Alfalfa mosaic virus (AMV):

Alfalfa mosaic virus is a widespread, common virus infecting a variety of plants and consisting of many strains that differ in symptomatology on various hosts (Hull, 1969). Among Umbelliferae, celery has long been recognized as a susceptible host (Snyder and Rich, 1942; Hollings, 1964). It has also been found to infect parsley and carrot (Campbell and Melugin, 1971; Wolf and Schmelzer, 1973; Douine, 1976).

Hollings (1960, 1964) studied the properties of five viruses of celery (*Apium graveolens*) in England. The isolates included cucumber mosaic and lucerne mosaic, both prevalent, celery yellow vein, celery yellow spot and celery yellow net viruses. Arabis mosaic and tobacco necrosis viruses were also recorded. Tomato aspermy and tobacco ringspot viruses were not found on celery, though affecting it in other countries. Three other viruses found had a restricted host range and difficult to purify and transmit. Celery yellow net was found occasionally in Suffolk and W. Midlands. Affected plants had bright yellow flecks and bands sharply defined along the major veins and a less pronounced yellow netting of minor veins. Plants were stunted and leaves were distorted.
Campbell and Melugin (1971) reported the natural occurrence of alfalfa mosaic virus (AMV) on two previously unrecorded hosts of family Umbelliferae viz., parsley (from which calico type of AMV was isolated) and carrot (from which an ordinary type of AMV was isolated). Both isolates produced chlorotic local lesions and systemic mottle in Chenopodium amaranticolor, chlorotic local lesions with etched rings followed by systemic mottle in Nicotiana spp. The viruses were purified by chloroform-butanol method with two cycles of differential centrifugation, followed by density gradient centrifugation. The isolates were identified as AMV on the basis of their serological reaction with AMV antiserum, particle morphology, aphid vector, host range and symptomatology.

Wolf and Schmelzer (1973) isolated alfalfa mosaic, arabis mosaic (AMV), carrot mottle, celery mosaic, cucumber mosaic, nasturtium ring spot and tobacco rattle (TMaV) viruses from carrot plants with spotting, mottling, mosaic, line and ring patterns, malformation and dwarfing symptoms. Aphid transmitted viruses were more common in GDR, while nematode transmitted AMV and TMaV were found only in the Spreewald region. Alfalfa mosaic, arabis mosaic, nasturtium ringspot, and tobacco rattle viruses were described for the first time on carrot plants. These viruses caused more than 50 per cent loss in carrot crops.

Douine (1976) isolated a new strain of lucerne mosaic virus from carrot in South Eastern France. It differed from
other strains of the virus in reaction on a range of differential hosts. The symptoms produced resembled closely that of cucumber mosaic virus. The virus was identified on the basis of electron microscopy and serological observations.

Carrot Mosaic Virus (CMY):

Chod (1965a, 65b, 66) described a new virus disease of carrot occurring in all parts of Bohemia and Moravia, Czechoslovakia. A distinct mosaic appeared in the second half of the first year, on outer leaves, inner leaves generally remained unaffected. Spots (1-2 mm), distinct in outline and varying in number were distributed all over the leaf blade, disappearing at the end of the year. In the second year the symptoms developed after 3-4 weeks growth of infected seedlings, middle and lower leaves were curled, red or orange spots sometimes appeared. Plants with large yellow spots had many weak stalks and umbels were often doubled over. Isolated yellow spots often appeared on stalks. Sometimes symptoms on older leaves gradually disappeared and the plant exhibited a typical yellow colour. Infected plants had a high turgor and remained upright in hot weather. Using cowpea as indicator plant, it was shown that the virus was unstable in crude sap and little infectivity was retained in dry leaves. The virus was transmitted by sap to 11 plants distributed in 4 families. The virus was transmitted by aphids viz. Acyrthosiphon pisum, Cavariella aegopodii and Myzus persicae to carrot and eight
other plant species in 4 families. Virus particles were filamentous, 750 nm long and appeared relatively in small concentration in carrot leaves. Symptoms, host range and the vector relationship confirmed that it was distinct from carrot motley dwarf virus and celery mosaic virus.

Kitajima camargo and Costa (1968) studied the morphology of carrot mosaic virus. In infected leaf preparations of various indicator plants spherical particles 25-30 nm, elongated particles 760 x 15 nm were observed. Characteristic inclusion bodies were observed in infected leaf tissue of coriander, a wild Apium and C. guinoa, which were less frequent and more difficult to detect in celery and parsley leaves. Presence of elongated particles 740 x 15 nm in the leaf preparations of infected indicator plants suggest that the virus belongs to potyvirus group.

Camargo et al. (1971a,b) made electron microscopic examination of cytoplasmic inclusions and cell modifications associated with carrot mosaic virus occurring in Sao Paulo, Brazil. The virus induced mosaic and malformation in carrot leaves and was transmitted by sap and aphid. Electron microscopy of thin sections of infected leaf tissue of carrot and coriander revealed the presence of cytoplasmic inclusions, in the form of dense bands 0.34 x 4 nm in size and were detectable even with light microscopy. The bands were built of thin lamellae 10-15 nm thick and closely apposed. Occasionally individual or group
of lamellae appeared free from bands producing pin wheels and rings. Carrot mosaic virus particles were sometimes observed in the vicinity of these inclusions, often parallel to their surface. These observations suggested that the virus belongs to potato virus Y group.

**Carrot Motley Dwarf Virus (CMDV):**

'Motley dwarf', an economically important virus disease of carrots, appears to have a world wide distribution. It was first reported in Australia (Stubbs and Grieve, 1944; Stubbs, 1948, 1952). Since then it has subsequently been recorded in Canada (Conners and Savile, 1948); Japan (Komuro and Yamashita, 1956); USA (Stubbs, 1956); New Zealand (Chamberlain, 1959); England (Watson, 1959, 1960); Denmark (Jorgensen, 1962); Scotland (Murant and Goold, 1964); North Ireland (Anonymous, 1964); Irish Republic (Anonymous, 1964); Germany (Heinze, 1964; Wolf, 1970; Wolf and Schmelzer, 1972). Carrot motley dwarf virus is actually caused by two viruses, carrot mottle and carrot red leaf viruses, which are transmitted together in a circulative manner by the willow aphid, *Cavariella aegopodii* (Scopoli).

Carrot motley dwarf virus was described as early as 1944 by Stubbs and Grieve from Australia. In young carrot plants it caused stunting and slight twisting of petioles, and reduction of leaflets which emerged soon after infection, followed by the appearance of a mosaic of light and dark green
areas on leaves. The petioles became twisted and the plants remained stunted and failed to produce marketable roots. Plants in an intermediate stage of development showed stunting of leaves and twisting of petioles; the outer leaves showed an irregular chlorotic mottle, replaced by marginal chlorosis, thus in turn giving way to marginal reddening. The inner leaves also showed some chlorosis. On plants approaching maturity, the petioles emerging soon after infection were distorted, twisted and shortened, so that the inner leaves had a rosette appearance. The petioles of the leaves were brittle and sometimes showed brown necrotic streaks. The disease was transmitted by Cavariella aegopodii, a common aphid pest of carrots. This virus diseases affected crops throughout Victoria, and also New South Wales, South Australia, Western Australia and Tasmania (Stubbs, 1948). The aphid vector (C. aegopodii) caused hundred per cent infection. The virus was also transmitted by grafting but not by sap or seed. Both wild and cultivated carrots were the only natural hosts. The host range included Apium ammi, A. australe, Conium maculatum, Ammi majus (dill) and Coriandrum sativum. All carrot varieties tested were susceptible. The aphid remained infective for 18 days, after 48 h of acquisition feeding. Infected roots when transplanted suffered a high rate of mortality in those which survived. Seed production was greatly reduced. The virus was named as 'carrot motley dwarf virus' (Stubbs, 1952). It was transmitted by C. aegopodii to tobacco
(Hickory pryor and Early Virginea), petunia, *Datura*, chilli and *Nemesia*. Local necrotic feeding lesions developed on older leaves after 8–13 days. Systemic symptoms developed after 20–40 days, consisted of vein clearing, distortion and downward cupping of lamina. The virus was successfully transmitted by sap from petunia to tobacco with the help of aloxite abrasive and from tobacco to tobacco and *Datura stramonium*. Carrot sap did not cause excessive injury to carrot leaves, nor did infection occur. The virus could not be recovered in carrot from infected solanaceous hosts by mechanical or aphid inoculations. Stubbs in 1956 recorded this virus disease from California, a new record for U.S.A. The disease was found in San Joaquin Valley, at Davis, in Salinas area, and in Ventura county. The virus appeared to spread far less rapidly than in Australia.

Komuro and Yamashita (1956) reported a virus disease of carrots from Kanto district of Japan. The disease appeared to be closely related to that caused by motley dwarf virus, reported by Stubbs (1948) from Australia. It was transmitted by the aphid *Brachycolus heraclei* but not by *Myzus persicae* or mechanically or by seed. The acquisition period was 1–24 h, and transmission occurred after 24 h feeding; the vector remained infective for 15 days, with no apparent latent period. Carrot was the only natural host found, however, celery was experimentally infected.
Watson (1959) isolated carrot motley dwarf virus from yellow, stunted carrot plants from Cambs, England. The virus was transmitted by an aphid in a persistent manner. The virus caused serious reduction of yield and quality of early crop (Chamberlain, 1959). It was found widely distributed in New Zealand. Carrot varieties Yates Top weight and Sweep Crop were field resistant while Chantenay, Early Nantes, Early Horn and Manchester Table were susceptible. Watson (1960) recorded the occurrence of this virus disease on carrot in 7 countries in England. Smith et al. (1960) suggested sowing of resistant varieties during carrot aphid (Cavariella aegopodii) flight periods and insecticide spraying as control measures for carrot motley dwarf virus. Early sowing caused losses up to 50 per cent in susceptible varieties.

Watson and Serjeant (1963) while studying the host range of CMDV recorded Trifolium incarnatum, Nicotina xanthi and Phaseolus vulgaris as the additional hosts of the virus. Parsnip mottle virus was found to be different from CMDV in being able to infect parsnip and celery, and being transmitted by aphids, without the aid of red leaf or another carrier virus. CMDV reduced Autumn King carrot yields by 18 per cent, Chantenay by 39 per cent and Sweet Crop by 46 per cent (Murant, 1963).
Tomlinson (1963a) studied the reaction of different carrot varieties to infection with a carrot motley dwarf virus (CMDV). Leaf symptoms were most severe on Early Nantes and Osborne Parke, and root yield was reduced by 42 and 40 per cent, respectively. The mildest symptoms were produced in All Season, Kurnella Strong Top and Dandecrop with 31, 30 and 23 per cent reduction in yield. CMDV was transmitted by Cavariella aegopodii after feeding for 15 min and even to some extent after 5 min (Tomlinson 1963b).

Watson et al. (1964) showed that motley dwarf disease of carrots is actually a disease complex, caused by carrot mottle virus (CMotV) and red leaf virus (RLV). CMotV was not transmitted to carrot by sap inoculations, but was transmitted to some species of Solanaceae, Leguminosae and Chenopodiaceae. On the other hand RLV had hosts only in family Umbelliferae, and was not sap transmissible, but was transmitted by grafting. C. aegopodii transmitted the CMotV from plants that also contained RLV. The aphids were unable to transmit CMotV alone from coriander plants, but after these plants were infected with RLV by aphids, the aphids acquired and transmitted both viruses from them. Aphids remained infective with both the viruses for 1-2 weeks and retained infectivity through molt. A minimum total of 9 h is needed for acquisition and transmission; vector efficiency increased with increase in the feeding times upto several days. However, Tomlinson (1963a,b) noted that CMDV was transmitted by C. aegopodii after feeding.
for 15 min and even to some extent after 5 min. Though sap inoculations transmitted the virus to *Nicotiana clevelandii*, it could not be transmitted to carrot either mechanically or through *Cuscuta subinetusa*. Symptoms were most severe on early Nantes and Osborne Parke and root yield was reduced by 42 and 40 per cent, respectively. The mildest symptoms were observed on All Season, Kurnella Strong Top and Dande Crop with 31, 30 and 23 per cent reduction, respectively, in yield. Murant and Goold (1964) while studying the reaction of carrot varieties to CMDV found that Chantenay type carrot, Clucas New Stump Rooted was more tolerant to CMDV.

Watson (1964) studied the transmission of parsnip mottle virus and CMDV by *Cavariella pastinacae*. The aphid transmitted the former but not the later. Goodman and Watson (1965) studied the change in carbohydrate concentrations in carrot plants due to CMDV infection. The concentrations of fructose, glucose and sucrose were increased in leaves. In roots the concentration of sucrose increased but fructose and glucose concentration dropped.

Heinze (1968) reported that reddening symptoms on carrot and celery leaves in Berlin area were caused by a virus complex with three different components, one carrot mottle virus, which was mechanically transmissible to several Umbelliferae but not carrot, and to some Solanaceae, Leguminosae and Chenopodiaceae. Also it did not infect parsnip and celery. Transmission of
this virus by *C. aegopodii* was possible only when the virus was combined with another component CRLV (probably seed borne, had only vector transmission and infected only Umbelliferous hosts). The third component parsnip mottle virus was transmitted mechanically and by *C. aegopodii* and *C. pastinancae*. It also infected celery and parsnip and induced symptoms in *Nicotiana clevelandii* and *Trifolium incarnatum*.

Murant et al. (1969) studied the properties of carrot mottle virus. The virus had a dilution end point of $10^{-3}$, thermal inactivation at 70°C and ageing *in vitro* at room temperature for 9-24 h. Electron microscopy of partially purified preparations and of ultrathin sections of *Nicotiana clevelandii* leaves revealed spherical particles of 52 nm diameter. In ultrathin sections, the particles were observed in the vacuoles associated with tonoplast. The particles were unlike any known plant virus and probably contained lipid.

Krass and Schlegel (1974) studied motley dwarf virus disease complex of California carrots and noted the presence of three morphologically distinct viruses. Samples were also taken from diseased fields in the Modesto and San Juan Bautista areas. The vector of the CMDV, *C. aegopodii* was found in infected carrot fields. In addition, large populations of *Dyaphis apiifolia* were found feeding on carrots. Aphids from diseased plants placed on healthy carrots transmitted a virus which produced yellow mosaic symptoms, although marginal
reddening associated with CMDV was lacking. The viruses were not transmitted through seed. Electron microscopic examination of partially purified virus revealed an abundance of 30 nm diameter particles, a few 50 nm diameter particles and long flexous rods (approx. 750 x 15 nm). Typical pinwheel inclusions were also observed in some ultrathin sections.

Howell and Mink (1974) observed numerous carrots with motley dwarf like symptoms in the Puget Sound, Columbia Basin and Yakima Valley areas of Washington and in West Oregon. The identity of the disease was confirmed by successful transmission by the aphid vector *C. aegopodii*.

Carrot mottle virus induced chlorotic local lesions in *Chenopodium quinoa*, dark brown local lesions in *Phaseolus*, yellow or slightly necrotic local lesions followed by systemic light and dark green mottle with slight stunting in *N. clevelandii* local silvery necrotic broken rings followed by systemic necrotic ring and line patterns in *N. tabacum* cv. xanthi NC and systemic chlorotic mottle or yellowing with necrotic flecking and moderate stunting in coriander (Murant, 1974). Species of Umbelliferae became infected with carrot red leaf virus and carrot mottle virus by aphid transmission. CMDV was transmitted by the aphid *C. aegopodii* but not by *C. pastinacae*, *C. theobaldii*, *Myzus persicae* and several other aphid species. The aphid acquired both the viruses after 30 min access to source plants, aphids given acquisition access feeds of 24 h
inoculated the virus to test plants in feeds of 2 min. In properties and particle morphology carrot mottle virus differed from all other well characterized plant viruses. It resembled tobacco mottle virus and ground nut rosette virus in its ability to be transmitted by sap and by aphids in a persistent manner, and its dependence on a helper virus for transmission by aphids. The virus had an average sedimentation coefficient of about 1.15. Spherical particles of C. 52 nm diameter were observed under electron microscope.

Murant (1975) reported natural occurrence of carrot mottle and red leaf components of carrot motley dwarf disease in Canada. He for the first time proved that both component viruses occur in North America.

Yellowing or red leaf of carrot was studied by Costa et al. (1975) in Brazil. The disease resembled carrot motley dwarf and carrot red leaf viruses, and caused losses up to 50 percent. The virus was transmitted by aphid (C. aegopodii), but not by sap or seed. Electron microscopy revealed isometric particles of 30 nm diameter and an elongated virus having 750 nm long particles. Control measures suggested include isolation of crops, vector control and the use of resistant varieties.

Howell and Mink (1976b) studied incidence of carrot thin leaf virus and carrot motley dwarf virus diseases in commercial carrots grown in Washington State during 1974 and 1975. Forty four carrot fields in 4 geographical regions were surveyed.
CMDV occurred at low incidence rates (0-16%) in all four regions, whereas carrot thin leaf virus often occurred at high rates (0-97%) in three regions surveyed in central Washington.

Elnagar and Murant (1978) studied the relations of carrot red leaf and carrot mottle viruses with their specific vector, Cavariella aegopodii. These studies confirmed the dependency of carrot mottle virus on carrot red leaf virus for transmission by the aphid. In winter, aphid transmission of both viruses was greatly increased, when the source plants received supplementary lighting, whereas carrot mottle virus infectivity of sap was not increased. C. aegopodii acquired carrot red leaf and carrot mottle viruses after minimum acquisition feeding of 2 min with a minimum latent period of 7-18 h. The viruses were retained by the aphid after molting, but were not transmitted to progeny insects. Aphids allowed 24 h acquisition, continued to transmit them at least for 12 days.

Murant and Roberts (1979) observed isometric particles of 22-25 nm diameter in ultrathin sections of leaves in the phloem tissue of chervil (Anthriscus cerefolium) infected with carrot red leaf virus. The virus particles were commonest in companion cells, occurred frequently in sieve elements and were found in phloem parenchyma.

Murant (1978) studied the transmission of two virus complexes by their aphid vector Cavariella aegopodii. The results showed the existence of a specific site of retention
in an aphid vector for the parsnip yellow fleck virus (PYFV) and its helper virus Anthriscus yellows virus (AYV). PYFV depended on some kind of helper factor produced in AYV infected plants and perhaps facilitated attachment of its particles to the site of retention or protected them against inactivation by the aphid saliva, whereas carrot mottle virus possibly depended on its nucleic acid becoming coated with carrot red leaf virus protein to prevent it from degeneration within the body of the aphid.

Ohki et al. (1979) observed spherical particles, 27 nm in diameter in the phloem tissue of carrot plants infected with carrot red leaf virus. Halk et al. (1979) made highly infective nucleic acid preparations of carrot mottle virus from N. clevelandii leaves. The infective material had the properties of a single stranded RNA. It had an apparent mol. wt. of about $1.5 - 1.6 \times 10^6$ in agarose-polyacrylamide gel. The RNA did not contain any considerable polyadenylate sequence or require an associated protein for infectivity.

CRLV was shown to be distantly related to barley yellow dwarf, beet western yellows, potato leaf roll, soybean dwarf viruses (Waterhouse and Murant 1980). CRLV was purified from red leaf virus infected, chervil by centrifuging the whole plant extract at low speed and incubating the resuspended pellets with driselase; the digest was then treated with 1 per cent triton X-100 and the virus concentrated by two high speed
centrifugations through a layer of 20 per cent sucrose. The preparation contained isometric particles C. 25 nm in diameter, had a sedimentation coefficient \( (S_{20, w}) \) of 140 S, a buoyant density in CCl of 1.403 g/cm\(^3\) and A260/A280 ratio of 1.62 (Waterhouse and Murant, 1981).

A leaf reddening of carrot and dill (Apium graveolens) was found associated with the presence of carrot red leaf virus (CRLV) in Australia (Waterhouse, 1985). The virus was identified on the basis of transmission by \( C. \) aegopodii, particle morphology, host range and serology. Carrot mottle virus was not found in any of the plants infected by CRLV.

**Carrot thin leaf virus:**

Carrot thin leaf virus, a very serious virus disease of carrot and other umbelliferae, has so far been reported only from U.S.A. (Howell & Mink, 1976a). They in the year 1973, isolated it from carrots (Daucus carrot var. Sativum) grown commercially in central Washington. Naturally infected carrot plants, had twisting and narrowing of leaves. Characteristic symptoms developed 2-3 weeks after inoculation and included distortion and narrowing of leaflet lobes of newly formed leaves, vein clearing, chlorotic spots and occasional faint mottling on some of the wider leaflet lobes of carrot and coriander. The virus infected Nicotiana elevelandii, Anthriscus cerefolium Apium australe, Coriandrum sativum, Daucus carota var. sativum,
Pastinaca sativa, Petroselinum hortense and 10 other species belonging to Chenopodiaceae, Compositae and Leguminosae. Nine species became infected, 2 displayed localised symptoms but the other seven were symptomless. The virus had a dilution end point greater than $10^{-5}$, longevity in vitro at $22^\circ C$ of 2 days, and thermal inactivation between $50-55^\circ C$. Both the aphids Myzus persicae and Cavariella aegopodii transmitted the virus in a non-persistant manner. The virus was purified using chloroform for clarification of the sap, PEG precipitation, differential centrifugation and finally by density gradient centrifugation. Electron micrographs of the virus showed flexuous rod shaped particles ranging between 550-820 nm (Modal length 736 nm). The virus was named as carrot thin leaf virus and was placed in PVY group on the basis of particle morphology, vector relationship and properties in crude sap.

Howell and Mink (1976b) studied incidence of carrot thin leaf virus (CTLV) and carrot motley dwarf virus (CMDV) diseases in commercial carrots grown in Washington state during 1974 and 1975. Forty four carrot fields in 4 geographical regions were surveyed. CMDV occurred at low incidence rate (0-16%) in all 4 regions, whereas CTLV often occurred at high rates (0-97%) in 3 regions surveyed in Central Washington. Occurrence of CTLV was also reported from Idaho. Howell and Mink (1977a & b) studied the role of aphids in the epidemiology of carrot virus diseases in Central Washington. The initial appearance of CTLV and CMDV in commercial carrot fields in
Central Washington was correlated with May and June flights of *Cavariella aegopodii* during 1974 and 1975. The subsequent spread was mainly attributed to the appearance of *Myzus persicae*, in carrot fields during July of both years. Secondary spread of CMDV apparently was limited by a wingless population of *C. aegopodii* to small group of plants located near the initial infection. Indexing surveys were also made throughout Central Washington during 1974-75 for natural weed hosts of CTLV and CMDV. CMDV was isolated only from wild carrot (*D. carota*) whereas CTLV was isolated from both poison hemlock and wild carrot. Gradients of CTLV and CMDV in commercial carrot fields adjacent to the infected weeds suggested that weeds were the primary source of both inocula in the Valla Valley. In the Columbia basin natural hosts for CTLV and CMDV were not found. However, volunteer carrots and overlapping growing seasons helped in the perpetuation of viruses.

Howell and Mink (1979) studied the effect of carrot thin leaf virus and carrot motley dwarf virus on the yield of carrots. Early infections with CMDV lowered Imperator 58 carrot root yield by 44-79 per cent and seed yields by 62-83 per cent in tests conducted in Washington from 1974-1976. CTLV reduced root yields by 14-28 per cent and seed yields by 24-28 per cent. No seed transmission of the virus was observed and germination percentage of the seeds was same, as of seeds from healthy plants.
Carrot thin leaf virus (%), (E/E, S/Ve/Ap.) infects several umbelliferae and a few species from other families, and is reported only from semi-desert areas of north-western U.S.A. The virus is transmissible by sap and by aphids (Myzus persicae and Cavariella aegopedii) in a non-persistent manner, but not by seed. The virus has a thermal inactivation point of 50-55°C, dilution end point beyond $10^{-5}$, and longevity in vitro of 2 days at 22°C. The particles are flexuous filaments 736 x 11 nm and have nucleic acid content of 4 per cent (based on 260/280 ratio 1:18). The virus appears to be weakly immunogenic (Homologous titre 1:64). The virus has many properties in common with members of the potyvirus group, but serologically unrelated to three potyviruses that infect umbelliferous crop plants (Clover yellow vein, parsnip mosaic, the type, poison hemlock and parsley strains of celery mosaic viruses) or to 10 other potyviruses (bean common mosaic, bean yellow mosaic, iris mild mosaic, peanut mottle, pea seed borne mosaic, potato virus Y, soybean mosaic, sugarcane mosaic, tobacco etch and turnip mosaic viruses). The disease causes twisting and narrowing of leaves of carrot and coriander (Howell and Mink, 1980). The virus can be detected in sap of infected plants by electron microscope serology.

**Celery Mosaic Virus (CeMV):**

Celery mosaic virus is the most prevalent of the virus diseases attacking Umbelliferous crops. The disease was first reported to occur in California infecting varieties of celeriac
and carrot (Severin and Freitag, 1938). The virus was found to be transmitted mechanically and by aphids, but no specific vector was detected. Eleven species of the aphids failed to transmit the virus. It had a thermal inactivation point of 60°C, tolerance of dilution to 1:4000 and resistance to ageing in vitro of 7 days. Six species not breeding on celeriac were capable of transmitting the disease, as well as 11 others (including Aphis gossypii, A. rumicis, Myzus persicae and M. circumflexus) found breeding on celery. Large, smooth Praque celeriac, dill (Anethum graveolens), curled chervil (Anthriscus cerefolium), caraway (Carum carvi), coriander (Coriandrum sativum), carrot (Daucus carota sativa) and single or plain parsley (Petroselinum hortense) were experimentally infected by sap and by aphids. The disease was confined to family Umbelliferae. They further reported the natural infection of celery by celery calico, celery yellow spot, celery crinkle leaf, celery yellows and tomato spotted wilt viruses. Natural infection of carrots by western celery mosaic virus was also recorded by Milbrath (1948).

Celery mosaic virus was first reported to occur in Germany by Golte (1957); in Japan by Iwaki and Komuro (1970); in Florida by Zitter (1970); in Canada by Kemp and Frowd (1975); in Hungary by Horvath et al. (1976); in Romania by Docea and Macovei (1979).

Iwaki and Komuro (1970) reported the natural occurrence of celery mosaic and cucumber mosaic viruses on carrot in Japan.
Plants infected with celery mosaic virus showed mosaic and occasionally fern leaf symptoms. Cucumber mosaic virus (ordinary strain) was found to infect carrots in Chiba and Hiratsuka province.

Wolf and Schmelzer (1973) isolated alfalfa mosaic (LMV), arabis mosaic (AMV), carrot mottle (MSV), celery mosaic (SMV), cucumber mosaic (GMV), nasturtium ringspot (TRMV) and tobacco rattle viruses (TMaV), from carrot plants with spotting, mottling, mosaic line and ring patterns, malformations and dwarfing symptoms. Aphid transmitted viruses were more common in GDR, while nematode transmissible AMV and TMaV were found only in Spreewald region. LMV, AMV, TRMV and TMaV were described for the first time on carrot plants. These viruses caused more than 50 per cent losses to carrot crops.

Kemp and Frowd (1975) isolated celery mosaic virus from stunted celery plants with vein clearing and leaf mottling, collected near Burlington, Ontario, Canada. The virus was identified on the basis of particle morphology, host range and serology.

Zitter and Tsai (1977) studied the transmission of three potyviruses viz. Fla strain of celery mosaic virus and 2 strains of water melon mosaic virus by *Liriomyza sativae*. The insect transmitted all the three potyviruses. Gracia and Feldman (1977) isolated celery mosaic virus from celery and *Conium maculatum* plants showing vein clearing and leaf mottling symptoms in
Mendoza, Argentina. Cucumber mosaic virus was also isolated from celery plants showing chlorotic mottling, line and ring patterns, leaf yellowing and reduction in leaf area with stunted or normal growth. The viruses were identified by electron microscopy, serology, host range and aphid transmission.

Buturac (1979) while studying this virus diseases of cultivated and wild Umbelliferae, recorded the natural occurrence of celery mosaic virus on celery, parsnip, coriander, carrot and parsley. In inoculation tests wild Umbelliferae were also infected.

Horvath (1979) studied the new artificial hosts and non-hosts and their role in the identification of bean common mosaic, celery mosaic (GMV) and Malva vein clearing viruses. In inoculation tests with GMV, a systemic and a new local lesion host (both Apiaceae) were found. About 13 plants useful in the separation of cucumber mosaic virus, which often occurred with CeMV were also identified.

Chod (1984) detected the natural occurrence of celery mosaic virus in Nantes carrot. The virus was transmitted mechanically to carrot, Chenopodium quinoa, C. amaranticolor, C. murale, Ammi majus and celery. Electron microscopy revealed flexuous filamentous particles of 760 nm. The isolate reacted with the antiserum to celery mosaic virus.
Walkey and Webb (1984) studied the relationship between bean yellow mosaic (BYMV), bean common mosaic ( BCMV), clover yellow vein (CYVV), lettuce mosaic (LMV), potato virus Y (PVY) turnip mosaic (TuMV) and Celery (Western) mosaic viruses using simple and relatively rapid electron microscopic serology decoration tests. A close relationship was observed between BYMV and CYVV and between BYMV. Celery mosaic virus was found quite closely related to BYMV and CYVV.

**Cucumber Mosaic Virus (CMV):**

Ronald (1961) isolated a yellow variant of CMV from carrot. The virus was identified on the manner of transmission, temperature inactivation and protection conferred on tobacco. It produced yellowish, minute confluent spots on 'Samsun' tobacco and did not resist a temperature of 20°C. The virus was named as *cucumis virus* 1 (CMV) var. *carota*.

Iwake and Komuro (1970) recorded the natural occurrence of celery mosaic and cucumber mosaic viruses on carrot in Japan. Cucumber mosaic virus (ordinary strain) was found to infect carrots in Chiba and Hiratsuka province, inducing mosaic symptoms.

Zitter (1970) found that all commercial varieties of celery grown in Florida were susceptible to cucumber mosaic (CMV) and Western Celery mosaic (WCMV) viruses. In the early stages of infection, both viruses produced similar symptoms, but
the symptoms were clearly distinguishable after 2-3 weeks. CMV produced vein clearing followed by necrotic specks, giving the plant a bronzed appearance. Petioles showed sunken brown spots and collapsed areas. Infected plants were most common during the fall and early winter months. Aphids were responsible for field spread of both the viruses. Weed hosts were the primary source of inoculum for CMV but not for WCMV, for which only celery was demonstrated as the source of inoculum.

Gracia and Feldman (1977) isolated celery mosaic virus from celery and Conium maculatum and cucumber mosaic virus from celery plants in Mendoza, Argentina. Celery plants infected with CMV showed chlorotic mottle, line and ring patterns, leaf yellowing and reduction in leaf area with stunted or normal growth. The viruses were identified by electron microscopy, serology, host range and aphid transmission.

Docea and Macovei (1979) isolated celery southern mosaic strain of cucumber mosaic virus from celery in Romania.

Kaniewski (1983) studied the properties of isolates of cucumber mosaic virus from Lupinus angustifolius and celery. The isolates were identified as distinct strains on the basis of electrophoretic mobility and isoelectric point, and on the basis of a slight difference in amino acids composition. The celery isolate contained more carboxyl groups in the protein subunit than that from lupin. Bedlan (1985) studied cucumber
mosaic virus symptoms and their variation with temperature on cucumber, melon, pepper, tomato, spinach, celery and lettuce. The importance of latent infection and weed hosts eg. Stellaria and Mentha spp. in transmission of this virus by aphids was also studied.

Some other viruses not very common on Umbelliferae:

Murant and Goold (1966) described a new disease of parsnip and proposed the name parsnip yellow fleck virus for the causal agent. Under electron microscope, particles of 29 nm diameter were observed. The virus had distinct serological relation to one from Anthriscus sylvestris. Murant and Munthe (1967) isolated another virus from parsnip, which they named as parsnip mosaic virus. It was transmitted non-persistently by Cavariella aegopodii, C. theobaldii and Myzus persicae and had a dilution end point of $10^{-3}$, a thermal inactivation point between 50-55°C and a life of 7 days at room temperature. It infected parsnip, Anthriscus sylvestris, coriander and carrot plants systemically. The virus had filamentous particles 750 nm long and failed to react serologically with antisera to other viruses with similar particles lengths.

Schmelzer and Wolf (1969) demonstrated the natural occurrence of Nasturtium ringspot virus in Indian bean (C. bignonioides) and carrot (Daucus carota). The virus was transmitted mechanically to Chenopodium spp. and various Solanaceae.
Parsnip mosaic virus (PMV) infects several umbelliferous species and a few species from Amaranthaceae, Chenopodiaceae and Scrophulariaceae. The virus is transmissible by sap and by aphids (Cavariella aegopodii, C. theobaldii and M. persicae) in a non persistent manner. The virus lost infectivity after dilution to $10^{-3}$-$10^{-4}$ or storage for 10 minutes at 55-58°C or 7-8 days at 18°C. The particles are flexuous filaments 736x14 nm. Infected coriander leaf cells contained abundant pinwheel and bundle inclusions resembling those described for potyviruses (Murant, 1972).

Fedotina (1977) observed bacilliform particles 46-52 x 23-27 nm, in the phloem tissue of yellows infected carrot containing mycoplasma like organisms.

Kralik and Limberk (1977) observed rhabdovirus like particles (265 x 90 nm), associated with cow parsnip mosaic, in ultrathin sections, as globular aggregates in the nucleus and within perinuclear space of leaf parenchyma cells of naturally infected Heracleum spondylium plants, or in leaves and flower petals of manually inoculated and infected parsley.

Bos et al. (1978) reported the occurrence of a new virus disease of celery from the Netherlands. The disease was symptomless in celery and 13 other celeriac cultivars. Of the 14 new hosts, it was latent in Anthriscus cerefolium, Nicotiana megalosiphon, pea, spinach and crimson clover. Five aphid species tested were unable to transmit it, seed transmission
of this virus was detected in cereliac (upto 34 per cent) Chenopodium quinoa (upto 67 per cent) and Amaranthus caudatus. The virus had a sedimentation coefficient of 161 S. Flexuous particles (av. length 885 nm) were found at low concentration in crude sap and at high concentration in purified sap preparation. Inclusion bodies were not observed by light microscopy or by electron microscopy.

Volvas (1978) investigated a line pattern of parsley, which was found to be caused by the RS strain of chicory yellow mottle virus. The infected plants had yellow linear spots, often associated with a yellowish green mosaic on the blade. The virus was identified on the basis of reactions to differential hosts, sedimentation in sucrose density gradient and serology.

Ohki et al. (1978) detected a new rhabdovirus infecting carrot in Kanto area of Japan. Infected plants sometimes showed vein clearing, 15-20 days after inoculation but later became symptomless. The virus was not transmissible by sap, but was transmitted by aphid Semiaphis heraclei, in a persistent manner to celery, Cryptolaenla japonica and carrot. Electron microscopy of ultrathin section of infected carrot leaf tissue revealed virus particles 220 x 70 nm, in cytoplasm and on the surface of the nuclei. The virus was named as carrot latent virus.

Srivastva et al. (1979) recorded the occurrence of a new virus disease of Ammi majus in India. Infected plants showed
bright yellow ring and line pattern mosaic on the leaves. The virus induced chlorotic local lesions in \textit{C. amaranticolor}, a transient mosaic in tobacco, mild mosaic in \textit{Zinnia} and a temporary vein yellowing in \textit{Datura stramonium}. Bos \textit{et al.} (1979) described parsley latent (PLV), a new seed transmitted virus, prevalent in the Netherlands. The disease was isolated from 38 out of 54 samples of seed parsley (\textit{Petroselinum crispum}), of 17 out of 24 cvs. and from all 5 European countries tested, but not from some samples from USA. It could easily be detected in seedlings and also in seeds germinated on moist filter paper but not in dry seeds or seeds soaked in water. The virus was symptomless in parsley and caused latent systemic infection in \textit{Gomphrena globosa}, three cvs. of \textit{Spinacea aleracea} and often weak and transient systemic infection in \textit{Chenopodium amaranticolor}, \textit{C. giganteum}, \textit{C. glaucum} and \textit{C. quinoa}. The virus could easily be transmitted mechanically, but not by 7 aphid species tested. The virus had a dilution end point between 100-1000, thermal inactivation point between 55 and 60°C and ageing in vitro of 7 days. Virus particles were spherical of Ca 27 nm diameter with sedimentation coefficient of 127.5 S, buoyant density of 1.449 gm/ml and had a RNA content of 36 percent.

Twardoweiz Jakusz \textit{et al.} (1983) reported the natural occurrence of peanut stunt virus on celery in Poland. The symptoms of celery included chlorotic discolorations readily spreading from the leaf blade base along veins. Chlorotic
spots and rings 2-3 mm in diameter were often observed. The virus had a thermal inactivation point between 75-80°C, a dilution endpoint between 1:5000-1:10,000 and longevity in tobacco sap, of 2-4 days and in pea sap of 9-10 days. The virus was identified on the basis of host range, biophysical properties and serology.

Dijk and Bos (1985) described a new virus disease of carrot, chervil, coriander, dill and some wild Umbelliferae from Netherlands. The disease was named as 'Viral dieback' and was identified as Anthriscus strain of parsnip yellow fleck virus (PYFV). Diseased carrot plants developed necrosis in axillary shoots, followed by death; the tap roots appeared normal. The virus was transmitted from field by C. aegopodii to chervil and or by sap to Ammi majus C. amaranthus, C. quinoa, Gomphrena globosa, N. bentha-miana and N. clevelandii. During 1981-84 seventy one seed plants and 73 ware plants of carrot having a wide range of symptoms were tested. Fifty seven isolates were identified as Anthriscus strains of PYFV, 36 of CRLV or CMotV. Plants containing PYFV often showed necrosis and yellowing in sprouts and umbels or apical leaves. Fibrous lateral roots and tip of the tap roots occasionally showed dieback.

Duifus et al. (1986) studied a new yellowing disease of lettuce, sugarbeet, carrot, cucurbit and other crops prevalent in the desert areas of SW USA (Arizona and California). The
virus, named as lettuce infectious yellows virus (LIYV), was transmitted by white fly (*Bemisia tabaci*) in a semipersistent manner, but not mechanically. It had a wide host range infecting 45 plant species distributed in 15 families, and caused economically significant losses in a number of important crop plants. The virus was purified by differential centrifugation and density gradient centrifugation. Purified virus had an A260/A280 ratio of 1.28 and electron microscopy revealed long flexuous particles of 13-14 x 1800-200 \text{nm}. The host range, particle size, insect transmission and serology distinguished LIYV from previously described viruses.