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REVIEW

Apple mosaic virus

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Summary. Apple mosaic, rose mosaic, plum and birch line pattern, horse chestnut yellow mosaic, and hazel mosaic are some of many diseases caused by one of the longest-known pome fruit viruses: *Apple mosaic virus*. The worldwide distribution of *Apple mosaic virus* and its negative impacts on fruit, nut and hop production are sufficient to cause international interest. This review endeavors to piece together all known information about the virus. We describe taxonomic position, virion structure, the host range and symptoms, modes of transmission and diagnostics for the virus, as well as distribution and control of the diseases it causes.

Key words: ApMV, hosts, diagnostics, transmission, control.

Introduction

Apple mosaic virus (ApMV) was named after the symptoms that were initially characterized in apples, and first described by White (1928), Bradford and Joley (1933) and Christoff (1934). The virus has been known by several synonyms, including: Apple infectious variegation virus, Rose infectious chlorosis virus, Rose mosaic virus, Plum line pattern virus, European plum line pattern virus, Mountain ash variegation virus, Birch line pattern virus, Birch ringspot virus, Dutch plum line pattern virus, Hop A virus, Horse chestnut yellow mosaic virus, Hop virus A, Hop virus C, Mild apple mosaic virus and Severe apple mosaic virus) (Fulton, 1972; Petrzik and Lenz, 2011). Because of serological similarities to *Prunus necrotic ringspot virus* (PNRSV), ApMV has commonly been incorrectly reported as a strain of PNRSV in hop (Bock, 1966, 1967; Barbara *et al.*, 1978; Smith and Skotland, 1986). However, as far back as 1992, Crosslin and Mink highlighted serological and biophysical similarities and differences between PNRSV infecting hop and other hosts. In their studies, sedimentation profiles of PNRSV isolates from hop were similar to the profile of ApMV from hops. Nucleoprotein analysis of PNRSV from hops produced bands that migrated more slowly than those from *Prunus* sp. or rose. When isolates were separated into electrophorotypes, PNRSV from hop was assigned to a group by itself, and antisera produced against PNRSV and ApMV from hop reacted strongly only with isolates from hop, indicating their serological distinction from PNRSV from other sources and their similarity to each other. Some confusion regarding virus nomenclature continues in the present molecular era, when an isolate of PNRSV was published as an ApMV isolate (Sánchez-Navarro and Pallás, 1994) and was later re-classified as a PNRSV isolate (Sánchez-Navarro and Pallás, 1997). In addition, the G isolate of ApMV was published as PNRSV (Guo *et al.*, 1995).

Taxonomic position

ApMV is a species of the *Ilarvirus* genus. This genus, together with the genera *Alfamovirus*, *Anulavirus*, *Bromovirus*, *Cucumovirus* and *Oleavirus*, constitute the family *Bromoviridae*, which contains 19 species (Anonymous, 2015). The species of *Illavirus*

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are divided into six major subgroups according to their serological characteristics: ApMV is classified into subgroup III together with PNRSV, *Blueberry shock virus* (BlShV) and *Humulus japonicus latent virus* (HJLV) (Roosinck *et al*., 2005). Within ApMV, the two *Ilarvirus* serotypes have been found commonly infecting commercial hops: the ApMV-hop serotype (ApMV-H) and the ApMV-intermediate serotype (ApMV-I). This reflects the distant relationship with PNRSV and the closer but phylogenetically distinct relationship with ApMV from *Malus* spp. (Crowle *et al*., 2003).

Virion properties

ApMV is a positive-sense single-stranded RNA virus (Roosinck *et al*., 2005). No information concerning the particle composition of this virus is available, but by analogy with PNRSV the particles probably contain l6% RNA (Fulton, 1972).

ApMV virions each consist of three quasi-spherical or slightly pleomorphic particles that range in diameter between 25 and 29 nm. Each particle contains one component of the tripartite RNA genome (approx. 2.1, 3.0 and 3.5 kb) (Barbara *et al*., 1978). All of the genomic components need to be present in the host plant for initiation of infection. The ApMV genome consists of RNA 1, RNA 2, RNA 3, and a subgenomic RNA 4 (Roosinck *et al*., 2005). The largest RNA (RNA 1) is 3476 nucleotides (nt) in length and encodes a single large polypeptide that is similar to the methyltransferase-like and helicase-like domains present in many plant RNA viruses. The RNA 2 nucleotide sequence is 2979 nt in length and contains a single large open reading frame (ORF) (Shiel and Berger, 2000). The RNA 2 ORF shares sequence homology with a motif found in most virally-encoded RNA-dependent RNA polymerases (Buck, 1997). RNA 3 is 2056 nt in length and contains two ORFs. The first of these encodes for a peptide that represents the movement protein (MP) (Shiel *et al*., 1995), which plays a role in cell-to-cell movement and is directly translated from RNA 3 (Alrefai *et al*., 1994). The second ORF is 654 to 669 nt in length and encodes for a peptide (Petrzik and Lenz, 2002) that constitutes the coat protein (CP) that is translated from the subgenomic mRNA (RNA 4), which is derived from RNA 3 (Alrefai *et al*., 1994). The CP of ilarviruses forms the shell for the three genome components and plays a major role in the multiple steps of replication and initiation, and in the propagation of infection (Jaspars, 1985; Ansel-McKinney and Gehrke, 1998; Bol, 1999).

The amino acid sequences encoded by RNA 1 and 2 exhibit similarity to the other ilarviruses for which sequence data are available; however, both are most closely related to *Alfalfa mosaic virus* (AMV). Points of similarity include the absence of ORF 2b, which is present on the RNA 2 of all previously characterized ilarviruses. The close relationship to AMV can also be observed in the movement protein, but not with the coat protein (Shiel and Berger, 2000).

Considerable attention is currently directed towards CP sequence variability among different ApMV isolates. The consensus ApMV CP sequence has been established as having 654 nt, but isolates with insertions of 6 to 15 nt after nt position 141 have been described. The RNA around the insertion point can potentially form a stable secondary structure with three hairpins. The insertions may stabilize this structure or may be neutral. The predicted folding of the translated protein is not influenced by the insertions or frame shift (Petrzik and Lenz, 2002). Phylogenetic analysis of the complete CP gene of the largest set of ApMV isolates discriminated two main clusters of isolates: one cluster includes Maloideae hosts and Trebouxia lichen algae hosts, while the second includes the hop, *Prunus*, and other woody tree hosts (Grimová *et al*., 2013). No correlation was found between the clusters and geographic origins of the virus isolates, and the positive selection hypothesis in distinct hosts was not confirmed. Purifying selection was therefore shown to have occurred in all of the virus populations. The GGT to AAT substitution that resulted in a Gly to Asn change inside the zinc-finger motif in the capsid protein was revealed to be specific for discrimination of the clusters and could influence host preference (Grimová *et al*., 2013).

Host range

ApMV has a wide host range, including woody and herbaceous plants. It is capable of infecting over 65 species in 19 families, by either experimental or natural routes (Fulton, 1952; Kristensen and Thomsen, 1963; Fulton, 1965 and unpublished data in the 1950s era). Horticulturally important host plants infected by this virus include apple (*Malus domestica*), pear (*Pyrus communis*), apricot (*Prunus armeniaca*), peach (*P. persica*), cherry (*P. avium*), plum (*P. domesti-*

ca), almond (*P. amygdalus*), strawberry (*Fragaria* sp.), raspberry (*Rubus idaeus*), blackberry (*R. occidentalis*), red currant (*R. rubrum*) and hazelnut (*Corylus avellana*). Other susceptible species include hop (*Humulus lupulus*), roses (*Rosa* sp.), wooly blackberry (*Rubus canescens*), *R. ursinus* and further *Prunus* species such as *P. cerasifera*, *P. instititia*, *P. mahaleb*, *P. salicina*, *P. serulata*, *P. triloba*, *P. cerasus* and blackthorn (*P. spinosa*). ApMV was also found in silver birch (*Betula pendula*), white birch (*B. papyrifiera*), yellow birch (*B. alleghaniensis*), European mountain ash (*Sorbus aucuparia*), horse chestnut (*Aesculus hippocastanum*), red horse chestnut (*A.* × *carnea*), sweet buckeye (*A. parviflora*), bottle brush buckeye (*A. flava*) and hawthorn (*Crataegus* sp.) (Fulton, 1972; Casper, 1973; Gotlieb and Berbee, 1973; Sweet and Barbara, 1979; Baumann *et al*., 1982; Nemeth, 1986; Polák and Zieglerová, 1997; Desvignes *et al*., 1999; Arli-Sokmen *et al*., 2005; Tzanetakis and Martin, 2005). ApMV has been detected in weeds that are often found in ApMV-infected hazelnut orchards, including shepherd's needle (*Scandix* sp.), mugwort (*Artemisia vulgaris*), bellflower (*Campanula* sp.), wild clary (*Salvia verbenaca*), hempnettle (*Galeopsis* sp.), self-heal (*Prunella* sp.) and old man´s beard (*Clematis vitalba*) (Arli-Sokmen *et al*., 2005). Moreover, lichens have been recently identified as ApMV hosts (Petrzik *et al*., 2014).

In addition to the natural host plants, several diagnostic experimental hosts are used as indicators in biological tests, including *Chaenomeles japonica*, *Cucumis sativus*, *Torenia fournieri*, *Vinca rosea*, *Vigna sinensis*, *Cyamopsis tetragonoloba*, *Petunia hybrida*, *Chenopodium quinoa*, *C. amaranticolor*, *Cucurbita maxima*, *C. pepo*, *Nicotiana benthamiana, N. megalosiphon*, and *Phaseolus vulgaris* (Fulton, 1972; Paunovic *et al*., 2011).

Geographical distribution

ApMV is distributed worldwide wherever its hosts are present (Fulton, 1972). Nevertheless, information is scanty on the geographical distribution of the virus in commercial plantings and in the wild. Many minor studies addressing ApMV monitoring in different regions and countries have been performed, and their results are presented below. However, their informative value concerning the health status of selected crops is disputable due to the restricted amount of tested plants, and could therefore represent a starting point for the investigation of ApMV incidence.

Distribution of ApMV in family Rosaceae

In Europe, the virus is more commonly present in *Prunus* spp. than in *Malus* spp. (Paunovic *et al*., 2011). ApMV is found often in mixed infections with PNRSV and *Prune dwarf virus* (PDV) on their common stone fruit hosts. In pome fruits, ApMV often occurs together with *Apple chlorotic leaf spot virus* (ACLSV), *Apple stem pitting virus* (ASPV), *Apple stem grooving virus* (ASGV), and other apple-infecting viruses, but the frequency of ApMV infection seems to be much less than those of the latter viruses (Petrzik and Lenz, 2011). In the Czech Republic, ApMV was detected in approx. 17% of 472 apple trees tested in nine selected orchards. The incidence of ApMV-positive trees was greater in old apple orchards, with 50% of trees infected (Svoboda and Polák, 2010). Petrzik and Lenz (2002) detected ApMV in 81.3% of 16 tested pear trees. Polák (2007) suggested that the incidence of ApMV in individually growing trees of the genus *Prunus* was negligible in the Czech Republic. ApMV occurred in 1% of 86 tested plum trees and 0.8% of 162 myrobalan trees. ApMV was also detected in 6% of 29 tested European mountain ash trees in the Czech Republic (Polák and Zieglerová, 1996).

In Romania, 6% of 17 tested apple trees were found to be infected with ApMV (Popescu *et al*., 2004), while only 0.7% of 140 tested apple trees were found to be infected in Albania (Myrta *et al*., 2004).

In southern Italy, the presence of ApMV in almond was reported to be as great as 45% (Savino *et al*., 1995; Di Terlizzi, 1998), whereas in the Valencia and Murcia regions of Spain, the ApMV infection rate of almond ranged from 14 to 17% (Llacer *et al*., 1986; Pallás *et al*., 1998). The results of a survey in Italy conducted for ApMV prevalence recorded that 21% of 323 plum trees, 20% of 370 apricot trees and 2% of 1397 peach trees were infected with the virus (Di Terlizzi *et al*., 1992). The results of the survey conducted in the Valencia region were 7% of both apricot and plum and 1% of peach (Llacer *et al*., 1986) and 6% of 450 apricot trees in the Murcia region in Spain (Dominguez *et al*., 1998). The incidence of ApMV in apple trees in different plantations in Greece ranged from 3 to 1% (Varveri and Bem, 1995). In Turkey, the incidence of ApMV infection in apple trees differed according to the region. Extensive surveys were conducted in the Van Province, but ApMV was not detected in any of the samples tested and the surveyed apple trees were apparently free of symptoms (Korkmaz *et al*., 2013). A low level of ApMV infection

was detected in apple samples from commercial orchards and nurseries in Malatya (9%) (Elibuyuk and Erdiller, 1998). In reports from other regions of Turkey that record pome fruit infections, rates of 29% of 205 plants were found in the Black Sea, Mediterranean, and Marmara regions (Dursunoglu and Ertunc, 2008), 15% in Southeast Marmara (Uzunogullari and Ilbagi, 2009), approx. 10% of 174 plants in the East Mediterranean region (Caglayan *et al*., 2006), and 68% of 461 total samples in Central Anatolia (Akbas and Ilhan, 2005). A total of 22% of 54 tested samples of blackthorn were ApMV-positive in the Trakya region (Ilbagi *et al*., 2008), while 7% of 22 collected samples from plums were identified as ApMV-positive (Akbas and Degirmenci, 2010). In the Lakes region of Turkey, 8% of 218 oil roses (*Rosa damascena*) from ten oil rose plantations were found to be ApMV-positive (Yardimci and Culal, 2009). Only 0.2% of 1077 tested apple trees from six governorates were infected by ApMV in Syria (Ismaeil *et al*., 2006), and only 4% apple trees from 100 collected samples were positive in Morocco (Afechtal *et al*., 2010). In Tunisia, 15% of 111 apple samples and 11% of 106 tested pear trees were positive for ApMV (Mahfoudhi *et al*., 2013). Surveys from Algeria showed an incidence of 3% for ApMV in stone fruits from the eastern regions (i.e., 1% of 109 peach trees, 2% of 98 plum trees, 2% of 91 apricot trees, 2% of 64 cherry trees and 3% of 24 tested myrobalan trees were ApMV-positive) (Rouag *et al*., 2008).

In the Indian states of Himachal Pradesh, Jammu and Kasmir, surveys revealed the presence of ApMV in 13 of 198 apple samples, indicating incidence of approx. 7% (Thokchom *et al*., 2009), and Lakshmi *et al*. (2011) detected ApMV in 26% of 78 tested apple trees.

In the Pacific Northwest of the United States, 4% of 19 *Rubus ursinus*, *R. idaeus* and *R. occidentalis* were found to be ApMV-positive. In a red raspberry (cv. Shoemann) field in Germany, latent infection by ApMV was found in 2% of 42 samples tested (Baumann *et al*., 1982).

Distribution of ApMV in family Fagales

ApMV was detected in 0.4% of 1045 tested birch trees in Germany (Gruntzig *et al*., 1996), and its presence, though not numerically evaluated, was also confirmed in Wisconsin in the USA (Gotlieb and Berbee, 1973). In the Czech Republic, the virus was found in 5% of 38 horse chestnut trees, in 2% of 18 red horse chestnut trees, in one of three *A. flava* and in one of five tested *A. parviflora* (Polák and Zieglerová, 1997).

Distribution of ApMV in family Corylaceae

Only 3% of 320 samples collected from hazelnuts cv. Negret in Spain were free from ApMV (virus incidence 97%) (Aramburu and Rovira, 2000). In Turkey, 73% of 150 ELISA-tested hazelnuts trees growing in 80 orchards in the Bartin, Duzce and Zongulldak provinces were found to be infected with ApMV (Akbas and Ilhan, 2005). Subsequently, an investigation into the incidence of ApMV in hazelnuts in the west Black Sea coast of Turkey revealed an average infection rate of 4% in the 1465 hazelnuts sampled and tested (Akbas and Degirmenci, 2009). Kobylko *et al*. (2005) showed the presence of ApMV infection only in two cultivars (i.e., Negret and a single tree of clone 104 E) of 27 hazelnut varieties and clones in Poland. Postman and Mehlenbacher (1994) performed a survey at the germplasm repository in Oregon in the USA and found that ApMV was present in 44% of 48 clones imported from Spain, 15% of 34 clones from Turkey and 8% of 65 clones from Italy. One of 28 German cultivars and one of three cultivars from the Republic of Georgia were also found as ApMV positive.

Distribution of ApMV in family Cannabaceae

Because of serological similarities of ApMV with PNRSV (Crowle *et al*., 2003), the reports about ApMV distribution based on serological diagnostics in hop (*Humulus lupulus* L.) gardens worldwide could be misleading. ApMV has been reported in the Pacific Northwest states of Washington, Oregon, and Idaho in the USA. An ELISA-based virus survey was conducted in 1994 to determine the virus incidence in the major hop cultivars grown, including Galena, Cluster, Nugget, Willamette, Chinook, Cascade, and Tettnanger. Twenty samples were collected randomly from each of a total of 160 hop gardens, and the incidence of ApMV was found to be 1% in the samples (Klein and S.D. Husfloen, 1995). Pethybridge *et al.* (2002a) demonstrated that the combined incidence of ApMV and PNRSV ranged from 1 to 6% of 80 plants of cv. Horizon in hop gardens of Washington State in the USA.

In the Riwaka district in New Zealand, 27 fields of hop mostly of the locally bred triploid cultivars Superalpha, Green Bullet, and Sticklebract, were monitored for ApMV presence. The incidence of the virus was found to be more than 81% in 1620 plants. Both the intermediate (I) and apple (H) serotypes of ApMV were detected (Hay *et al*., 1992). ApMV (H + I) was also detected in New Zealand, occurring in 41 of the 60 gardens surveyed. The virus was detected in all ten assessed cultivars, namely Pacific Gem, Pacific Hallertaue, Pacific Jade, Nelson Sauvin, Motueka, New Zealand Hallertaue, Green Bullet, Cascade, Super Alpha, Southern Cross (Pethybridge *et al*., 2009).

At Gunns Plains, Tasmania, Australia, ApMV incidence (both H and I serotypes) in five gardens of cv. Victoria was 85–100%. Virus incidence in 13 'Pride of Ringwood' gardens, 10-19 years old and initially established from material of unknown virus status, was 0–77%, and in an 8-year-old trial, in which 3/4 cultivars were planted with elite material, incidence of ApMV in Victoria was 98%, in cv. T11 58% and in cv. Opal 31% (Pethybridge *et al*., 2000).

Polák and Svoboda (1989) used an ApMV antiserum and obtained positive reactions in almost all Czechoslovak and foreign hop cultivars and clones growing in that country, including Osvalďs clones (6% infected of 58 plants), cv. Zlatan (9% of 17 plants), Žatecký Červenák (100% of seven plants), cv. Aromat (three of four plants), Sirem (100% of three plants), Blato (100% of seven plants), Lučan (100% of three plants), Petham Golding (40% of 20 tested clones), Early Prolific (30% of ten), Sacramento (five of ten), Cobbs Golding (one of two), Early Bird Golding (one of two), Wye Early Bird Golding (one of two), Atlas (one of one), Hüller Bitterer (one of one), Northern Brewer (three of three), Brewers Gold (one of one), Smooth Cone (one of one), Shinshu Wase (one of one), Fuggle N (four of four), and *Humulus lupulus* var. *neomexicanus* (two of two tested plants). The ApMV occurrence in the Czech cultivars was later confirmed by Svoboda (1993).

Symptomatology

The symptoms caused by ApMV are variable on different host plants and differ in expression with different virus strains.

Although most currently popular apple cultivars remain asymptomatic after infection, sensitive varieties react with a variety of leaf symptoms (Figure 1) (Fulton, 1972; Svoboda and Polák, 2010). ApMV isolates causing symptoms in apple ranging from mild to severe have been described (Fulton, 1972). ApMV-infected apple trees can develop pale yellow to bright cream-coloured irregular spots or bands along the major veins on new leaves as they expand in spring. These lesions may change in the affected leaves after exposure to summer sun and heat (Dursunoglu and Ertunc, 2008), and symptomatic leaves drop prematurely. The distribution of symptomatic leaves may be erratic throughout individual trees or limited to a single limb. Whether uneven symptom expression is caused by uneven virus distribution in individual hosts has not been recorded. The number and severity of symptomatic leaves also depends on temperature, with more severe symptoms in years with moderate spring temperatures. Most commercial apple cultivars can be affected, but the symptoms vary in severity (Posnette and Cropley, 1956). ApMV has been shown to reduce tree growth and fruit yield and to adversely affect fruit quality (Posnette and Cropley, 1956, 1959). Chamberlain *et al*. (1971) demonstrated that the reduction in growth of ApMV-infected trees, as measured by weight of wood produced during the 12 y life of the trees (from 1958 to 1968), amounted to 42%. The average total yield per tree over a 9 y cropping period (from 1960 to 1968, with exception of 1961, when the yield was not recorded) was 530 kg for ApMV-free trees and 390 kg for infected trees, representing a 7% reduction caused by disease. No significant effect on fruit size or quality was reported. Wood *et al*. (1975) showed that at the end of an 8 y trial with ApMV infected trees, severely infected trees (extensive mosaic on leaves) in full bearing produced only one-third as much fruit as mosaic-free trees. According to these authors, moderate and severe strains of the virus also caused statistically significant reductions in the average trunk circumferences and weights of individual fruit.

Ringspots have been described in pear trees after mechanical inoculation of the cultivar Beurré Hardy, but the association of ApMV with these symptoms was not confirmed, because the authors were not able to retransmit the virus from pear (Kristesen and Thomsen, 1963). According to Petrzik (2005), ApMV infection on pears is usually asymptomatic. Sporadically, faint yellow ringspots can be observed on some pear leaves, however the virus was not transmitted

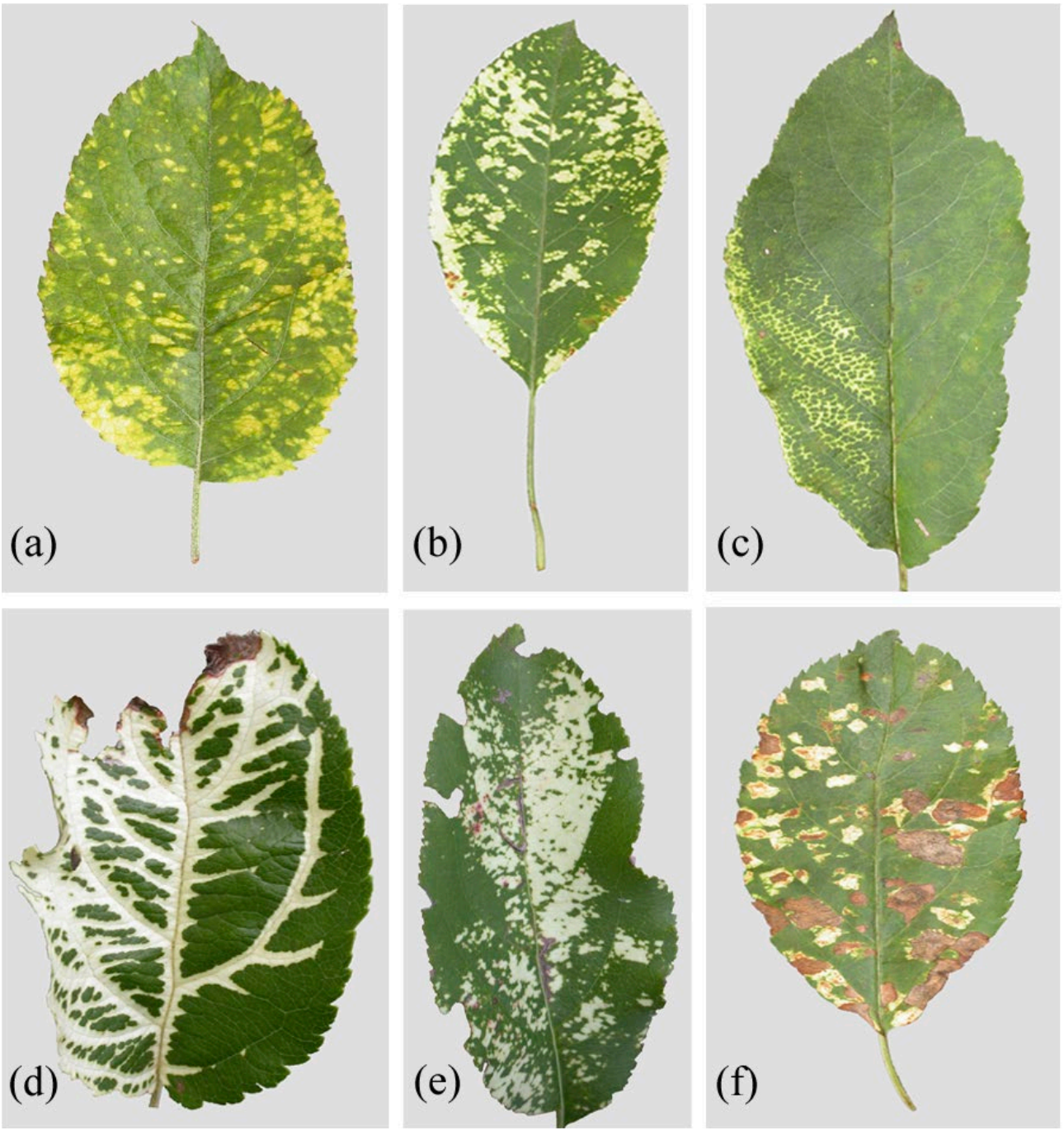

Figure 1. Symptoms associated with infection by ApMV in the form of line patterns and/or rings on apple leaves.

further to other host and thus the presence of other pathogens in the examined samples cannot be excluded.

Pale yellow to bright cream-coloured irregular patterns have been observed in European mountain ash leaves after experimental inoculation by

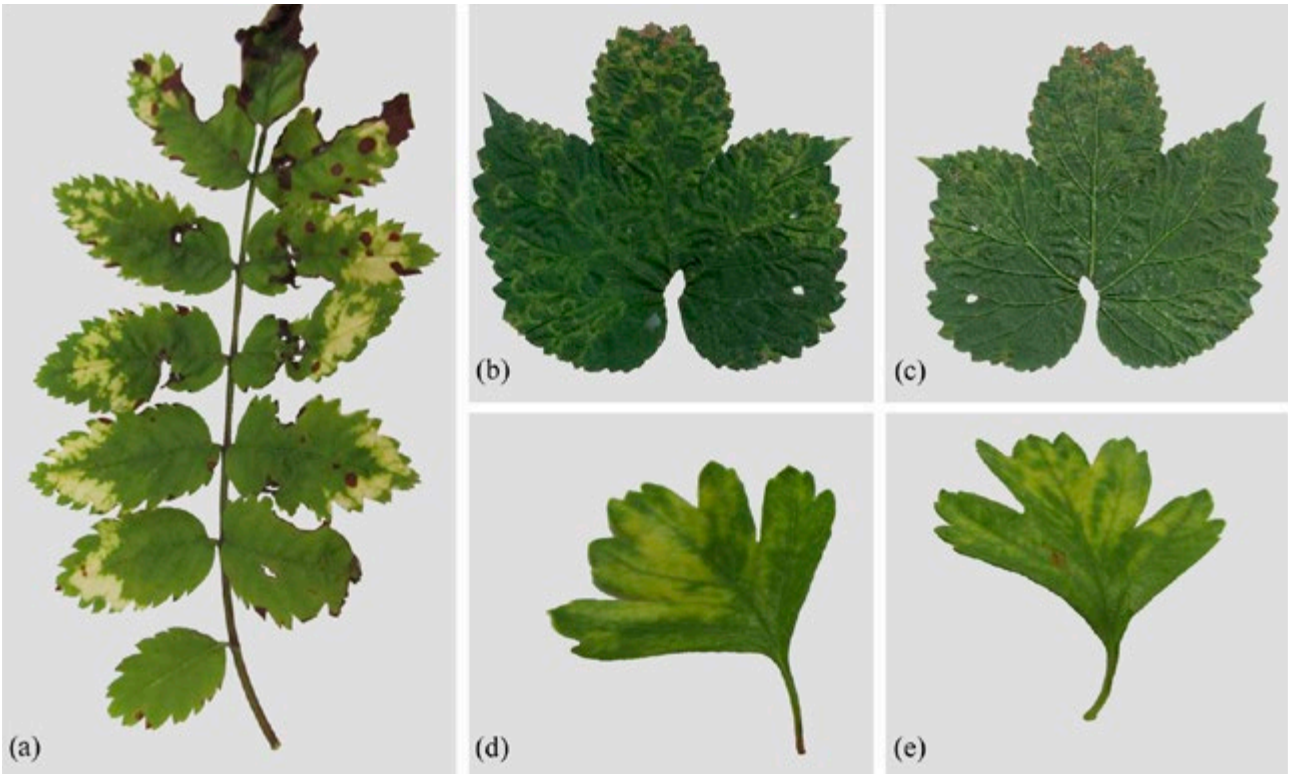

Figure 2. Symptoms associated with infection by ApMV in the form of line patterns on leaves of European mountain ash (a), hop (b) and (c) and hawthorn (d) and (e). Photos (b) and (c) provided by Dr Petr Svoboda from Hop Research Institute Co., Ltd. (Zatec, Czech Republic).

bud grafting (Figure 2) (Grimová *et al*., unpublished data). Similar symptoms varying from well-defined ringspots, ring and line patterns to variegation consisting of whitish spots of various size and shape on ApMV-infected *Sorbus* plants were described by Polák and Zieglerová (1996).

The leaves of infected stone fruit trees (i.e., apricot, peach, and cherry) show typical yellow line patterns, bright yellow blotches, rings, bright yellow vein clearing, and oak-leaf patterns (Posnette and Ellenberger, 1957; Canova, 1960; Ellenberger, 1962; Nemeth, 1986; Diekmann and Putter, 1996). The symptoms generally appear at the beginning of summer and in some cases are present only on a limited number of leaves randomly distributed on affected plants (Paunovic *et al*., 2011). However, the symptomatology is generally not of diagnostic significance, because similar symptoms may be produced on *Prunus* spp. by other ilarviruses (e.g., PNRSV and *American plum line pattern virus*, APLPV) or by further pathogens. Some peach cultivars may fail to display any symptoms (Choueiri *et al*., 2001). Characteristic symptoms in ApMV-infected plum trees include line and oak leaf patterns with chlorotic lines and rings in leaves. The disease evoked by ApMV in plums has been named European plum line pattern disease and has be found in many regions of the world (Németh, 1986). Bright chrome yellow discolourations on leaves in the form of patchy or more or less widespread mottling, ringspot, and line patterns are caused by ApMV in almond trees (Savino *et al*., 1995). In some sensitive cultivars, the virus induces the failure of blossoming and leaf bud growth, commonly referred to as almond leaf failure (Diekmann and Putter, 1996; Desvignes *et al*., 1999). Average losses of 25% were quantified by Martelli and Savino (1997) in almonds trees affected by almond mosaic, a complex disease in which ApMV plays a major etiological role.

Postman and Cameron (1987) described ApMV symptoms on hazel trees as chlorotic ringspots and

line patterns on the older foliage. By early summer, this older foliage was hidden by new symptomless growth. Symptoms were sometimes expressed on a single branch or on one side of each affected tree, but the virus was detected throughout the tree. Akbas and Degirmenci (2009) described symptoms associated with ApMV infection that were similar to those reported by previous authors, and added new symptoms including the formation of yellow flecking, oak leaf patterns and broad vein banding. Various leaf symptoms associated with ApMV infection on hazelnut trees are shown in Figure 3. Akbas and Degirmenci (2009) found ApMV only in symptomatic trees; however, Aramburu and Rovira (2000) found ApMV symptoms in fewer than 10% of the 311 sampled trees, while the remainder of the ApMV-infected hazel trees was completely asymptomatic. Postman and Cameron (1987) validated these results by confirming the presence of ApMV in asymptomatic plants. According to Kobylko *et al*. (2005), the presence of disease symptoms may be attributed to the average and maximum daily temperatures in the first half of May (spring).

ApMV is a labile virus, so its concentration can be negatively affected by high temperatures (Matthews, 1991; Zotto and Nome, 1999). This causes masking of symptoms caused by the virus (Aramburu and Rovira, 1998) and/or reduction of viral titre in plants. However, it is not easy to precisely determine the causes of these divergences in virus load in plant tissues, specifically whether they are caused by true changes in virus concentrations or the reliability of detection methods is influenced by inhibitors.

Kobylko *et al*. (2005) reported high yield differences between healthy and ApMV-infected hazelnut trees (healthy trees yielded 77% more than infected trees) and the absence of significant differences in nut quality traits. Marenaud and Germain (1975) observed a 10 to 25% reduction in yield in France when they compared heavily-infected with less-infected 'Negret' hazelnut trees. This yield decrease was attributed to a reduction in fruit numbers. However,

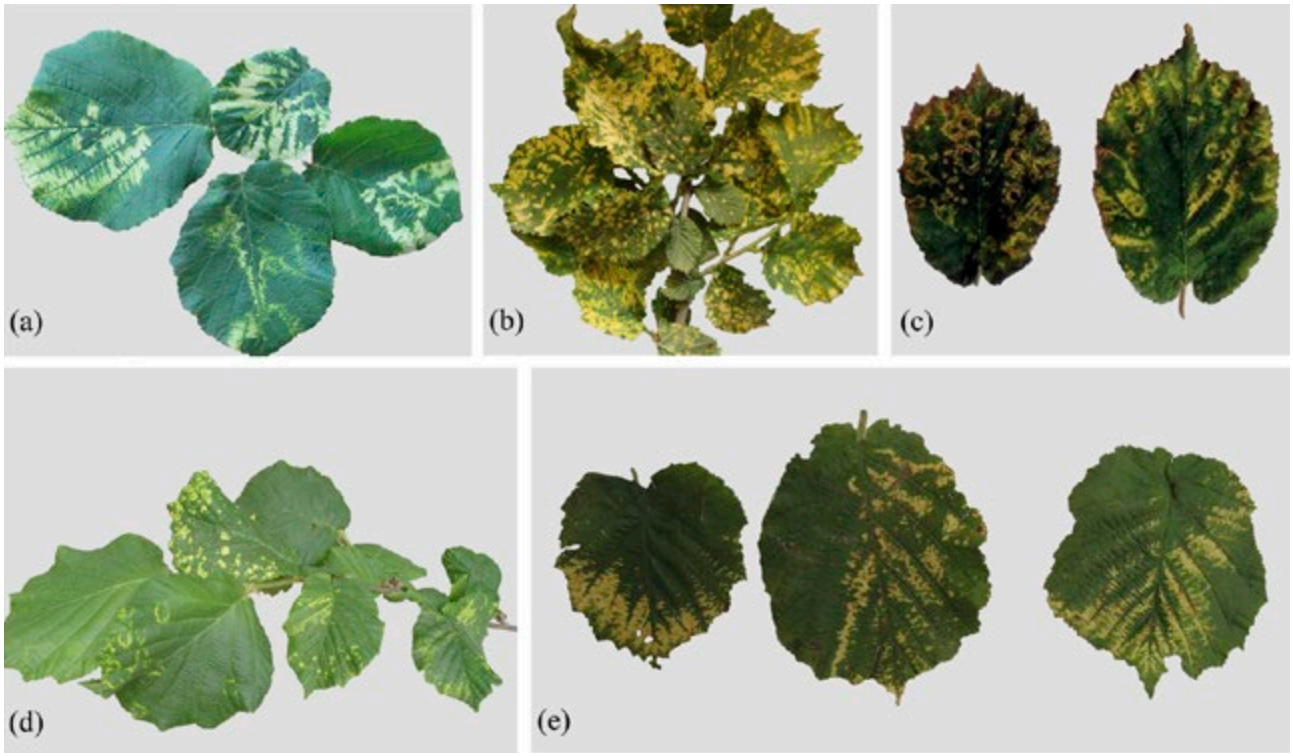

Figure 3. Symptoms associated with infection by ApMV in the form of line patterns and/or chlorotic rings on hazelnut leaves. Photos (a) and (d) provided by Dr Mercè Rovira from IRTA (Constantí, Turkey) and photos (b), (c) and (e) provided by Dr Miray Sokmen from Ondokuz Mayis University (Samsun, Turkey).

Akbas and Degirmenci (2009) reported that nut cluster weight was reduced (on average by 28%) in ApMV-infected hazelnuts, but nut size was also decreased and many clusters contained empty nuts. These authors also described the marked influence of ApMV on the slightly reduced growth of infected shrubs (as estimated visually), which was more prominent plant older than 6.

ApMV has been associated with yellow mosaic disease of horse chestnut (*Aesculus* x *carnea* and *A. hippocastanum*). The symptoms occur throughout the canopy on some trees, whereas on other trees the symptoms are restricted to sectors of the canopy. Spatial and temporal patterns of flowering were not substantially affected by ApMV infection (Figure 4) (Sweet and Barbara, 1979).

Most of the woolly blackberry (*R. canescens*) plants found in Turkey and infected by ApMV showed symptoms of yellow flecking (Arli-Sokmen *et al*., 2005). Baumann *et al*. (1982) described some ApMV-infected raspberry plants (*R. idaeus*) growing in Germany that exhibited apparent line patterns and/or yellow speckling. In contrast, they found that ApMV-infected berry plants (i.e., *R. canescens, R. idaeus* and *R. ursinus*) in North America were symptomless. Leafroll symptoms observed in strawberries have also been attributed to ApMV infection (Tzanetakis and Martin, 2005).

Figure 4. Symptoms associated with infection by ApMV in the form of line patterns on chestnut tree leaves (Sweet and Barbara, 1979). Photo provided by *Annals of Applied Biology*.

Symptoms of ApMV in hop range from chlorotic ringspots (Figure 2) that can become necrotic to oakleaf line patterns to latency. Symptoms are usually found after a rapid change in temperature (Barbara *et al*., 1978). ApMV is regarded as one of the most deleterious viruses to hop production, but the effect is dependent upon the cultivar. Some ApMV-infected varieties have been reported to possess lower alpha and/or beta acids and/or lower yields. It has been reported that ApMV-free cv. Fuggle plants had greater alpha (0.5%) and beta (0.3%) acids and yields (8%) than infected plants (Anonymous, 1976). Cultivar Cascade plants free of ApMV had 2.8% greater alpha acids, 1.4% greater beta acids, and 20% greater yields. Plants of cv. Bullion had 1.5% more alpha acids and 20% greater yields when ApMV-free, but beta acids were not affected. Neve and Lewis (1975) showed that cones from ApMV-free plants grown in the United Kingdom had 0.7 to 2% greater alpha acid content than infected plants, but with no effect of the virus on cone yield. Among three cultivars, Neve and Lewis (1977) found ApMV caused an average decrease in yield of 16% and an 11% reduction in alpha acids. Similarly, ApMV reduced cone yield by 32% and alpha acid levels by 8% in cv. Wye Northdown. Neve and Thresh (1984) summarized much of this research and showed that five cultivars freed of ApMV infection had increases in yield of 3 to 21% and alpha acid levels of 4 to 16%.

In Germany, comparisons between plots of ApMV-infected and virus-tested cv. Hüller Bitter over three growing seasons consistently had reductions in alpha acid content of, respectively, 21, 18, and 22% in each year. In a later study, the alpha acid content of ApMV-infected cv. Hüller Bitter and Northern Brewer was 18 to 26% less than in virus-tested plants (Kremheller *et al*., 1989). In Australia, ApMV has been associated with an increase in softwood cutting mortality following propagation and a decrease in cone yield and levels of brewing organic acids. In cv. Nugget, plants infected by ApMV-I had 11% less alpha acids and 7% less beta acid levels. In cv. Opal, reductions in yield and alpha acid levels were reported with both ApMV serotypes when present in co-infections with the carlaviruses *Hop mosaic virus* and *Hop latent virus*. In cv. Victoria, infection by the individual viruses had no effect in the first year of the study, but significant reductions from some virus

combinations were reported, suggesting an exacerbation of the deleterious effects of viruses as plants become older (Pethybridge *et al*., 2002a). Yield reductions from ApMV-H have also been reported in cv. Super Alpha in New Zealand. In this study, these effects were attributed to reductions in individual cone weight and the numbers and weights of bracteoles, and lower alpha acid levels resulted from a smaller number of bracteoles with fewer lupulin glands (Hay *et al*., 1989).

Basit and Francki (1970) described ApMV symptoms on *Rosa* spp. as line patterns, chlorotic ringspots, chlorotic mottle, and vein banding. In many cases, all of these symptoms occurred on different leaves of individual plants. Wong *et al*. (1988) surveyed field-grown roses for visual symptoms to determine the incidence and the expression of virus infection. The expression of viral symptoms varied from year to year among the 19 surveyed cultivars. Infected rose foliage demonstrated chlorotic patches, puckering and distortion. No effect of ApMV on flower production and quality was observed.

Line pattern symptoms consisting of chlorotic lines forming oak-leaf designs, irregular rings or linear flecks sometimes accompanied by a mild mosaic were observed in white (*B. papyrifiera*) and yellow birches (*B. alleghaniensis*) (Gotlieb and Berbee, 1973). Emerging leaves on infected trees generally remained symptomless until after they became fully expanded. Leaf symptoms never developed on some of the infected trees, but on others a few leaves sometimes exhibited symptoms. Leaf symptoms rarely occurred throughout the tree crowns, but more commonly were restricted to a few leaves on a few branches. During mid-summer, chlorotic leaf tissues turned almost white, and leaf symptoms persisted throughout the growing season. Leaves displaying symptoms, leaves without symptoms on infected trees and leaves on healthy trees all abscised at about the same time in the autumn (Figure 5) (Gotlieb and Berbee, 1973).

Most of the ApMV-infected weed species were asymptomatic, but some showed virus-like symptoms (i.e., leaf chlorosis on *Salvia verbenaca* and oak leaf pattern on *Prunella* sp.) (Arli-Sokmen *et al*., 2005).

Transmission

The virus is graft-transmissible and persists in vegetative propagation material from infected trees, which most likely constitutes the main source of inoculum. The virus can be experimentally sap-transmitted by mechanical inoculation (albeit not easily) to several herbaceous plants, such as *Cucumis sati-*

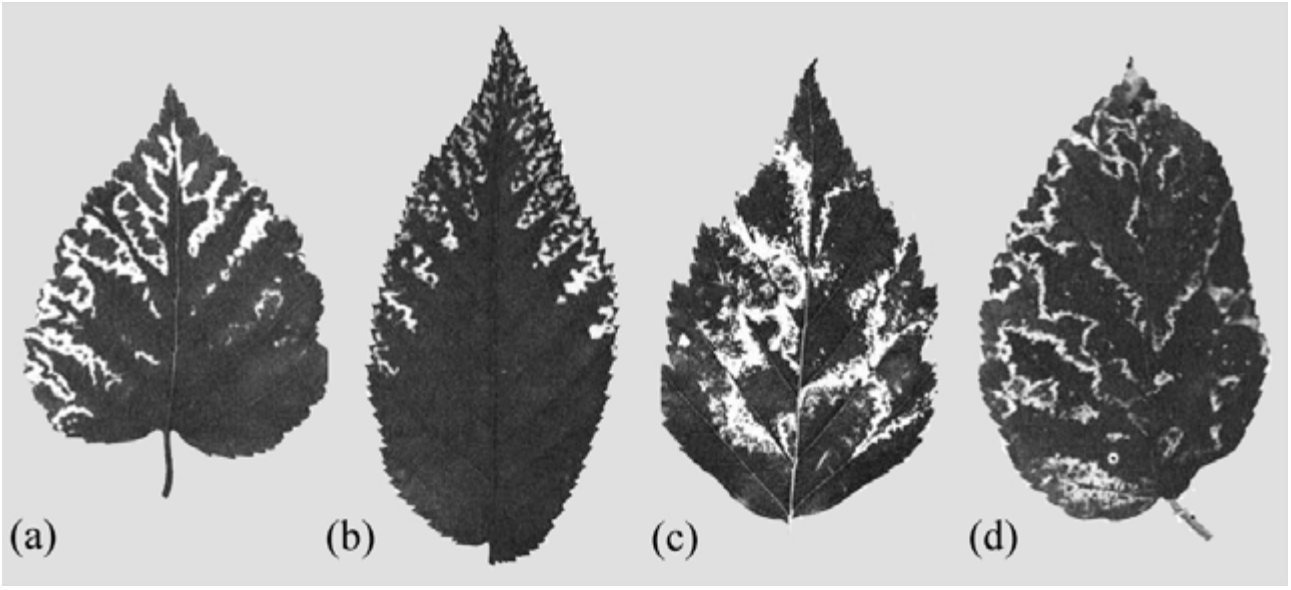

Figure 5. Symptoms associated with infection by ApMV in the form of line patterns and/or chlorotic rings on white birch leaves (a), (b), (c) and (d) (Reproduced with permission of the copyright owner from Gotlieb and Berbee, 1973).

vus, *Torenia fournieri*, *Vinca rosea*, *Vigna sinensis*, *V. unguiculata*, *Cyamopsis tetragonoloba*, *Petunia hybrida*, *Chenopodium quinoa*, *C. amaranticolor*, *C. capitatum*, *Cucurbita maxima*, *C. pepo*, *Nicotiana benthamiana*, *N. megalosiphon* and *Phaseolus vulgaris* cv. Pinto and cv. Black Turtle (Fulton, 1972; Baumann *et al*., 1982; Arli-Sokmen *et al*., 2005; Paunovic *et al*., 2011). The efficiency of mechanical transmission differs in individual hosts (at the levels both of species and cultivars), and/or depending on inoculation technique (Basit and Francki, 1970; Sweet and Barbara, 1979; Ragozzino, 1980; Bauman *et al*., 1982; Sano *et al*., 1985; Pethybridge *et al*., 2002b; Akbas and Ilhan, 2005; Arli-Sokmen *et al*., 2005).

Transmission of ApMV was also demonstrated by pruning with scalpel blades contaminated after slashing infected hop plants (Pethybridge *et al*., 2002b). ApMV transmission by dodder plants has not been confirmed; the tested plants included *Cuscuta campestris, C. gronovii, C. subinclusa* (Fulton, 1952; Gilmer, 1958) and *C. reflexa* (Nagaich and Vashishth, 1963).

Little information is available concerning the spread of ApMV in the field. However, a number of studies in different countries have reported increases in the incidence of ApMV in established apple orchards, hazelnut and rose plantations and hop gardens. Although ApMV has a wide host range, no natural vectors of ApMV are known. It is notable that other species from the genus *Ilarvirus* are commonly transmitted in association with thrips (Jones, 2005). Moreover, phylogenetic analysis of sequences of the protein products encoded by ApMV RNA1 and 2 and the putative movement protein encoded by RNA3 suggested that ApMV is more closely related to *Alfalfa mosaic virus* (AMV; genus *Aflamovirus*, family *Bromoviridae*) than to other ilarviruses (Shiel and Berger, 2000). AMV is non-persistently transmitted by 14 species of aphids (Jaspars and Bos, 1980). However, the aphids *Myzus persicae*, *Macrosiphum euphorbiae*, and *Eriosoma lanigerum* failed to induce ApMV transmission in vector transmission studies (Hunter *et al*., 1958). One possible explanation for ApMV infection could be that ApMV may have an unidentified slow-moving arthropod vector.

Another plausible means of the natural spread of ApMV could be root grafting, which is a well-known phenomenon in many horticultural crops (Graham and Bormann, 1966). Hunter *et al*. (1958) confirmed this route of ApMV transmission in apple trees. Further experiment focused on ApMV transmission through root grafting was conducted with roses (Golino *et al*., 2007). Using multiple tests to determine whether the virus would spread naturally, these authors demonstrated that an average of 10% of the healthy bushes that were planted in close proximity to virus-infected bushes became infected over the course of one year. Two years after inoculation, the infection rate ranged to 48% depending on the rose variety and virus accession. In general, the rate of virus spread within the rootstock *R. multiflora* was greater than in the varieties tested. These differences correlated with the cultivars relative root vigour. To test for the occurrence of root grafting, trials were conducted using the systemic herbicide glyphosate. A role for root grafting in ApMV transmission was also suggested by Pethybridge *et al*. (2002b), who demonstrated that root contact of hop plants did not transmit ApMV, whereas root grafting of deliberately injured and joined plants did. The presence of root grafts in a commercial hop garden was also suggested.

As mentioned above, Arli-Sokmen *et al*. (2005) detected ApMV in several weed species. The transmission of ApMV from woody hosts to herbaceous plants is usually difficult by mechanical means (Shiel and Berger, 2000). However, weed species collected during surveys were found to be growing in very close proximity to hazelnut bushes with ApMV-type symptoms. Each bush has 5–10 stem roots that can spread horizontally for approx. 1 m. Therefore, one explanation for the ApMV infection of weeds could also be attributed to root grafting, even though the species were unrelated. It was not clear whether this transmission occurred due to prolonged contact or organic connections between these species. However, it was assumed that this type of grafting would not form a functional graft union (Fulton, 1966).

Transmission of ApMV was also shown to occur via plant to plant contact by hop shoot intertwining (Pethybridge *et al*., 2002b).

Confirmation of transmission by seed and pollen is problematic, because these studies are demanding in terms of time and space requirements. No evidence was found for seed and pollen transmission in apples and roses (Golino *et al*., 2007). However, Cameron and Thompson (1986) demonstrated seed transmission of ApMV in hazelnut trees, and suggested that the occasional occurrence of mosaic symptoms on apple seedlings may also be due to this route of

transmission. Postman and Mehlenbacher (1994) demonstrated that infected female parents of hazels produced an average of 6% infected progeny (range from 1 to 2%) in 516 tests on 4- to 7-y-old seedlings from several crosses. Aramburu and Rovira (2000) reported that 15% of the un-grafted hazel trees were infected with ApMV 10 y after planting. The inoculum source was attributed to infected pollen, because it was possible to detect the virus in 8% of the seeds from healthy trees.

ApMV was detected in the embryoa and endosperms of unripe seeds of *A. hippocastanum* and *A. carnea*, but was apparently lost during seed maturation and no evidence of seed transmission was found, although ApMV was present in the anthers of *A. carnea.* However, the possibility of pollen transmission remained undetermined (Sweet and Barbara, 1979).

Diagnostic procedures

In general, the detection of plant viruses is greatly aided by the expression of disease symptoms in infected plants, but it is more difficult in plants that do not show visible symptoms or for virus infections with non-specific symptoms. Symptoms induced by ApMV vary widely depending on the natural host, the specific isolate present and the climatic conditions. Although ApMV may induce symptoms, it can also be latent or the symptoms can be masked by higher temperatures (Aramburu and Rovira, 1998). Moreover, the symptoms can be mistaken for nutritional deficiencies, toxicities or damage caused by other pests. Therefore, symptomatology alone is not reliable for definite virus identification, and further diagnostic methods are needed for ApMV detection, including biological assays using indicator plants, serological methods and molecular detection.

For woody field indexing, Fulton (1972) suggested that *Malus silvestris* cv. Lord Lambourne and Jonathan be used, on which ApMV causes prominent mosaic symptoms. Mink *et al*. (1987) further included the apple cultivar Golden Delicious. Cultivars Lord Lambourne and Golden Delicious are also recommended for indexing trees in woody tests in the field by an EPPO certification scheme relevant to the pathogen-tested material of *Malus* (Anonymous, 1999). No similar indexing recommendations exist for pears; however, the detection of ApMV in pear cultivars was reported following testing using apple seedlings as rootstocks and an apple cultivar as an indicator (Wood, 1997). ApMV can be detected in stone fruit plants by grafting onto GF 305 peach seedlings or peach cv. Elberta in the field, but testing in temperature controlled greenhouses is recommended. Both of these inoculated indicator plants display light green, yellowish, or bright yellow rings, spots, bands, or oak-leaf patterns on their leaves (Desvignes, 1976; Nemeth, 1986). The plum cv. Ersinger has been recommended in addition to the two abovementioned species within the EPPO certification scheme for almond, apricot, peach and plum (Anonymous, 2000a; Anonymous, 2000b). *Rosa multiflora* cv. Burr is a recommended indicator plant for roses (Anonymous, 2002). The herbaceous indicator plants used for biological indexing following mechanical inoculation are *Cucumis sativus*, *Chenopodium quinoa*, *C. amaranticolor*, *Cucumis sativus*, *C. pepo*, *Phaseolus vulgaris*, *Petunia hybrida*, *Torenia fournieri*, *Vinca rosea* and *Vigna sinensis* (Fulton, 1972; Arli-Sokmen, 2005; Paunovic *et al*., 2011). However, biological indexing on herbaceous indicators is of limited sensitivity, and therefore is not reliable enough for precise virus detection (Paunovic *et al*., 2011).

Specific monoclonal and/or polyclonal antibodies are available for routine diagnosis of ApMV. Original serological detection of the virus was by gel diffusion assays (De Sequeira, 1967), but the routine technique for detection is now ELISA (Clark and Adams, 1977). However, these tests are reliable only during a short period of the year, because ApMV, as a member of *Ilarvirus* group, is a labile virus (Llácer, 1978) whose titres in its hosts can be negatively affected by high temperatures within growing seasons (Matthews, 1991; Zotto and Nome, 1999). Moreover, the minimal growth of shoots and leaves during the hot season does not favour increases in virus titres. Torrance and Dolby (1984) reported that ApMV could be reliably detected in apple trees by ELISA from April to June. Svoboda and Polák (2010) reported that the highest relative concentration and therefore the greatest credibility of virus detection was obtained with leaves in April before flowering; moreover, leaves sampled later mostly showed a continuous decreases in relative concentrations of ApMV until reaching zero values in July. Another aspect that could also affect the ELISA results is the variation in the type and age of the plant organs used for virus detection. Torrance and Dolby (1984) discovered that absorbance values using ELISA for ApMV detection were greater for young leaves than for ma-

ture ones. Svoboda and Polák (2010) reported high virus concentrations in young leaves and flower petals, as did Turk (1996). In contrast, Matic *et al*. (2008) showed that ApMV was detected by ELISA in most samples of dormant buds collected in November from stone fruit trees grown under Mediterranean climatic conditions, while only 10% of the leaf samples from these trees tested positive during the hot season in spring and summer. The reason for these divergences could be due to the dissimilar regional climatic conditions that affect ApMV thermolability. There is also the possibility that ApMV may be unequally distributed in its hosts. Although Torrance and Dolby (1984) did not find evidence for uneven distribution of the virus in host plants, Gruntzig *et al*. (1994) classified ApMV in a viral group with only partly systematic distribution in plum varieties. In agreement with previous authors, Aramburu and Rovira (2000) described erroneous negative results obtained from ApMV-infected hazels, although the field surveys were conducted in spring when pathogen detection in leaves was demonstrated to be more reliable.

Another possibility for ApMV detection is the use of more sensitive molecular techniques such as RT-PCR to amplify sections of the viral genome. Reports have described the use of a standard two-step RT-PCR (Rowhani *et al*., 1995), multiplex RT-PCR (Saade *et al*., 2000; Menzel *et al*., 2002) and one-step RT-PCR (Sanchéz-Navarro *et al*., 2005), or their use in combination with serological detection of the amplified products (RT-PCR-ELISA) (Candresse *et al*., 1998). Molecular detection can be optimized through the automatic purification of nucleic acids from pathogens by columns or robotics. Tests on apple trees showed that these techniques based on "*in vitro*" amplification of the viral genome are reliable for longer periods of the year compared to serological techniques, with reliable detection extending from early spring to autumn. Akbas and Degirmenci (2010) preferred RT-PCR to ELISA for ApMV detection in hazelnut trees. However, the low ApMV titres in the tested material still constitute the limiting factor in these analyses. It was reported that plant defense mechanisms that specifically target viral RNA (silencing) are more active at high temperatures (Szittya *et al*., 2003; Chellappan *et al*., 2005). Therefore, the combination of this factor with the alleged lower viral replication during the hot months of the year may explain the differential virus behaviour throughout the year. Another possibility for more sensitive detection and quantification of ApMV from biological material is real-time PCR, which can be accomplished via absolute or relative quantification (Bustin and Nolan, 2004; Gadiou and Kundu, 2012).

A non-isotopic molecular hybridization assay has been developed to detect ApMV by Herranz *et al*. (2005), and a tissue print (TP) hybridization assay has successfully been used (Matic *et al*., 2008). The disadvantage of TP is that the method analyzes only a small portion of the host tissue (only the printed area), which could result in a false negative if the potentially uneven virus distribution in trees is taken into consideration. The more recently described oligonucleotide microarray hybridization has also been applied for the detection of ApMV. The indirect labeling method showed the greatest specificity among the different types of fluorescently labeled targets (Lenz *et al*., 2008). However, diagnostic chips are not currently commercially available.

Control

Preventive measures to avoid planting of contaminated material are of the greatest importance in the context of integrated approaches to control of diseases caused by ApMV. Among such measures, testing of planting material for pathogen-free status is an important, although not exclusive, method for controlling viral diseases of plants. Because many viruses remain latent in the planting material or occur in low numbers, detection methods with high sensitivity, specificity and reliability are required (López *et al*., 2003).

Various techniques have been developed to eliminate viruses from various plant species. In the past, heat treatment in growth chambers was the most common method used to eliminate virus pathogens, because many viruses are sensitive to elevated temperatures (Kassanis and Posnette, 1961). Thermotherapy was performed on the apple cultivar Jonathan. Buds from virus infected trees were grafted to healthy trees, which were then placed in a greenhouse and incubated at 37°C for 28, 30, 36 and 40 d. The trees did not show any symptoms in the following year, although symptoms were present on trees that were the sources of the buds (Hunter *et al*., 1959). However, the plants were not tested after the first year; this could represent a problem because some authors have demonstrated that symptoms

do not appear until the second year after grafting (Posnette and Cropley, 1956). Bhardwaj *et al*. (1998) described that complete ApMV inhibition was observed with hot water treatment of wood scions at 47°C for 30 min and 50°C for 15 min. This finding is in agreement with Pandey *et al*. (1972), who reported that bud take was greater at 50°C when exposed for a short duration compared with a long one. Baker (1962) suggested that some causes of plant damage from treatment in water at the temperatures necessary for therapy included leaching, water soaking and asphyxiation of host tissue. Additionally, such treatments may break or increase the plant dormancy period. Pandey *et al*. (1972) reported that exposure to 50°C for 8 to 12 min was completely effective in inactivating ApMV. The 15 min treatment also inactivated the virus, but the resulting bud take was poor. Similar results of ApMV inactivation were revealed by ELISA testing when plants were exposed to temperatures of 40 or 50°C for, respectively, 30 and 15 min. Navarro (1988) showed that virus-free plants could be produced by thermotherapy of infected plants at 37°C for 3 to 4 weeks. Following dry heat treatments, a decrease in the survival percentage was observed concomitant with the increase in temperature and duration of exposure. The observations in several studies have suggested that the survival of infected plants in the leaf chambers was affected by plant age, time since potting and seasonal affects (Bolton, 1967; Kassanis, 1954; Nyland, 1960). Lower temperatures and shorter duration heat treatments failed to eliminate the virus from the plants. The exposure of plants to hot air at 37°C for 4 weeks and 40°C for 2 weeks eliminated ApMV from plants, as determined by ELISA. Other studies have reported similar results (Posnette and Cropley, 1956; Holmes 1960; Hansen and Denby, 1978; Lenz *et al*., 1983; Bhardwaj *et al*., 1998). Vine and Jones (1969) applied heat therapy to hop, exposing plants for 2 to 4 weeks at 35°C and raising tips of up to 5 mm long in a culture medium to obtain plants free of ApMV. Adams (1972) also obtained ApMV-free plants by growing excised tips (1 to 5 mm) from plants treated at 35°C for 10 d.

Tissue culture therapy has partly replaced heat treatment to eliminate crop viruses. Meristem tip cultures have been frequently used to obtain virus-free plants, because the virus titre has been observed to be low or absent in meristematic regions in a large number of plant species (Laimer *et al*., 1988; Welander and Huntriesier, 1991). According to Bhardwaj *et al*. (1998), ApMV could be detected in cultures raised from meristems longer than 0.2 mm, which represents the preferred length to enhance the rate of meristem survival and increase shoot regeneration.

Barba *et al*. (1992) compared *in vitro* micrografting to thermotherapy to evaluate which method was more effective for ApMV elimination from stone fruits, and found that ApMV was more easily eliminated by micrografting. Plum plants and some peach varieties suffered from the heat treatment, and the apical shoots were not suitable for grafting onto healthy rootstocks; in contrast, no problem arose during micropropagation.

Recovery by tissue culture was also tested on red raspberry plants. During experiments with meristematic cultures, it was demonstrated that a correlation existed between the elimination of virus and explant size. The recovery was successful only with small axillary buds (0.5 mm) where it reached 87%, but with large buds (2 to 3 mm) only 25% of plants were virus-free. Complete elimination of ApMV was achieved using apical meristems. However, the number of established plants used in the study was small (Theiler-Hedtrich and Baumann, 1989). Virus-free plants can also be produced by *in vitro* shoot tip grafting (Navarro, 1988; Barba *et al*., 1992). Micro-shoot tips approximately 0.2–0.8 mm in length and each consisting of a meristematic dome plus a few leaf primordia were used to eliminate ApMV from infected roses. However, the survival and rooting in tissue culture was low and variety dependent. Out of twelve varieties, only half were regenerated. Virus testing showed that 72% of 57 plants tested negative for ApMV (Golino *et al*., 2007).

Cross protection, a type of induced resistance developing in plants against viruses, during which a prior infection with one virus affords protection against closely related ones, has been tested on the apple cultivar Jonathan (Chamberlain *et al*., 1964). Healthy trees were infected by mild strains of ApMV, and these trees were then exposed to viral infection by moderate and severe strains. Trees infected by mild strains were resistant to further attacks by the virus

Concluding remarks

Although most ApMV-infected hosts remain asymptomatic, the presence of the virus can cause se-

vere damage, either by itself, by acting as a predisposing factor for another pathogen infection, and/or by decreasing resistance to stress factors. The wide host range of ApMV constitutes an inconspicuous virus reservoir for virus spread through plant populations. However, the mechanism behind virus dissemination is still not fully understood. This factor should be taken into consideration when designing and implementing certification schemes to restrain or eliminate potential contamination of stocks with this virus to preserve healthy tree populations worldwide.

Although ApMV was first described more than 80 years ago, much of the information concerning the virus is lacking or supported by contradictory evidence. Areas where conclusive evidence of relevant information is lacking include the mechanisms by which an extremely wide range of unrelated hosts (including lichens) are infected, the modes of transmission, virus distribution within individual plants, seasonal changes in its concentration and detectability, resistance or tolerance of hosts, and interactions with other viruses, bacteria or fungi.

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