is especially so for systemic plant virus infections, where the host plants such as sweetpotato are 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 infested sweetpotato sap onto carborundum-dusted leaves of *Nicotiana tabaccum*, *N. rustica*, and *N. benthamiana* (Tugume et al. 2010b). SPMMV is also easily graft transmissible in sweetpotato and the indicator host, *I. setosa* (Brunt et al. 1996; Ssamula et al. 2019; Tugume et al. 2010b). The ease of sap or 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 with SPCSV, which 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 enzyme-linked immunosorbent assay and RT-PCR (Clark et al. 2012; Mukasa et al. 2006). However, both SPFMV and SPMMV become easy to detect 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 (Cuellar 2008; Cuellar et al. 2008, 2009; Mukasa et al. 2006; Untiveros et al. 2007; Y. Wang et al. 2021). 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 (Alves-Júnior et al. 2009; Moreno and López-Moya 2020). In various host plants, the infecting begomoviruses, all of which are whitefly transmitted, are reported to be phloem-limited (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 (Levy and Czosnek 2003; Sudarshana et al. 1998; Wege et al. 2001). Both single and mixed infections of tomato yellow leaf curl virus and tomato yellow leaf curl Sardinia virus 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 rather than in shoots (Harper et al. 2014). Pea enation virus 2 complements PLRV mechanical transmission and facilitates its systemic infection (cell-to-cell movement and inside-the-phloem movement) (Ryabov et al. 2001).

In sweetpotato, most cultivars in eastern Africa are naturally resistant to SPMMV, where single virus infection may not incite major symptoms, sometimes to undetectable levels (Clark et al. 2012). This resistance is probably comparable with the ipomovirus CBSV resistance observed in two elite South American cassava genotypes, DSC167 (highly resistant and immune) and DSC260 (which restricts the virus replication to stems and roots only) (Sheat et al. 2019). The resistance in these cassava lines is not a restriction of long-distance movement but is due to preventing virus unloading from the phloem into parenchyma cells for replication, thus restricting the CBSV to the phloem cells only (Sheat et al. 2021). Only a low CBSV signal was found in phloem tissue of DSC 167, indicating that there is no replication in this genotype, while the intense CBSV signals in the phloem of DSC 260 provided evidence for CBSV replication in companion cells. Neither of the two genotypes showed evidence of virus replication outside the phloem tissues, indicating that, in resistant cassava genotypes, CBSV is confined to the phloem tissues only, in which virus replication can still take place or is arrested (Sheat et al. 2021).

Relatedly, one hypothesis is that SPMMV in resistant sweetpotato clones is restricted within the phloem in a mechanism similar to CBSV in resistant cassava genotypes but that a coinfection with SPCSV breaks this restriction, allowing widespread and enhanced replication in other nonvascular tissues. For example, the begomovirus abutilon mosaic virus (AbMV) is phloem limited in single infections; however, coinfection with CMV changes tropism of AbMV to be no longer phloem limited (Wege and Siegmund 2007). Infections of tomato yellow spot virus (ToYSV) in *N. 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 toward mesophyll tissues. These examples are of DNA viruses; however, in a coinfection of SPFMV and SPCSV, both of which are RNA viruses, SPCSV enhances the multiplication and increases titer of SPFMV in non-phloem tissues and abundance in leaves, causing severe SPVD symptoms (Karyeija et al. 2000a). Whether enhanced SPMMV titers and ease of detection in the sweetpotato coinfected with SPCSV is coupled with 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 has not been studied. Earlier experiments showed that, although infecting viruses may be detected in all tissues of sweetpotato and 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 and 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 assessments of any of the studied viral distributions across the different organs were made.

The new sweetpotato crop is normally initiated by planting vines of 15 to 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 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, after 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 roots; 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 similar or different scenarios with respect to translocation between roots and shoots. In sweetpotato, 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 tubers infected with potato potexvirus X (PVX), potato Andean mottle comovirus, PVY (jointly with PVX), or PLRV 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 on 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 showed high genetic diversity in comparison with other sweetpotato viruses (Mukasa et al. 2003b; Tairo et al. 2005; Tugume et al. 2010b) and other members of genus *Ipomovirus* (Adams et al. 2005, 2011; Webster and 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 (Chare and Holmes 2004; da Silva et al. 2020; Forrester et al. 2012; Mauck et al. 2019; Mondal and Gray 2017; Power 2000).

It is reasonable that tuber-mediated transmission 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 for 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 shoot sprouts from 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 the storage roots of SPMMV-infected sweetpotato. As such, SPMMV-infected but symptomless sprouts arising from tuberous roots may occasionally get included in the farmer-selected planting materials together with the sweetpotato vines that have reverted from SPMMV. Therefore, in this context, it may be speculated that tubers and their sprouts generate diverse virus populations, providing new alleles to the SPMMV metapopulation, and the diversity can be constrained by host reversion and random subsampling when SPMMV is spreading in the field through vector transmission. Due to high nonsynonymous versus synonymous variability, diversifying selection was detected in the P1 protein of SPMMV whereas purifying selection was implicated for the HC-Pro-, P3-, 6K1- and CP-encoding regions (Tugume et al. 2010b). If SPMMV HC-Pro mediates vector transmission as hypothesized earlier under this review, the purifying selection in this protein would make sense. An extensive analysis of evolutionary signatures in SPMMV should be possible when additional genomic data are available. Nonetheless, the high genetic diversity observed in these isolates coupled with recombination and adaptive evolution (Mukasa et al. 2003b; Tugume et al. 2010b) and infectivity of a broad host range (Brunt et al. 1996; Hollings et al. 1976) may imply an ongoing fixation of better-fit genotypes in the SPMMV population.

East African sweetpotato genotypes most frequently revert from SPMMV infection. Absence of viral infection in plants that were previously infected (reversion) was reported in some eastern African sweetpotato varieties that had been infected with SPFMV (Gibson et al. 2014; Mwanga et al. 2013; Ssamula et al. 2019). Similar reports were made in cassava infected with UCBSV and CBSV (Mohammed et al. 2016). Furthermore, reversion from virus infection was observed on storage root sprouts infected singly with SPFMV, whereas those infected with SPCSV alone showed reversion, and none of the storage root sprouts infected by both viruses showed reversion (Adikini et al. 2019). In cassava, UCBSV-infected plants had a higher rate of reversion when compared with plants infected with CBSV (Mohammed et al. 2016), supporting another line of evidence of the devastating nature of CBSV (Alicai et al. 2016). Ssamula et al. (2019) provided the first evidence of sweetpotato reversion from SPMMV. Moreover, the highest reversion rates were observed from SPMMV and SPLCV, both from sweetpotato genotypes from east Africa and the United States, and this was postulated to explain the comparatively low field prevalence of SPMMV observed in east Africa (Aritua et al. 1999; Ateka et al. 2004b; Mukasa et al. 2003a; Njeru et al. 2008; Tairo et al. 2004) and SPLCV in the United States (Clark et al. 2012). Observations by Ssamula et al. (2019) showed that reversion from viral infection in sweetpotato was virus specific and also affected by environmental factors, as well as whether or not the virus was in single or mixed infections. Furthermore, there was host genotypic predisposition to reversion in sweetpotato cultivars from both east Africa and the United States. Higher rates of reversion from SPMMV are certainly beyond the host and environment alone because these are uniform variables with other viruses in the sweetpotato pathosystem of eastern Africa. It is likely that higher reversion rates from SPMMV have to do with unknown virus-specific variables, which require extended studies.

GENETIC VARIATION AND EVOLUTIONARY TRAJECTORY OF SPMMV

Genetic variability in isolates of SPMMV. Only 32 isolates of SPMMV have been sequence characterized to date (Tairo et al. 2005; Tugume et al. 2010b). Most of the genetic sequence information is available for the 3′ end of the virus' genome (except for 16 isolates also characterized for their 5′ genomic ends) compared with 3 complete (10,818, 10,832, and 10,864 nucleotides [nt]) and 13 nearly complete (7,679 to 7688 nt) sequences (Colinet et al. 1998; Mukasa et al. 2003b; Tairo et al. 2005; Tugume et al. 2010b). In total, 8 of the 13 nearly complete sequences of SPMMV are of isolates from wild plants belonging to five species (Tugume et al. 2010b). 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. 2010b). 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. 2010b). In the absence of enough complete genomes, the CP gene is used for demarcation of *Potyviridae* virus species (Shukla et al. 1994) and, as such, suggests the occurrence of a single species of SPMMV (Tugume et al. 2010b). Phylogenetic analysis of a limited number of isolates from wild plants formed a separate cluster, suggesting host-driven evolution of SPMMV isolates (Tugume et al. 2010b). However, this is difficult to ascertain with greater precision due to limited sequence information because phylogenetic 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 the ability to be transmitted by vectors to sweetpotato plants.

Selection pressure and recombination are some of the key drivers of plant virus diversity and evolution (Elena et al. 2014; García-Arenal et al. 2001, 2003; Pagán 2018; Stobbe and Roossinck 2016). Therefore, studies showed that the majority of amino acid sites in the characterized protein-encoding genomic regions of SPMMV were under strong purifying selection, although there were a few amino acid sites in which genes were found to be under adaptive evolution (Tugume et al. 2010b). Similarly, strong

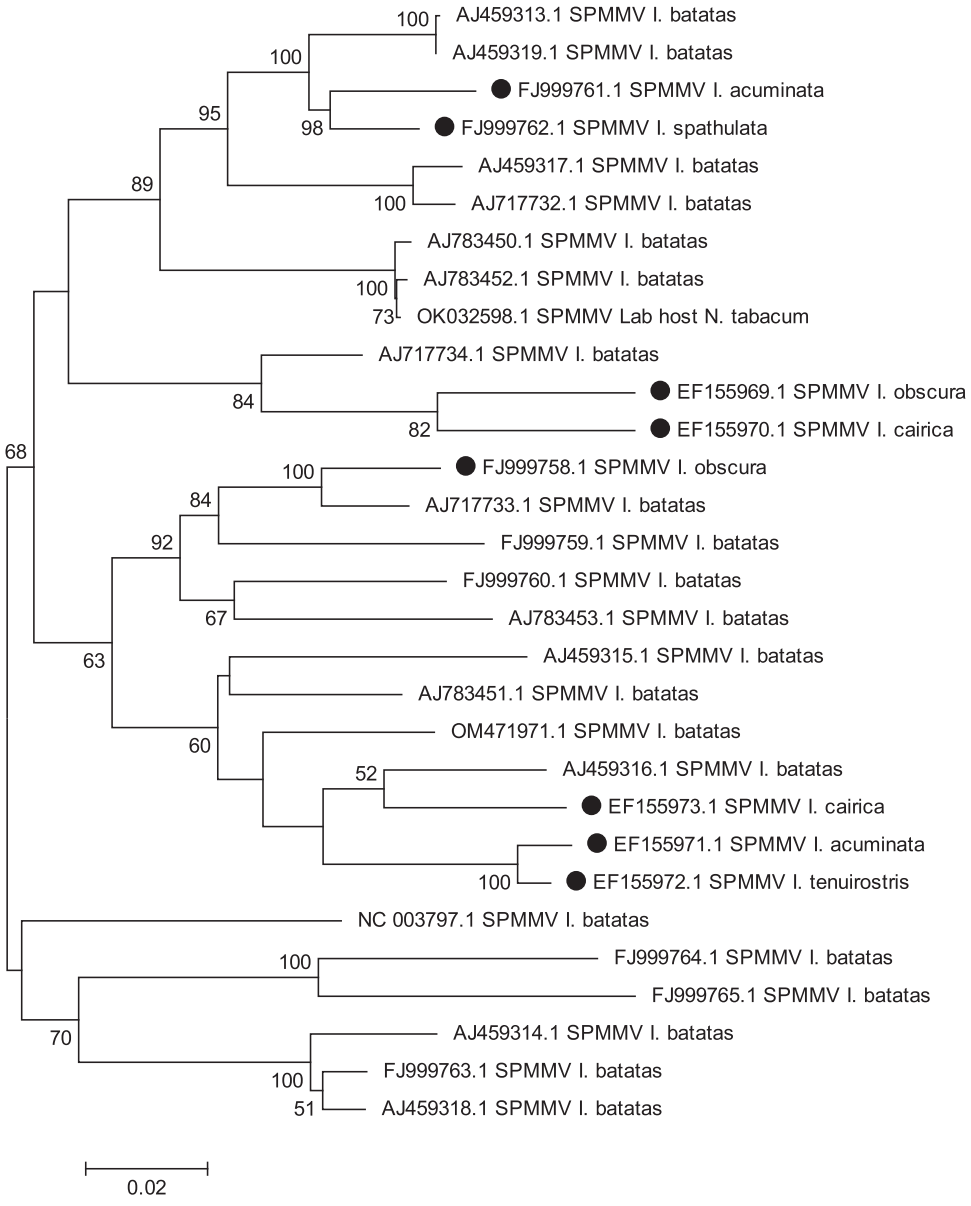

Fig. 2. Molecular phylogenetic tree generated using 30 nucleotide sequences (approximately 1,800 nucleotides) encoding the 3′ end of sweet potato mild mottle virus genome containing partial NIb, complete coat protein, and 3′ untranslated region. Only isolates with this genomic region sequenced were included in the analysis. 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 the symbol • preceding the sequence accession number). Phylogenetic clustering was inferred by using the maximum-likelihood method based on the Tamura-Nei model (Tamura and Nei 1993). Values shown at the branch nodes represent bootstrap values of 1,000 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; therefore, there were 1,775 positions in the final dataset. Phylogenetic analysis was conducted in MEGA7 (Kumar et al. 2016).

purifying selection was also reported in the CBSIs (Mbanzibwa et al. 2009b, 2011b) but a different study indicated that CBSV was evolving faster than UCBSV and had many sites under positive selection as compared with 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 with what would be expected of viruses with small genomes, which is the case with ipomoviruses and other viruses in the family *Potyviridae*.

Recombination events were detected in the 5′ proximal genomic end of 14 isolates (from wild plants and sweetpotato) and for the 3′ end of genomes of 29 isolates (Tugume et al. 2010b). The lack of evidence for major and minor parent-like sequences in that study was in favor of the argument that SPMMV originated in eastern Africa and has been evolving there (Tugume et al. 2010b). No SPMMV isolates have been detected or characterized outside this region. Recombination events have been detected in other ipomoviruses, the CBSIs with eastern Africa geographical restrictions (Mbanzibwa et al. 2011b). Analyzing complete genomes of CBSV and UCBSV revealed recombination events in the 3′ proximal region of CBSV and in P1-, CI-, VPg-, Nib-, HAM1h-, and CP-encoding regions and the 3′ untranslated region, respectively (Mbanzibwa et al. 2011b; Ndunguru et al. 2015). Therefore, recombination appears to be the main driving force for the evolutionary diversification of SPMMV and CBSIs. These three ipomoviruses, while embracing strong purifying selection, allow exchange of genetic material through homologous recombination as a means of maintaining genetic variability.

Genomic differences and similarities between SPMMV and other ipomoviruses. SPMMV and CBSIs are geographically and taxonomically related because they are both important ipomoviruses of economically important, vegetatively cultivated crops in eastern Africa. The typical genome of SPMMV is structurally similar to that of potyviruses and translates into a polyprotein that autocatalytically cleaves into 10 mature proteins (Adams et al. 2005; Colinet et al. 1998; Cui and Wang 2019; Valli et al. 2006, 2007). However, SPMMV differs from its cogners in the 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 (Casinga et al. 2021; Mulenga et al. 2018; Rey and Vanderschuren 2017). First, 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). Second, the size of SPMMV P1 (83 kDa) is nearly twice as large as P1s of CBSIs (42 kDa) (Adams et al. 2005; Colinet et al. 1998; Mbanzibwa et al. 2009a; Valli et al. 2007). Third, the CBSIs have encoded am Maf/HAM1-like sequence, which is recombined between the replicase and CP domains in the 3′ proximal part of their genomes. Only two other viruses, the potyvirus euphorbia ringspot virus (Knierim et al. 2017; Mbanzibwa et al. 2009a) and the torradovirus cassava torrado-like virus (Leiva et al. 2022), infecting euphorbiaceous plants have encoded this class of sequence in their genomes. Fourth, 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. 2010b). Different sites for cleavage of SPMMV CP from NIb have been proposed and it could be that there is more than one site because the sizes of CP reported differ. Whereas Hollings et al. (1976) reported the size of the SPMMV CP to be 37.7 kDa, Tugume et al. (2010b) demonstrated that SPMMV CP size was 35 kDa. 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 (Goh and Hahn 2021; Mbanzibwa et al. 2009a; Palani et al. 2021; Tomlinson et al. 2019a, b).

The P1 protein of CBSIs, tomato mottle mosaic virus (ToMMV), and SPMMV are not duplicated compared with those of other known ipomoviruses (Cui and Wang 2019; Valli et al. 2006, 2007). The much larger P1 protein of SPMMV compared with 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 CocMoV and to P1a and P1b of CVYV, SqVYV, and CocMoV (Table 1). However, CI (50.9 to 55%) and NIb (51.6 to 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 the other groups (Fig. 3). Amino acid similarities with SPMMV are highest with ToMMV (Table 1). The close sequence

TABLE 1
Percent nucleotide sequence identities of protein encoding sequences of sweet potato mild mottle virus (SPMMV) (accession NC_003797) with homologous proteins of other known ipomoviruses[a]

Protein	CBSV	UCBSV	ToMMV	SqVYV	CVYV	CocMoV
P1	20.6	21.1	17.8	–	–	–
	–	–	–	20.6[b]	22.5[b]	20.6[b]
	–	–	–	16.9[c]	15.7[c]	17.6[c]
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

[a] Comparisons were made using the ClustalW method implemented in MEGA7 (Kumar et al. 2016). CBSV = cassava brown streak virus and UCBSV = Ugandan cassava brown streak virus. Squash vein yellowing virus (SqVYV), cucumber vein yellowing virus (CVYV), and coccinia mottle virus (CocMoV) have duplicated P1 proteins in form of P1a and P1b instead of a single P1 protein. Other viruses, except SPMMV and tomato mottle mosaic virus (ToMMV), lack the helper component protease (HCPro). Dashes (–) indicate the absence of protein in a given virus genome.
[b] Percent identities of P1a nucleotide sequences of these viruses with the P1 of SPMMV.
[c] Percent identities of P1b nucleotide sequences of these viruses with the P1 of SPMMV.

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, 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 (Giner et al. 2010; Mbanzibwa et al. 2009a; 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 (Ateka et al. 2017; Colinet et al. 1998; Mukasa et al. 2003b; Tugume et al. 2010b) yet 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 the PTK motif in the SPMMV HCPro has not been evaluated for SPMMV trans-

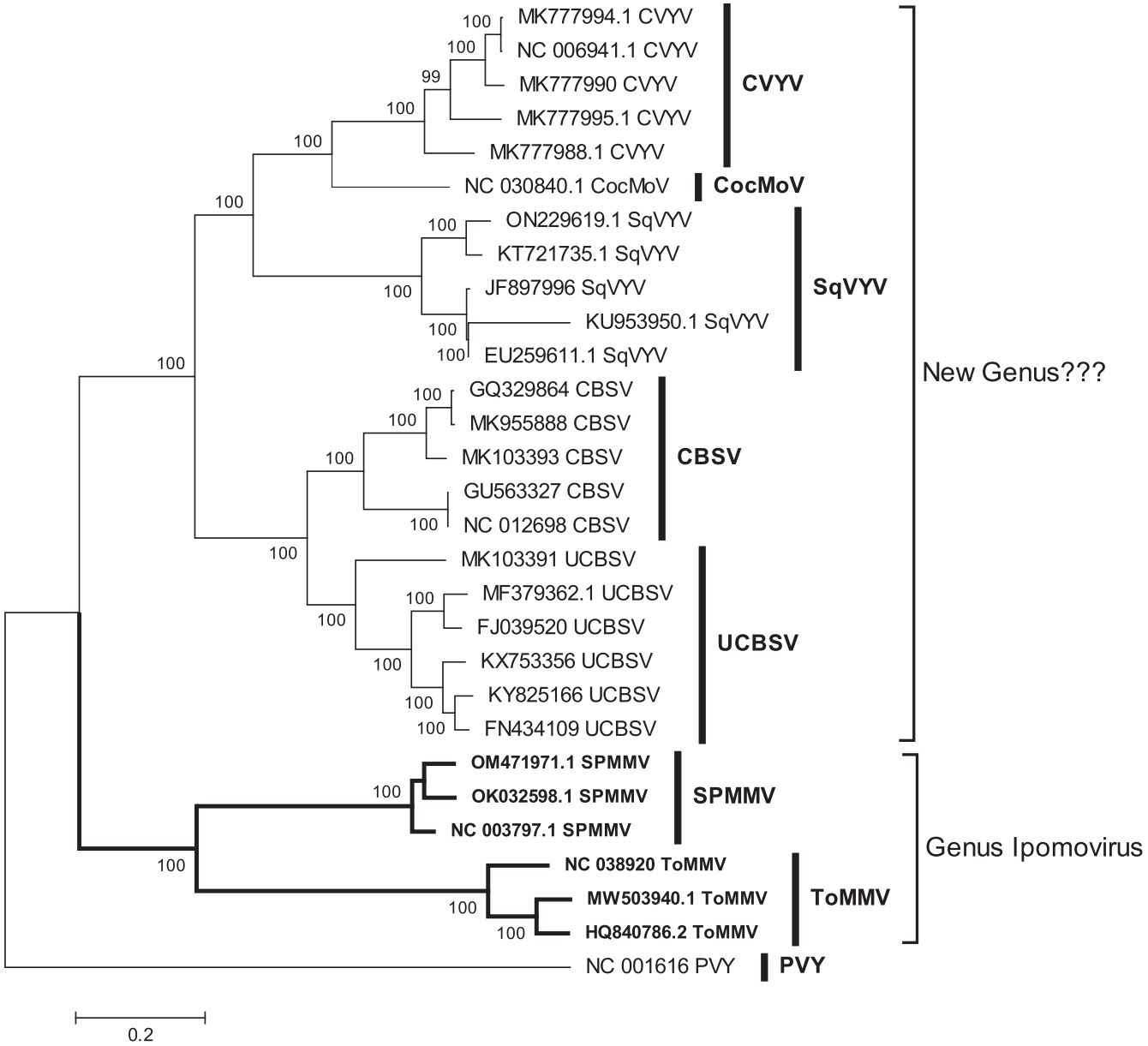

Fig. 3. Molecular phylogenetic tree generated using 29 representative complete genome sequences of ipomoviruses. Currently, the International Committee on Taxonomy of Viruses recognizes only seven virus species. In the analysis, each species was represented by at least one viral sequence: Ugandan cassava brown streak virus (UCBSV), cassava brown streak virus (CBSV), squash vein yellowing virus (SqVYV), coccinia mottle virus (CocMoV), cucumber vein yellowing virus (CVYV), sweet potato mild mottle virus (SPMMV), and tomato mottle mosaic virus (ToMMV). Accession numbers are shown for each sequence before the abbreviations of virus names. Potato virus Y (PVY; *Potyvirus*) was used as an outgroup. 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 and Nei 1993). Values shown at the branches represent bootstrap values of 1,000 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 7,554 positions in the final dataset. Phylogenetic analysis were conducted in MEGA7 (Kumar et al. 2016).

mission by aphids. Recently, Lindenau et al. (2021) demonstrated that the N-terminal region of CVYV CP (between amino acid positions 93 and 105) functions in whitefly transmission. This region shows conserved amino acid 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 noncanonical nucleoside triphosphates into DNA and RNA (Galperin et al. 2006; Mbanzibwa et al. 2009a), as determined previously (Noskov et al. 1996; Takayama et al. 2007). However, no experimental evidence that the CBSV *HAM1* gene protected CBSV's genome from mutations was found (Tomlinson et al. 2019a). These discrepancies in the presence 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 with that of CBSIs, the causative agents of 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 (Ano et al. 2021; Bigirimana et al. 2011; Casinga et al. 2021; Chipeta et al. 2016; Hillocks and Maruthi 2015; Koima et al. 2018; Mulimbi et al. 2012; Munganyinka et al. 2018; Rey and Vanderschuren 2017). The rapid westward and southward spread of CBSIs in Africa projects a heavy presence of CBSIs and CBSD by 2030 (Ano et al. 2021; Jarvis et al. 2012; Mero et al. 2021; Rey and Vanderschuren 2017).

Although we have 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 (Amisse et al. 2019; Mbanzibwa et al. 2011a, b). 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 for our understanding of the ecologies of these viruses and their potential of reemergence. For instance, during colonial rule, CBSD was eradicated from Uganda only to reemerge in the late 1990s (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 that CBSD incidence increases with increased whitefly populations (Shirima et al. 2020). This could also be due to occurrence of unknown alternate hosts of the virus and arrival of new cassava genotypes generated during the efforts to eradicate cassava mosaic disease (CMD). This argument is supported by the fact that the new cassava genotypes, with resistance to CMD, are susceptible to CBSD (Alicai et al. 2007; Beyene et al. 2016). Although, today, SPMMV is less of a problem, especially in single infections, in the future it can, through some circumstances, become a threat to sweetpotato production just like CBSV and UCBSV to cassava production in the region.

EMERGENCE OF SPMMV VIA "NEW ENCOUNTER SCENARIO" IN EASTERN AFRICA

Endemism versus exoticism of SPMMV in eastern Africa. The endemism of SPMMV in eastern Africa is a hypothesis arising from the lack of evidence of the virus outside the region, postulating that SPMMV invaded the newly introduced sweetpotato in eastern Africa via a "new encounter scenario" approximately 400 years ago (Clark et al. 2012; Lindenau et al. 2021; Tairo et al. 2005; Tugume et al. 2010b). New encounter scenarios describe situations in which plants (in this case, sweetpotato) are introduced into new areas, which allows them to come into contact with viruses with which they have not interacted before and to which they express no resistance (Jones 2009, 2020, 2021; Jones and Coutts 2015). It also refers to situations where plant viruses are transferred from their indigenous hosts to cultivated hosts or are transported to other areas as new disease agents. In this sense, plant viruses and their principle hosts should have common centers of origin, unless the viruses were derived from new encounter scenarios (Jones 2009, 2020, 2021; Jones and Coutts 2015; Lovisolo et al. 2003). Sweetpotato originated in tropical America (Austin 1975, 1988; Roullier et al. 2013) and dispersed around the world, mainly via human-mediated migration, toward the start of the 16th century (Lebot 2010). An exception is Australasia and the South Pacific, where there is evidence for prehistoric sweetpotato cultivation (Huang and Sun 2000; O'Brian 1972; Switek 2013; Yen 1963; Zhang et al. 2004). Indeed, evidence shows that the introduction of sweetpotato into Polynesia predates human colonization of the region by thousands of years, probably via long-distance mediated buoyant seed dispersal by ocean currents (Muñoz-Rodríguez et al. 2018). The crop was introduced to Africa by the Portuguese later in the 16th century (Zhang et al. 1999, 2004), probably first into eastern Africa in Tanzania, then from eastern to western Africa (Lebot 2010). Accordingly, west African sweetpotato germplasm was derived from east African sweetpotato and would imply codispersal with SPMMV from eastern Africa to western Africa. However, SPMMV is absent in western Africa (Clark et al. 2012; Gutierrez et al. 2012; Tibiri et al. 2020). In addition, the lower diversity of sweetpotato in east Africa than in west Africa suggests that west African sweetpotato is not simply a subsample from eastern Africa but might have been independently introduced into west Africa later on (Glato et al. 2017).

In contrast, the hypothesis of "exoticism of SPMMV in eastern Africa" is derived from the second definition of new encounter scenarios; that is, transport of the viruses in plants (in this case, sweetpotato) to distant areas as new disease agents there (Jones 2009, 2020). Under this hypothesis, SPMMV or its progenitors existed in the wild plants in tropical America prior to their domestication as sweetpotato in their center of origin and were later dispersed across the world, including introduction to eastern Africa. It is believed that, prior to plant domestication, plant viruses coevolved or coexisted with their natural wild host plants in the plants' centers of origin (Jones 2009, 2020, 2021; Lovisolo et al. 2003) but this coevolutionary "balance" was drastically altered following the domestication of wild plants and agricultural intensification (Diamond 2002; Jones 2009, 2020; Jones and Coutts 2015; Purugganan 2019). This disruption was further exacerbated by the gradual dispersal of crops away from their original centers of domestication to other regions

(Harlan 1965, 1971; Lovisolo et al. 2003), which created opportunities for new encounter scenarios between host plants and viruses (Jones 2009, 2020). However, absence of SPMMV outside eastern Africa even when globally most or all sweetpotato lines originate from one center 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 center of origin in tropical America (Lefeuvre et al. 2019; Stobbe et al. 2012). Wild relatives of sweetpotato from Uganda in eastern Africa are the only alternative hosts that have been analyzed for SPMMV infectivity, indicating close genetic identities with isolates from cultivated plants (Tugume et al. 2010b); 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 Africa in wild *Ipomoea* spp. or other closely related taxa (Agnew and Agnew 1994; Blundell 1992; Tugume et al. 2008; Verdcourt 1963) as primary hosts and invaded sweetpotato when that plant was introduced from tropical America. To date, there are only a few reports, mostly in Australia, of plant viruses still restricted to wild plants and natural ecosystems (Jones 2009, 2020; Jones and Coutts 2015; Vincent et al. 2014), which is in contrast with 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, 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. 2010a, 2013; Wokorach et al. 2020), although subsequent reports show the presence of this strain elsewhere. Similar observations are made for SPCSV, which has only two contrasting strains, of which the EA strain is the only strain prevalent in eastern Africa (Qin et al. 2013a, c; Tairo et al. 2005; Tugume et al. 2013) and only lately detected elsewhere. Isolates of the EA strain of SPCSV from wild plants and sweetpotato in 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; Qin et al. 2013c; Tugume et al. 2013; L. Wang et al. 2021), indicating the unique variability of isolates of the 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 were 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 strains are more common than others in eastern Africa. The geographical range of plant viruses and successful perpetuation within an agroecosystem is constrained more by virus–vector than by virus–host plant relations (Power 2000, 2008; Power and Flecker 2003, 2008) and this may influence dominance of one or more strains over the others. Therefore, the optimization of virus–host and virus–vector interactions may be an ongoing process in unique viral genotypes in eastern Africa, with an ongoing 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, in addition to sweetpotato. In addition to the CBSIs already mentioned above in cassava, the virulent recombinant strain of cassava mosaic begomoviruses (CMBs) exhibited a gradient of decreasing prevalence from east to south in Africa (Bull et al. 2006; Legg 1999; Ndunguru et al. 2005; Patil and Fauquet 2009). Rice yellow mottle virus (RYMV; genus *Sobemovirus*) showed phylogenetic congruence with geographical origin of isolates on an east-to-west transect across Africa and decreased nucleotide diversity westward across Africa (Fargette et al. 2004, 2006; Jones 2020; Ochola et al. 2015; Ramathani et al. 2021; Traore et al. 2005; Traoré et al. 2009). 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; Pinel-Galzi et al. 2015; Traore et al. 2005; Traoré et al. 2009; Trovão 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 (Burgess et al. 2007; Dimitrov et al. 2012; Küper et al. 2004; Myers et al. 2000; Skarbek 2008). The climate in these areas is modulated by the Indian Ocean, promoting variabilities of microclimatic gradients away from east Africa (Blau and Ha 2020; Marchant et al. 2007; Nicholson 2017) 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 contribute to crop disease pandemics (Kriticos et al. 2020). The southwest 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 that distinct species of these viruses are found in mainland eastern Africa (the Lake Victoria basin) and Indian Ocean coastal areas (Mbanzibwa 2011; Mbanzibwa et al. 2009b, 2011b; 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 the 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 21st century agriculture (Amari et al. 2021; Jones 2009, 2020, 2021). Because virus emergence is a fundamental result of climate change effects on both agroecosystems and natural ecosystems (Jeger 2020, 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, create a series of irregular microclimatic gradients away from the Indian Ocean coastal areas (Blau and Ha 2020; Marchant et al. 2007) that 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 such as 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 from the 1930s, when it was first reported in eastern Africa, until the 1990s to mid-2000s, when the disease "exploded" (Alicai et al. 2007; Hillocks and Jennings 2003; Monger et al. 2001a, b; Storey 1936). CBSD remains the single most destructive threat to cassava farming in Africa (Ano et al. 2021; Pennisi 2010). Whereas efforts to contain CBSD in eastern Africa were at first successful, the disease has already extended its borders and is fast spreading westward, with projections to affect western Africa by 2030 (Ano et al. 2021; Jarvis et al. 2012). Using this analogy, SPMMV was first reported on sweetpotato in eastern Africa in the 1970s (Hollings et al. 1976; Tairo et al. 2005; Tugume et al. 2010b). 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 1990s, 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 a virus disease complexes of sweetpotato (Mukasa et al. 2006; Tugume et al. 2010b). However, as demonstrated for CBSIs in eastern Africa, and numerous other viruses elsewhere (Jones 2020, 2021; Jones and Naidu 2019), 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. 2010b). Due this high likelihood SPMMV reemergence, it is critical that we build essential, basic scientific information on this virus in advance, which has remained vague or absent now for half a century. Lack of accurate information about a virus and the associated diseases limits effectiveness of measures used in disease management: hence, the following information is most needed for the effective management of any disease outbreaks associated with SPMMV.

First, vector transmission dynamics, including determining the actual vectors that transmit 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 "opportunistic transmission of SPMMV by aphids using SPFMV as a helper virus" may be tested through sequential aphid feeding (or inoculation-access) events between plants infected by single SPFMV (or its encoded protein), SPMMV, dual SPFMV + SPMMV, or triple SPFMV + SPMMV + SPCSV. The second hypothesis, "SPMMV transmission being modulated by synergism with SPCSV", may be tested by transmission experiments where source plants are singly or coinfected with SPCSV, regardless of the candidate vector. It is noteworthy that vector transmissibility is often lost or compromised during serial mechanical passages of plant viruses (Garcia et al. 2019; Gray and Banerjee 1999; Legavre et al. 1996). Therefore, it is essential that the transmission experiments use SPMMV isolates directly from nature or those that have not stayed for long outside the natural environment.

Second, the transmission dynamics of SPMMV need to be evaluated under field conditions in association with other sweetpotato viruses. Most surveys conducted in eastern Africa have provided only 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 closest to 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 symptom severity and reduced yield when there was coinfection of SPLCV with SPCSV and SPFMV; however, whether or not a coinfection of SPMMV with SPLCV results in reduced yields and enhanced disease symptoms is not known.

Third, histolocalization of SPMMV, and whether or not this localization is variable according to the multiplicity of infection, should be profiled. This may be combined with study of the feeding behavior of the vectors 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 growth habits and propagation means similar to those of sweetpotato, shows organ-based accumulation of PVY (Kogovšek et al. 2011). The exception is that sweetpotato plants 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 will require extended analysis at the individual host plant level prior to meaningful extrapolation on the 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 the complete SPMMV genomic space as well as comprehensive geographical coverage is urgently needed. This will be 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 (Clark et al. 2012; Fuglie 2007; Zhang et al. 2020). Therefore, all of 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.

ACKNOWLEDGMENTS

We thank our host institutions for giving us ample time to assemble this review, Richard Edema (Makerere University Regional Centre for Crop Improvement [MaRCCI]) for technical and financial support, Abubakar Sadik Mustafa (Department of Plant Sciences, Microbiology and Biotechnology, Makerere University) for

his assistance in the assembly of Figure 1, and the anonymous reviewers for their constructive comments that improved this review.

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