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Review paper

Current status, potential threat and challenges of *Turnip Mosaic Virus* (TUMV) management on Brassica crops

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Abstract

Turnip Mosaic Virus (TuMV) is the widest spread viral disease causing 30-90% yield losses in oilseeds *Brassica* crops at about 35 countries in the world especially when it is associated with *Cauliflower Mosaic Virus* (CaMV), and *Beet Western Yellows Virus* (BWYV). Its origin is from a virus of wild orchids, which acquired the pathogenicity on *Allium* spp. and then through wild *Brassica* plants became pathogenic to cultivated *Brassica* and *Raphanus*. The TuMV is a member of genus Potyvirus in the family potyviride has flexcious filamentous particles 135 Å wide with a model length of 729 nm, containing a single copy of a single stranded positive sense RNA (+ssRNA) genome. Virions are 720 x 15-20nm, flexcious rods and are composed of 95 % coat protein (CP) and 5 % RNA. Under field conditions, it is transmitted by more than 89 species of aphids in non-persistent transmission mode, however, mainly by *Myzus persicae* and *Brevicoryne brassicae*. In general symptoms of TuMV infection are vein clearing, chlorotic mottling, leaf distortion, mosaic, necrosis, and plant stunting and in severe cases host death. Symptom variation in different *Brassica* species may be observed influenced by environmental conditions, virus strains, aphid vector activity, host genotypes, crop growth stage, and association of other viruses. Its host range is very wide infecting more than 318 species of 156 genera in dicots and monocots including several field crops, ornamentals and weeds. Pathogenic variability in TuMV has been recorded from more than 20 counties in the form of strains/pathotypes and phylogenetic groups infecting different hosts. The molecular mechanisms of host infection and pathogenesis have been observed through identification of effectors and determinant genes during host-virus interactions. The effectors alter the host metabolism to suit viral replication to increase its capability to become more virulent for increased cell infection and pathogenesis. Host resistance to TuMV in *Brassica* crops governed by both qualitative and quantitative genes. In *B. rapa* (A) genome, 15 dominants and 6 recessive genes have been mapped to provide resistance to different isolates/pathotypes of TuMV. Five dominant genes and QTLs have been mapped in A and C genome of *B. napus*. One dominant and 3 recessive genes have been mapped in A genome of *B. juncea* lines. In *B. oleracea* (C) genome, one dominant gene TuRBO2 has been mapped to provide broad-spectrum resistance to TuMV isolates. The *Raphanus sativus* cv. Daikan has resistance to pathotypes 1 and 8, while cv. Sparkler has extreme resistance to pathotype 1, 7, 8 of TuMV. In *Arabidopsis thaliana* (A) genome, 5 dominant genes and 2 recessive genes have been mapped in different ecotypes to provide resistance to TuMV isolates/pathotypes. During *Arabidopsis*-TuMV interaction, upregulation of GSTs as well as cellular and apoplastic GGT with GR activities limits TuMV replication to exhibit resistance. It is difficult to control TuMV because of its wide host range as reservoirs of inoculum, high variability, numerous insect vectors, and development of resistance to insecticides in aphid vectors to make them ineffective. Use of host resistant cultivars is the most effective management method. The use of integrated approaches with precautionary measures to prevent introduction and spread of virus through early warning system for virus incidence can help in effective management TuMV.

Keywords: *Brassica crops, management, Turnip Mosaic Virus, transmission vectors*

Introduction

The *Turnip Mosaic Virus* (TuMV) is the first virus disease discovered on *Brassica rapa* in 1921 in the USA (Gardner and Kendrick, 1921; Schultz, 1921) out of more than thirteen viral diseases known to occur on crucifer's host species. However, typical symptoms of flower breaking in annual stock (*Matthiola incana*) by TuMV infections were described in France in 1862 (Tompkins, 1939). In a survey of virus diseases of vegetable crops made in 28 countries, TuMV has been ranked at second position after *Cucumber Mosaic Virus* (Tomlinson, 1987). It also infects several non-*Brassica* crops, ornamentals, and several weeds including model plant *Arabidopsis thaliana* widely used for molecular biological studies. Its origin is believed to be from a virus

of wild orchids in the Mediterranean region or Middle East approximately about 1000 years ago and spread via southern Europe to Asia Minor region and adapted to wild cultivated *Brassica* crops. During the last 104 years (1921-2024) after discovery of TuMV, lot of data has been generated on aspects of pathogen, its taxonomy, genome, phylogenetics, serology, transmission, pathogenic variability, infection and pathogenesis, host-pathogen interaction, identification of effectors genes, the disease, its symptomatology, distribution, host range, economic losses, epidemiology, sources of disease resistance, genetics and molecular mechanisms of host resistance, identification of *R*-loci, transfer of resistance, and disease management practices (Tomlinson, 1970; 1987; Shattuck, 1992; Walsh and Jenner, 2002; Nellist *et al.,* 2022). The historical events in the discovery of TuMV have been given in the tabular form (Table 1). These historical discoveries had been a great source of inspiration for the *Brassica* crops scientists to set the pathways of research through discoveries on TuMV as pathogen, its interactions, and effects on host and device strategies for disease management. The TuMV ability to infect *Arabidopsis* makes an excellent model to study plant virus host-pathosystem to reveal molecular mechanisms of host resistance and viral pathogenesis to breed durable resistant cultivars of *Brassica* crops. The TuMV infection is positively associated with *Cauliflower Mosaic Virus* and *Beet Western Yellows Virus* but it is negatively associated with *Turnip Yellows Mosaic Virus* on crucifers' hosts. The TuMV symptoms on the host are diverse and in general include vein clearing, mosaic, necrosis, plant stunting and host death. Under field conditions, mixed infection of TuMV with other *Brassica* viruses is very common.

The TuMV is a potential threat to production and productivity of *Brassica* crops, since it can cause yield losses in the range of 30-90 per cent under congenial epidemic conditions. Its management is challenging because of wide host range, non-persistent transmission by large number of insect vectors, development of resistance to insecticides in the insect vectors, and evolution of new strains/pathotypes with new virulence by the virus. However, the prospective management strategies to manage this notorious virus should be to transfer *R*-genes through conventional and molecular approaches to breed durable resistant cultivars of *Brassica* crops.

Table 1: Investigations of historical importance on *Turnip Mosaic Virus*

Historical events	Year	References
Discovery of TuMV on *B. rapa* in USA	1921	Gardner and Kendrick (1921)
Transmissible mosaic disease of Chinese cabbage	1921	Schultz (1921)
TuMV causes serious loses in *Brassica* crops	1940	Ling and Yang (1940)
Aphid transmission of non-persistent TuMV	1953	Sylvester (1954)
Purification of TuMV	1960	Shepherd and Pound (1960)
Identification of TuMV strains on the basis of symptoms on *B. oleracea* and *N. glutinosa*	1963	Yoshii (1963)
Identification of broad-spectrum resistance to TuMV in *B. rapa*	1980	Provvidenti (1980)
Radish mosaic- a new virus disease caused by TuMV in India	1984	Ahlawat and Chenulu (1984)
Identification of QTLs in *B. oleracea*	1986	Pink *et al.* (1986)
Genetics of host resistance to TuMV in *B. napus*	1989	Walsh (1989)
The complete nucleotide sequence of TuMV RNA	1992	Nicolas and Laliberte (1992)
Identification of amino acid with aphid-transmissibility of TuMV	1993	Nakashima *et al.* (1993)
Arabidopsis thaliana – TuMV host pathosystem as model for molecular biological studies	1994	Martinez-Herrera *et al.* (1994)
Host gene silencing defense to virus	1997	Ratcliff *et al.* (1997)
Determination of TuMV cDNA clones' infectivity and transcripts on the host *A. thaliana*	1998	Sanchez *et al.* (1998)
Mapping of QTLs (*TuRBO2*) in the C genome of *B. napus*	1999	Walsh *et al.* (1999)
The first TuMV resistant dominant gene *TURBO1* in a line Wester from *B. napus*	1999	Walsh *et al.* (1999)
Identification of serotypes in TuMV	1999	Jenner *et al.* (1999)
Identification of HC-Pro effector to suppress post transcriptional gene silencing	2000	Dalmay *et al.* (2000)

Identification of cylindrical inclusion gene of TuMV as pathogenic determinant	2000	Jenner *et al.* (2000b)
Identification of P3 and C1 as avirulence determinants	2002	Jenner *et al.* (2002a)
Ecology of TuMV in wild *Brassica* species	2003	Raybould *et al.* (2003)
Use of TuMV as biosafe viral vector	2008	Tourino *et al.* (2008)
Phylogenetic relationship of TuMV	2010	Gibbs and Ohshima (2010)
Ability of TuMV to use alleles from *B. rapa* for translation	2010	Jenner *et al.* (2010)
TuMV moves systemically through vascular tissues	2015	Wan *et al.* (2015)
Structure of TuMV	2019	Cuesta *et al.* (2019)
Cell to cell movement of TuMV via plasmodesmata	2021	Wang (2021)
Two TuMV strain from India shared identity with World B- pathotypes and sub-pathotype world B3	2022	Singhal *et al.* (2022)

The TuMV as a pathogen

The TuMV as a pathogen is very important infecting broad range of dicotyledonous and monocotyledonous plants and transmitted by >89 aphid species in a non-persistent manner to develop in epidemic form in a very short period after infection. It is a member of the genus Potyvirus (Type species Potato Virus Y) in the family potyviride. It is only single potyvirus which infects *Brassica* crops all over the globe. Amongst the viral pathogens of *Brassica*, TuMV has been studied extensively. The TuMV has flexuous filamentous particles 135 Å wide with a model length of 720 nm, containing a single copy of a single stranded positive sense RNA (+ssRNA) genome. Virions are 720 x 15-20 nm flexuous rods and are composed of 95 per cent coat protein (CP) and 5 per cent RNA. Under field conditions TuMV is transmitted by aphids as vector but it is also readily sap transmitted mechanically to use in research investigation by the TuMV scientists (Edwardson and Christie, 1986; Walsh and Jenner, 2002; Nellist *et al.,* 2022). It is believed that TuMV probably first acquired the pathogenicity on *Allium* plants and then through wild *Brassica* plants became pathogenic to cultivated *Brassica* and *Raphanus*. The TuMV infection symptoms depend upon the virus strains, host plant species, aphid vector species and environmental conditions and their interaction time. The symptoms of TuMV infection under field condition may be severe and confusing when plants are simultaneously infected with other viruses like mixed infection of TuMV with *Cucumber Mosaic* or/and *Tomato Virus*; TuMV with CaMV, *Broccoli Necrotic Yellows Virus* and *Beet Western Yellows Virus.* From India, the biological, serological and coat protein properties of potyviride causing mosaic disease of crucifers have been investigated (Chiu and Chang, 1982; Walsh and Tomlinson, 1985; Hardwick *et al.*, 1994; Jenner and Walsh, 1996; Haq *et al.,* 1994).

Transmission of TuMV

The virus can be transmitted readily by mechanical sap inoculation but not by *Cuscuta* species. In general, its thermal inactivation point is below 62^{O}C, the dilution end point in sap is between 10^{-3} and 10^{-4} and infectivity is retained at 20^{O}C for 3-4 days. Infective sap kept at 2^{O}C retains infectivity for several months. Seed transmission is host and isolate specific. TuMV isolate 12 is transmitted through *Brassica* seeds up to 14 per cent. TuMV is mainly transmitted by aphids (> 89 species) under field conditions from infected plants to healthy plants. The generalist aphis *Myzus persicae* and specialist's aphid *Brevicoryne brassicae* with all instars can transmit the virus. It can be acquired in less than one minute and transmitted in less than one minute. There is no latent period and it is retained in some vectors for less than 4 hrs. In Australia, TuMV is transmitted non-persistently by the aphid species mainly *Myzus persicae*, *B. brassicae* and *L. pseudobrassicae* who colonize *Brassicae* hosts. Many other aphid species which do not colonize *Brassica* species are also potential vectors. Under Asian conditions, the specialist aphid *Lipaphis erysimi* is an important vector. The virus strain from India have been reported to be transmitted in a non-persistent manner by *Myzus persicae, Brevicoryne brassicae* and *Aphis gossypii.* The protein ORF, HC-Pro functions as helper component (HC) for aphid transmission and protease activity. Critical amino acids for aphid transmissibility of the virus have been identified in this protein. This protein acts as multimer to aid binding of viral coat protein to the aphid stylet A lysine motif (KITC) located within the N-terminal cysteine –rich domain of HC-Pro along with another HC-Pro motif named PTK is essential for aphid transmission. Flea battles (*Phyllotreta* spp.) and thrips (*Thrips angusticeps*) can also transmit the virus. (Tompkins, 1939; Sylvester, 1953, 1954; Kennedy *et al.,* 1962; Edwardson and Christie, 1986; Haq *et al.*, 1994; Wang and Pirone, 1999; Nellist *et al.,* 2022).

TuMV genome and particle structure

The genome of TuMV isolate UK 1 consists of +ssRNA molecules 9830-9833 nucleotide in length. Most of the isolates have polyprotein coding regions of 9492

nucleotides. Between the isolates genome sequence nucleotide identifies are >76 per cent. The 5' terminus of genome RNA is capped with single covalently attached molecules of the genome linked viral protein (VPg). The 3'-terminus consists of a poly A tail of variable length similar to potyviruses. The regions encode the protein (P1), helper- component proteinase protein (HC-Pro), protein 3 (P3), potyviride ORF (PIPO), 6 KDa 1 protein, cylindrical inclusion (C1) protein, 6 KDa 2 protein, VPg nuclear inclusion a–proteinase protein (Nia-Pro), nuclear inclusion of b protein (Nib), and coat protein (CP) with length of 1086, 1374, 1065, 177, 156, 1932-1935, 159, 573- 576, 729, 1551 and 864-867 nucleotides respectively. The structural organization of TuMV isolates is similar to other potyviruses known so far along with all motifs. More than 80 per cent TuMV isolates are recombinants. On the basis of polyprotein-coding sequences of non-recombinant isolates, they have been partitioned into six major phylogenetic groups as Asian BR, basal B, basal BR, Iranian, orchis and world B (Basso *et al.*, 1994; Sanchez *et al.*, 1998; Tan *et al.,* 2004; Ohshima *et al.,* 2007; Kawakubo *et al.,* 2021; Nellist *et al.,* 2022). The complete genome sequences of two TuMV world-B3 strains infecting yellow and black mustard in India were investing through high - throughput RNA sequencing subjecting ribosomal RNA depleted mRNA revealed that viral genomes of the two isolates were 9817 and 9829 nucleotides long. They featured two open reading frames (ORFs), one of which encoded a polyprotein comprised of 3164 amino acids and the other of which encoded a PIPO protein of 62 amino acids (Singhal *et al.,* 2022).

The particle structure of TuMV has been viewed through cryo-electron microscopy at a resolution of 5 Å. The empty structure of virus-like particles has also been resolved. The virions are non- membranous, elongated and flexuous, 135 Å wide with a model length of 720nm. Virions display a left–handed helical arrangement of > 2000 copies of CP, which enclose a single +ss RNA molecules. Regions of the filamentous stretch and shrink with an aptitude of around 2 Å per turn. The wall of the flexuous tube is made up of core domains of the capsomers and central regions of the CPs. In the boundary between CP sub-units there is a network of protein-RNA and protein-protein interactions that supports the proper orientation of the flexible N-terminal arm. The participation of flexible N- and C terminal arms in the interaction between Cp subunits is the structural basis for the flexible nature of the virions. The N-terminal domain of each capsomers is projected towards the exterior of the tube, whereas the C- terminal domain is internally aligned with vertical axis. The N-terminal arm of each TuMV CP interacts with another two subunits. After a 90^{O} turn, the N-terminal arm reacts to other subunits in the next turn of the helix. The ssRNA resides in a groove at the folded central domain, just next to the last helix. The two ends of the flexuous particles are not identical. One of the them holds the S' end of the viral RNA, which is covalently linked to a viral protein VPg. This tip presents a protruding structure associated with VPg and HC-Pro. TuMV particles are made up of protein RNA, sugars, and phosphates. The studies conducted in India reveals that the Average size of the virus particles in a purified preparation was 740 nm × 12 nm. The SDS-PAGE analysis of the viral coat protein showed two major bands of approximately 37 kDa and 31 kDa, a pattern very similar to that of a reference isolate of turnip mosaic virus (TuMV) from the USA. In Western-blot immunoassay assay, an antiserum to TuMV reacted with both the coat protein bands of the Poty-Rape isolate and the TuMV, but not with the coat proteins of four other potyviruses. The high-performance liquid chromato-graphic profile of tryptic peptides from the coat protein of Poty-Rape was found to be very similar to that of the reference TuMV, but differed substantially from those of four other potyviruses as reported from other parts of the world. So, the Poty-Rape isolate is considered to be a distinct strain of TuMV in India (Haq *et al.,* 1994; Sanchez *et al.,* 1998; Torrance *et al.,* 2005; Cuesta *et al.,* 2019; Kawakubo *et al.,* 2021; Nellist *et al.,* 2022).

The disease and symptomatology

The disease caused by TuMV are known by the association of virus name with group of crops/hosts by prefix TuMV as *Turnip Mosaic Virus* of oilseed crops (group of crops) and TuMV of *Brassica napus* (host). The symptoms produced by TuMV infection may vary depending on host plant virus strain and environmental conditions prevalent at the time of interaction with growth stage of the host. In general symptoms include vein clearing, chlorotic mottling, leaf distortion, mosaic, necrosis, plant stunting and host death. In *Brassica* crops symptoms appear at the seedling stage in the form of chlorotic spots, leaf mottling followed by vein clearing, mosaic, necrosis, leaf distortion, and stunting. Symptoms expression in *Brassica* crops are greatly affected by the TuMV strain and temperature at the initial stages of the host virus interaction. In *B. napus* genotypes some isolates of virus develop progressive necrosis of leaves, petioles and stem leading to host death. Dry spots and mosaic at seedling stage. Siliquae of *Brassica* crops at severe infection stage are reduced in size and number, some are malformed without seed. The seed size is reduced with poor yield of diseased plants. Seed viability is also affected. The temperature influences type of symptoms on the host. In *B. oleracea* plants mottle symptoms are more pronounced at 28^{O}C than plants grown at 16^{O}C. The cytoplasm of diseased leaf epidermal, mesophyll and phloem cells contain

cylindrical cytoplasmic inclusions consisting of pin wheels, bundles, scrolls and laminated aggregates. Mosaic and shrinking at seedling stage. In India, infection on yellow and black mustard exhibit leaves puckering and mosaic like symptoms with 100 percent severity (Pound and Walker, 1945; Walsh and Tomlinson, 1985; Edwardson and Christie, 1986; Sanchez *et al.,* 2015; Nellist *et al.,* 2022). Characteristic symptoms on different hosts by TuMV strains are given in Table 2. It shows how virus/ strain express itself on different hosts under the influence of host-environment interaction at different growth stages of *Brassica* hosts and aphid vector activity. Under field conditions, sometimes simultaneous infection of more than one virus my cause severe and confusing symptoms. TuMV infection often occurs mixed with *Cucumber Mosaic* and *Tobacco Virus*. The mixed infection of TuMV with CaMV, *Broccoli Necrotic Yellows Virus* and *Beet Western Yellows Virus* has also been observed (Chiu and Chang, 1982; Walsh and Tomlinson, 1985; Hardwick *et al.,* 1994; Jenner and Walsh, 1996; Singhal *et al.,* 2022).

Table 2: Characteristic symptoms of *Turnip Mosaic Virus*/ strain specific infection on different host species

Host species	Characteristic symptoms	References
Arabidopsis thaliana	Stunting, deeply serrated leaves, flower with narrow sepals, split carpel's, aborted anthers and sterile plants. Flower stalk elongation cell wall alterations	Kasschau *et al.* (2001) Sanchez *et al*. (2015); Lopez-Gonzalez *et al.* (2020
Brassica juncea	Veins clearing near leaf base, few or no flower production. Siliquae poorly filled and shriveled	Ling and Yang (1940)
B. napus	Dry spots and mosaic at seedling stage. Leaf necrosis, host death	Nellist *et al.* (2022)
B. napus	Strip spots, wheel spots and dot spots at plant stage. Mosaic, leaf distortion, stunting, net brown necrosis in the leaves.	Walsh and Tomlinson (1985); Walsh and Jenner (2002)
B. oleracea	Mottling, black necrotic spots, ring spots	Hunter *et al.* (2002)
B. oleracea	Internal necrotic spots	Nellist *et al.* (2022)
B. rapa ssp. *perviridis*	Leaf distortion, leaf mosaic	Nellist *et al.* (2022)
B. rapa	Leaves puckering and mosaic	Singhal *et al.* (2022)
B. nigra	Leaves puckering and mosaic	Singhal *et al.* (2022)
Erysimum sp.	Flower breaking	Tompkins (1939)
Matthiola incana	R. Br.Flower breaking	Tompkins (1939)

Host range

The TuMV has very wide host range of cultivated crops and weeds. It infects more than 318 species of 156 genera in dicots including Crucifereae, Compositae, Chenopodiaceae, Leguminaceae and Caryophyllaceae with large number of monocots in families Amaryllidaceae, Araceae, Commelinaceae, Iridaceae, Liliaceae, Musaceae and Orchidaceae. The virus strain in India readily infected 4 of the 5 plant species in the family *Brassicaceae* in which it induced severe systemic mosaic symptoms. It also causes chlorotic and necrotic local lesions in *Chenopodium amaranticolor*, but failed to infect 4 other species of *Chenopodiaceae*. However, the virus infects 20 other plant species belonging to the family of Amaranthaceae, Apiaceae, Canabinaceae, Compositae, Cucurbitaceae, Euphorbiaceae, Leguminosae and Solanaceae. The TuMV is most damaging to cruciferous crops causing yield losses of up to 70 per cent in several countries all over the globe. These crucifers include all species of *Brassica napus, B. rapa, B. oleracea, B. juncea, Eruca sativa, B. carinata, B. nigra, Raphanus sativus* including a weed model host plant *Arabidopsis thaliana*. Other important field crops infected by TuMV are pea, chickpea, and coriander (Edwardson and Christie, 1991; Haq *et al.,* 1994; Li *et al.,* 2018; Palukaitis and Kim, 2021).

Geographical distribution

The virus is widely distributed in the areas where *Brassica* crops including Oilseeds and vegetables are grown all over the world. It is endemic in temperate and tropical regions wherever these crops are grown. Although TuMV was reported long back in 1862 in France but on *Brassica* crops, it was first reported in USA in 1921 followed by UK in 1935. The center of origin of TuMV is believed to be Mediterranean and Middle East countries then spread to other countries of the world. The virus is severe and most damaging in the countries like Australia, Belgium, Brazil, Canada, China, Czeck- Republic, Denmark, Europe, France, Germany, Greece, India, Iran, Italy, Japan, Kenya, Myanmar, Netherlands, New Zealand; Poland, Portugal,

Russia, Serbia, Spain, South Korea, Taiwan, Thailand, Turkey, Ukraine, UK, USA, Uzbekistan, and Vietnam. Apart from these countries TuMV occurs in mild to severe form on *Brassica* crops in several regions of the countries from Europe, Asia, Africa and New world (Tomitaka and Ohshima, 2006; Korkmaz *et al.,* 2008; Farzadfar *et al.,* 2009; Nguyen *et al.,* 2013a; Yasaka *et al.,* 2015; Nguyen 2021; Kawakubo *et al.,* 2021, Nellist *et al.,* 2022). In-spite of first report of TuMV on *Brassica* crops in nineteenth century; it was reported quite late from many countries of the world (Table 3). It seems that either the virus is of minor effect or there is lack of resources like finance and technical men power from where the occurrence of TuMV is awaited.

Yield losses

Amongst viral diseases of *Brassica* crops, TuMV disease is the most damaging disease on *Brassica* oilseeds and vegetable crops. Its infection at early growth stage of the crops has adverse effect on host crop growth and production. The severely infected plants are stunted in growth with a smaller number of siliquae, poorly filled, twisted and have shriveled seeds. Such plants produced a smaller number of branches. Seeds of severely infected siliquae are smaller in size and have reduced viability and oil contents. Yield losses from severely infected *B. napus* crop have been recorded from 30-90 per cent from different countries of the world. Yield losses of 50 per cent has been recorded from Kenya in *B. oleracea* var *capitata* (Table 4). The TuMV in association with BWYV produces internal necrotic spot in white cabbage and reduce the yield and quality of crops. The TuMV induces major development traits and flower stalk elongation in model host plant *Arabidopsis* (Shattuck and Stobbs, 1987; Hardwick *et al.,* 1994; Hunter *et al.,* 2002; Spence *et al.,* 2007; Sanchez *et al.,* 2015; Milosevic *et al.,* 2015).

Table 3: First report of *Turnip Mosaic Virus* on *Brassica*

Host species	Year	Country	References
Brassica rapa	1921	USA	Gardner and Kendrik (1921); Schultz (1921)
Brassica oleracea	1935	UK	Smith (1935)
Brassica napus	1940	China	Ling and Yang (1940)
Brassica oleracea	1959	Australia	Conroy (1959)
Brassica napus	2002	Iran	Shahraeen *et al.* (2002)
Brassica spp.	2003	Iran	Shahraeen *et al.* (2003)
Brassica spp.	2004	Spain	Moreno *et al.* (2004)
Brassica spp.	2007	Turkey	Korkmaz *et al.* (2007)
Brassica spp.	2015	India	Singh *et al.* (2015, 2018)
Brassica rapa	2015	India	Singh *et al.* (2015, 2018)
Brassica spp.	2016	Serbia	Milosevic *et al.* (2015)
Brassica spp.	2018	Ukraine	Shevchenko *et al.* (2018)
Brassica juncea	2020	India	Kapoor *et al.* (2020)
Raphanus sativus	1984	India	Ahlawat and Chenulu (1984)

Table 4: Yield losses caused by TuMV in different crops

Yield loss (%)	Crops	Country	References
30	*Brassica napus*	Canada	Shattuck and Stobbs (1987)
70	*Brassica napus*	UK	Hardwick *et al.* (1994)
90	*Brassica napus*	China	Wei *et al.* (1960)
70-79	*Brassica napus*	Serbia	Milosevic *et al.* (2015)
50	*B. oleracea* var. *capitata*	Kenya	Spence *et al.* (2007)
46-84	*B. juncea*	Australia	Jones *et al.* (2021)

TuMV infection and multiplication

The host infection by TuMV takes place by aphid vectors while after feeding on infected host, they visit healthy plants and start probing or feeding. Aphid vectors acquire virus from diseased host and introduce the virus in plant cell via the stylet of aphids in the typical non-persistent transmission mode during aphid feeding and probing. Once in the host cell, the virus particles are uncoated and the genome replication/ multiplication start to produce more numbers of virus particles. The TuMV establish relationship with host cells and tissues similar to (+) RNA viruses. RNA is a messenger RNA of viral genome from which the final functional proteins are auto-proteolytically released after encoding a large polyprotein. The other mechanism involves polymerase

slippage, which is responsible for the production of a mRNA encoding an additional fusion protein, PIPO. To interact with eukaryotic as a factor for translation/ multiplication initiation 6K2 and VPg proteins are covalently linked to the 5' of the viral RNA. The template specificity to prevent the viral multiplication of host mRNAs is believed to present in the sequence of 3' end of the viral RNA. The first protein of the ORF, P1 is very basic and has the ability to bind single stranded nucleic acids (RNA or DNA) and dsRNA. It functions in RNA translation and /or the transport of nucleic acids between cells, perhaps by altering the size exclusion limits of plasmodesmata. Multiplication/replication of TuMV occurs in association with membranous structures present in the cellular endo-membrane system. After TuMV host cell entry, its 6K2 protein is responsible for the membrane proliferation. New viral RNA progeny is encapsulated, which are mostly accumulated in vacuoles (Jenner *et al.,* 2010; Grangeon *et al*., 2012; Jin *et al.,* 2018; Wu *et al.,* 2020).

The movement of the virus after infection takes place by the process of inter and intra-cellularly from the initial infected cells. For this process membranous 6K2-containing replication trait accumulates pre-nuclearly, and the n move intercellularly towards the cell periphery like organelle cytoplasmic streaming. In this complex process numbers of proteins are involved. The C1 protein is crucial in forming inclusions bodies and recognized as hallmarks of potyvirus infected cells. Cell plasmo-desmata are modified to serve as places for simplistic movement, possibly for virions or ribonucleoprotein complexes. The apoplastic movement of virus is in extracellular space. The virus moves systemically through vascular bundles. The TuMV replication complexes are present in both phloem sieve elements and xylem vessels. Photosynthetic sink parts of the infected plants are the first target to reach especially roots. Systemic virus accumulation is strongly influenced by plant growth period like bud formation for inflorescence during which no increased accumulation in systemically invaded leaves with negligible viral particles in the roots (Lunello *et al.,* 2007; Wan *et al.,* 2015; Movahed *et al.,* 2017, 2019 a, b; Lopez-Gonzalez *et al.,* 2020; Wang, 2021).

Pathogenic variability of TuMV

The pathogenic variability in TuMV has been observed in the form of strains and pathotypes along with their phylogenetic groups differentially infecting crucifers and other hosts from more than 20 countries all over the world (Table 5). The variability in TuMV may arise through point mutation and recombination. RNA polymerases lack 3'-5' exonucleolytic proof reading activity, and mismatch repair cannot occur on single stranded progeny genomes. As result, there is high rate of mis-incorporation error typically 0.1 -10 mutations for 10Kb molecules per replication cycle. In addition, replication slippage in the 5' region of the TuMV genome has been observed. The rapid evolution ability of TuMV makes it more virulent to infect wide range of host species. The strains and pathotypes of TuMV have been designated based on various schemes infecting specific host species, varieties, or cultivars. The first scheme was used by Yoshii (1963) to identify two strains on the basis of symptoms type on cabbage and *Nicotiana glutinosa*. Seven strains as pathotypes Tu1 to Tu7 were identified on the basis of symptoms types and disease severity index on *Brassica* spp. by Liu *et al.,* (1990). Differentials lines of *B. rapa* were used to distinguish 4, 5 and 6 strains by Provvidenti (1980), Green and Deng (1985) and Stobbs and Shattuck (1989) respectively. The TuMV strains were designated as pathotypes 1-12 on the basis of four *B. napus* differentials by Jenner and Walsh (1996). The pathotypes 1,3, and 4 are most predominate. Later on, Walsh and Jenner (2002) characterized resistance genes in these differentials along with virulence effectors of the virus. Four host specific isolates were identified as i- 'B' isolate which do not infect *Raphanus* but infect *Brassica* species with systemic mosaic symptoms, ii- ('B') isolates that do not infect *Raphanus* but sporadically infect (often latently) *Brassica* plants. iii- B (R) isolates infect *Brassica* species with systemic mosaic symptoms but occasionally infect *Raphanus* plants latently and iv- 'BR' isolates that cause systemic mosaic in both *Brassica* and *Raphanus* plants (Ohshima *et al.,* 2002; Tomimura *et al.,* 2003; Nguyen *et al.,* 2013b). Provvidenti (1986) identified strain CL which is unable to infect *B. rapa* cv. Tropical delight. European pathotype 1 is unable to infect *B. rapa* line R4 (Jenner and Walsh, 1996).

In Australia, TuMV isolates/pathotypes *viz*., AU1/8, NSW 1/7, NSW 2/1, 7; WA-AP1/8; 12.1, 12.5/-; AUST 19/-; AUST 23/- have been identified infecting *Brassica* cultivars differentially. Isolates 12.1 and 12.5 were identified as most virulent on *Brassica* cultivars. The sequences of Australian isolates of TuMV were in phylogenetic groups I and II of world B, II of Basal -BR, and IV of Basal B. The most virulent isolates 12-1 and 12-5 (resistance breaking) were in separate groups II of World-B than other two isolates, AUST 19 and AUST 23 (Yasaka, 2017; Nyalugwe *et al.,* 2015b; Guerret *et al.,* 2017).

Isolates of TuMV have also been identified as genetic clusters in four groups by Sanchez *et al*. (2003), and Guerret *et al.* (2017) *viz*., i- *Brassica* isolates on analysis of coat protein gene as MB genetic clusters) ii- Radish isolates on analysis of coat protein gene as MR genetic clusters; iii- Intermediate between *Brassica* and Radish as IBR genetic clusters and iv- Outside *Brassica* and

Radish clusters as OBR genetic clusters. Other isolates group include i- Orchis group from Germany consisting of sister lineage of orchis viruses (Nguyen *et al.,* 2013b; ii- Iranian group on the basis of time scale of emergence and spread of TuMV (Yasaka *et al.,* 2017), and iii-MR and JPN1 serotype on the basis of correlation between genetic cluster and serotype (Sanchez *et al.,* 2003; Table 6). After analysis of 41 isolates from different hosts and geographical origins with a panel of 30 MAbs, three groups of isolates (Serotypes) were identified (Jenner *et al.,* 1999). Isolates of TuMV identified from different sources have been placed equivalent to pathotypes and phylogenetic groups (Table 5).

Table 5: Identification of isolates/pathotypes of *Turnip Mosaic Virus* from different countries

Country	Isolates/Pathotypes	Phylogenetic group	References
Australia	AU1/8, NSW 1/7, NSW 2/1, 7, WA-AP1/8, 12.1, 12.5, AUST 19/-, AUST 23/-	World B	Walsh and Tomlinson (1985); Ohshima *et al.* (2002); Guerret *et al.* (2017)
Belgium	BEL1/7	OBR	Sanchez *et al.* (2003)
Canada	CDN1/4	World B	Tomimura *et al.* (2003, 2004);
	CDN2 (aka Q-) Ca/3		Wang *et al.* (2009a,b)
China	CHN1/1	IBR	Sanchez *et al.* (2003); Ohshima *et al.* (2002);
	CHN 2, 3, 4, 5, 12/3	World B	Tomimura *et al.* (2004)
Czech Republic	CZE1/3	World B	Ohshima *et al.* (2002)
	CZE 5, 18/5,4	World B	Tomimura *et al.* (2004)
Denmark	DNK 2,3/5; DNK4/3	World B	Tomimura *et al.* (2004)
Europe	NSW 1, NSW 2; WA –Ap/1, 7, 8	Pathotypes 1-12 from European isolates	Jenner and Walsh (1996) Nyalugwe *et al.* (2016); Jenner and Walsh (1996);
France	FRA 2/4	World B	Tomimura *et al.* (2004)
Germany	DEU 1/5	World B	Tomimura *et al.* (2004)
	DEU 2/4	IBR	Sanchez *et al.* (2003)
	DEU 4/1	Basal BR	Tomimura *et al.* (2004)
	DEU 5/4	World B	Tomimura *et al.* (2004)
	FRD 1/1	World B	Tomimura *et al.* (2004)
	PV 376/4	World B	Ohshima *et al.* (2002)
Greece	GK 1/9	Basal B, B2	Ohshima *et al.* (2002); Tomimura *et al.* (2004)
	GRC 2, 6, 12, 17, 18, 31, 32/1	World B	Tomimura *et al.* (2004)
	Sister lineage of orchis	Orchis group	Nguyen *et al.* (2013a, b)
India	Radish isolate	-	Ahlawat and Chenulu (1984); Kapoor *et al.* (2020)
	Identity of world B pathotypes and sub-pathotype of world B3 different from other isolates of Asian BR- type	World- B and world -B3	Singhal *et al.* (2022)
Iran	All isolates	Iranian group	Yasaka *et al.* (2017)
Italy	ITA1/6	Basal- B	Ohshima *et al.* (2002)
	ITA 3/10	Basal B, B2	Tomimura *et al.* (2004); Wang *et al.* (2009a)
	ITA 4/5	Basal- B2	Tomimura *et al.* (2004)
	ITA 5, 6 /3	Basal- B2	Tomimura *et al.* (2004)
	ITA 6/3	OBR	Sanchez *et al.* (2003)
	ITA 7/1	Basal- BR	Ohshima *et al.* (2002); Tomimura *et al.* (2004)
	PV 377/2	OBR	Sanchez *et al.* (2003)

Japan	JPN 1/7	MR	Sanchez *et al.* (2003)
Kenya	KEN 1/1	World -B	Ohshima *et al.* (2002)
Netherlands	NLD 1, 2/1	World -B	Tomimura *et al.* (2004)
Poland	POL 1,2,4/4	World -BT	Omimura *et al.* (2004)
Portugal	PRT 1/1	IBR, World -B	Sanchez *et al.* (2003); Tomimura *et al.* (2004)
United Kingdom	GBR 7/1	World -B	Ohshima *et al.* (2002)
	GBR 8/4	World –B	Tomimura *et al.* (2004)
	UK 1/1	World –B	Tomimura *et al.* (2004)
USA	USA 1/1	World –B	Ohshima *et al.* (2002); Tomimura *et al.* (2004)
	USA 4/5	World -B	Tomimura *et al.* (2004)
Uzbekistan	UZB1/7	Basal -B	Ohshima *et al.* (2002)

Pathotypes and phylogenic group designated by Jenner and Walsh (1996)

Table 6: Schemes /basis of identification of pathotypes/strains of *Turnip Mosaic Virus*

Pathotypes/ strains isolates	Schemes basis of identification	References
Two Strains	Symptom type on cabbage and *Nicotiana glutinosa*	Yoshii (1963)
7 strains (Tu 1-7)	Symptom types and disease severity indexes on *Brassica* species	Liu *et al.* (1990)
4 Strains (1-4)	Differential lines of *B. rapa*	Provvidenti (1980)
5 Strains (1-5)	Differential lines of *B. rapa*	Green and Deng (1985)
6 Strains (1-6)	Differential lines of *B. rapa*	Stobbs and Shattuck (1989)
12 pathotypes (1-12)4	Differential lines of *B. napus*	Jenner and Walsh (1996)
Host specificity types 'B'	IsolateIsolate infect *Brassica* but do not infect *Raphanus*	Ohshima *et al.* (2002)
Isolates '(B)'	Isolates do not infect *Raphanus* but infect latently *Brassica* plants)	Nguyen *et al.* (2013b)
Host type 'B (R)'	Isolate cause systemic mosaic symptom in *Brassica* species and latent infection in *Raphanus*	Tomimura *et al.* (2003)
Host type 'BR' isolates	Isolate cause systemic mosaic symptoms in *Brassica* and *Raphanus*.	Tomimura *et al.* (2003)
Strain CL	Unable to infect *B. rapa* cv. Tropical delight	Provvidenti (1980)
European Pathotype 1	Unable to infect *B. rapa* line R4	Jenner and Walsh (1996)
MB genetic cluster	*Brassica* isolates on analysis of coat protein gene	Sanchez *et al.* (2003)
MR genetic cluster	Radish isolates on analysis of coat protein gene	Sanchez *et al.* (2003)
IBR genetic cluster	Intermediate between *Brassica* and radish clusters	Sanchez *et al.* (2003)
OBR genetic cluster	Outside *Brassica* and radish clusters	Sanchez *et al.* (2003)
Orchis group from Germany	Sister lineage of Orchis viruses	Nguyen *et al.* (2013a)
Iranian group	Time scale of emergence and spread of TuMV	Yasaka *et al.* (2017)
MR and JPN1 serotype	Correlation between genetic clusters and serotype	Sanchez *et al.* (2003)
Predominant serotype (30 isolates)	From *Brassica*/Europe/new World group based on CP amino acid homologies	Lehmann *et al.* (1997)
JPN1 serotype	Isolates from the radish and Asian group	Lehmann *et al.* (1997)

Phylogenetic groups

The TuMV is phylogenetically related to poty-viruses' group being a member of lineage of potyviruses. In the potyvirus group more than 11 major phylogenetic groups are known including the TuMV group. Other than crucifer's viruses, some sweet potato potyviruses are more closely related potyviruses to the TuMV group. The phylogenetic groups of TuMV consist of *Japanese Yam Mosaic Virus, Narcissus Late Season Yellow*

Viruses, Narcissus Yellow Strip Virus, Scallion Mosaic Virus, Wild Onion Symptomless Virus and TuMV. Apart from TuMV, all the species in the TuMV phylogenetic group have been recorded from Monocots host plants including one isolate of wild Orchid, which are biologically and phylogenetically different from *Brassica* isolates. The major phylogenetic groups of TuMV are basal B (*Brassica*), Iranian basal – BR (*Brassica/Raphanus*), Asian- BR and World B groups. Some groups have been splitted into sub-groups like basal- B into – B1 and B2, Iranian group into Iranian 1 and Iranian 2, and the world B group into World B1, B2 and B3 sub-groups. Studies conducted in India reveals that two TuMV strains shared identity with the world-B pathotype and sub-pathotype world B3 which is its emergence first time in South Asia. This study indicates that other isolates reported previously from South Asia having Asian-BR pathotypes, and present report indicates that these are differ in their phylogeny. It indicates that it is first instance of TuMV association with black mustard naturally. Their geographical prevalence justifies a lower degree of genetic differentiation and higher rate of gene flow calculated between the World-B and Asian-BR pathotypes. This study provides insights knowledge on population structuring, expansions and evolution, level of genetic heterogeneity and variability of worldwide available isolates of TuMV (Gibbs and Ohshima, 2010; Nguyen *et al.,* 2013a, b; Yasaka *et al.,* 2015, 2017; Ohshima *et al.,* 2018; Gibbs *et al.,* 2020; Kawakubo *et al.,* 2021; Nellist *et al.,* 2022; Singhal *et al.,* 2022).

Epidemiology

The epidemic development of TuMV on *Brassica* crops is governed by several ecological factors. The major factors include the i). Wild *Brassica* populations in the near vicinity; ii). The TuMV association with other viruses during infection; iii). Age of the host plant at the time of infection, iv). The TuMV isolates/pathotypes involved in infection, v). Level of vulnerability in the *Brassica* population in the areas around vi). Evolution of TuMV isolates/pathotypes, vii). Initial source of virus inoculum, viii). Crop growth stage at primary infection, ix). Distance of primary source of inoculum, x). Influence of weather conditions (Temp. and rainfall on vector population, and xi). Species and population of aphids involved in virus transmission. The variations in the TuMV incidence have been recorded on wild *B. oleracea* populations in UK. There is a positive association of TuMV with *Cauliflower Mosaic Virus* and *Turnip Yellows Virus* but a negative association with the *Turnip Yellow Mosaic Virus*. The incidence of TuMV is higher in younger plants than older plants. The TuMV infection and severity is higher in wild *B. oleracea* plants causing mortality and reduced seed yield. With such plants, TuMV isolates belonging to pathotypes 1 have been observed. In Iran, high incidence of TuMV has been recorded on wild populations of *Rapistrum rugosum* and *Sisymberium loeselii*. The population of aphid vector, *M. persicae* and *L. erysimi* difference in non-infected and infected host plants. It is believed that introgression of virus resistance with major genes or a transgenic from a crop from increases host fitness in the natural population of *B. oleracea*. Serial passage of two TuMV isolates in arrange of less and more tolerant genotypes/ecotypes of *Arabidopsis* indicated that more tolerant genotypes promoted virus multiplication and reduced the effect of infection on plant mortality but not on plant fecundity (Jenner and Walsh, 1996; Walsh and Jenner, 2002; Raybould *et al.,* 2003; Farzadfar *et al.,* 2009; Adachi *et al.,* 2018; Montes *et al.,* 2021; Nellist *et al.,* 2022).

Global changing climate is likely to induce alterations in epidemics of TuMV on *Brassica* crops around the world. The direct and indirect effects of climate change factors may be on plant growth, vector population and movement, and virus transmission and multiplication due to induced diversity in plant virus pathosystem. The probable effects are i). Modification of virus epidemic components resulting in congenial epidemics of higher magnitude, ii). Emergence of new and different kinds of TuMV vectors, iii). Evolution of new more virulent TuMV isolates/pathotypes, iv). Difficulties arising in management of virus diseases, v). Effect on *Brassica* plant virus research programmes, and vi). Effect on development of virus disease forecasting system/model. The changing climate variables include rainfall, temperature, wind velocity fluctuations of climate variables, and elevated levels of greenhouse gases (CO_2, methane and others), which have major influences on virus epidemiology and *Brassica* crops yield losses. However, experimental data on each parameter of climate change effecting *Brassica*-TuMV host pathosystem are lacking (Jones and Barbetti, 2012; Jones, 2009, 2016, 2020, 2021).

Molecular mechanism of infection and pathogenesis

The infection and pathogenesis of TuMV on *Brassica* is related to several effectors/determinant genes after interactions of the viruses with host plants. The effectors alter the metabolism of the host to suit viral replication to increase its capability to become more virulent for increased cell infection and pathogenesis. In the infection process VPg-NLa complex induces in protoplasts the decreased host translation through altered host metabolism. Similarly, C1 interacts with Histone H3 to affect host transcriptional shut down during infection. The sequestration of host chloroplast

components necessary for viral replication takes place by interaction of CP with a host protein 37 KDa located in chloroplasts. Hc-Pro is an important virulence factor for viral pathogenesis through long distance movement and maintenance of genome replication. It acts as a suppressor of post transcriptional gene silencing factors. Its mechanisms of action are by elimination of the accumulation of small interfering RNAs by a cal-modulation related cellular protein as in tobacco (rgs-CaM). The *C1* and *P3* genes are the virulence determinants for the breakdown of resistance genes *TuRBO1, TuRBO1b, TuRBO5*, and *TuRBO4* in *B. rapa* cultivars. Amino acid (aa) 279 in the C-terminal of the *P3* gene is a determinant for *Arabidopsis* stalk development when challenged with TuMV isolates UK1 and JPN1. A single amino acid change results in different sub-cellular location of P3 gene and different types of cell wall alterations depending upon virus strains. Narrow stem area and defects in secondary cell wall are due to TuMV strains JPN1 infection. This strain also reduces endothecium lignification. Infections with UK1 strain induce severe floral cell and organ development alterations along with a general transcriptional decrease of most regulatory genes. The infection of TuMV induces accumulation of PR1 protein which acts as virulence factors and allows more multiplication of virus in the host cells. The expression of genes by TuMV such as *RbohD* and *RbohF* is responsible for most ROS production during infection which promotes virus multiplication in the host plants. Plant NADPH oxidases, the respiratory burst-oxidase homologues (RBOHs) are a major source of reactive oxygen species (ROS) during host-pathogen interactions. NADH oxidases and RbohF are crucial in the regulation of the TuMV infection cycle in *Arabidopsis*. Systemic virus infection in *Arabidopsis* is induced by AtGSTU1 and AtGSTU24 which is correlated with significant downregulation of GSTs (glutathione transferases) and cellular and apoplastic GGT (γ-glutamyl transferase with GR/glutathione reductase) activities. The genus *AtGSTU19* and *AtGSTU24* are important in modulating the response to TuMV in *A. thaliana* (Mc Clintock *et al.,* 1998; Plante *et al.,* 1999; Tempo *et al.,* 1999; Kasschau and Carrington, 2001; Vance and Vaucheret, 2001; Jenner *et al.,* 2002a; Suehiro *et al.,* 2004; Sanchez *et al.,* 2015; Lopez-Gonzalez *et al.,* 2020; Otulak-Koziel *et al.,* 2020, 2022, 2023; Table 7).

Table 7: Identification of effectors/ determinants genes of *Turnip Mosaic Virus* for infection and pathogenesis

Effectors / determinants genes	Mechanisms /Effects	References
VPg-NLa complex	Altered metabolism decrease host translation	Plante *et al.* (1999)
Interaction of C1 and Histone H3	Shut down host transcription for infection	Tempo *et al.* (1999)
Interaction of CP with 37KDa host protein	Sequestration of host chloroplast components for viral replication	Mc Clintoch *et al.* (1998)
HC-Pro suppress PTGS	Help long distance movement and maintenance of viral genome replication. Elimination of small interfering RNAs	Kusschau and Carrington (2001); Vance and Vaucheret (2001)
C1 and P3	Systemic non-necrotic infection in *B. napus* resistant lines)	Jenner *et al.* (2002a)
P3- coding region	Systemic infection and regulation of virus accumulation and long-distance movement	Suehiro *et al.* (2004)
P3 cistron	Developmental and cell wall alterations in *Arabidopsis*	Sanchez *et al.* (2015); Lopez-Gonzalez *et al.* (2020)
PR1 protein	More multiplication of virus	Otulak-Koziel *et al.* (2020)
RbohD, RbohF	Produces ROS during infection. Promotes virus replication	Otulak-Koziel *et al.* (2020)
AtGSTU1, AtGSTU24	Systemic virus infection	Otulak-Koziel *et al.* (2022, 2023)
AtGSTU19, AtGSTU24	Modulates response to TuMV	Otulak-Koziel *et al.* (2022)

Host resistance

The management of TuMV under field conditions is very difficult through the spray of insecticides to control aphid vectors since they are not very effective because the virus is transmitted persistently and aphid have ability to evolve and develop resistance mechanisms against commonly used insecticides. The best way to manage TuMV is through host resistance. Initially sources/ resistance gene against TuMV were identified in *Matthiola incana* (recessive *rm* gene), *Lactuca sativa* (Dominant *Tu* gene), and *Cichorium intypus* (most accessions). In the *Brassica* species both qualitative

(dominant) and quantitative (recessive) genes have been identified to confer resistance to TuMV. Sources of resistance to TuMV with dominant and recessive genes have been identified and molecularly mapped on the chromosomes of *B. rapa, B. napus, B. oleracea, B. junce*a, and *A. thaliana*. The genetic inheritance of sources identified has been determined to breed durable resistance to TuMV. Most of the TuMV resistance genes are present in A genome of *B. rapa* (Chinese cabbage) along with some genes in A or C genome of *B. napus, B. oleracea, B. juncea* and *A. thaliana* (Table 8, 9).

(i) *Brassica rapa*: In *B. rapa* (A) genome 15 dominant genes and 6 recessive genes have been mapped to provide resistance to different isolates/pathotypes of TuMV. The dominant resistant genes identified are *TuRBO1b, COnTRO1, BcTuR3, TuRBH01, TuRBO7, TuMV-R, R3, R4, R6*, *TuRBCSO1, Tu1, Tu2, Tu3, Tu4,* and *RNT1-1*. The recessive genes include *retrO1*, *rnt 1-2, rnt 1-3, retr O2* and *trs*. The dominant gene *TuRBO1b* from TD 34-S1 line of *B. rapa* is effective against pathotype 1 to provide resistance to TuMV. A dominant gene *COnTRO1* from RLR 22 line of *B. rapa* provides broad spectrum resistance to seven pathotypes of TuMV (Pathotypes 1, 3, 4, 7, 8, 9, 12). The dominant gene *BCTu*R3 identified from cv. Duanbaigeng of Chinese cabbage shows hypersensitive response upon infection and is known as classic R-gene. The *TuRBCHO1* gene from line Q048 of *B. rapa* has been mapped to confer resistance to isolate TuMV-C5. The *TuRBO7* gene provides resistance to TuMV isolate C-4 mapped from lines VC1/VC-40 of *B. rapa*. The TuMV R gene from line VC-40 of *B. rapa* provides resistance to isolate TuMV-C-4. It has four classic R-genes, *CC-NLR* genes and two pathogenesis related 1 gene. Another dominant gene *TuRBCSO1* identified from line 8407 of *B. rapa* also confer resistance to TuMV-C4 isolates. In *B. rapa* line Y195 -93, dominant genes *R3*, *R4* and *R6* have been mapped to confer resistance to TuMV–C4 isolate. Four dominant QTLs identified from *B. rapa* provide resistance to isolate TuMV-C3 (TuR1, TuR2, TuR3, TuR4) and three QTLs to isolate TuMV-C4 (Tu1, Tu2, Tu3), respectively. A dominant gene *Rnt* 1-1 from *B. rapa* line A 59 provides resistance to TuMV pathotype UK1. This gene (*Rnt* 1-1 resistance and necrosis to TuMV 1-1) is allelic or closely linked to the recessive gene *rnt* 1-2 and incompletely recessive to *rent 1-3* (Tables 8, 9; Rusholme, 2000, Rusholme *et al.,* 2007; Zhang *et al.,* 2008a, b, 2009; Ma *et al.,* 2010; Xinhua *et al.,* 2011; Fujiwara *et al.,* 2011; Chung *et al.,* 2014; Jin *et al.,* 2014; Li *et al.,* 2015).

The broad-spectrum resistance recessive genes *retr* 01 has been mapped in *B. rapa* which is epistatic to a dominant gene *CoTRO1* in a cross between *B. rapa* ver. *Pekinensis* and *B. rapa* spp. *trilocularis*. Both *retr01* and *COnTRO1* have been identified as different copies of the isoform of eukaryotic translation initiation factors 4E, BraAelF (iso) 4E.a and BraAelF (iso) 4E.c, respectively. The gene *retr* O1 mapped to *B. rapa* line K185 has broad-spectrum extreme resistance to TuMV. The resistance provided by this gene is effective against five isolates of TuMV (UK1, CZE1, GBR6, POL1, CDN1) representing major resistance breaking isolates /pathotypes 1, 3, 4.

A single recessive gene *reteO2* from *B. rapa* line BP 8407 provides resistance to *TuMV*-C-4 isolate. Another recessive gene *trs* mapped to *B. rapa* line SB 18/SB 22 provides broad spectrum resistance to four isolates CHN2, CHN3, CHN4 and CHN5 of TuMV. This gene may be tightly linked to recessive gene *retr* O1 or another allele (Table 8, 9; Rusholme, 2002, Rusholme *et al.,* 2007; Fujiwara *et al.,* 2011; Qian *et al.,* 2013; Kim *et al.,* 2013; Walsh *et al.,* 2023.)

(ii) *Brassica napus*: It is an important oilseed crop in many countries yielding quality canola oil. *B. napus* is thought to have multiple origins resulting from independent natural hybridization events between *B. oleracea* and *B. rapa* having both genome (AACC). The TuMV strains are most virulent on the crop and cause severe losses. Five dominant genes and QTLs have been mapped in A (*TuRBO1,* 3, 4, 5) and C (*TuRBO2*) genome of *B. napus* to provide resistance to different isolates/pathotypes of TuMV. The dominant gene *TuRBO1* has been mapped in *B. napus* line N-O-9 and it provides resistance to TuMV pathotype 1. The other dominant gene *TuRBO2* from the same line (N-O-9) of *B. napus* is effective against isolates CHN2 and PN1 of TuMV. Third dominant gene, *TuRBO3* identified from *B. napus* line 225 is effective to TuMV isolate CDN1. Fourth gene, *TuRBO4* mapped from line 165 of *B. napus* has broad spectrum resistance to TuMV isolates 1 and 3 (Table 8, 9; Walsh *et al.,* 1999; Hughes, 2001; Jenner *et al.,* 2002a, 2003).

Resistance in *B. napus* Australian cultivars or breeding lines to TuMV isolates/pathotypes under artificial inoculated conditions has been characterized as phenotypes. The different categories of phenotypes used were i). The +phenotype denotes susceptibility, ii). The RN/+ localized necrosis with systemic spread without necrosis, iii). The RN localized necrosis without systemic spread, iv). The +N systemic movement with necrosis, and v). The R localized resistance to systemic movement without necrosis. Twenty-two cultivars or lines segregating for different types of resistant phenotypes (+N, R and /or RN). None of the cultivars or lines showed extreme resistance phenotype (O). the resistance breaking TuMV isolates 12.1 and 12.5 showed susceptible phenotype (+) in 19 cultivars and one breeding line. The other isolates/pathotypes WA-

AP1/*, NSW 1/7, and NSW 2/1 on inoculation showed four different resistant phenotypes (O, RN, R, and +N) either singly or segregating in different combination. The functional mechanisms of *R*-genes against TuMV isolates/pathotypes is very complex and influenced by R-genes combinations in the genotypes developed under different climatic situation and viral virulence presser. In the presence of a dominant gene *TuRBO1b* in *B. napus* cultivar, pathotype 3 showed +N types of phenotypes when two dominant genes *TuRBO1* and *TuRBO3* are present in the cultivar. The TuMV pathotypes 1 and 3 showed O type phenotype. When *R*-genes are used singly in the cultivars, then phenotype O develops in the presence of dominant gene *TuRBO4* and phenotype RN with dominant gene *TuRBO5*, while both together show phenotype O to pathotype 3. In the presence of a dominant gene *COnTRO1* and a recessive gene *etroO1* phenotype R develops against several pathotypes of TuMV (1, 3-4, 7-9 or 12) (Hughes *et al.,* 2002; Coutts *et al.,* 2007; Guerret *et al.,* 2017; Jones *et al.,* 2021).

(iii) *Brassica juncea*: It is a natural amphidiploid derived from crosses between *B. rapa* and *B. nigra* with AB genome. All the resistance genes have been mapped to A genome. One dominant and three recessive resistant genes have been identified to confer resistance to TuMV isolates. A dominant resistance gene *TuRBJVO1* mapped to *B. juncea* line oasis C18 provides resistance to TuMV isolate WA-AP1. One recessive gene *retr* O3 mapped to *B. juncea* line VCO29 is effective against TuMV isolate Z1. The gene *retr* 03 is an allele of the eukaryotic translation initiation factor 2B-beta (alF2Bβ), and has been identified to provide new mechanisms of resistance to TuMV. The other three recessive genes, *retro* 04 mapped to TWBJ 14 and TWBJ20, *retr*05 to TWBJ14, *retr06* to TWBJ 20 line of *B. juncea* provide broad-spectrum resistance to TuMV isolates UK1, vVIR 24, CDN1 and GBR6 equivalent to pathotypes 1, 3, 4, and 5 respectively. An alternative oxidase (AOX) gene *BjAOX1a* of *B. juncea* has been cloned by RT-PCR. This gene contains several metal binding regions, α- helical regions and cysteine reduces similar to other AOX1 proteins. The AOX1 protein alleviates reactive oxygen species (ROS) and enhances resistance of *B. juncea* plants to TuMV.

The F2 progeny plants of *B. juncea* cross JMO6006 (+only) and Oasis C1 (+ND only) inoculated with pathotype 8 isolate WA-API showed segregation ratios of 3:1 (systemic necrosis: susceptibility) at an early stage of infection, but at late stage of infection, the segregation ratio was 1:2:1 (+ND: N: +). It indicated that a single incompletely dominant gene *TuRBJUO1* responsible for expression of phenotypes +ND and +N in the homozygous and heterozygous conditions. The resistance gene *TuRBJuO1* is not temperature sensitive when tested at 16^{o}C and 28^{o}C. However, the gene *TuRBJuO1* is strain specific as it was less effective to strains breaking resistance to *B. napus* and ineffective against NSW-3. The mechanisms of systemic hypersensitive resistance (SHR, phenotype +ND) in *B. juncea* gene *TuRBJuO1* is elicited on TuMV challenge was found to be associated with phloem necrosis, xylem occlusion, lignification and hydrogen peroxide accumulation when viewed through light microscopy and histochemical analysis of cross section (Table 8,9; Zhu *et al.,* 2012; Nyalugwe *et al.,* 2015a, 2016; Shopan *et al.,* 2017; Bramham *et al.,* 2022).

(iv) *Brassica oleracea*: It is an important *Brassica* species with 9 varieties as rich sources of vegetable and fodder crops to feed both human and animal populations of the world. In the search of several cvs. and breeding lines of white cabbage only field resistant was observed initially to the very destructive virus TuMV. Resistance in Brussels sprouts to TuMV was identified due to a partial dominant's gene. Subsequently four QTLs were identified which contributed resistance to TuMV. One dominant gene *TuRBO2* has been mapped to *B. oleracea* (C) genome which provides broad- spectrum resistance to TuMV isolates. Resistance to pathotypes 1, 7, and 8 has been observed in cultivars of cauliflower, cabbage and broccoli with different levels from extreme to systemic resistance but genes have not been identified (Table 8, 9; Tomlinson and Ward, 1981; Pink *et al.,* 1986; Walsh *et al.,* 1999; Nyalugwe *et al.,* 2015a; Guerret *et al.,* 2017).

(v) *Brassica carinata* and *B. nigra*: In these two species of *Brassica* mapping and identification of TuMV resistance is lacking. Although one cv and 8 accessions of *B. carinata* (BBCC) genome for resistance to pathotype 1 of TuMV were analyzed but they showed different patterns like resistance to systemic infection (One accession), segregation for systemic resistance (3 accessions), segregation for systemic resistance and /or extreme resistance (4 accessions), and segregation for systemic resistance with or without local necrosis (3 accessions). However, testing with TuMV pathotypes /7 on two of these accessions showed similar but not identical segregation patterns. The *B. nigra* (BB) genome using five accessions have been analyzed with TuMV pathotype 3 but none of the accessions showed any kind of resistance (Kehoe *et al.,* 2010; Nyalugwe *et al.,* 2014; Sardaru *et al.,* 2018).

(vi) *Raphanus sativus*: To identify resistance to TuMV in *Raphanus* on artificially synthesized *Raphanobrassica* hybrid (RRCC) genome was used and resistance identified on the chromosome from Kale *B. oleracea* (C) genome in a monosomic addition line (2n=19). Two QTLs have been identified in an F2 population using two radish-inbred lines. Two cloned

genes Rs el4E and RselF (iso) 4 E are involved in resistance to TuMV of radish. Extreme resistance in cv Daikan has been observed to pathotypes 1 (UK1) and 8 (JN1) of TuMV. Radish cv Sparkler has extreme resistance to pathotypes 1, 7, and 8 of TuMV. In radish lines G07-12P1 and KBO7-IOP2, two QTLs related to resistance has been observed (Kaneko *et al.,* 1996; Li, 2009; Cheng, 2013; Lopez-Gonzalez *et al.,* 2017; Palukaitis and Kim, 2021).

Table 8: Crucifers sources of resistance to *Turnip Mosaic Virus*

Host	Sources/genes	References
Cichorium intypus Chicory	Most accessions	Provvidenti *et al.* (1996)
Impatiens balsamina Garden balsam		Provvidenti *et al.* (1982)
Lactuca sativa Lettuce	Dominant Tu gene	Zink and Duffus (1970); Robbins *et al.* (1994)
Matthiola incana Stocks	Recessive rm gene	Johnson and Barmhart (1956)
Brassica oleracea	QTLs C4	Pink *et al.* (1986)
B. napus	*TuRBO2*	Walsh *et al.* (1999)
B. napus line Wester and Rafal	*TuRBO1*	Walsh *et al.* (1999); Walsh (1989)
B. napus line 165	*TuRBO3, TuRB04, TuRBO5*	Hughes (2001); Jenner *et al.* (2002b)
B. rapa	*TuRBO1b*	Rusholme (2000)
B. rapa	Lines BPO79, BPO58	Walsh *et al.* (2002)
B. rapa line RLR22	Dominant (*CanTRO1*); recessive (*retr* 01)	Rusholme (2000)
B. rapa	*BcTuR3*	Ma *et al.* (2010)
	TuRBCHO1	Xinhua *et al.* (2011)
	TuRBO7	Jin *et al.* (2014)
	TuMV-R	Chung *et al.* (2014)
	TuRBCSO1	Li *et al.* (2015)
	Tu1, Tu2, Tu3, Tu4 QTLs	Zhang *et al.* (2008b)
	Rnt 1-1, rnt1-2, rnt 1-3	Fujiwara *et al.* (2011)
	retrO1, ConTRO1	Rusholme *et al.* (2007)
	Retr O2	Qian *et al.* (2013)
	trs	Kim *et al.* (2013)
B. rapa	Line K185, *retrO1*	Walsh *et al.* (2023)
B. rapa line	R-O-18	Haj Kassem and Walsh (2008)
B. rapa lines	Jong Bai N02, Jin G55	Hughes *et al.* (2003)
	Line R 54 (QTLs)	Graichen and Schliephake (1996); Graichen and Rabenstein (1996)
B. oleracea	*TuRBO2*	Walsh *et al.* (1999)
Arabidopsis thaliana	TuiN1 (*RGX, RG2, RG3*)	Liu *et al.* (2015)
Ecotypes	Bay-0, Di-O, Er-O, Or-O, UK1	Martin *et al.* (1999)
	One dominant gene	Kaneko *et al.* (2004)
	Lsp1	Lellis *et al.* (2002); Duprat *et al.* (2002)
	pcap1	Vijayapalani *et al.* (2012)
	RTM3	Rubio *et al.* (2019)
B. juncea	*TuRBJUO1, retr O3*	Nyalugwe *et al.* (2016); Shopan *et al.* (2017)
B. juncea	*Retr O3, retrO4 (TWBJ14, TWBJ20)*	Bramham *et al.* (2022)
	retrO5 (TWBJ14), retrO6 (TWBJ20); TuBRJUO1	Nyalugwe *et al.* (2015b)
Raphanus sativus	cvs. Daikan, Sparkler	Palukaitis and Kim (2021)

Table 9: Identification of *R*-genes to *Turnip Mosaic Virus* isolates/pathotypes

Resistance genes/ QTLs genome	Host/lines	Effective against pathotypes/isolates	References
Tu	*Lactuca sativa*	-	Robbins *et al.* (1994)
rm	*Matthiola* spp.	-	Johnson and Barnhart (1956)
TuRBO1	*Brassica napus* (A) N-0-9	1	Walsh *et al.* (1999)
TuRBO1 b	*B. napus* (A) TD34-S1	1	Rusholme (2000); Lydaite *et al.* (2014)
TuRBO2	*B. napus* (C) N-0-9	CHN1, IPN1	Walsh *et al.* (1999)
TuRBO3	*B. napus* (A) 225	CDN1	Hughes (2001, 2003)
TuRBO4	*B. napus* (A) 165	1,3	Jenner *et al.* (2002a, 2003)
TuRBO5	*B. napus* (A) 165	1,3	Jenner *et al.* (2002a, 2003)
retro1	*B. rapa* (A) RLR 22	1,3,4,7,8,9,12	Rusholme (2000, 2007)
ConTRO1	*B. rapa* (A) RLR 22	1,3,4,7,8,9,12	Rusholme (2000, 2007)
BcTurR3	*B. rapa* var. *chinensis* (A) Duanbaigeng	-	Ma *et al.* (2010)
TuRBCHO1	*B. rapa* var. *chinensis* (A) Q048	C5	Xinhua *et al.* (2011)
TuRBO7	*B. rapa* (A) VC1/VC40	C4	Jin *et al.* (2014)
TuRBCSO1	*B. rapa* (A) 8407	C4	Li *et al.* (2015)
Tu1, Tu2, Tu3, Tu4	*B. rapa* (A) 91-112	C4	Zhang *et al.* (2008 a, b)
TuR1, TuR2, TuR3, TuR4	*B. rapa*	C3	Zhang *et al.* (2008a)
Tu1, Tu2, Tu3,	*B. rapa*	C4	Zhang *et al.* (2009)
Rnt 1-1	*B. rapa* (A) A59	UK1	Fujiwara *et al.* (2011)
retrO1	*B. rapa*	-	Rosholme *et al.* (2007)
ConTRO1	*B. rapa*	-	Rosholme *et al.* (2007)
retrO2	*B. rapa* (A) BP 8407	C4	Qian *et al.* (2013)
R3, R4, R6	*B. rapa* (A) Y195-93	C4	Zhang *et al.* (2009)
trs	*B. rapa* (A) SB18/ SB22	CHN2, CHN3, CHN4, CHN5	Kim *et al.* (2013)
TuMV -R	*B. rapa* (A) VC-40	C4	Chung *et al.* (2014)
retrO1	*B. rapa* K185	UK1, CZE1, GBR6, POL1, CDN1 (Pathotypes 1, 3,4)	Walsh *et al.* (2023)
TuRBJUO1	*B. juncea* (A) Oasis C18	(WA-Ap1)	Nyalugwe *et al.* (2015a, b, 2016)
retrO3	*B. juncea* (A) VC 029	Z1	Shopan *et al.* (2017)
Retr04 (TWBJ14, TWBJ20), retr 05 (TWBJ14), retr06 (TWBJ20)	*B. juncea* TWBJ14, TWBJ20	UK1, vV/R24, CDN1, GBR6 (Pathotype 1,3,4,4)	Bramham *et al.* (2022)
TuRBO2	*B. oleracea* (C)	-	Walsh *et al.* (1999)
TuN1 (RGX, RG2, RG3)	*A. thaliana*	-	Liu *et al.* (2015)
lsp1	*A. thaliana*	-	Lellis *et al.* (2002)
pcap1	*A. thaliana*	-	Vijayapalani *et al.* (2012)
RTM3 region	*A. thaliana*	-	Rubio *et al.* (2019)
Ecotype Bay -O- Di-O, Er-O, Or-O	*A. thaliana*	UK 1	Martin *et al.* (1999)
Ecotype ber	One dominant gene	-	Kanko *et al.* (2004)

(vii) *Arabidopsis thaliana*: The *Arabidopsis* has been widely used as a model plant to study *Brassica* host-pathosystem through which molecular mechanisms of host resistance and pathogenesis has been revealed against major pathogens (Saharan *et al.,* 2022). In a screen of 106 ecotypes of *Arabidopsis*, ecotypes bay-O, Di-O, Er-O, and Or-O were found resistant to systemic infection of TuMV isolate UK1. One ecotype Bay –O also has resistance to cell-to-cell movement of the virus. A single dominant gene in the *A. thaliana* ecotype erO provides resistance to TuMV for systemic venial necrosis. A dominant gene *TuN1* in the *NLR*-R gene cluster has been mapped for resistance to TuMV. This gene is a complex of three genes with RGX being the primary determinants of resistance and RG2 and RG3 are involved in regulation of TuN1- mediated necrosis. A recessive gene *lsp1* also provides resistance to TuMV. Another potential recessive gene PCaP1 provides resistance through a cation –binding protein that attaches to the plasma membrane. The protein P3N-PIPO interacts with PCaP1 gene through a genome wide association study in 317 accessions of *Arabidopsis.* RTM3 region has been identified as potential domain for resistance to TuMV by blocking long distance movement of virus, molecular mechanisms of host resistance in *Arabidopsis*–TuMV pathosystem has been revealed. (Table 8, 9; Martin *et al.,* 1999; Lellis *et al.,* 2002; Kaneko *et al.,* 2004; Vijayapalani *et al.,* 2012; Liu *et al.,* 2015; Rubio *et al.,* 2019).

Respiratory burst oxidase homologes (Rbohs) have very essential roles during host plant-TuMV interaction and produces reactive oxygen species for development, growth, and response to stress. Increased rboho/c-TuMV reaction functions for a highly dynamic increase in total cellular and apoplastic glutathione content to induce expression of *AtGGT1*, *AtGSTU13* and *ATGSTU19* genes. The upregulation of GSTs as well as cellular and apoplastic GGT with GR activities limits TuMV replication exhibit resistance. Glutathione participants in the reactive oxygen species (ROS) dependent signaling pathway under biotic stress conditions. Most of the glutathione-s-transferases (GSTs) are induced in cells during the defense responses of host plants through highly specific glutathione-binding abilities and signaling functions. The over-expression of the genes *GSTU19* and *GSTI13* in *Arabidopsis* limits TuMV to provide resistance (Otulak-Koziel *et al.,* 2020, 2022, 2023).

TuMV disease management through host resistance

It is very difficult to manage TuMV since it is transmitted by >89 aphid species in a non-persistent manner under natural conditions. The aphid vector introduces the TuMV into plant cells by their stylet in a non-persistent transmission mode during probing or feeding. The wide host range, high genetic/pathogenic variability and transmission by wide range of vectors make challenging to manage TuMV by chemical control measures. Insecticides can control some species of aphids but not all and soon aphid species are replaced to continue infection. The most effective, and environmentally friendly method is use of host resistant varieties. It can be achieved in *Brassica* crops by transformation of R-genes into crops, molecular marker-assisted selection (MAS) breeding for resistance, pyramiding of R-genes, and host induced gene silencing (HIGS) approaches to breed viral resistant cultivars of *Brassica* crops.

(I) Transformation of *R*-genes into *Brassica* crops: It has been observed that W95L, K150L and W95L/K150L amino acid mutations of *B. rapa* elF (iso) 4E interrupted the interaction with TuMV VPg. The over expression of these mutants of elF (iso) 4E in the susceptible Chinese cabbage cv can confer resistance to multiple strains of TuMV. The resistant genes *retrO1* and *retrO2* can encode elF (iso) $E in *B. rapa* and the different copies of elF (iso) 4E from a resistant to *B. rapa* line have been transformed into an el (iso) 4E knockout line of *A. thaliana*. A recessive resistance *retrO3* gene cloned from *B. juncea* resistant line has been transformed into susceptible line to confer resistance mechanisms to TuMV. Therefore, genetic engineering approaches can be employed to improve resistance in *Brassica* crops to TuMV (Kim *et al.,* 2014; Nellist *et al.,* 2014; Shopan *et al.,* 2017).

(ii) Molecular marker-assisted breeding in *Brassica* crops: In the recent past, there has been great improvement in marker types used and molecular mapping approaches to breed *Brassica* with MAS. In the past RAPD, AFLP, and RFLP markers were being used which were less efficient. During this century, use of SNP based markers like BSA-developed markers, competitive allele-specific PCR (KASP) markers, and markers from genome–wide association studies with high throughput approaches of mapping have become very popular (Walsh *et al.,* 1999; Rusholme *et al.,* 2007; Zhang *et al.,* 2008a, 2009; Qian *et al.,* 2013; Li *et al.,* 2016; Cheng *et al.,* 2016).

(iii) Pyramiding of *R*-genes in *Brassica* for durable resistance to TuMV: Breeding with single isolate–specific R-gene is highly effective but this kind of resistance can be easily broken with the evaluation of new virulence and with the effect of climate changes. Polygenic resistance governed by QTLs may be more durable than qualitative resistance. However, its effectiveness varies between cropping seasons with the influence of environmental conditions. Therefore, pyramiding of major genes (*R*-genes) with very high level of quantitative resistance in *Brassica* crops will be

an ideal approach to maximize the durability of resistance. At present more than 26 dominant and 10 recessive gene have been mapped in *Brassica* crops in addition to QTLs and sources of resistance in *Brassica* lines, cultivars and ecotypes of *Arabidopsis* (Table 8, 9). Therefore, these sources can be easily utilized by the breeders for pyramiding *R*-genes for durable resistance to TuMV in *Brassica* crops (Rusholme *et al.,* 2007; Qian *et al.,* 2013; Shopan *et al.,* 2017; Li *et al.,* 2019; Palukaites and Kim, 2021).

(iv) Use of host-induced gene silencing approach in *Brassica*: This approach allows the use of pathogen genes to develop resistance in *Brassica* crops against TuMV via HIGS. To confer resistance to virus, the CP gene of TuMV has been used via HIGS to inhibit virus multiplication / replication in the host cells. It has been demonstrated that the fusion of viral segments to DNAs can confer resistance to multiple viruses. The broad–spectrum resistance to TuMV in *B. rapa* has been achieved through CP gene of TuMV using this approach. The other molecule P3 protein of TuMV is also associated with avirulence when it interacts with *B. napus* dominant genes *TuRBO3* and *TuRBO4* to provide resistance to TuMV isolate CDN1. When a wild type CP protein of TuMV interacts with dominant R-gene, *TuRBO*5, an HR-like phenotype with mutation at position +5447 in the C1 gene breaks to resistance conferred by *TuRBO5* (Jenner *et al.,* 2002b; Hughes *et al.,* 2003; Nowara *et al.,* 2010).

Integrated management of TuMV in *Brassica* crops

The use of integrated disease management approach effective against non-persistently aphid born viruses have been suggested to control TuMV on *Brassica* crops. The various approaches include; i). Deployment of non-host barrier crops, ii). Promotion of early crop canopy development and high plant density to reduce aphid landing rates, iii). Sowing into standing stubbles, iv). Avoiding or eliminating potential virus reservoirs with herbicides, v). Plant breeding to enhance the TuMV resistance in *Brassica* crops with additional *R*-genes for protection against wide range of TuMV isolates/ pathotypes, vi). Incorporation of R-genes with suitable combinations for durable resistance, vii). Identification of resistance breaking isolates/pathotypes and incorporation of effective *R*-genes against such virulence's, viii). Searching new sources of resistance effective against large number of virulent pathotypes, ix). A strategy to slow spread of virulent pathotypes, x). Manipulation of date of sowing to avoid infection, xi). Preventing movement of infected crop residues in adjoining non-infected areas (Jones, 2001; Guerret *et al.,* 2017; Jones *et al.,* 2021).

Major precautions to manage TuMV: To avoid the introduction and spread of TuMV new virulence's some precautionary measures are required at country level. i). to impede introduction of *Brassica* viruses, steps should be taken for strict plant biosecurity regulation, ii). Avoiding the entry of infected plant material and their vectors from importing countries, iii). Preventing the establishment and spread of viruses in the country, iv.) Pre-arrival inspection and plant health certification of planting material, v). On arrival inspection at air-port and sea-port for virus infection, vi). To develop reliable early warning system based on historical data on vector and virus load on *Brassica* crops, and vii). Eradication of virus infected material to avoid further spread. Future research efforts are required in development of more accurate and cost-effective diagnosis and surveillance approaches to help avoid establishment of damaging viruses and their vectors within each country (Rodoni *et al.,* 2010; Jones, 2016, 2020, 2021).

Protection from resistance breaking TuMV strains

The resistance breaking strains of the virus are likely to evolve when *Brassica* cv. With a single dominant gene are grown in large area for very long duration. Therefore, it is essential to monitor *Brassica* crops and nearby weeds to search for resistance-breaking virus strains. Precautions should also be taken to avoid their introduction from other sources and nearby countries (Guerret *et al.,* 2017; Jones *et al.,* 2021).

Conclusion

The TuMV belongs to the Potyvirus genus within the potyviride family. It possesses flexuous filamentous particles that are 135 Å wide and have a model length of 729nm. These particles contain a single copy of a single-stranded positive-sense RNA (+ssRNA) genome. The virions are 720 x 15-20nm in size and are composed of 95% coat protein (CP) and 5% RNA.

The determination of its genome constituents and particle structure has been accomplished. In field conditions, it is transmitted by over 89 species of aphids, with Myzus persicae and Brevicoryne brassicae being the primary vectors. Typical symptoms of TuMV infection include vein clearing, chlorotic mottling, leaf distortion, mosaic patterns, necrosis, plant stunting, and, in severe cases, host death. The manifestation of symptoms may vary among different *Brassica* species due to factors such as environmental conditions, virus strains, aphid vector activity, host genotypes, crop growth stage, and the presence of other viruses. Its host range is remarkably extensive, infecting more than 318 species from 156 genera in both dicots and monocots, including various field crops, ornamentals, and weeds. The transmission of TuMV to the host occurs through aphid vectors, which acquire the virus from infected

hosts. These vectors then feed on and probe healthy plants, introducing the virus into host cells through the stylet in a non-persistent transmission mode. Once inside the host cell, the virus particles shed their outer coat and begin replicating the genome, resulting in an increased number of virus particles. TuMV has shown pathogenic variability, with strains/pathotypes and phylogenetic groups infecting various hosts, documented in over 20 countries. Host resistance to TuMV in *Brassica* crops governed by both qualitative and quantitative genes.

Controlling TuMV is a highly challenging task due to various factors. These include its ability to infect a wide range of hosts, making it difficult to control the reservoirs of the virus. Additionally, the virus exhibits high variability, which allows it to evolve resistance-breaking isolates. Furthermore, there are numerous insect vectors that can transmit the virus in a non-persistent mode. Moreover, the development of resistance in aphid vectors to insecticides renders them ineffective in controlling the virus. However, the most effective and cost-efficient method to manage TuMV is through the use of host resistant cultivars. This approach involves continuously strengthening the resistance in cultivars against new strains of the pathogen. This can be achieved through various approaches, including the transfer of *R*-genes, molecular markers assisted breeding, pyramiding of *R*-genes, and the use of host-induced gene silencing. To effectively manage TuMV, it is crucial to adopt integrated approaches that incorporate precautionary measures. This includes preventing the introduction and spread of the virus and developing an early warning system to detect its occurrence. By implementing these strategies, the management of TuMV can be significantly improved.

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