https://doi.org/10.17221/51/2025-PPS

The role of seed transmission in the spread of cereal viruses: Global challenges and prevalent threats in Ukraine

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Citation: Snihur H., Shevchenko T., Shevchenko O., Kyrychenko A. (2025): The role of seed transmission in the spread of cereal viruses: Global challenges and prevalent threats in Ukraine. Plant Protect. Sci., 61: 201–221.

Abstract: The transmission of plant viruses through seed plays a fundamental role in virus spread, persistence, and survival, particularly in economically important crops. Besides its considerable ecological significance, seed transmission influences plant and virus evolution. Virus contamination of the seed also has critical epidemiological implications, especially when combined with subsequent or additional insect vector spread. Plants grown from contaminated seeds serve as primary viral inoculum sources, facilitating the introduction of viruses into new regions and triggering disease outbreaks with substantial economic losses for growers. Changes in environmental conditions increasingly influence plant virus epidemiology by affecting vector populations, host susceptibility, and transmission dynamics, thus increasing virus transmission risks in cereal crops. This review explores the mechanisms of seed transmission and its consequences, with a focus on key cereal viruses in Ukraine: barley stripe mosaic virus, wheat streak mosaic virus, High Plains wheat mosaic virus, sugarcane mosaic virus, and maize dwarf mosaic virus. Hereby, the biological properties of these viruses, the risks posed by seed transmission, and the economic impact on crop production are discussed. Given the widespread distribution of these pathogens, presented data will also be valuable for other cereal-growing regions, particularly those bordering Ukraine and engaged in seed import/export. This review underscores the global need to manage seed-transmitted viruses to safeguard cereal crop productivity and food security. Future research should focus on developing resistant cultivars and advanced diagnostics to control their spread.

Keywords: seed-borne viruses; insect vectors; epidemiology; climate change; economic impact

Seed-transmitted viruses pose significant economic threats to cereal crops worldwide and hold particular importance for Ukraine, where grain production is a vital part of the agricultural economy and forms the basis for food security and the country's export potential. The ability of virus-

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Supported by the project No. PAN.BFB.S.BWZ.407.022.2024, being implemented through the Long-term program of support of the Ukrainian research teams at the Polish Academy of Sciences carried out in collaboration with the US National Academy of Sciences with the financial support of external partners.

es to persist in seed represents a crucial survival strategy as seeds serve as a unique link between cropping seasons, enabling the virus to survive between planting cycles. It is also pivotal for efficient virus spreading among plants. This role is especially important for viruses with a narrow host range or those transmitted by insect vectors with low mobility (*Frankliniella occidentalis* (western flower thrips) or in a non-persistent manner. For instance, the barley stripe mosaic virus (BSMV, species *Hordeivirus hordei)*, a major threat to *Triticum aestivum* (wheat) production, does not have any known insect vectors and hence primarily relies on seed transmission for its spread from year to year (Mink 1993; Jiang et al. 2021; Sandra & Mandal 2024). Moreover, some other non-cereal seed-transmitted viruses including cucumber mosaic virus (CMV, species *Cucumovirus* CMV), potato virus Y (PVY, *Potyvirus yutuberosi)*, and pea seed-borne mosaic virus (PSbMV, *Poytyvirus pisumsemenportati)* also heavily rely on seed transmission to establish new hotbeds of infection, as the primary method for their long-term survival (Gutiérrez-Sánchez et al. 2023; Escalante et al. 2024). These viruses, although also efficiently transmitted by vectors, can persist across seasons in contaminated seed stocks, possibly causing damage to future crops even in the absence of vectors.

In addition to ensuring the survival of viruses within the seed over long periods (years), seed transmission plays a pivotal role in the long-distance spread of viruses. This mechanism enables viruses to cross geographical boundaries and invade regions and distant continents. This is important for the epidemiology of plant viruses and the global transportation of agricultural products (Mink 1993; Albrechtsen 2006; Sastry 2013; Shanmugam et al. 2024). Beyond its epidemiological significance, virus contamination of the seed leads to deterioration in crop quality and yield losses, with far-reaching economic consequences. Viruses can affect germination rate, plant growth, and grain development, often resulting in reduced productivity and crop failure (Johansen et al. 1994; Sastry 2013; Aveling 2014). In some cases, seed-borne viruses are responsible for reducing seed viability and causing secondary infections that interfere with the development of healthy crops, thus threatening food security. To improve clarity, we provide a comparative summary (Table 1) highlighting the key differences between seed-borne and seed-transmitted viruses. A more detailed discussion of such viruses follows in the subsequent sections.

While early studies on plant viruses mainly focused on their general ability to persist in seed, subsequent research has concentrated on the mechanisms of seed transmission and the physiological and molecular factors influencing transmission efficiency (Mandahar 1981, 1985; Mink 1993; Maule & Wang 1996; Sastry 2013; Hamelin et al. 2016;

Table 1. Comparison of properties of seed-borne and seed-transmitted viruses

Main characteristic of the viruses	Viruses	
	Seed-borne	Seed-transmitted
Localisation	Present in certain parts of the seed (seed coat, rarely – endosperm, embryo) or reproductive tissues	Localised in embryo (typically) or other seed tissues
Infection of progeny	Not necessarily	Yes, by definition
Mechanism	Seedling contamination occurs through microlesions on the seed coat during harvest, storage, or processing; may persist in seed tissues without infecting the embryo or seedling	Invade plant reproductive tissues, enabling infection during germination; transmitted vertically to the next generation
Epidemiological significance	May serve as a source of inoculum for other transmission modes (e.g., insect vectors); limited direct impact on seedlings unless transmitted	Causes direct infection in seedlings, reducing yield and quality; significant for cereal crops due to early disease onset
Detection	May be detected by serological or molecular methods in seeds	Requires both seed testing and seedling infection assays
Examples	*Tritimovirus tritici* (wheat streak mosaic virus, WSMV), *Poacevirus tritici* (triticum mosaic virus, TriMV), *Machlomovirus zeae* (maize chlorotic mottle virus, MCMV), etc.	*Hordeivirus hordei* (barley stripe mosaic virus, BSMV), *Potyvirus sacchari* (sugarcane mosaic virus, SCMV), *Potyvirus zeananus* (maize dwarf mosaic virus, MDMV), etc.

Pagán 2022; Gutiérrez-Sánchez et al. 2023; Escalante et al. 2024).

Despite significant advances in understanding the virus-host interactions that underlie seed transmission, there remains much to be learned about the factors influencing transmission rate, the genetic variability within both virus populations and host plants, and the long-term ecological consequences of seed-transmitted viral infections (Maule & Wang 1996; Pagán 2022).

Given the global scope of the issue, understanding the dynamics of viruses transmitted through seeds in cereals is critically important for developing better strategies to control these pathogens. This review aims to contribute to the growing body of knowledge by examining the key seed-transmitted cereal viruses circulating in Ukraine as a major global seed producer, focusing on their global distribution, transmission mechanisms, and impacts on crop health and productivity.

WHY IS SEED-TRANSMITTED VIRUS INFECTION SO DANGEROUS?

More than 231 characterised viruses, representing approximately one-third of all known plant viruses, have been reported to be transmissible through seeds of various cultivated plants and weeds (Singh & Mathur 2004; Sastry 2013; Shanmugam et al. 2024). Advancements in detection methods, enabling more accurate identification, increased the number of known seed-transmitted viruses (Pagán 2022). For instance, begomoviruses, which were once thought not to be transmitted vertically through seed, are now recognised as capable of seed transmission. This has significantly altered our understanding of the epidemiological significance of begomovirus diseases and the potential role of seed transmission in the spread and emergence of the diseases they cause (Gomathi Devi et al. 2023; Sandra & Mandal 2024).

Seed-transmitted viruses present a substantial threat to agribusinesses and seed companies because of their ability to initiate infections and spread silently, often going undetected at early stages. This silent transmission poses a considerable risk to the yields of many economically important crops, as infections may not be evident until later in the growing season, making early detection and management more difficult (Escalante et al. 2024).

One of the most disturbing aspects of the seed transmission of viruses is that even low transmission rates can lead to the rapid spread of infection, given the number of seeds used for sawing. The initial number of infected plants in a population plays a crucial role in disease progression. While plant viruses are most commonly spread from primary infection sources by arthropod vectors, vertical transmission represents a significant vector-independent route, especially when vector populations are low in numbers or inefficient in virus transmission. For example, even if only 1% of seeds carry the virus, this can result in the emergence of 10^5–10^7 infected plants per hectare (Maule & Wang 1996). Despite such low transmission rates, the viruses, particularly in the presence of efficient insect vectors, can spread rapidly to new regions and cause outbreaks or, under favourable conditions for vector multiplication, can lead to epiphytoties. For example, Mikel et al. (1984) reported that a seed transmission rate as low as 0.0045% [one maize dwarf mosaic virus (MDMV, species *Potyvirus zeannanus*) – contaminated seed among 22.189 tested] was sufficient to introduce the virus into a new area. Similarly, even maize chlorotic mottle virus (MCMV, species *Machlomovirus zeae*), which also has an extremely low seed transmission rate of 0.004–0.100%, can still significantly impact *Zea mays* (maize) production (Jensen et al. 1991; Kimani et al. 2021; Bernardo et al. 2023). This highlights that even small amounts of infected seed can introduce viruses into new agricultural areas (often distant ones), where the viruses may find favourable conditions for their rapid spread.

The epidemiological significance of virus transmission through seed is particularly important when it is highly efficient. For example, the seed transmission efficiency of potyviruses such as MDMV, cowpea aphid-mosaic virus (CABMV, species *Potyvirus vignae*), and bean common mosaic virus (BCMV, *Potyvirus phaseovulgaris*) ranges 3–95%, while the transmission efficiency of PSbMV can be as high as 100% (Kyrychenko et al. 2020; Shanmugam et al. 2024).

The danger of seed-transmitted virus infections is evident from the significant yield losses ranging 4–100%, which can be caused in various crops (Stacie-Smith & Hamilton 1988; Holkar et al. 2020; Sandra & Mandal 2024). Seed-transmitted cereal viruses, for instance, BSMV in *Hordeum vulgare* (barley) and MDMV in maize, lead to significant yield and quality losses and are notable for their economic impact (Jeżewska & Trzmiel 2009). In Australia, wheat streak mosaic virus (WSMV, species *Tritimovirus tritici*) is prevalent and poses an ongoing challenge due to its potential to cause substantial yield losses. In contrast, virus-contaminated seed stocks and infected volunteer wheat seedlings play a crucial role in initiating new viral epidemics (Jones et al. 2021).

VIRUSES: SEED-BORNE OR SEED-TRANSMITTED?

In early studies, the appearance of symptoms on seedlings was often interpreted as evidence of seed-borne transmission of the virus, leading to the synonymical use of the terms "seed-borne" and "seed-transmitted". For instance, several *Daucus carota* (carrot) viruses, including carrot mottle virus (CMoV, species *Umbravirus maculacarotae*) and carrot red leaf virus (CTRLV, species *Poleovirus* CTRLV), were repeatedly classified as seed-transmitted since seedlings germinated from the seed of virus-infected plants exhibiting characteristic viral symptoms (Mink 1993). However, subsequent attempts to transmit these viruses from symptomatic seedlings to susceptible plants using aphid vectors were unsuccessful. Consequently, these pathogens have been classified as seed-borne, indicating that while present in certain parts of the seed or reproductive tissues, they do not necessarily infect the next generation of plants. Examples of non-cereal seed-borne viruses include CMV, PSbMV, soybean mosaic virus (SMV, species *Potyvirus glycitessellati*), zucchini yellow mosaic virus (ZYMV, species *Potyvirus cucrbitaflavitesselati*), various tobamoviruses and several begomovirus species (De Assis Filho & Sherwood 2000; Dombrovsky & Smith 2017; Holkar et al. 2020; Fortes et al. 2023; Sandra & Mandal 2024). For instance, PSbMV has been detected in the embryo (Wang & Maule 1994), CMV has been found in both the seed coat and embryo of pepper seeds (Ali & Kobayashi 2010), while tobacco mosaic virus (TMV, species *Tobamovirus tabaci*) is commonly associated with the seed coat (Sastry 2013). Some begomoviruses, such as tomato leaf curl New Delpji virus (ToLCNDV, species *Begomovirus solanumdelhiense*), have been detected in the seed coat, endosperm, and embryo of infected seeds. However, transmission efficiency varies among host-virus combinations (Sandra & Mandal 2024). Among cereal viruses, seed-borne examples include WSMV, Triticum mosaic virus (TriMV, species *Poacevirus tritici*), and MCMV (Gautam et al. 2023; Bernardo et al. 2023), whereas seed-transmitted viruses include BSMV, sugarcane mosaic virus (SCMV, species *Poacevirus sacchari*), and MDMV (Table 1) (Kannan et al. 2018; Sastry et al. 2019; Escalante et al. 2024).

Conversely, seed-transmitted viruses can be passed from the seed to the growing plant, leading to systemic infection in the next generation (Jones et al. 2005; Sandra & Mandal 2024). These viruses invade plant reproductive tissues at specific developmental stages, ensuring vertical transmission. The invasion can occur through two primary pathways: *(i)* pre-fertilisation infection of reproductive structures such as megaspores, seed embryos, microsporocytes, and pollen, or *(ii)* post-fertilisation direct invasion of the embryo (Johansen et al. 1994; Wang & Maule 1994). Many viruses utilise both routes, although the regulatory mechanisms governing each pathway differ and are controlled by distinct regions of the host genome (Mink 1993; Maule & Wang 1996; Pagán 2022; Escalante et al. 2024).

The transmission of seed-borne viruses to seedlings typically occurs through microlesions on the seed coat that form during germination, mechanical wounds, or insect feeding (Maule & Wang 1996; Fortes et al. 2023). However, true seed-to-seed transmission from an infected seed coat is relatively rare, except for viruses in the *Tobamovirus* group, which can persist on seed surfaces and be effectively managed through seed disinfection (Davino et al. 2020; Shanmugam et al. 2024). The low transmission efficiency of most seed-borne viruses is attributed to their instability during seed dehydration, harvesting, and storage. Furthermore, in many cases, the virus may not come into contact with the emerging seedling without mechanical means or vectors (Maury et al. 1987; Johansen et al. 1994).

A major challenge in distinguishing seed-borne and seed-transmitted viruses lies in detection methodologies. The presence of viral genome or proteins in the seed, as determined by sensitive molecular assays, does not necessarily indicate subsequent successful transmission of the virus to progeny plants (Mink 1993; Johansen et al. 1994). This highlights the need for comprehensive testing to differentiate between viral contamination and active infection in the next generation. Modern detection techniques have significantly improved our ability to identify and study seed-transmitted

https://doi.org/10.17221/51/2025-PPS

viruses. Various approaches are employed, including biological assays (grow-out test), microscopy-based methods (confocal laser scanning microscopy), hybridisation-based techniques (Southern and dot blot hybridisation), molecular and serological assays such as PCR-based techniques (nested PCR, multiplex PCR, real-time PCR), loop-mediated isothermal amplification, next-generation sequencing, DAS-ELISA and immunosorbent transmission electron microscopy (Albrechtsen 2006; Kaur et al. 2020; Patel et al. 2023; Sandra & Mandal 2024). These advancements have increased our understanding of virus transmission mechanisms and facilitated the implementation of effective management strategies, which play a crucial role in mitigating the risk of introducing seed-transmitted viruses into new regions and preventing their impact on global agriculture.

MOLECULAR BASIS OF SEED TRANSMISSION

Virus transmission efficiency through seed (vertical transmission) varies among host plants. It depends on factors such as the host cultivar, virus characteristics, environmental conditions, plant physiology, developmental stage at the time of inoculation, and potential synergism in case of mixed virus infections – all reflecting the complexity of the biological processes involved (Maule & Wang 1996; Montes & Pagan 2019; Gutiérrez-Sánchez et al. 2023; Shanmugam et al. 2024).

The molecular mechanisms underlying seed transmission involve complex interactions between the virus, the maternal host, and the progeny (Maule & Wang 1996). Despite ongoing research, this area remains insufficiently explored. Understanding the genetic factors that influence seed transmission rate and identifying viral genes responsible for this process are essential for determining why certain viruses are transmitted through seed while others are not, providing valuable insights into the mechanisms involved (Sastry 2013; Escalante et al. 2024).

Knowledge of the functions of plant genes involved in seed transmission remains very limited. However, studies are confirming that host genetic factors also play a significant role in determining seed transmission efficiency. In barley, seed transmission of BSMV is controlled by a single genetic locus. In contrast, in legumes such as *Pisum sativum* (pea), *Medicago sativa* (alfalfa), and *Glycine max* (soybean), multiple loci regulate seed transmission of PSbMV, alfalfa mosaic virus (AMV, species *Alfamovirus* AMV), and SMV (Liu & Ding 2024). In soybeans, seed transmission of SMV is controlled by genes homologous to *Arabidopsis* DCL3 and RDR6, which are involved in the RNA silencing defence response (Domier et al. 2011). A genome-wide association study identified loci on chromosomes 3 and 9 associated with SMV seed transmission, including genes related to RNA-mediated transcriptional gene silencing and virus movement restriction (Liu et al. 2019). In barley and pea, BSMV and PSbMV seed transmission is controlled by unidentified loci with quantitative effects (Pagán 2022).

Several viral genetic determinants have been identified encoding proteins that either facilitate or hinder seed transmission (Carroll 1979; Bowers & Goodman 1982; Mink 1993; Johansen et al. 1996; Hull 2014; Pagán 2022; Escalante et al. 2024). For instance, the γb protein of barley stripe mosaic virus acts as a viral suppressor of RNA silencing (VSR) and plays a key role in seed transmission (Edwards 1995). This protein regulates the expression of BSMV genes involved in virus movement and replication. Similarly, cysteine-rich proteins, such as the 12K protein of pea-early browning virus (PEBV, species *Tobravirus pisi*) and the helper component protein (HC-Pro) of PSbMV function as VSRs and are implicated in seed transmission (Johansen et al. 1996; Sandra & Mandal 2024). The HC-Pro of PSbMV, particularly, has pleiotropic effects on virus replication and movement. Gene products involved in virus movement, including coat protein, HC-Pro, and cylindrical inclusion protein, influence the virus's ability to invade gametes or developing embryos, directly affecting seed transmission efficiency (Carroll 1979; Edwards 1995; Wang 1997; Cobos 2019).

Environmental conditions also play a significant role in modulating seed transmission rates. Factors such as temperature, humidity, rainfall, light intensity, and CO_2 levels can affect the physiological state of the host plants, virus stability within the seed, and ultimately, the efficiency of seed transmission (Johansen et al. 1994; Montes & Pagán 2019; Gutiérrez-Sánchez et al. 2023). Stress conditions, such as drought or high temperatures, may either enhance or suppress seed transmission, depending on the host-virus system (Montes & Pagán 2019).

Understanding these molecular interactions and specific genetic resistance mechanisms is essential for developing strategies to manage seed-transmitted viral diseases. Identifying specific nucleotide sequences or gene products associated with the efficiency of seed transmission can be used in breeding programs aimed at developing plant varieties with lower/close-to-zero seed transmission rates and improving seed health management practices.

EFFECTS OF SEED-BORNE/-TRANSMITTED VIRUSES ON POLLEN AND SEED FORMATION

Viruses transmitted through seed can significantly impact pollen and seed formation, affecting plant health, productivity, and crop yields. While these viruses often induce mild symptoms in their hosts, certain virus-host interactions can result in severe disease manifestations (Johansen et al. 1994). In case of intense viral infection, plants may exhibit altered shoot development, reduced flower formation, and prevented or delayed seed development. These disruptions can directly influence virus transmission efficiency through seed, as a weakened reproductive system may reduce the seed set or limit the number of viable, virus-contaminated seeds.

Viral infections often affect both pollen and ovules and can cause morphological or genetic changes in the seed embryo, reducing fertility and leading to poor seed quality and lower yields. Some viruses, such as tomato aspermy virus (TAV, species *Cucumovirus* TAV), are not seed-transmitted exactly because they disrupt the meiosis process, causing sterility of both pollen and ovules (Mandahar 1981; Johansen et al. 1994).

Pollen is often more severely affected by viral infections than ovules, making it a critical factor in limiting virus transmission. Plants infected by pollen-transmitted viruses tend to produce less viable pollen with reduced germination capacity and often generate fewer fertile pollen grains, reducing their overall reproductive success (Mandahar 1981; Johansen et al. 1994; Fetters & Ashman 2023). However, despite the negative impact of viral infection on pollen viability and seed production, these effects are often insufficient to eliminate the epidemiological importance of pollen transmission. Many viruses, including BSMV, remain capable of highly effective transmission despite reproductive challenges (Mandahar 1981; Mink 1993).

SEED-TRANSMITTED VIRUSES OF CEREAL CROPS IN UKRAINE

Seed transmission is a significant mode of dispersal for at least eight known cereal viruses, although transmission rates and epidemiological importance vary widely among viruses and host plants (Table 2).

These viruses belong to different taxonomic groups, exhibit varying degrees of seed transmission efficiency, and infect a diverse range of susceptible cereal crops. Five of these viruses circulate in Ukraine and have a substantial impact on cereal production: BSMV (Snihur & Shevchenko 2013; Snihur et al. 2018), HPWMoV (Pasichnyk et al. 2020; Snihur et al. 2020; Pozhylov et al. 2022), MDMV, SCMV (Snihur et al. 2021; Vlasova et al. 2024), and WSMV (Mishchenko & Bojko 2004; Mishchenko 2009; Snihur & Shevchenko 2013; Pozhylov et al. 2022). Most of these viruses are consistently detected in agroecosystems across Ukraine and pose a significant threat to cereal production. Their ability to reduce grain yields affects agricultural productivity and has broader commercial implications for the country and global economy.

BARLEY STRIPE MOSAIC VIRUS

Taxonomy and structure. *Hordeivirus hordei* (barley stripe mosaic virus, BSMV) belongs to the genus *Hordeivirus* within the family *Virgaviridae* (ICTV 2023). BSMV virions are rigid, rod-shaped particles measuring approximately 112–150 nm in length and 18–24 nm in diameter. The BSMV genome consists of a linear, positive-sense, single-stranded RNA comprising three genome components designated as α, β, and γ in decreasing order of molecular weight (Jackson et al. 2009). This tripartite genome encodes proteins involved in replication, movement, and virus-host interactions, facilitating the virus's systemic infection.

Host range and distribution. BSMV is widespread in all major cereal-growing regions worldwide, naturally infecting wheat, barley, *Secale cereale* (rye), maize, *Avena sativa* and *A. fatua* (cultivated and wild oats), as well as several experimental *Poaceae* and non-*Poaceae* hosts (Jackson et al. 2009; Jiang et al. 2021; Jezewska 2022; EPPO 2025a). Historically, BSMV was listed as a quarantine pest under the European and Mediterranean Plant Protection Organization (EPPO) A2 list but

Table 2. Seed-borne and seed-transmitted viruses of cereal crops

Virus species name (acronym)	Seed transmission rate (%)	Host plants	Geographical distribution	Key references
Hordeivirus hordei (BSMV)	0.600–100.0000	*Hordeum vulgare, Avena sativa, Secale cereale, Triticum aestivum, T. durum, Zea mays*	all continents	Timian 1974; Sastry et al. 2019; Jiang et al. 2021; EPPO 2025a
Tritimovirus tritici (WSMV)	0.1000–3.1000	*Triticum* sp., *A. sativa, H. vulgare, S. cereale, Z. mays*	all continents	Coutts et al. 2014b; Singh et al. 2018; Hasan et al. 2025
Emaravirus tritici (HPWMoV)	0.0001–4.0000	*H. vulgare, T. aestivum, Z. mays*	Argentina, Australia, Canada, Iran, USA, Ukraine, New Zealand	Coutts et al. 2014a; Alemandri et al. 2017; Pozhylov et al. 2022; Bragard et al. 2022; Nourbakhsh et al. 2023
Potyvirus sacchari (SCMV)	0.5000–4.8000	*Z. mays, Saccharum* spp., *Ranunculus japonicus* Thunb., *Poaceae grasses*	all continents	Braidwood et al. 2019; Lu et al. 2021; Regassa et al. 2021; Alabi 2022; Xu et al. 2023; EPPO 2025b
Potyvirus zeananus (MDMV)	0.0070–6.5000	*Z. mays, Sorghum bicolor, S. halepense, Saccharum officinarum*	all continents	Hill et al. 1974; Mickel et al. 1984; Rosenkranz 1987; Trzmiel & Jezewska 2008; Kannan et al. 2018
Machlomovirus zeae (MCMV)	0.0400–0.3300	*H. vulgare, T. aestivum, Z. mays*	North and South America, East Africa, East Asia	Jensen et al. 1991; Regassa 2021; Bernardo et al. 2023; Isabirye & Rwomushana 2016
Potexvirus setariae (FoMV)	1.0000	*A. sativa, Z. mays, H. vulgare, S. cereale, T. aestivum, T. durum, S. bicolor*	USA	Paulsen & Niblett 1977
Poacevirus tritici (TriMV)	7.0000–12.0000	*H. vulgare, A. sativa, A. fatua, S. cereale,* × *Triticosecale* Wittmack, *Poaceae* grasses	USA, Canada, Argentina	Seifers et al. 2010; Gautam et al. 2023; Tatineni et al. 2025

was removed in 1999 due to its widespread occurrence in barley-growing regions, which made quarantine measures less effective. Despite removing the virus from the list, it remains under phytosanitary monitoring to prevent its spread via contaminated seed, the primary transmission mode.

Symptoms and pathogenic effects. BSMV infection in barley typically manifests as chlorotic streaks and yellow or whitish chlorosis that initially appear near the base of the youngest leaves. As the infection progresses, systemic chlorotic and mosaic patterns develop on older leaves (Figure 1). Advanced infection stages can manifest through leaf desiccation, stunted growth and reduced tillering. At the late stages of infection, certain virus strains may induce lethal necrosis, significantly impacting plant viability (Sastry et al. 2019).

Infected barley plants produce fewer and smaller grains per spike, with seeds of reduced weight and shrivelled appearance due to poor nutrient transport and virus-induced stress, ultimately leading to impaired plant growth and development (Nutter et al. 1984). BSMV infection delays or inhibits plastid development and the formation of photosynthetic apparatus (Harsányi et al. 2002). Infected barley leaves have less chlorophyll than non-infected leaves, as the mosaic pattern shows. In wheat, BSMV infection causes leaf mosaic, mottling, and chlorotic stripes and patches similar to those observed in barley (Tatineni et al. 2022). In oats, the virus induces milder symptoms with delayed onset compared to wheat, suggesting that wheat cellular machinery supports BSMV replication and movement than oats. Symptom severity varies depending on virus strain, host genotype, and environmental conditions. Furthermore, plants grown from contaminated seeds may not always exhibit visible symptoms, making it challenging to detect and eliminate infected breeding material (Sastry 2013). This asymptomatic transmission

https://doi.org/10.17221/51/2025-PPS

Figure 1. Barley stripe mosaic virus in winter wheat in Ukraine
(A) leaf striping symptoms; (B) wheat spikelets of infected (left) and healthy (right) plants; (C) virus morphology (photographs from the authors' research)

underscores the importance of rigorous seed testing and the introduction of molecular diagnostic approaches for effective disease management.

Transmission and epidemiology. BSMV has no known biological vectors and spreads in barley crops from primary infection foci originating from contaminated seed mainly through mechanical transmission, making contaminated seed stocks the crucial source of inoculum (Lawrence & Jackson 1999). Depending on host species and environmental factors, the virus is seed-transmitted at varying rates, reaching up to 90–100% in susceptible cultivars (Timian 1974; Sastry et al. 2019). In barley, seed transmission occurs through two primary mechanisms: *(i)* direct infection of the embryo via the egg and *(ii)* indirect infection via infected gametes (Mandahar 1981). The ability of BSMV to persist in the embryo significantly enhances the seed transmission efficiency, enabling the virus to spread effectively across cereal crops. The conditions of embryo maturation can influence virus replication, affecting seed transmission rate (Johansen et al. 1994). Although virus particles have been detected in the pollen tube, zygote, endosperm, persistent synergid, and nucellus, indicating that pollen can infect maternal plant tissues, this mode of transmission has not been experimentally confirmed (Brlansky et al. 1986; Jackson et al. 2009). Therefore, pollen transmission is not considered a significant factor in the spread of this virus.

Economic impact. BSMV is considered to be the only hordeivirus of economic importance, causing severe yield losses, particularly in barley, with reported yield losses ranging from 20–50%, depending on the time of infection, the proportion of infected plants, virus strain pathogenicity, and crop variety (Carroll 1980; Lawrence & Jackson 1999). In the case of 90% BSMV incidence in barley, yield reductions of 35–40% were observed, though losses ranging from 19–62% have been reported depending on barley variety. The grain losses are primarily attributed to reduced productive tillers, lower seed weight per spike, increased floral sterility, poor seed set, seed shrivelling, and reduced seedling viability (Nutter 1984). BSMV is also highly resistant to various physical and chemical treatments. In *H. vulgare*, BSMV remains viable and infectious even after 19 years of seed storage (Sastry 2013).

The high levels of BSMV seed transmission and the global transportation of contaminated seeds have been responsible for the distribution of BSMV worldwide. Additionally, BSMV presents a serious challenge for cereal breeding programs due to high seed transmission rate and persistence. However, since seed transmission is essential for virus survival from season to season, BSMV can be controlled through sensitive diagnostic screening to identify and eliminate contaminated seed stocks (Jackson et al. 2009). The development of sensitive virus detection methods allowed for pre-planting seed testing, thus reducing virus circulation and even eradicating several countries across the Americas and Europe. Currently, many countries have implemented strict seed testing and certification protocols [EPPO Phytosanitary Procedure PM 3/34(1)] to prevent seed transmission and limit the spread of BSMV (OEPP/EPPO 1991). These measures, along with the development of resistant cultivars through genetic breeding programs, have significantly reduced the need for quarantine regulations.

https://doi.org/10.17221/51/2025-PPS

Figure 2. Diverse symptoms of wheat streak mosaic virus infection observed in winter wheat in Ukraine (photographs from the authors' research)

WHEAT STREAK MOSAIC VIRUS

Taxonomy and structure. *Tritimovirus tritici* (wheat streak mosaic virus, WSMV) is the typical of *Tritimovirus* genus in the family *Potyviridae* (Inoue-Nagata et al. 2022). WSMV has filamentous particles of approximately 700 nm in length and 13 nm in diameter and has a single-stranded positive-sense RNA genome with characteristic features of a typical potyvirus (Gautam et al. 2023).

Host range and distribution. WSMV is common in all major cereal-growing regions worldwide and infects a wide range of cereal crops, including winter and spring wheat, barley, rye, oats, *Panicum miliaceum* (millet), maize, *Sorghum bicolor* (sorghum), and weedy grasses of *Poaceae* family (Singh et al. 2018; Byamukama 2022). Various weed species, such as *Setaria italica* (foxtail millet), *S. viridis* (green foxtail), *Echinochloa crus-galli* (cockspur grass), *Stipa* spp. (feather grass), *Aegilops* spp. (goatgrass), *Digitaria* spp. (crabgrass), *Lolium perenne* (ryegrass), *Bromus* spp. (bromes), and *Eriochloa villosa* (cupgrass) act as reservoirs for WSMV, contributing to its persistence and spread in agroecosystems (Kyrychenko et al. 2023). In the fall, these alternative hosts provide a "green bridge" for virus transmission by vectors to wheat crops (Christian & Willis 1993).

Symptoms and pathogenic effects. The virus typically causes leaf streaking, varying from light green to bright yellow, but symptoms can vary depending on the host species, environmental conditions and infection severity. In wheat, WSMV infection leads to leaf yellowing, mosaic patterns, streaking, chlorosis, shoot necrosis, and plant stunting and can result in substantial or even complete yield loss. Infected plants are often severely stunted, with symptoms frequently progressing to leaf tissue necrosis and plant death (Kyrychenko et al. 2023). Symptoms in winter wheat vary among cultivars with characteristic fine chlorotic streaks or discontinuous light green stripes of different widths running parallel to leaf veins (Singh et al. 2018). The visual differences in viral symptoms highlight the challenges in diagnosing WSMV solely based on visual symptoms (Figure 2).

In maize, WSMV infection induces leaf yellowing and a characteristic mosaic pattern with longitudinal chlorotic streaks along leaf veins (French & Stenger 2004). However, symptom severity in maize varies, with some hybrids exhibiting only mild discolouration and reduced vigour (Trzmiel & Szydło 2012). Variability in symptom expression suggests that maize hybrids differ in susceptibility to WSMV.

Transmission and epidemiology. WSMV is transmitted mainly by the *Aceria tosichella* Keifer (wheat curl mite), a microscopic eriophyid mite that facilitates virus spread within and between fields. Although WSMV is not insect-transmitted, seed transmission plays an important role in the long-term survival of the virus and its spread by the vector from the primary inoculum reservoir to new areas. The seed transmission rate of WSMV depends on wheat genotype and is usually very low, ranging from 0.2–0.5% to 1.5% (Jones et al. 2005; Singh et al. 2018). However, McKelvy et al. (2023) reported a significantly higher seed transmission rate in spring wheat, averaging 3.1%, five times greater than the 0.6% rate observed in winter wheat. Importantly, WSMV is not transmitted through seeds of barley, oats, and certain annual grasses (Coutts et al. 2014b). Although such low seed transmission rates are unlikely to drive WSMV epidemics within an individual field, the global exchange of contaminated germplasm enhances the risk of virus introduction into new regions (Singh et al. 2018; Hasan et al. 2025).

Economic impact. WSMV significantly reduces grain production depending on disease intensity, with annual yield losses ranging from 2–5% to nearly 100% in case of severe outbreaks (Singh et al. 2018; Hasan et al. 2025). In North America, WSMV is responsible for annual yield losses of approximately 5%, though under conditions favouring vector proliferation, localised epidemics can result in complete crop failure (Jones et al. 2005; Xie et al. 2022).

In Ukraine, WSMV is distributed across all wheat-growing regions and nowadays is considered the most harmful virus affecting cereals. Crop losses due to WSMV can reach 60% in some regions and may escalate to 80–100% when the infection is widespread early in the crop life (Mishchenko et al. 2018; Kyrychenko et al. 2023). The overall severity of the virus depends on weather conditions, plant species/variety and cultivation practices. Yield reduction primarily results from damage to the plant's reproductive organs, leading to deterioration of seed quality and reduced germination energy and seed germination rates by up to 50% (Pozhylov et al. 2022).

Efforts to manage WSMV focus on integrated strategies, including planting resistant cultivars, implementing cultural practices to break the virus transmission cycle, and controlling wheat curl mite populations. A crucial component of WSMV management is the removal of "green bridge" hosts – summer weed reservoirs favouring the survival of mite populations and hence facilitating virus persistence between cropping seasons (Gautam et al. 2023).

Wsm1 and *Wsm2* resistance genes have been introduced into commercial wheat cultivars to control WSMV (Jones et al. 2011). Although these resistance genes do not completely prevent seed transmission, they significantly reduce transmission of WSMV through contaminated seeds. Still, they are ineffective against TriMV, which is often found in mixed infection with WSMV. The resistant varieties have accumulated TriMV and WSMV similarly to susceptible ones, most likely due to breaking *Wsm1*- and *Wsm2*-mediated resistance at temperatures above 18°C. Even with reduced accumulation levels in the seed coat, these viruses pose a significant threat to agriculture, emphasising the importance of planting virus-free seed stocks and the need to identify/develop new sources of resistance for disease prevention (Gautam et al. 2023). Ongoing breeding programs continue to seek new genetic sources of resistance to enhance WSMV control and mitigate its economic impact on cereal production.

HIGH PLAINS WHEAT MOSAIC VIRUS

Taxonomy and structure. *Emaravirus tritici* (High Plains wheat mosaic virus, HPWMoV, also known as High Plains virus or wheat mosaic virus), is a member of the genus *Emaravirus* within the family *Fimoviridae* (Mielke-Ehret & Mühlbach 2012; ICTV 2015; Kuhn et al. 2023). The viral particles are enveloped, quasi-spherical to ovoid, and measure 80–200 nm in diameter. HPWMoV has a segmented, single-stranded negative-sense RNA genome composed of eight RNA segments, with a total genome size of approximately 18.5 kb. The virus was first identified and described in the United States in 1993 as the causal agent of high plains disease, primarily affecting wheat and maize (Jensen et al. 1996).

Host range and distribution. HPWMoV has been reported in Iran, Australia, New Zealand, South America, Canada, the USA (where it's frequently found in mixed infection with WSMV and/or TriMV), and in Ukraine in wheat and maize across several regions (Snihur et al. 2020; Bragard et al. 2022; Pozhylov et al. 2022; Nourbakhsh et al. 2023).

Although HPWMoV has not been officially reported in European countries other than Ukraine, due to the occurrence of its vector, wheat curl mite (same as for WSMV), and numerous susceptible hosts, there is a potential risk of virus spread in Europe. The European Food Safety Authority (EFSA) has assessed the potential risks of HPWMoV introduction and establishment within the European Union (EU) and recommends phytosanitary measures to prevent virus entry and distribution. These measures should consider biological and technical factors that may limit their effectiveness (Bragard et al. 2022).

In nature, HPWMoV infects wheat, maize, and several other cultivated or wild *Poaceae* species [*Setaria lutea* (yellow foxtail), green foxtail, *Bro-*

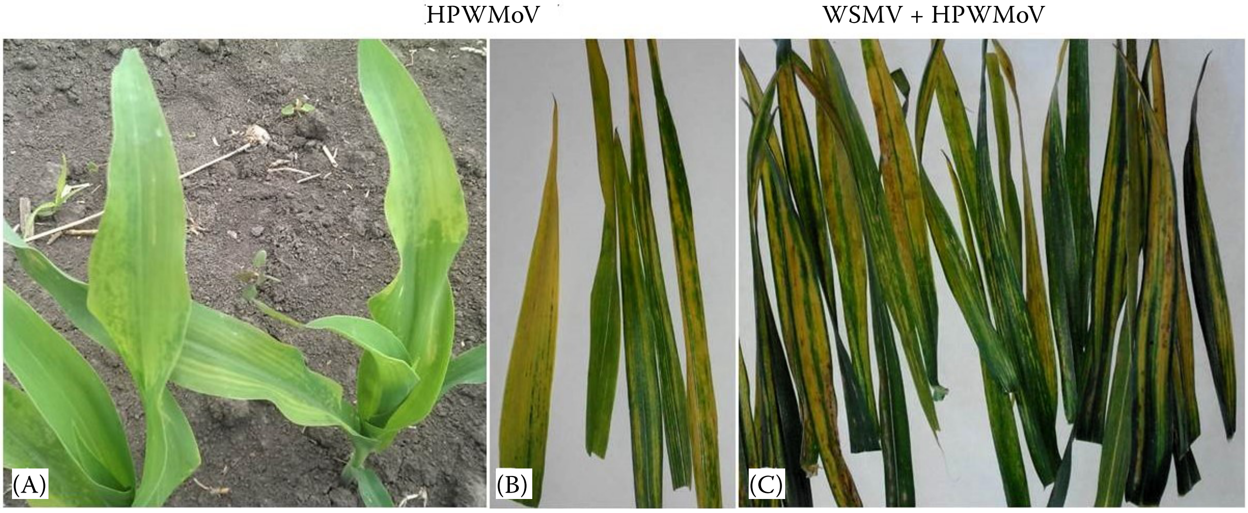

Figure 3. Symptoms of leaf mosaic
(A) in maize; (B, C) winter wheat naturally infected with high plains wheat mosaic virus alone (A, B); (C) mixed infected with wheat streak mosaic virus (photographs from the authors' research)

mus tectorum (downy brome), and *Hordeum jubatum* (foxtail barley)] (Snihur et al. 2020; Redila et al. 2021; Bragard et al. 2022; Pozhylov et al. 2022). Under experimental conditions, additional species such as oats, barley, rye, and *Festuca pratensis* (meadow fescue) were successfully infected using wheat curl mites as a vector (Bragard et al. 2022). Some wild grasses and other cereal crops may also serve as secondary or reservoir hosts, and thus contribute to virus persistence between growing seasons and its subsequent spread. The role of these alternative hosts should be considered in disease management strategies.

Symptoms and pathogenic effects. The symptoms of HPWMoV often resemble those caused by WSMV, varying from mild to severe depending on environmental conditions and host susceptibility (Burrows et al. 2009; Kyrychenko et al. 2023). In wheat, symptoms include mosaic, chlorosis and necrosis, while infected maize plants show stunting, red striping, and chlorotic mosaic or streaking (Figure 3) (Jensen et al. 1996; Tatineni & Hein 2021; Bragard 2022). Severe infections, particularly in susceptible *Zea mays* var. saccharata (sweet maize), can lead to plant death when infections occur at early growth stages.

HPWMoV can cause both mono- and mixed infections with TriMV and WSMV, with mixed infections generally leading to more severe disease outcomes. Mixed infections of HPWMoV and WSMV are common in both wheat and maize, often resulting in more severe symptoms (Burrows et al. 2009; Mielke-Ehret & Mühlbach 2012). Distinguishing HPWMoV from other mosaic-inducing viruses based on symptoms alone is challenging and unreliable (Figure 3). In maize, mixed infections with WSMV result in more severe symptoms, leading to higher crop losses (Burrows et al. 2009).

Transmission and epidemiology. HPWMoV is primarily transmitted by the wheat curl mite, which also serves as the vector for WSMV and TriMV. Limited experimental evidence suggests that HPWMoV can also be seed-transmitted in sweet maize at a rate ranging from very low to 2–4% (Forster et al. 2001; Blunt & Hill 2004; Nischwitz 2020). However, there is no conclusive evidence of seed transmission in wheat or other hosts and no information on the transmission of emaraviruses with pollen (Mielke-Ehret & Mühlbach 2012; Bragard et al. 2022). The virus epidemiology and disease cycle are closely linked to wheat curl mite populations and behaviour, as well as to the presence of "green bridge" host plants that allow the viruses and viruliferous mites to survive in the absence of susceptible crops between cropping seasons (Bragard et al. 2022). This factor plays a significant role in virus transmission and spread. Unlike many plant viruses, HPWMoV is not mechanically transmissible through sap inoculation. However, vascular puncture inoculation has been successfully used to infect maize embryos in experimental settings (Seifers et al. 2004; Louie et al. 2006). Due to its potential for seed transmission, even at low rates, HPWMoV poses a risk of long-distance dispersal through international seed trade (Tatineni & Hein 2021).

Economic impact. HPWMoV, WSMV, and TriMV form the wheat mosaic disease pathogen complex, with all three viruses being vectorised by the wheat curl mite. Mixed infections with different combinations of these viruses frequently intensify disease severity, sometimes resulting in complete crop failure and substantial yield losses under field conditions (Mahmood et al. 1998; Burrows et al. 2009; Tatineni & Hein 2021). In Ukraine, HPWMoV was detected in the country's eastern regions, primarily affecting winter wheat crops. The virus was predominantly found in mixed infection with WSMV, while mono-infection of HPWMoV was detected in only ~9% of infected plants (Pozhylov et al. 2022).

The economic importance of HPWMoV is largely tied to its role in mixed infections. Therefore, yield losses from HPWMoV alone are difficult to calculate. However, reported losses range 27–79.6% in wheat, up to 75% *Zea mays* var. indentata (dent maize), and up to 100% in sweet maize (Bragard et al. 2022). Mixed infections can affect HPWMoV epidemiology and significantly exacerbate yield losses already caused by WSMV alone (Seifers et al. 2002; Hein et al. 2012; Bragard et al. 2022).

Although HPWMoV seed transmission occurs at relatively low rates, this mode remains epidemiologically significant due to its potential for international spread (Tatineni & Hein 2021). The effective disease management strategy includes implementing routine seed testing for HPWMoV, breeding wheat cultivars resistant to the wheat curl mite, and implementing phytosanitary measures to prevent its introduction into new regions via contaminated seed. These measures can mitigate direct damage from mite feeding and losses caused by the virus complex it transmits (Tatineni & Hein 2021).

SUGARCANE MOSAIC VIRUS

Taxonomy and structure. *Potyvirus sacchari* (sugarcane mosaic virus, SCMV), a positive-sense single-stranded RNA (+ssRNA) virus, belongs to the *Potyvirus* genus of the *Potyviridae* family and is an important pathogen affecting *Saccharum officinarum* (sugarcane) cultivation worldwide (ICTV 2023; Xu et al. 2023; Magarey 2024). The morphology of SCMV virions is typical for the members of the *Potyviridae* family, with non-enveloped, flexuous-filamented virions 750 nm long and 13 nm wide (Viswanathan et al. 2018).

Host range and distribution. SCMV is widely distributed and reported across six continents, including Africa, Asia, the Americas, and Europe, making it a cosmopolitan virus (Braidwood et al. 2019; Alabi 2022; EPPO 2025b). Mosaic disease caused by SCMV has been widely observed in most sugarcane-growing regions worldwide and has become a pandemic in many countries or regions (Lu et al. 2021).

SCMV has a broad host range. While sugarcane is the natural host, SCMV also infects more than 100 species in 40 genera of the *Poaceae* family, including maize, sorghum, and various wild grasses (Rosenkranz 1987; Viswanathan et al. 2018; Lu et al. 2021; Regassa et al. 2021; Xu et al. 2023; Magarey 2024).

In Ukraine, SCMV has been primarily detected in maize, where it caused significant damage to crops. The virus was first reported in the Kyiv region in 2018, and its spread in Ukrainian fields posed a significant risk (Snihur et al. 2021; Vlasova et al. 2024). The increasing prevalence of SCMV in maize is closely linked to the widespread distribution of its primary vector, the aphid *Rhopalosiphum padi* (bird cherry-oat aphid), which is prevalent across Ukraine (Pozhylov et al. 2022). Serological testing has confirmed that maize in Ukraine is infected by SCMV exclusively in mono-infection, with no other viruses detected so far in mixed infection with SCMV. However, mixed infections of SCMV with other maize-infecting viruses, particularly with barley yellow dwarf virus-PAV (BYDV-PAV, species *Luteovirus pavhordei*), which is widely distributed across the country, is very likely and may complicate disease management in the future (Vlasova et al. 2024). Thus, the spread of SCMV in Ukrainian agroecosystems is believed to occur through a combination of seed and vector-mediated transmission (Snihur et al. 2021).

Symptoms and pathogenic effects. The symptoms of SCMV in maize include irregular light/dark green mosaic patterns on the leaves, alternating white or yellow areas, spots, and stripes (Figure 4). However, similar symptoms can also be caused by at least five other known 24 maize viruses – sorghum mosaic virus (SrMV, species *Potyvirus sorghitessellati*), sugarcane streak mosaic virus (SCSMV, species *Poacevirus sacchari*), maize yellow mosaic virus (MaYMV, species *Poleovirus* MAYMV), sugarcane mild mosaic virus (SCMMV, unclassified *Ampelovirus)*, maize striate mosaic virus (MSMV, species *Mastrevirus*

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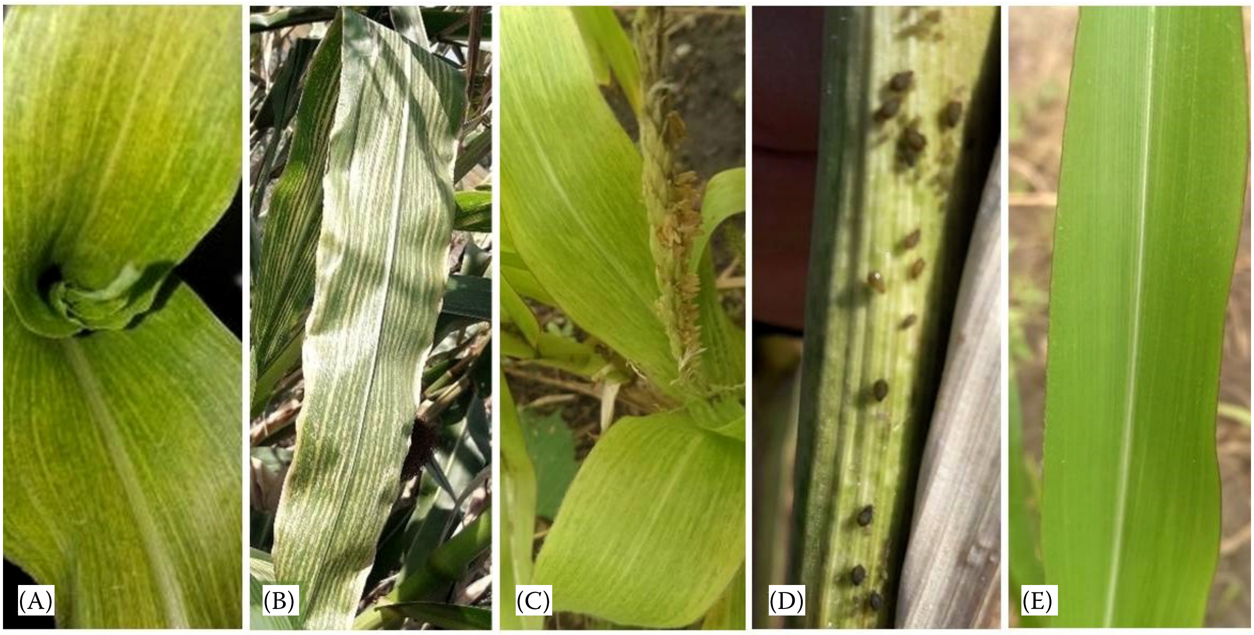

Figure 4. Detection of sugarcane mosaic virus in *Zea mays* in Ukraine

(A) mosaic symptoms on naturally infected plants in early June; (B) middle of September; (C) mosaic symptoms on artificially inoculated plants; (D) *Rhopalosiphum padi* aphids on naturally infected plants; (E) healthy plant (photographs from the authors' research)

striatus) (Daugrois et al. 2023; Krishna et al. 2023), making highly unreliable accurate diagnosis based on visual symptoms alone, especially in case of mixed infection. These viruses are responsible for mosaic disease, one of the most important and, in some regions, the most damaging diseases of sugarcane (Lu et al. 2021). The specific causative virus (SrMV, SCMV or SCSMV) of sugarcane mosaic disease varies from country to country, and the severity of disease symptoms depends on the plant species, virus strain and environmental conditions.

Transmission and epidemiology. SCMV is transmitted by various species of aphid vectors in a non-persistent manner, through mechanical inoculation, and by seed, albeit at a low rate (< 1%) (Regassa et al. 2021; Bernardo et al. 2023). However, seed transmission rates as high as 3.9–4.8% have been reported in susceptible maize breeding lines, as determined by seed grow-out tests and ELISA (Li et al. 2007, 2011). SCMV can infect maize embryos through both female and pollen pathways, with the maternal route being the main pathway for virus entry. Pollen-mediated seed transmission is insignificant at 0.04–0.10% (Li et al. 2007). Additionally, the embryo development stage affects seed transmission efficiency, where late-stage infections may not result in seed transmission due to the absence of the suspensor at this stage (Escalante et al. 2024).

Economic impact. SCMV is among the most economically damaging plant viruses affecting sugarcane, maize, sorghum, canna, and other monocot species worldwide (Rybicki 2015; Muhammad et al. 2022). Mixed infections involving SCMV tend to be more virulent than single-virus infections, suggesting virus synergy (Wu et al. 2012). Mosaic disease caused by SCMV and associated viruses leads to significant yield losses in sugarcane, ranging 0.5–80.0% in different countries. This depends on the susceptibility of the cultivar, virus strain, climate conditions, and insect vector population (Singh et al. 2003; Krishna et al. 2023).

The disease directly affects the photosynthesis and growth of sugarcane, leading to a substantial decrease in cane yield, sucrose and juice content, and crystallisation rate (Lu et al. 2021). The increasing incidence of mosaic disease in China and India, where infection rates have reached 100% in some sugarcane-growing areas, is attributed to the cultivation of highly susceptible varieties, prolonged use of self-propagated material, continuous cropping, frequent introductions, and inadequate management practices (Lu et al. 2021).

Reported yield reductions due to SCMV infection range 10–35% in sorghum and sugarcane and

20–50% in maize (Viswanathan & Balamuralikrishnan 2005; Braidwood et al. 2019). In India, SCMV causes a progressive decline in yield and sugarcane juice quality, with losses ranging 0.5–45.0% (Singh et al. 2003). In Brazil, sowing of 100% SCMV-contaminated sugarcane seed resulted in a yield reduction of up to 71% in some cultivars. Early SCMV infection in maize has also resulted in 18–46% yield losses in several countries, particularly affecting susceptible hybrids (Alabi 2022). The losses caused by SCMV are mainly due to reduced yield, decreased crop vigour, and, in severe cases, complete crop failure. Although SCMV has been known for decades, new strains and genetic variations continue to emerge worldwide, posing an ongoing threat to sugarcane and maize production (Wu et al. 2012).

Since aphids naturally transmit SCMV in a non-persistent manner, control of SCMV vectors is ineffective. The most effective and economical way to prevent SCMV outbreaks lies through cultivating resistant or tolerant varieties and using healthy seedlings (Dussle et al. 2002; Wu et al. 2012). Although the virus is transmitted with seed at a low rate, SCMV-specific molecular assays are important for ensuring virus-free vegetative planting material and seed stocks to mitigate field-level spread of the virus (Smith & van de Velde 1994).

MAIZE DWARF MOSAIC VIRUS

Taxonomy and structure. *Potyvirus zeananus* (maize dwarf mosaic virus, MDMV) belongs to the genus *Potyvirus* of the family *Potyviridae* (ICTV 2023). MDMV virions exhibit the characteristic morphology of potyviruses, consisting of filamentous, flexuous particles with a length of 750 nm and a 12–15 nm diameter. The genome of MDMV is a monopartite, linear, +sense ssRNA, about 9 500 bp long, with a VPg (viral genome-linked protein) at the 5' end and a poly(A) tail at the 3' end (Shukla et al. 1992).

Host range and distribution. MDMV is a serious pathogen of maize and, along with SCMV, is responsible for maize dwarf mosaic disease, which occurs worldwide wherever maize is cultivated. The disease incidences were reported in Africa, the United States, Asia, and Europe (Gordon et al. 1981; Achon & Alonso-Duenas 2009). Unlike SCMV and its strains, MDMV can infect both diploid (2n) and tetraploid (4n) *Sorghum halepense* (Johnson grass), which serves as a key distinguishing factor between these viruses (Toler 1985). MDMV is a unique potyvirus capable of infecting Johnson grass easily, contrary to other cereal viruses (Mohammadi & Hajieghrari 2009; Haji et al. 2022).

MDMV has an unusually wide host range, with over 250 plant species susceptible to infection through mechanical inoculation or aphid transmission (Toler 1985). Under natural conditions, the virus infects numerous annual crops, small grains, and perennial grasses of the *Poaceae* family, acting as reservoirs during winter in some areas (Thongmeearkom et al. 1976; Toler 1985; Rao et al. 1996). The primary overwintering host and natural reservoir of MDMV is Johnson grass, the only host playing a significant role in virus epidemiology (Achon 1999). Infected plants are generally assumed to be a persistent source of MDMV inoculum, which is transmitted by aphid vectors to susceptible crops, particularly maize and sorghum (Achon et al. 2001).

In Ukraine, MDMV was first detected in 1970–1971 in maize fields in Kyiv, Dnipropetrovsk, and Kherson regions (Snihur et al. 2021). The incidence of the virus was related to the widespread distribution of the main virus vector, *R. padi*, and other aphid species, such as *R. maidis* (corn leaf aphid) and *Schizaphis graminum* (green bug). The virus is particularly damaging in the regions of the expansion of its natural reservoir, Johnson grass. This weed was first recognised as a quarantine pest in Ukraine in 2003, initially detected in the Odesa region across 55 hectares, with subsequent expansion to 760 hectares by 2006 (Fedorenko & Pylypenko 2012; Parker 2022). In addition to Johnson grass, other MDMV reservoir hosts in Ukraine include several annual and perennial grasses such as *Bromus arvensis* (field bromegrass), *B. secalinus* (rye bromegrass), *Panicum capillare* (hairy millet), and green foxtail.

Symptoms and pathogenic effects. The symptoms of mosaic disease caused by MDMV in maize are similar to those of SCMV, causing considerable confusion in the taxonomy of these viruses in earlier literature. It is now recognised that MDMV strain B (MDMV-B) is, in fact, a strain of SCMV (Shukla et al. 1992). Early symptoms of MDMV infection in maize appear particularly near the base of the youngest leaves as small chlorotic spots along the veins, which subsequently develop into a characteristic mosaic pattern as fine or coarse patches with alternating light and dark green shades, depending on the plant genotype (Tsai & Brown 1989; Kannan et al. 2018).

Infected leaves may develop yellow streaks along their edges, which can fade in hot weather, leading to common chlorosis in subsequent growth. In older plants, chlorosis is limited to upper leaves, and red streaks may develop in late infections. Additional symptoms include mottling, necrotic lesions, and intensified mosaic, fleck, and ring patterns. Severe infections result in continuous chlorosis and reddening, leading to leaf desiccation and drying (Kannan et al. 2018). The severity of symptoms varies depending on host genotype and environmental conditions. The red-purple streaks observed in symptomatic plants are attributed to anthocyanin accumulation induced by chlorophyll degradation or activated anthocyanin biosynthesis in response to infection (Luo et al. 2021).

Oryza sativa (rice), maize, sorghum, and Johnson grass react systemically to the virus artificial inoculation. Infected rice plants show yellowing and severe mosaic, while inoculated maize and sorghum plants exhibit severe streak, yellowing, and necrosis at the leaf apex. *S. bicolor* develops severe mosaic, yellowing, striation, and necrosis at the leaf apex, whereas infected Johnson grass develops yellow streaks, turning orange and red at the later stage (Haji et al. 2022).

Transmission and epidemiology. MDMV is transmitted in a non-persistent manner by more than 20 species of aphids, several of which are prevalent in Ukraine: *R. maidis*, *R. padi*, *S. graminum*, and *Sitobion avenae* (English grain aphid) (Knoke & Louie 1981). Aphids acquire the virus within 10–30 s of feeding on infected plants and retain the virus for an average of 15–20 min, with a maximum retention time of up to 240 min in *M. persicae* (Thongmeearkom et al. 1976; Berger et al. 1983; Kannan et al. 2018). The efficiency of transmission is influenced by several factors, including aphid species and biotype, virus concentration in the plant, age of the virus source, and virus strain (Tu & Ford 1971).

MDMV is also transmitted via infected seed, though at a low rate (Chauhan 1985). Seed transmission rates range 0.007–0.400% in maize (Shepherd & Holdeman 1965; Williams et al. 1968; Hill et al. 1974; Mickel et al. 1984). However, much higher rates, ranging 1.1–6.5%, depending on the maize variety, were recorded in Poland (Jeżewska & Trzmiel 2009). The primary pathway for seed transmission appeared to be through female rather than male gametophytes, as MDMV is found in unfertilised kernels, silk, glumes, and anthers but not in pollen grains or mature embryos (Mickel et al. 1984; Li et al. 2007; Kannan et al. 2018).

Although seed transmission occurs at a low frequency, it plays a crucial role in the epidemiology of MDMV, particularly in combination with insect-mediated transmission. Long-distance virus spread is facilitated by virus-carrying aphids transported by wind over 300–1 500 m (Parry 2013). Consequently, integrated cereal aphid management requires an area-wide approach rather than field-based control alone. For this purpose, understanding and predicting aphid movements at different spatial scales is vital.

Economic impact. The economic damage caused by MDMV, which can reach up to 70% or even 90% in susceptible maize hybrids, is mainly related to the reduction in the photosynthesis rate and the increase in respiration, resulting in direct yield losses. Symptoms such as stunting, lower ear size, and poor grain filling contribute to lower yield quality (Kannan et al. 2018; Rai et al. 2023; Jones & Ohlson 2024). Severe infections can lead to partial or complete sterility. The yield losses vary and significantly depend on the genotype and the time of infection. Even in MDMV-resistant hybrids, where plants may remain asymptomatic or display only mild mosaic symptoms, yield losses of approximately 5% have been reported (Jones & Ohlson 2024). The virus is particularly damaging in mixed infections with other plant viruses, intensifying symptoms and yield losses (Mickel 1984).

CONCLUSION

The transmission of plant viruses through seed is a highly complex biological process that integrates host plant physiology, development, and virus replication mechanisms, many of which remain understood. Despite this complexity, seed transmission plays a crucial role in virus epidemiology, serving as a primary inoculum source and contributing to plant viruses' long-term persistence and spread.

In Ukraine, three of the five cereal viruses known to be seed-transmitted – wheat streak mosaic virus, barley stripe mosaic virus, and maize dwarf mosaic virus – have been documented since the 1970s. In contrast, the other seed-transmitted pathogens, High Plains wheat mosaic virus and sugarcane mosaic virus were detected only after 2015. It is likely

https://doi.org/10.17221/51/2025-PPS

that seed transmission, along with the intensive exchange of seed material, has contributed to the spread of these viruses across the country.

Though prevalence varies, the challenges of Ukraine with seed-transmitted viruses are mirrored in neighboring cereal-producing countries (e.g., Poland, Slovakia, Hungary, Romania, Moldova). BSMV, with seed transmission rates up to 50% in Ukrainian barley fields, is reported in Poland, Slovakia, Romania, and Hungary, with a likely presence in Moldova due to seed trade. MDMV and SCMV are documented in Poland, Romania, and Hungary and likely occur in Moldova due to its maize-centric agriculture. Specific studies on MDMV and SCMV are lacking in Slovakia, but its maize production and seed import from Ukraine suggest potential risks. The rise of maize as a key crop in these countries, combined with aphid vectors and seed exchange, likely contributes to the spread of MDMV and SCMV. In contrast, major producers like the United States and China report higher SCMV incidence due to intensive maize monoculture, mitigated in the US by diversified rotations and transgenic resistant varieties, which Ukraine and its neighbours could emulate.

The potential introduction of HPWMoV to the European Union is a significant concern. Despite this virus being only documented in Europe in Ukraine, its vector, the wheat curl mite, is widespread in the EU, where cultivated and wild hosts are abundant. Seed trade, particularly maize and wheat seed, represents a key entry pathway for HPWMoV, posing a high risk of its establishment and economic impact on EU cereal production. Robust phytosanitary measures, including seed testing and quarantine protocols, are essential to prevent its spread.

Understanding virus transmission through seed is essential for developing integrated disease management strategies to minimise virus-induced yield losses. The primary focus of control methods should be preventing infected seeds from entering the field. This can be achieved through early detection and removal of contaminated seed using chemical or physical seed disinfection techniques, in line with the development of advanced molecular diagnostics tools (PCR, ELISA, high-throughput sequencing) to enable early and precise virus detection in the seed. Implementing seed certification programs and quarantine regulations is crucial to prevent the introduction and spread of seed-transmitted viruses into new regions. Ukraine is actively working to strengthen its virological research. It is investing efforts to enhance its role in regional seed certification programs by collaborating with EU neighbours to harmonise phytosanitary standards and with non-EU countries to improve seed testing. Such cooperation can mitigate the risk of seed-transmitted viruses and enhance the protection of cereal crops across Eastern Europe and beyond.

Conflict of interest. The authors declare that they have no conflict of interest.

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Received: March 29, 2025
Accepted: May 26, 2025