VIRUS DISEASES OF CHRYSANTHEMUMS IN THE MANAWATU

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ABSTRACT

A survey of the virus diseases of chrysanthemums in the Manawatu revealed that tomato aspermy virus (TAV) and chrysanthemum stunt were present in most cultivars grown for cut-flowers, often resulting in unmarketable flowers. Latent infection of both viruses was revealed in most cultivars tested by sap or graft indexing to suitable indicator plants.

Rod-shaped particles of the leaf mottle group were observed in leaf-dip electron microscopic examinations from most cultivars examined, although leaf symptoms were not widespread.

Characterisation of TAV revealed that the virus could be sap and graft transmitted and was aphid transmitted in a stylet-borne manner. Host range and *in vitro* properties of TAV did not differ sufficiently from overseas reports to warrant the designation of a new strain of TAV. The addition of DIECA to chrysanthemum triturates markedly increased the infectivity of TAV in chrysanthemum sap.

Graft indexing for chrysanthemum stunt, using the cultivar 'Mistletoe' was found to be more reliable than sap inoculation to either 'Mistletoe' or cineraria, or the starch local lesion test. Defoliation of the 'Mistletoe' scions reduced the latent period of chrysanthemum stunt from 4 to 2 months.

The presence of rod-shaped particles ca. 690 x 15 nm in leaf-dips from many chrysanthemums, and from inoculated petunia leaves showing yellow local lesions suggested the presence of one or more of the leaf mottle viruses. Graft indexing with 'Good News' chrysanthemums gave symptoms of mosaic infection similar to those described in the U.S.A., suggesting a relationship between the mosaic and leaf mottle groups. The leaf mottle virus infecting chrysanthemums in the Manawatu was found to be aphid transmitted

and of the non-persistent or stylet-borne type. Erratic symptom expression in Petunia hybrida Vilm. necessitated the use of leaf-dip examinations to determine the presence of virus particles in the inoculated petunia leaves during the assessment of the in vitro properties of the leaf mottle group.

Suggested control measures include indexing to obtain virus-tested chrysanthemum stock plants, and the prevention of re-infection from outside sources by the aphid vectors. Preliminary investigations showed that meristem-tip culture is feasible for elimination of TAV and viruses of the leaf mottle group from totally infected chrysanthemum cultivars, but that treatment during "hardening off" is critical for plantlet survival.

CHAPTER 1.

INTRODUCTION

Chrysanthemums are one of the most popular ornamental crops grown. The cultivated chrysanthemum grown for cut-flower production belongs to the species Chrysanthemum morifolium Ram. but most cultivars have been extensively hybridised with Chrysanthemum indicum L. and Chrysanthemum nipponicum Hort. The intensive culture of chrysanthemums has resulted in increased flower numbers and better quality flowers, and attention is now being given to virus diseases and their effect on flower quality and plant vigour.

There are at present some 20 viruses or virus-like agents known to infect chrysanthemums, and some of these are reviewed in the following section.

1.1 A REVIEW OF THE LITERATURE ON VIRUSES RECORDED IN CHRYSANTHEMUMS

1.1.1 Aster Yellows Group */*:*/*:*/*:S/Au

One of the first reports of a disease of chrysanthemums attributed to a virus or virus-like agent, was by Nelson (95). He described symptoms appearing in chrysanthemums, similar to those described for aster yellows, and concluded that one of the yellows-type infectious agents was present. He called this disease chrysanthemum yellows. Kunkel (83) demonstrated that the causal agent of aster yellows was also present in some chrysanthemum cultivars and appeared to cause symptoms similar to those described by Nelson (95). These included the production of small green flowers and weak spindly lateral shoots, while thin

weak basal shoots with shortened internodes were shown to be signs of infection in plants which flowered normally. The infected plants usually died within a few months, but cuttings from recently infected plants often exhibited weak terminal growth and shortened internodes (15).

Similar symptoms have been reported in England by Hollings (51), where green flowers developed on infected 'Balcombe Perfection' chrysanthemums, together with spindly growth and root necrosis. Graft transmission of the causal agent was possible, but Hollings reported no leaf-hopper transmission. When symptoms of flower distortion did not appear on grafting infected plants to 'Blazing Gold', Hollings concluded that the symptoms were typical of the aster yellows group rather than flower distortion and called the disease chrysanthemum green flower (51).

In recent years aster yellows appears to have disappeared from commercial crops, possibly because of efficient control of the leaf-hopper vector, and the self-eliminating tendency (through unsuccessful propagation) of the infected cuttings.

The causal agents of 'yellows-type' diseases have long been thought to be viruses, but now there is considerable evidence casting doubt on this. In 1967, Doi et al (33) in Japan, and subsequently workers in other countries, reported mycoplasma-like organisms (MLO) in the phloem of plants exhibiting a variety of symptoms, including yellowing. There are numerous reports of MLO being associated with aster yellows and with the leaf-hopper vector (after feeding on diseased plants) (5, 88). Symptoms of aster yellows are reported to regress if plants are treated with certain tetracycline antibiotics. Similar reports have been made for a number of other diseases where MLO have been implicated (71).

It would be surprising if chrysanthemum yellows (sensu Nelson) and chrysanthemum green flower (sensu Hollings) do not prove to belong to this category too.

1.1.2 American Flower Distortion */*:*/*:*/*:A/Au

Symptoms of flower distortion, attributed to a virus infection, were recorded in England by Bewley and Richards (4) and Prentice (101), and later in the U.S.A. by Brierley (11).

In England, the organism causing this distortion of flowers was shown in most cases to be a strain of tomato aspermy virus (TAV) usually in association with at least one other virus (46).

In the U.S.A., however, Brierley (11) compared symptoms expressed in chrysanthemums infected with TAV, chrysanthemum mosaic, and those showing flower distortion symptoms, and he reported that the presence of flower distortion symptoms was not always related to infection with TAV and consequently attributed these symptoms to a flower distortion virus. Further work by Brierley (12) demonstrated that the pathogen causing flower distortion was transmissible by grafting and by dodder. Root necrosis, symptomatic of 'yellows-type' diseases, generally resulted in death of the infected plants, and Brierley proposed that this American flower distortion was in fact caused by one of the 'yellows-type' organisms.

Grafting infected plants to the chrysanthemum 'Blazing Gold' was shown to be the best means of identification of flower distortion. Symptoms of tight rosetting and deformation of the younger leaves appeared within 2 months and small distorted flowers were produced (21).

1.1.3 Tomato Aspermy Virus R/I:*/*:S/S:S/Ap

This virus (TAV) causes distinctive symptoms in tomatoes, including apical chlorosis, leaf mottling and distortion, the development of a bushy habit, a reduction in fruit numbers, and as the name of the virus implies, poor seed development (109).

Blencowe and Caldwell (6) demonstrated that these symptoms in tomatoes could be caused by a virus also present in chrysanthemums. Bewley and Richards (4) and Skillman (106)

described symptoms of leaf mottling, flower distortion and colour-breaking in chrysanthemums and named the disease chrysanthemum mosaic. However, Prentice (101) showed that mosaic symptoms were not always associated with distorted flowers and suggested that a better name for this disease would be chrysanthemum flower distortion.

Hollings (46) indexed several hundred chrysanthemums with various flower and leaf abnormalities and showed that many were infected with TAV, often in conjunction with other viruses. He named the disease aspermy flower distortion to distinguish it from the American flower distortion, and suggested that chrysanthemum mosaic (sensu Bowley and Richards) was in fact caused by a combined infection of TAV and one or more viruses of the leaf mottle group.

Smith (109) has called the virus involved in the production of aspermy flower distortion, chrysanthemum aspermy virus, and this is regarded as a strain of TAV (63).

Symptoms attributed to TAV in chrysanthemums include a reduction in flower size, untidy distorted flowers, and colour break in some of the darker flowered cultivars (97,108).

Several workers (23,37,46) have reported that Myzus persicae Sulz., together with other aphid species, in particular Aulacorthum solani Kltb. and Macrosiphoniella sanborni Gill., transmitted TAV from chrysanthemum to chrysanthemum.

The most satisfactory means of identification of TAV is the use of indicator plants, such as Petunia hybrida and Nicotiana glutinosa L. (46).

The properties of TAV in crude plant sap have been recorded overseas by several workers (23,37,46,97,99).

The results of studies on TAV present in chrysanthemums in the Manawatu are shown in chapter 3.

1.1.4 Tomato Spotted Wilt Virus R/*:*/*:S/S:S/Th

This virus, first reported in New Zealand in 1946 by Chamberlain (25), has been reported overseas as infecting chrysanthemums (1,18,80).

Symptoms in young chrysanthemum cuttings include pale chlorotic areas on the leaf tips, later spreading and turning bronze and necrotic (15).

Ie (69) described tomato spotted wilt virus (TSWV) as being an isometric particle 70-90 nm in diameter, with a thermal inactivation point of 40-46 C, a longevity *in vitro* of only a few hours and a dilution end point of 10^{-2}-10^{-3}. He also reports that TSWV has a wide host range and is mechanically transmissible, although difficult to purify.

Spread of TSWV in the field can occur by means of the thrips vector (109), although efficient insect control and roguing of young infected cuttings could account for the virtual disappearance of this virus in commercial chrysanthemum crops in the Manawatu.

1.1.5 Leaf Mottle Group

In 1952 Noordam (97) reported the presence of chrysanthemum virus B (CVB) infecting chrysanthemums in Holland, which gave chlorotic local lesions when sap inoculated to *Petunia hybrida*. Electron microscopic examination revealed the presence of rod-shaped particles ca. 600 x 30 nm. Symptoms observed in infected chrysanthemums included both flower and leaf abnormalities. Later work by Brierley and Smith (19) in the U.S.A. showed the presence of a similar virus which also gave chlorotic lesions on *P.hybrida*. They concluded that Noordam's virus B was present in a number of cultivars and was responsible for causing a disease they called chrysanthemum mosaic.

The flower symptoms attributed by Noordam to virus B were also observed by Hollings (46) in England but the presence of TAV in most plants studied made the effect of virus B on

chrysanthemum flowers difficult to determine. Subsequently, Hollings (48) reported that virus B was seldom isolated from TAV-free chrysanthemums with flower abnormalities. In these few instances he attributes the flower distortion to cold weather during bud opening rather than virus B infection. The leaf symptoms in many cultivars prompted Hollings (48) to suggest the name mild mosaic for the disease caused by virus B.

During studies on chrysanthemum virus B Hollings identified a further virus, chrysanthemum vein mottle which gave chlorotic lesions on P.hybrida and of similar particle size to that later accepted for chrysanthemum virus B (ca. 700 x 17 nm). However, vein mottle was distinguished from virus B on the basis of host range and the failure of virus B strains to afford cross protection (48). Chrysanthemum vein mottle also differed from CVB in that graft inoculation to the CVB-infected chrysanthemums 'English Blazing Gold', 'Pink Mistletoe', 'Penrod', 'Balcombe Perfection' and 'Imperial Pink', gave severe leaf mottle symptoms after 4-11 weeks, and no local lesions developed on Vicia faba L. when inoculated with chrysanthemum vein mottle (48).

The justification for such a distinction on these bases could be questioned, at least until further characterisation is conducted and serological studies made.

In 1958 Brierley and Smith (22) distinguished 8 mosaic viruses in the U.S.A. on the basis of slight difference in lesion development on P.hybrida, symptom expression on the grafted 'Good News', 'Mistletoe' and 'Dynamo' chrysanthemums, and aphid transmission characteristics.

Similar differences in symptom expression in naturally infected chrysanthemums and in inoculated plants led Hollings (57) to suggest the formation of a leaf mottle group, comprised of chrysanthemum virus B, chrysanthemum vein mottle, chrysanthemum necrotic mottle, and chrysanthemum dwarf mottle, all of which had rod-shaped particles ca. 700 nm long and gave chlorotic lesions on P.hybrida. The possible inclusion in this leaf mottle group of Brierley and

Smith's mosaic viruses and Keller's virus Q (78), which also gives chlorotic local lesions on P.hybrida (19) could result in up to 13 diseases of chrysanthemums being attributed to viruses of the leaf mottle group.

Studies have shown that "chrysanthemum virus B", i.e. one of the leaf mottle group, is serologically related to the carnation latent virus and potato viruses S and M (42), and serological tests have been used by several workers to detect 'CVB' in chrysanthemums (40,99).

The variability of the symptom expression in naturally infected chrysanthemums (48), test cultivars (22) and in inoculated P.hybrida (48) makes the possibility of these leaf mottle viruses being related to, or even strains of chrysanthemum virus B, a feasible proposition. For the purposes of this study, the viruses in this group were considered as one entity and no attempt was made to distinguish the constituent components of the leaf mottle group.

In 1970, Hollings and Stone (61) reported unusual leaf symptoms in 'Golden Sundance' chrysanthemums. These included leaf flecking and chlorotic spotting, the latter areas becoming necrotic and falling out, causing a "shot-hole" effect. On grafting to 'Mistletoe' and 'Good News', severe vein mottle symptoms developed. Electron microscopic examination revealed rod-shaped particles ca. 700 nm in length, of the vein mottle type.

The relationship of this "chrysanthemum shot-hole" virus to chrysanthemum vein mottle is not known, but it is possible that the symptoms expressed in infected 'Golden Sundance' could be due to a hypersensitive reaction to chrysanthemum vein mottle, or that vein mottle is one constituent of a mixture of viruses acting to produce these symptoms.

1.1.6 Chrysanthemum Virus Q

Another virus infecting chrysanthemums and causing symptoms on P.hybrida similar to the vein mottle group, is chrysanthemum virus Q. This virus was originally reported by Keller (78) and was first seen in the cultivar 'Blanche', where symptoms of severe leaf distortion and white leaf flecking were induced when symptomless 'Blanche' was grafted to stunt-infected chrysanthemums. Grafting stunt-infected chrysanthemums to 'Blanche' gave more severe stunt symptoms and suggested the possibility of a symptomless virus in 'Blanche' which acted synergistically with stunt to cause the leaf distortion and flecking symptoms.

Brierley and Smith (19) and Miller (89) compared CVB and virus Q, and concluded that either CVB was a mild form of virus Q, or virus Q was a complex involving CVB as one of its constituents.

1.1.7 Chrysanthemum Stunt */*:*/*:*/*:S/*

The first report of chrysanthemum stunt was by Dimock (29) in the U.S.A. in 1947. The disease was first thought to be a physiological effect of continuous vegetative propagation resulting in degeneration and debilitation. A Deuteromycete fungus was also suggested as the causal organism, but further investigations revealed no relationship between the fungus and stunt symptoms (79). In 1949 Brierley and Smith (16,17) reproduced the stunt syndrome through graft and mechanical transmission from chrysanthemum to chrysanthemum and suggested that stunt was caused by a virus. Studies by Keller (79) showed that although stunt was highly infectious there were no satisfactory local lesion hosts. The most reliable indexing method involved grafting to 'Mistletoe' chrysanthemums, where distinctive white spots and flecks (measles) developed after 6-8 weeks.

Symptoms of chrysanthemum stunt have been reported in several countries including Holland (97), England (51) and Canada (80) and the disease is undoubtedly present in most countries where chrysanthemums are grown.

The symptoms of chrysanthemum stunt can vary with the cultivar infected and time of year, with symptoms being more easily recognised during periods of rapid growth - in the summer and spring (109). Three diagnostic features of stunt infection in chrysanthemums have been described (51,79,80).

> The whole plant can be up to half the normal size, with leaves and internodes proportionally smaller. The plant does not appear deformed, merely reduced in size. This difference in size is particularly noticeable during rapid forcing of the plants, in comparison with healthy plants.
>
> Flowers on infected plants develop 7-10 days earlier than on healthy plants.
>
> The flowers are smaller than usual.

Other flower symptoms, including bleaching of darker flowered cultivars, have been reported although the bleaching is usually entire rather than streaked as is often the case with TAV-infected flowers (18). In some instances, particularly with winter glasshouse crops, stunted plants may fail to flower because of their reduced vigour (15).

Some chrysanthemum cultivars have been found to give distinctive leaf symptoms when infected with stunt, and these have been used as test plants.

'Vibrant' and 'Seagull' give light pin-point flecking 1 month after graft-inoculation, and 2-3 months after sap-inoculation with stunt (79).

'Blazing Gold' gives a diffuse yellow veining in the young leaves, 6-8 weeks after graft-inoculation (15).

'Mistletoe' produces distinctive chlorotic leaf flecking when infected with chrysanthemum stunt (18).

Virus Q-infected 'Blanche' gives pronounced leaf distortion when infected with chrysanthemum stunt, and was used by Keller (79) in his studies of chrysanthemum stunt. He found that the latent

period in 'Blanche' could be reduced from 3-5 months to 2 months by removing most of the leaves above the graft union.

Hollings (51) reported that symptoms similar to those described by Keller (79) had been observed in chrysanthemums in England since 1952. However, the causal agent of stunt in these plants was less infectious than that described in America. Furthermore, Hollings was able to eliminate stunt from the English chrysanthemums by heat treatment, a result he was unable to achieve with "American" stunt. He also reported an increase in the incidence of the "American" stunt in England and attributed this to the increase in popularity of chrysanthemum cultivars bred in America (51).

Serological studies (51,79) have shown a lack of serological activity of preparations from stunt-infected plants and this fact combined with work on purification of the causal agent of stunt (52,67) have led Hollings to hypothesise that the causal agent is not a virus, although many properties suggest this (113). Using extraction procedures similar to those used by Diener and Raymer (28) in their characterisation of potato spindle tuber, Hollings and Stone (61) have succeeded in extracting infectious material similar in properties to potato spindle tuber (28). Incubation of the infectious extract with ribonuclease destroyed infectivity, the infective principle was not precipitated with 8% polyethylene glycol plus 2% NaCl, and no virus-like particles could be seen in electron microscope examinations of infective preparations. These results support the possibility that chrysanthemum stunt 'virus' is an uncoated nucleic acid (61).

1.1.8 Other "Viruses" Infecting Chrysanthemums

Most of the other reports of virus-like diseases of chrysanthemums involve the occurrence of symptoms in a few chrysanthemum cultivars, and much still remains to be established on the distribution, prevalence and characteristics of the causal agents.

Factors influencing the identification of these causal organisms as viruses, include the variability of symptom expression in different chrysanthemum cultivars, e.g. "shot-hole" symptoms in 'Golden Sundance', the possibility of strains of a virus giving different reactions on test plants, e.g. TAV, and the presence of 2 or more viruses acting together to give distinctive symptoms different to those caused by the constituent viruses, e.g. chrysanthemum stunt mottle.

Until the conditions of Koch's Postulates are satisfied as far as is possible with obligate parasites, the identification of these organisms must remain in doubt.

Some of these viruses or virus-like organisms reported to infect chrysanthemums but about which little is known, are recorded below.

Chrysanthemum stunt mottle */*:*/*:*/*:S/*

Symptoms in chrysanthemum of vein mottling, vein clearing and occasional leaf distortion, accompanied by plant stunting were described by Welsh (115). He also showed that the causal agent could be graft transmitted and proposed that the lack of early flowering symptoms, together with leaf mottling symptoms, distinguished the disease from chrysanthemum stunt, and he proposed the name chrysanthemum stunt mottle. Smith (109) however, lists chrysanthemum stunt mottle as a synonym for chrysanthemum stunt, and it is possible that the presence of one of the leaf mottling group, together with chrysanthemum stunt could give the symptoms described by Welsh.

Chrysanthemum latent "virus" */* */* */* S/*

This "virus" is apparently rare in chrysanthemums in England and has not been recorded in the U.S.A. or Europe. Symptoms on P.hybrida are similar to those expressed by TAV, but the "virus" does not go systemic. Latent "virus" differs from the leaf mottle group in host range, physical properties, and is not aphid transmitted (48).

Chrysanthemum ringspot "virus" */* */* */* S/*

This was first reported by Brierley and Smith (20) in the U.S.A. when some cultivars developed large, chlorotic ring patterns. The causal agent was shown to be graft transmitted but not aphid-borne. Grafting infected plants to 'Good News' resulted in yellow spots developing after 6 weeks and yellow blotching with leaf distortion after 6 months. Symptoms on Senecio cruentis D.C. included severe dwarfing, but attempts to seperate the causal agent from that of chrysanthemum stunt failed - the two were always found together - and it is possible that symptoms on S.cruentis were the result of a mixed infection (20). No vector for chrysanthemum ringspot has been reported (102).

Hollings (50) has reported similar symptoms in a few chrysanthemums in England and he has called the causal agent chrysanthemum ringpattern "virus".

Chrysanthemum "virus" C */* */* */* S/*

This "virus" has been reported in Holland by Noordam (97) but was found in only one plant and no further reports have been made. The infected plant showed mottling and circular spotting on the leaves, with slight distortion of the flowers. Sap inoculation to P.hybrida gave grey necrotic spots, characteristic of TAV, and also concentric ring lesions. Systemic symptoms included ringspots and yellow-white line patterns.

Chrysanthemum "virus" D */* */* */* S/*

Symptoms of flower distortion on chrysanthemums, similar to those caused by TAV, were reported by Prentice (102). Inoculation to Chenopodium amaranticolor Coste. & Reyn. gave distinctive chlorotic and ring local lesions after 3 weeks. Later work by Hollings (51) showed that chrysanthemums infected with a mixture of "virus" D and vein mottle, when grafted to 'Good News', gave symptoms of leaf mottling, flower distortion and floret necrosis within 6-8 weeks. In New Zealand, chrysanthemum "virus" D has been recorded in the chrysanthemum cultivars 'Peach Blossom' and 'Actress', by Thompson (113).

Chrysanthemum "virus" E */* */* */* S/*

This "virus" has been reported in England by Hollings (46) and causes symptoms similar to those induced by virus D, including reduction in flower size and short tangled florets often tightly packed in the centre. Flowers can be bleached and may fail to open. Grafting to 'Blazing Gold', 'Good News' and 'Mistletoe' all give similar symptoms of leaf mottling, and no vectors have been reported for this virus (105).

Chrysanthemum rosette group */* */* */* S/*

Brierley and Smith (22) reported the presence of a group of "viruses" infecting chrysanthemums which, when grafted to the cultivars 'Good News' and 'Golden Mistletoe' gave yellow mottling and leaf dwarfing, sometimes accompanied by rosetting.

The first disease in this group was observed in the cultivar 'Ivory Seagull' and the causal agent could be re-isolated to chrysanthemums from inoculated S.cruentis plants which remained symptomless (18). Kemp (80) reported a disease similar to Ivory Seagull rosette in the cultivar 'Wilsons White'.

The other disease in this group, Yellow Rayonante rosette, was first observed in the cultivar 'Yellow Rayonante' by Brierley and Smith (22). They considered that Yellow Rayonante rosette differed from Ivory Seagull rosette in that re-isolation from inoculated cineraria plants was not possible.

Chrysanthemum chlorotic mottle

Chlorotic mottle, first reported in the cultivar 'Yellow Delaware' in 1967 by Dimock and Geissinger (30) caused mild mottling of the young leaves, often followed by general chlorosis and dwarfing. Delayed flower development was also noted. Frequently these symptoms were only transient. The causal organism was shown to be graft transmissible, and electron microscopic examination of infected sap failed to show any virus-like particles (30,31).

Sowbane mosaic virus

Hollings, Stone and Bouttelli (64) have reported the presence of a virus serologically related to sowbane mosaic virus and carnation 689 virus in 26 out of 29 English chrysanthemum cultivars, and suggest that pollen transmission of the virus may occur (58).

Another virus, similar to sowbane mosaic virus has been reported in the chrysanthemum cultivar 'Rose Harrison' which had undergone heat treatment and meristem-tip culture. Purified preparations of this virus contained isometric particles 25-30 nm diameter, and gave local lesions followed by systemic mottling and distortion symptoms in Chenopodium quinoa Willd. This virus was not found to be serologically related to sowbane mosaic virus, but exhibited the same property of being difficult to transmit in crude sap, from chrysanthemum to Chenopodium spp, but in partially purified and concentrated preparations transmission to Chenopodium spp was possible (58).

The salient features of this review are listed in tables 1 and 2.

Table 1 is a summary of virus or virus-like organisms occurring naturally in chrysanthemums, including synonyms, methods of transmission, and therapy of infected plants.

Table 2 is a summary of the properties of the three main virus or virus-like diseases occurring naturally in chrysanthemums, viz. tomato aspermy virus, chrysanthemum stunt and viruses of the leaf mottle group.

TABLE 1. Viruses and virus-like organisms occurring naturally in chrysanthemums; synonyms, methods of transmission, and therapy of infected plants.

Virus or Virus Group - Synonyms		Transmission*		Therapy**	
YELLOWS GROUP					
American flower distortion	(11)	G.	(12)	H.	(21)
Aster yellows virus	(83)	G. L.	(15)	H.	(109)
- Chrysanthemum yellows virus	(95)				
- Chrysanthemum green flower virus	(51)	G.	(51)	H.	(111)
LEAF MOTTLE GROUP					
Chrysanthemum virus B	(97)	G.S.A.	(97)	H.M.	(41)
- Chrysanthemum mild mosaic virus	(48)				
- Noordam's chrysanthemum virus B	(48)				
Chrysanthemum dwarf mottle	(57)	G.S.	(61)		
Chrysanthemum mosaic virus group	(22)	G.S.A.	(22)	H.	(22)
Chrysanthemum necrotic mottle	(48)	G.S.	(61)		
- Chrysanthemum "shot-hole"	(61)	G.	(61)		
Chrysanthemum vein mottle	(48)	G.S.A.	(48)		
CHRYSANTHEMUM VIRUS C	(97)	S.	(97)		
CHRYSANTHEMUM VIRUS D	(102)	G.S.	(51)		
CHRYSANTHEMUM VIRUS E	(51)	G.	(109)		
CHRYSANTHEMUM LATENT VIRUS	(48)	S.	(48)		
CHRYSANTHEMUM RINGSPOT VIRUS	(20)	G.S.	(20)	H.	(55)
- Chrysanthemum ringpattern virus	(50)				
CHRYSANTHEMUM ROSETTE GROUP					
Ivory Seagull rosette virus	(18)	G.S.	(22)		
Yellow Rayonante rosette virus	(22)	G.S.	(22)		
CHRYSANTHEMUM STUNT	(29)	G.S.	(79)	H + M	(60) (62)
- Marmor chrysanthemi sp.nov.	(79)				
Chrysanthemum stunt mottle	(115)	G.S.	(109)		
Chrysanthemum chlorotic mottle	(30)	G.	(31)		
TOMATO ASPERMY VIRUS	(6)	G.S.A.	(63)	H.M.	(14) (55) (91)
- Chrysanthemum mosaic virus	(4)				
- Lycopersicon virus 7	(6)				
Chrysanthemum aspermy virus	(109)	G.S.A.	(46)		
- Aspermy flower distortion	(46)				
- Cucumis virus 1 strain chrys.	(97)				
- Chrysanthemum virus A	(97)				
Chrysanthemum mild mottle	(114)	G.S.A.	(114)		
TOMATO SPOTTED WILT	(18)	S.Th.	(69)	H.	(109)

* G. = graft transmission L. = leaf-hopper transmission
S. = sap transmission A. = aphid transmission
Th. = thrips transmission.

** H. = hot air treatment M. = meristem-tip culture.

TABLE 2. Some characteristics of chrysanthemum stunt, tomato aspermy virus and viruses of the leaf mottle group.

Characteristic	Chrysanthemum stunt	Tomato aspermy virus	Leaf mottle group
Cryptogram	*/* */* */* S/*	R/1 */* S/S S/Ap	*/* */* E/E S/Ap
Symptoms on chrysanthemums	Stunting, early flowering, bleaching in darker flowers (51,79)	Flower distortion (46,97,108)	Occasional leaf mottle or leaf fleck (19,48,57,97)
Diagnostic species	'Mistletoe' - yellow leaf fleck 2-8 months after grafting (79)	N.glutinosa-leaf mottle C.amaranticolor-dot lesions P.hybrida-leaf mottle (37,46,63)	P.hybrida-yellow local lesions T.expansa-yellow lesions (19,48,89)
Physical properties	TIP - 96-100 C DEP - 10^{-2}-10^{-3} LIV - 55 days @ 21 C (79)	TIP - 50-60 C DEP - 10^{-4}-10^{-5} LIV - 2-6 days (63)	TIP - 65-70 C DEP - 10^{-2}-10^{-4} LIV - 24 hours (48,97)
Particle characteristics	Possible uncoated RNA particle (60,61)	RNA - single stranded (?) isometric 25-30 nm diam. (42,65,114)	Rod-shaped particles ca. 700 x 17 nm (48,61)
Control	Heat - 14-37 weeks @ 35 C then meristem-tip culture (60,62)	Heat - 4 weeks @ 37 C or meristem-tip culture (14,55,91)	Heat - 8 weeks @ 37 C or meristem-tip culture or both (41)

1.2 CHRYSANTHEMUM VIRUS SITUATION IN NEW ZEALAND

There is very little information published on the viruses present in chrysanthemums in New Zealand. Tomato aspermy virus has been recorded in tomatoes in New Zealand (113) and Dingley (32) lists TAV as infecting chrysanthemums in New Zealand. Thomson (113) has reported the development of symptoms attributed to chrysanthemum virus D on sap inoculated C.amaranticolor leaves. These results were obtained with inoculations from two chrysanthemum cultivars, 'Peach Blossom' and 'Actress'.

The introduction of cultivars from overseas, and the prevalence of virus infection reported overseas, (54,97,99) particularly of TAV, chrysanthemum stunt and viruses of the leaf mottle group, suggest that these viruses in particular could be present in cultivars grown in the Manawatu.

1.3 OBJECTIVES OF STUDY

A study of viruses naturally infecting chrysanthemums was undertaken to determine:

a) the incidence of viruses in chrysanthemums grown commercially for cut-flowers in the Manawatu;

b) the identity and characteristics of the more commonly occurring viruses in chrysanthemums in the Manawatu;

c) approaches to control of the viruses found in chrysanthemums in the Manawatu, based on the general methods of chrysanthemum virus control outlined in chapter 6 and the virus characteristics determined in this study.