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## Virus Disease of Small Fruits

R. H. Converse

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## Dedication

*The Editorial Committee dedicates this handbook to  
Dr. Norman W. Frazier*

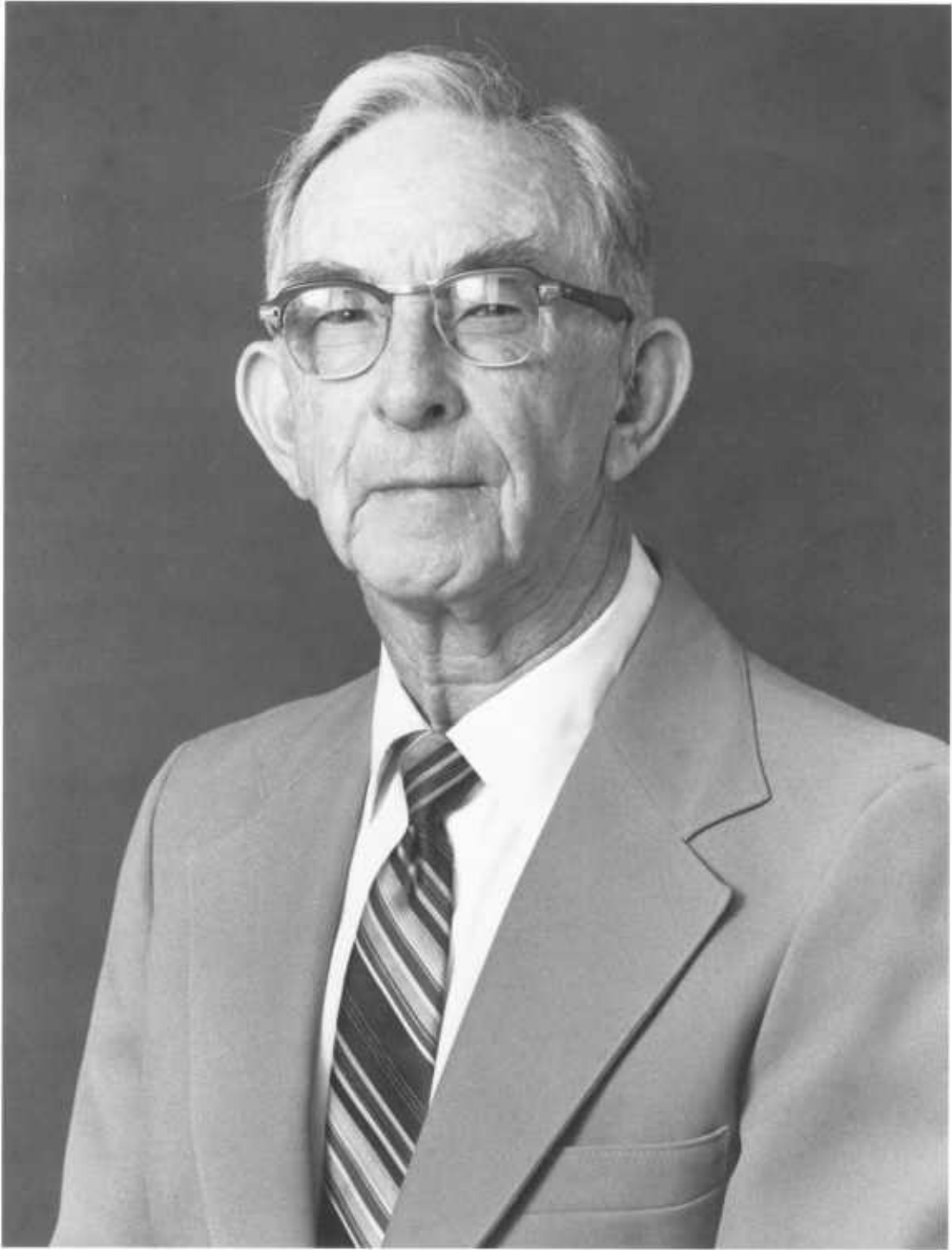
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and

Chairman, Editorial Committee, Virus Diseases of Small Fruits and Grapevines, 1970  
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Corvallis, Oregon  
August 1987



*Dr. Norman W. Frazier (1907- )*

**United States  
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Agriculture**

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Research  
Service

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# **Virus Diseases of Small Fruits**

R. H. Converse, Editor



## Abstract

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This illustrated handbook was compiled by international authorities on virus and viruslike diseases of small fruits. Crops covered are in the plant genera *Fragaria* (strawberry), *Vaccinium* (blueberry and cranberry), *Ribes* (currant and gooseberry), and *Rubus* (blackberry and raspberry). The history, geographic distribution, importance, symptoms, transmission, cause, detection, and control of virus and viruslike diseases attacking these crops are discussed.

**Keywords:** virus, viruslike disease, small fruit, soft fruit, *Fragaria*, *Vaccinium*, *Ribes* and *Rubus*, strawberry, blueberry, cranberry, currant, gooseberry, blackberry, raspberry

## Preface

This handbook is concerned with virus and viruslike diseases of cultivated *Fragaria*, *Ribes*, *Rubus*, and *Vaccinium* and is intended to supersede "Virus Diseases of Small Fruits and Grapevines" edited by N. W. Frazier and published in 1970 by the University of California Division of Agricultural Sciences. This handbook, however, considers only the virus and viruslike diseases of strawberry (*Fragaria*), blueberries and cranberries (*Vaccinium*), currants and gooseberries (*Ribes*), and blackberries and raspberries (*Rubus*). Readers interested in the virus and viruslike diseases of grapevines (*Vitis*) are directed to the recent review by J. K. Uyemoto et al. 1978, "Grapevine (*Vitis*) Virus and Virus-Like Diseases," Set 1, 29 p. In O. W. Barnett and S. A. Tolun, editors, Plant Virus Slide Series, College of Agricultural Sciences, Clemson University, Clemson, S.C. 29631. Many of those involved in writing this handbook were also authors in the 1970 handbook. Free use has been made of the 1970 handbook material in preparing this handbook; however, the present authors take full responsibility for their articles. This handbook was prepared under the auspices of the Small Fruit Virus Working Group of the International Society for Horticultural Science (ISHS).

For most papers in this handbook, reviews of the literature were completed in March 1981.

It is the intention of those of us involved in preparing this handbook to provide:

1. Information and illustrations to facilitate the identification, management, and control of small fruit virus and viruslike diseases of the major small fruit crops.
2. Citations to the important primary literature on these diseases.
3. Notes on major gaps in current knowledge of small fruit virus and viruslike diseases in the hope of encouraging additional needed research.
4. Information on nonviral disorders or abnormalities that may mimic or obscure small fruit virus and viruslike diseases.

It is the expectation of the Small Fruit Working Group of ISHS that sufficient progress will have been made in the decade following publication of this handbook to warrant a new handbook. Therefore, readers are urged to make suggestions for improvement and to correct obvious errors and omissions in this handbook to the senior editor.

I wish to express my thanks and to compliment the many specialists from all over the world who have contributed to this handbook. Their knowledge and hard work and that of the three section editors and Howard Sherman, ARS technical editor, who worked with me have made this handbook possible.

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## 24 Section 1. Virus and Viruslike Diseases of *Fragaria* (Strawberry) //

### Introduction

By R. H. Converse

The period between the publication of "Virus Diseases of Small Fruits and Grapevines" (Frazier 1970b) and the publication of this present handbook has been one of major advances in our knowledge of strawberry viruses and viruslike diseases. A few important new viruslike diseases have been described, like strawberry rickettsia yellows and mycoplasma yellows in Australia. (See "Strawberry Rickettsia Yellows and Mycoplasma Yellows," p. 41.)

Important advances have also been made in biology, characterization, detection, and control of many major strawberry virus diseases. (The term "virus" will often be substituted hereafter for the more accurate but cumbersome phrase "virus and viruslike diseases.") Important and economically damaging interactions among viruses in strawberry have been discovered, for example, between pallidosis agent and several viruses, particularly strawberry mild yellow-edge virus. (See "Strawberry Pallidosis," p. 55, and "Strawberry Mild Yellow-Edge," p. 25.) Vectors have been identified for viruses that can infect strawberry. Important virus-vector relationships have been discovered, as for instance, the sites of nepovirus attachment in the alimentary canals of some vector nematodes. (See Murant "European Nepoviruses in Strawberry," p. 46.) For another example, the ability of nonvector aphids to transmit strawberry crinkle virus that has been injected into their hemocoel has been demonstrated. (See "Strawberry Crinkle," p. 20.)

The discovery that mycoplasma-like organisms and rickettsialike agents are associated with a number of leafhopper-borne yellows diseases of plants led to their description in a number of the yellows diseases of strawberry. (See the chapters on strawberry leafhopper-borne diseases, p. 31.)

The viruslike particles associated with a number of the major strawberry virus diseases have been observed. These include: strawberry mottle (p. 10), strawberry mild yellow-edge (p. 25), strawberry crinkle (p. 20), and strawberry vein banding virus, whose relationship to the caulimovirus group was also confirmed by serological studies (p. 16).

In 1970, among the viruses infecting strawberry, only the nepoviruses could be detected by serological studies (p. 46). At present, serodetection is also possible for strawberry mild yellow-edge (p. 25), strawberry vein banding (p. 16), and tobacco streak virus in strawberry (p. 57), and promising results have been achieved for the serological detection of strawberry green petal disease agent in Great Britain (M. F.

Clark, D. J. Barbara, and D. L. Davies, unpublished data). The technologies of enzyme-linked immunosorbent assay (ELISA) and immunospecific electron microscopy (ISEM) have been added to existing methods of radioimmunoassay (RIA) to provide increased sensitivity for the detection of strawberry viruses against which specific antisera have been developed. (See "Detection and Elimination of Virus and Viruslike Diseases in Strawberry," p. 2.)

The control of strawberry virus diseases has been improved since 1970 by the application of improved methods of heat therapy and shoot apex culture for virus eradication from clones, by the development of *Fragaria* indicator clones of increased sensitivity and superior methods of leaf grafting to assess the virus status of suspect clones, and by the application of ELISA to virus detection in strawberry clones where suitable antisera had been developed. (See "Detection and Elimination of Virus and Viruslike Diseases in Strawberry," p. 2.) Preliminary steps have also been taken toward the development of strawberry cultivars that will not support or only poorly support colonization by aphid vectors of specific viruses, as well as to develop cultivars that possess genetic immunity or tolerance to virus infection. (See "Detection and Elimination of Virus and Viruslike Diseases in Strawberry," p. 2.)

Despite the progress made in the last decade in strawberry virus research, rapid methods have not yet been developed to supplement bioassay detection procedures for the detection of most of the aphid-borne viruses and leafhopper-borne viruslike diseases of strawberry. The natural means of spread, aside from clonal propagation, have not been identified for pallidosis and chlorotic fleck diseases, nor have the causal agents associated with these and a number of other strawberry diseases been characterized.

I wish to take this opportunity to thank the many research workers around the world who contributed to the preparation of the strawberry virus section of this handbook. To those who wrote, who supplied photographs, and who provided data from their unpublished research, the readers of this section are in your debt.

For animal taxa, the following sources were used:

- Aphids: Eastop, V. F., and D. H. R. Lambers. 1976. Survey of the world's aphids. W. Junk, Publishers, The Hague. 537 p.
- Cicadellidae: Nielson, M. W., 1968. The leafhopper vectors of phytopathogenic viruses (Homoptera, Cicadellidae)



taxonomy, biology, and virus transmission. U.S. Department of Agriculture Technical Bulletin No. 1382, 386 p.

Nematodes: Southey, J. F. 1978. Plant nematology. Ministry of Agriculture, Fisheries and Food. Her Majesty's Stationery Office, London. 440 p.

Arthropods: Sutherland, D. W. S., Chairman. 1978. Common names of insects and related organisms. Entomological Society of America. 132 p.

For plant taxa, the following source was used:

Kelsey, H. P., and W. A. Dayton, editors, 1942. Standardized plant names. 2d ed. J. H. McFarland, Harrisburg, Pa. 673 p.

I wish also to express my thanks to P. W. Oman, Sr., Emeritus Professor of Entomology, Oregon State University, for his considerable assistance with arthropod taxa during the preparation of this handbook.

## <sup>245</sup> **Detection and Elimination of Virus and Viruslike Diseases in Strawberry** By R. H. Converse

### **Introduction**

Wherever cultivated strawberry (*Fragaria X ananassa* Duch.) is grown, virus and viruslike diseases cause major losses in the quantity and quality of the crop. More than 28 virus and viruslike diseases (hereafter collectively called virus diseases) are discussed in the strawberry section of this handbook. This number probably is only a portion of the total number of such diseases affecting this crop worldwide (Nouriseau 1979). As noted in the description of individual diseases that follow in this section, not all are major or occur everywhere strawberries are grown. Strawberry cultivars differ markedly in their genetic susceptibility to these diseases, ranging from completely susceptible through tolerant to immune. Both tolerance and immunity to plant viruses and to their vectors can be selected. Strawberry plant breeding programs utilizing such groups of genes are being investigated at present in several laboratories (Barritt and Shanks 1980; Barritt and Daubeney 1982; Crock and Shanks 1982).

The economic loss from virus diseases as measured in decrease in yield and quality of fruit per unit area has been determined by a number of workers in studies that differ in viruses, cultivars, and locations chosen. Under the heading "Economic Importance" in each of the individual chapters of this section, data bearing on this question are presented. It is important to remember that few strawberry viruses act singly or exert their effects under ideal environmental circumstances. The interactions among cultivars, viruses in combinations or strains, and stressful environments can profoundly influence the degree of economic loss. However, even when strawberry virus diseases are so severe that yield is negligible, it is usually impossible to determine by inspecting the plants what causal agents are present. Therefore, a number of direct and indirect detection procedures (table 1) have been developed to make correct diagnoses. Careful reviews of this subject have been prepared (Fulton and McGrew 1970; Fulton 1977).

### **Methods of Virus Detection**

**Self-indicating diseases and false symptoms.** Some diseases like aster yellows, green petal, lethal decline, rickettsia yellows, mycoplasma yellows, leafroll, witches'-broom, and multiplier plant cause characteristic symptoms in strawberry cultivars. (See these specific chapters.) Problems may arise in the detection of these diseases in daughter plants that are taken from recently infected mother plants. Infected daughter plants will usually exhibit characteristic symptoms but may not do so at digging time in early spring. Lethal decline disease (Schwartz and Frazier 1964), for instance, sometimes requires extensive roguing of mother plant-daughter plant systems in late autumn and winter in the Pacific

**Table 1.—Recommended methods of detection and identification of strawberry viruses and viruslike diseases**

Disease	Diagnostic symptoms in cultivars? <sup>1</sup>	Antiserum available for ELISA or other tests?	Sap transmissible to herbaceous hosts?	Preferred indicator(s) for leaf graft transmission <sup>2</sup>	Transmission by vector useful in diagnosis?	Thermotherapy properties useful in diagnosis?	Notes <sup>2</sup>
Aphid borne:							
Crinkle	No	No	No	4, 5	No	No	Petal streak.
Latent C	No	No	No	5, EMC	No	No	
Mild yellow-edge	No	Yes <sup>3</sup>	No	4, 5	No	No	UC-6 latent
Mottle	No	No	Yes	4, 5	Yes	Yes <sup>4</sup>	by Cf
Pseudo mild yellow-edge.	No	No	No	4, 12, Alp.	Yes	No	10, 11 latent.
Vein banding	No	Yes <sup>5</sup>	No	6, 12	No	No	
Leafhopper borne:							
Aster yellows	Yes	No	No	—	No	No	Distinguish
Green petal	Yes	No	No	—	No	No	on herbaceous hosts.
Lethal decline	Yes	No	No	—	No	No	
Mycoplasma yellows	Yes	No	No	—	No	No	Distinguish
Rickettsia yellows	Yes	No	No	—	No	No	by electron microscopy.
Nematode borne:							
Arabis mosaic	No	Yes	Yes	—	No	No	
Raspberry ringspot	No	Yes	Yes	—	No	No	
Straw. latent ring-spot	No	Yes	Yes	—	No	No	
Tomato black ring	No	Yes	Yes	—	No	No	
Tomato ringspot	No	Yes	Yes	4, 5 Alp.	No	No	
Fungus borne:							
Tobacco necrosis	No	Yes <sup>3</sup>	Yes	—	Yes	No	Root sap inoculated to Cq.
Vector unknown:							
Chlorotic fleck	No	No	No	EMB, EMK	No	No	
Leafroll	Yes	No	No	5	No	No	
Witches'-broom	Yes	No	No	4, 5	No	No	
Multiplier plant	Yes	No	No	—	No	No	
Feather-leaf	Yes	No	No	Alp., 4, 1	No	No	
June yellows	Yes <sup>4</sup>	No	No	—	No	No	
Pallidosis	No	No	No	10, 11	No	Yes	
Tobacco streak	No	Yes	Yes	Alp., 4	No	No	

<sup>1</sup>The cultivar itself develops symptoms that enable the causal agent to be identified.

<sup>2</sup>Abbreviations for strawberry indicators: Numbers are for UC indicators 1-12; Alp. = *F. vesca* var. *semperflorens* 'Alpine'; EMB, EMC, EMK = various clones of *F. vesca* 'East Malling clone'; Cq = *Chenopodium quinoa*; Cf = *Chaetosiphon fragaefolii*.

<sup>3</sup>From roots only.

<sup>4</sup>Exceptions noted.

<sup>5</sup>Heterologous antiserum has been used successfully.

Northwest of the United States where this disease is a threat in nursery fields so that infected but symptomless daughter plants can be culled out.

When diagnosing viruslike symptoms in the field, another problem confronts the grower. A number of nontransmissible conditions, including mineral deficiencies and imbalances; fungus-, bacteria- and pesticide-induced symptoms; and symptoms caused by arthropod pests, may mimic, complicate, or obscure the diagnosis of transmissible virus disease symptoms. Several chapters in this strawberry section are devoted to these abnormalities so as to assist readers in distinguishing among them.

**Virus detection by grafting methods.** Although the first reports of strawberry virus diseases were made in the 1920s' (Plakidas 1926, 1927), it was not until Harris (1932) developed the technique for intergrafting stolons that a ready method of detection of such strawberry diseases by grafting became available. At first, susceptible cultivars were used as indicators. Harris and King (1942) demonstrated the sensitivity of *Fragaria vesca* L. 'East Malling Clone' or 'EMC' to many viruses in strawberry cultivars. 'EMC' was widely used as an indicator plant to detect virus diseases by the stolon grafting method. Unfortunately, 'EMC' was infected with the latent A strain of crinkle virus (Frazier 1953) (see "Strawberry Crinkle," p. 20), so that cultivars that had been stolon-graft indexed on 'EMC' became infected with latent A.

A number of other indicator clonal lines have been developed for strawberry virus detection. These include:

‘EMB’, ‘EMK’, and ‘HEMV’ clones of ‘EMC’ that have been freed of the latent A strain of crinkle virus (Frazier 1974b; Fulton 1960; Converse 1979).

‘Alpine’, *F. vesca* var. *semperflorens* (Duch.) Ser., a runnerless, seed-propagated clonal line (Harris and King 1942; Frazier 1955a).

‘UC-1’, *F. vesca*, a runnering seedling of ‘Alpine’ (Frazier and Posnette 1958).

‘UC-3’, *F. vesca* seedling (Frazier 1974b).

‘UC-4’, *F. vesca* x *F. virginiana* hybrid (Frazier 1974b).

‘UC-5’, a complex hybrid of *F. vesca*., *F. chiloensis*, and *F. virginiana* (Frazier 1974b).

‘UC-6’, *F. vesca* x *F. virginiana* hybrid (Frazier 1974b).

‘UC-10’, *F. virginiana* (Frazier 1974b).

‘UC-11’, *F. virginiana* (Frazier 1974b).

‘UC-12’, *F. virginiana* (Frazier 1974b).

‘FV 72’, *F. vesca* (McGrew 1967).

‘M1’, *F. virginiana* — (King and von Ruden 1962) carrying pseudo mild yellow-edge disease (Frazier 1966b).

The development of the leaf grafting technique (Bringhurst and Voth 1956) provided a relatively simple grafting system whereby the donor plant would not be cross-infected by the indicator plant. The petiole-insert leaflet grafting technique has been evaluated by several workers (Cropley 1958; Fulton 1957a; Jorgensen 1957; Miller 1958). It is now generally used for graft transmission of strawberry viruses. Several of these investigators reported that successful leaf graft unions from known infected donors did not always result in characteristic symptom development in the indicators. Frazier (1974a), however, demonstrated that transmission efficiency could be improved, and the length of incubation time until the appearance of symptoms in the indicator could be lessened if all leaves except the grafted ones were removed from the donor at the time of grafting.

Since the improved leaf grafting techniques developed by N. W. Frazier are best seen to be learned, figure 1 can be used to follow details of the procedure. Because of its elasticity and coherence, Sealtex brand tape is widely used for leaf grafting. For the delicate job of splitting the indicator petiole and shaping the donor petiole (fig. 1F), surgical scalpels are satisfactory but become dull quickly. Small pieces of new razor blades broken off to form a sharp point and held in a suitable tool (figs. 1D and F) are frequently used instead of scalpels. One to three leaflet grafts are generally made per indicator plant.

After the grafted plants are held in a mist chamber or a humid atmosphere in the greenhouse for about 1 week, long enough for graft union to occur, they are placed on the greenhouse bench and evaluated periodically for symptom development for 4 to 35 wk, depending on the disease (Converse 1979). A subtle but essential part of successful disease detection by leaflet graft is the maintenance of indicators in a vigorous

state of growth before and after grafting. Moderate temperatures and light intensities, such as are encountered in spring and autumn in most greenhouses, are key factors in growing good indicator plants. The presence of tobacco necrosis virus and its soil fungus vector, *Olpidium brassicae* (Wor.) Dang., appears to cause premature death of older leaves of *F. vesca* var. *semperflorens* ‘Alpine’, mimicking one of the major symptoms of mild yellow-edge virus. (See “Tobacco Necrosis Virus in Strawberry,” p. 64, and “Strawberry Mild Yellow-Edge Virus,” p. 25.)

*Fragaria virginiana* L. clones ‘UC-10’, ‘UC-11’, and ‘UC-12’ have been reported by Frazier (1974b) to develop symptoms when graft inoculated with pallidosis disease agent. (See “Strawberry Pallidosis,” p. 55.) No *F. vesca* clones have been found that produce distinct symptoms when graft inoculated with pallidosis agent. *F. virginiana* indicators are generally poorer than *F. vesca* indicators for the detection of other virus and viruslike diseases of strawberry. Therefore, it is necessary to use both *F. vesca* and *F. virginiana* indicators to detect the known strawberry virus and viruslike diseases by leaf grafting. A list of currently popular strawberry virus indicator clones and their specific uses is found in table 1.

In several situations, indicators that are already infected with a virus can be used advantageously to detect mild strains of other viruses that are challenge inoculated into these indicator plants. Several virus combinations act synergistically to produce more severe symptoms than the sum of their individual symptoms. Examples are latent A strain of crinkle virus acting synergistically with mottle virus; synergism between crinkle and vein banding viruses; and synergism between mild yellow-edge virus and pallidosis agent. (See individual virus chapters in the “Strawberry” section for further discussion of individual synergistic reactions.) Cross-protection between strains of the same virus can also be utilized in the identification of strawberry viruses. For example, ‘EMC’ infected with the latent A strain of crinkle virus is a poor indicator for the detection of other strains of crinkle virus (Frazier and Posnette 1958). On the other hand, clones of ‘East Malling’ *F. vesca* that have been freed of latent A, like ‘EMB’, ‘EMK’, and ‘HEMV’ are reasonably satisfactory for crinkle detection.

Some *Fragaria* indicator clones fail to express symptoms when inoculated with certain viruses, and these relationships can also be used to advantage in graft analysis. For example, ‘UC-6’ is symptomlessly infected by most strains of mild yellow-edge virus that have been tested so far, and ‘UC-10’ and ‘UC-11’ are symptomlessly infected by pseudo mild yellow-edge disease. Other examples of symptomlessly infected indicators can be found in the individual chapters in this section and in the paper by Frazier (1974b).

The analysis of multiple virus and viruslike infections in a single strawberry plant is in an unsatisfactory state. Aside from the viruses like the nepoviruses, tobacco streak, and,

possibly, strawberry vein banding virus and strawberry mild yellow-edge virus, (see chapters on these viruses) which can be detected serologically, all of the other virus and viruslike diseases of strawberry that do not cause diagnostic symptoms in fruiting cultivars must presently be detected and identified by a combination of graft and vector transmissions to indicator hosts. In the cases of multiple infected strawberry plants that are severely weakened by these viruses, it may be difficult to obtain leaflets of sufficient vigor and size to make leaflet grafts to indicators. Moreover, there is no assurance, even if grafts survive, that the entire complement of viruses and their strains will become established in the indicator plant. The same holds true when vector transmission is used. Conversely, it is not possible to predict with confidence that a disease complex can be generated by leaflet grafting a number of virus isolates into a test plant. Evaluation in the field is still the method of choice for rating strawberry cultivars and selections for their tolerance to virus and viruslike diseases, not only because of the technical difficulties connected with experimental inoculation but because of the interplay of pathogenic strains and environmental stresses that comes with field testing.

**Vector transmission.** Aphids, leafhoppers, and nematodes have been found to transmit various virus and viruslike diseases to strawberry. Details are given in the "Natural and Experimental Transmission" portions of the individual chapters of this section. Because of difficulties in handling them and the long incubation periods involved, transmission by leafhoppers and nematodes is seldom used as a diagnostic tool for identification of strawberry virus and viruslike diseases. Where the aphids of the genus *Chaetosiphon* are vectors, transmission properties are often useful in the detection of these diseases. As detailed in the various "Natural and Experimental Transmission" portions of individual chapters, aphids like *C. fragaefolii* (Cock.) can be used to acquire and transmit certain viruses to suitable indicator plants. This is the method of choice for strawberry mottle virus identification. Where a complex of aphid-borne diseases is present in a strawberry clone and detection of the causal agents involved is desired, sequential feeding of aphids from this source plant on a succession of indicator plants may facilitate the separation of the component causal agents by chance or by differences in their retention and transmissibility by the vector.

**Sap transmission to herbaceous plants.** Several viruses causing diseases of strawberry can be transmitted by sap inoculation to various herbaceous host plants. As detailed under "Natural and Experimental Transmission" portions of these virus chapters, the nematode-borne viruses, tobacco streak virus, tobacco necrosis virus, and strawberry mottle virus are sap transmitted. *Chenopodium quinoa* Willd. is a good herbaceous test plant for such sap transmissions, especially when it is growing vigorously under mild greenhouse temperature and light conditions. Strawberry plants are usually at their best as sap inoculation sources when in the early shock stages of infection. Various buffers

are used, but 2 to 3% nicotine alkaloid, often with various additives (Converse 1979), and 2% polyvinylpyrrolidone (mol. wt. 10,000) in 0.05M phosphate pH 7 (Martin and Converse 1982a) are useful buffers. Symptoms usually develop within 2 wk, but are seldom diagnostic. As described subsequently, however, sap from infected herbaceous indicator plants can be used for diagnostic serological tests when such antisera are available.

In the case of strawberry mottle virus, *Chaetosiphon fragaefolii* will transmit this virus to *Chenopodium quinoa*, producing diagnostic chlorotic spots on inoculated leaves within 2 wk after inoculation (Frazier 1968b).

**Serological detection methods for strawberry viruses.** At present, only 9 of the 28 named virus and viruslike diseases of strawberry have had specific antisera prepared against them, or antisera that will react with them. These are the nepoviruses, tobacco streak virus, tobacco necrosis virus, strawberry mild yellow-edge virus, and strawberry vein banding virus. One of the critical needs in present-day strawberry virology is the development of specific antisera against the many economically important strawberry viruses for which such sera are lacking. This author is of the opinion that satisfactory detection of viruses and viruslike diseases in strawberry is dependent upon the production of a complete set of antisera that span the range of these economically important diseases or upon the development of alternate, rapid, sensitive, biochemical detection procedures for the causal agents of these diseases. The application of monoclonal antibody techniques (Kennett et al. 1980) to strawberry viruses offers increased opportunity for developing new and effective antisera.

Where antisera do exist and are available to the investigator, a number of serological tests are available that detect these viruses rapidly and with great sensitivity. Enzyme-linked immunosorbent assay (ELISA) is a technique that is increasing in popularity for the detection of viruses in perennial crops, including strawberry (Clark 1981; Barbara and Clark 1982; Converse and Martin 1982; Johnson et al. 1984). Agar gel double diffusion serology and immunospecific electron microscopy are also useful or promising serological techniques for strawberry virus detection (Milne and Luisoni 1977; Converse 1981).

**Nonserological biochemical methods of detection of strawberry viruses.** In the rapidly developing field of nucleic acid biochemistry, several techniques have become available that offer opportunities for biochemical detection of strawberry viruses and viruslike diseases by other than serological means. This approach may become important for strawberry virus detection because of the general difficulty of purifying viruses directly from strawberry tissue or transmitting them to more manageable hosts from which purification could be more readily accomplished. The isolation of disease-specific nucleic acids, such as double-stranded RNA





(Morris and Dodds 1979) or viroid RNA (Morris and Smith 1977), has required some modifications of the existing procedures because of the high content of polysaccharides in strawberry extracts. The potential for using rapid nucleic acid detection methods for routine diagnosis of disease agents where serology is impractical was dramatically illustrated using cloned DNA for the detection of viroid in potatoes (Owens and Diener 1981). The potential for using such procedures for routine diagnosis of strawberry viruses and viruslike disease offers new opportunity for understanding the disease complexes and possibly controlling the diseases.

## Methods of Virus Elimination in Strawberries

**Seed transmission and its elimination.** As noted in specific chapters dealing with these viruses, the nepoviruses viruses, tobacco streak virus, and June yellows are all seed transmitted in varying percentages in strawberry when the seed or pollen parent is infected. None of the aphid-borne, leafhopper-borne, nor other strawberry virus diseases for which vectors are not known have so far been found to be seedborne. Viruses spread in strawberry seed rarely produce recognizable symptoms in the resulting seedlings; however, all of these viruses (but not June yellows) can be detected serologically so that the parents involved in controlled pollinations can be tested serologically to avoid the production of symptomless, infected seedling progenies. Seed from open-pollinated crosses can be evaluated by ELISA for tobacco streak virus (Johnson et al. 1984), and probably many of the nepoviruses can be similarly detected by direct assay of the dormant seed samples.

## Methods of elimination of viruses and the causal agents of viruslike diseases in strawberry clones.

**Chemotherapy.** Although there is a growing literature in the chemotherapy of plant viruses (Matthews 1981) the few reports of chemotherapy of strawberry viruses have either been negative (Miller and Vaughan 1957; Miller and Garren 1966) or have not been repeated (Fulton 1954). Additional research on strawberry virus chemotherapy using promising, recently studied chemicals should be attempted. The chemotherapy of the viruslike diseases June yellows and rickettsia yellows is reviewed in the chapters in this section on those diseases.

**Thermotherapy.** Some viruses and viruslike agents are eliminated in plant tissues that are grown at continuous elevated temperatures around 37° C or do not develop in plant cells formed at this temperature (Nyland and Goheen 1969). Posnette (1953b) was the first to eliminate a virus from strawberry by hot air therapy. Hot water therapy has also been studied (Miller 1953). Hot air therapy alone and in conjunction with tissue culture of shoot apices, as noted subsequently, has been successfully used to develop clones of many strawberry cultivars that are free of known virus diseases (table 2). (See individual strawberry chapters for numerous examples.)

Strawberry plants to be heat treated are generally grown in large pots without transplanting for several months prior to treatment to develop a large root system with good carbohydrate reserves. Inserting the pot into a somewhat larger pot and filling the intervening space with peat moss may help reduce damage to the root system during thermotherapy. Growth chambers are frequently programmed for 18 hr/day of 20,000 lux (fluorescent plus incandescent lights) at the leaf surface for the heat treatment. Plants are often placed in the growth chamber at greenhouse ambient temperature, and the growth chamber temperature is then raised a few degrees a day up to 37° C or higher (Bolton 1967).

Strawberry viruses and viruslike diseases differ markedly in their tolerance to thermotherapy. Strawberry mottle virus strains (with an exception) are readily inactivated in entire plants by being grown at 37° C for 10 to 14 days (Mellor and Frazier 1970b). At the other extreme, strawberry leafroll disease was not eliminated from plants exposed to 41°C for 20 days (Bolton 1970). Strawberry vein banding virus and strawberry green petal disease are easily eliminated from whole strawberry plants by growing them at a constant 37°C or higher for a few weeks (table 2).

Two horticultural techniques have been useful in improving the rate of success of elimination of virus and viruslike diseases by thermotherapy. Intentional destruction of the growing point of a plant during thermotherapy causes adventitious buds to develop that can be excised when 3 to 20 mm long and rooted under intermittent mist in the greenhouse (Mellor and Fitzpatrick 1961). In a second technique, after thermotherapy the crown is surface-sterilized and sliced into transverse sections. These sections when placed in sand or peat in the greenhouse will develop adventitious buds from which shoots and new plants can be obtained (Smith and Harland 1958; Posnette and Jha 1960).

Certain viruses and viruslike diseases have been eliminated from strawberry clones that were grown outdoors in a hot climate (Fulton 1956; Frazier et al. 1965). This "natural thermotherapy" eliminated some viruses like crinkle, which are difficult to eliminate in the growth chamber, but did not as readily eliminate strawberry mottle virus, which is easily inactivated in the growth chamber.

Prolonged exposure to cold temperatures has eliminated potato spindle tuber viroid from infected potato cultivars (Lizárraga et al. 1980). There is a claim that a similar treatment freed several cultivars from three aphid-borne viruses (Kacharmazov and Izvorska 1974). This approach may be worth trying for the elimination of viruses that are otherwise difficult to eliminate from desirable clones.

*Tissue culture for the elimination of viruses and viruslike diseases from strawberry clones.* Belkengren and Miller (1962) were the first to eliminate a virus (latent A strain of

**Table 2.—Thermotherapy and tissue culture for elimination of viruses and viruslike agents in strawberry**

Treatment	Disease	References
Eradication from whole plants after a few weeks at 37°C.	Green petal Latent A strain of crinkle Mottle Vein banding	Posnette and Ellenberger 1963. McGrew and Scott 1964b. Mellor and Frazier 1970. McGrew and Scott 1964. Bolton 1967.
Shoot apex tissue culture only.	Crinkle Latent C Mild yellow-edge Mottle  Pallidosis  Vein banding	McGrew 1980. J. R. McGrew, unpublished data. Mullin et al. 1974. McGrew 1980; Mullin et al. 1974. Mullin et al. 1974, 1975; McGrew 1980. McGrew 1980; R. Mullin, unpublished data.
Plants grown at 37 °C for several weeks, followed by crown bud propagation or shoot apex tissue culture.	Crinkle  Feather leaf Mild yellow-edge	Posnette and Cropley 1958; Posnette and Jha 1960; Mellor and Fitzpatrick 1961.  McGrew 1970a. Posnette and Jha 1960; Mellor and Fitzpatrick 1961; Miller and Belkengren 1963; Mullin et al. 1974; Mullin et al. 1976.
Treatments ineffective in eliminating the disease.	June yellows  Leafroll Lethal decline (X disease agent <sup>1</sup> )	Wills 1962; East Malling 1969; East Malling 1970. Bolton 1970. R. H. Converse, unpublished data. Nienhaus and Sikora 1979.

<sup>1</sup>X disease agent is possibly related to lethal decline disease agent.

strawberry crinkle virus) from a *Fragaria* clone by a combination of heat therapy and excision of runner tips 0.2 to 10 mm long. They developed whole plants from these tips in a sterile culture medium that was based on previous tissue culture work with other plants (White 1943). A number of specific media utilizing agar (Miller and Belkengren 1962; Miller and Belkengren 1963; McGrew 1965; Boxus 1974) and filter paper bridges in liquid media (Mullin et al. 1974, 1976) have been reported.

The regeneration of strawberry plants in tissue culture from small (500 micrometers (µm)) excised shoot tips is now a routine practice in the development of strawberry cultivars free from known viruses and viruslike diseases. McGrew (1980) has reviewed these techniques and has described the system used in his laboratory.

Although thermotherapy alone and tissue culture alone are sufficient to eliminate many virus and viruslike diseases in strawberry cultivars, the combination of these two techniques

is sometimes advantageous in developing clones free from a given virus or viruslike agent (table 2). As a routine procedure, this author currently heat treats all new USDA-OSU advanced strawberry selections for 8 wk at 37°C and excises several 500 µm shoot apices into tissue culture. Whole plants are allowed to develop without proliferation in culture and are potted and held in a humidity cabinet in the greenhouse when they are sufficiently rooted. Established plants are indexed for diseases by standard methods. They are evaluated for trueness to horticultural type, and a typical clone is then increased to supply official certification programs in insecticide-treated, screened enclosures by ordinary runner plant propagation. By this means, clones may be developed that have an increased likelihood of being free from all virus and viruslike diseases described and as yet undescribed.

The advent of rapid micropropagation in tissue culture (Boxus 1974; Boxus et al. 1977) has made it possible to maintain and increase a large number of strawberry cultivars



in tissue culture (Mullin and Schlegel 1976). However desirable this may be for the commercial nurseryman, there is enough uncertainty about the stability of some strawberry cultivars during long-term tissue culture micropropagation (Anderson et al. 1982; Swartz et al. 1981) to suggest to this author the desirability of maintaining the basic clones of superior virus-tested strawberry stocks by means of traditional runner propagation methods.

## Aphid-Borne Diseases

### Strawberry Mottle

By F. C. Mellor and H. Krczal

#### Additional Common Names

Mild crinkle (Prentice and Harris 1946) or virus 1 (Prentice 1948) was later identified as mottle virus (MV) combined with the latent A strain of crinkle virus (see "Strawberry Crinkle," p. 20). Type 1 (Demaree and Marcus 1951) was identified by McGrew (1956) as MV with either latent A or latent C (see "Strawberry Latent C," p. 29).

#### History and Geographic Distribution

Mottle is the most widespread of the viruses of strawberry, occurring wherever strawberries are grown. Until virus-tested stock was provided by heat therapy, most of the older cultivars were universally infected.

In early reports of virus diseases of strawberry, there was some confusion between MV and mild strains of crinkle virus (CV). This was because symptoms of MV were first described on a clone of *Fragaria vesca* L. cv. 'EMC' that was infected with latent A, a very mild strain of CV and, when MV and CV occur together, crinkle symptoms predominate. Clone 'EMC' had been selected as an indicator at East Malling in Great Britain because of its superior sensitivity to strawberry viruses (Harris and King 1942).

When Harris (1938) first transmitted a virus to 'EMC' by grafting from apparently normal plants of the strawberry cv. 'Royal Sovereign', symptoms on the indicator resembled those of the mild CV described and illustrated by Zeller (1933). Although Harris recognized that the disease might be etiologically distinct from severe CV, he referred to it as "mild crinkle," and for many years this name was used both for mild strains of CV and for combined infection with MV and latent A.

MV was subsequently transmitted to 'EMC', not only from apparently normal cultivars, but also from plants showing severe degeneration. By serial transfers of infective aphids to a succession of indicator plants, Prentice (1946) separated the viruses causing yellow-edge of 'Royal Sovereign' into "mild crinkle," which persisted in the vector for only a few hours, and mild yellow-edge, which persisted for several days. Similarly, Wood and Whitehead (1947) and Mellor and Fitzpatrick (1951) separated virus complexes causing the diseases known as "severe crinkle" and "yellows" into nonpersistent and persistent components. Vector relations of the component viruses distinguished the nonpersistent "mild crinkle" or MV from the persistent components, crinkle and mild yellow-edge. It was not until Frazier (1953) detected the









found that alate or apterous adults, and all stages of nymphs, were equally efficient as vectors, with 93 to 97% transmission.

Eulensen (1981) reported for *C. fragaefolii* a minimum acquisition period for MV of 15 min, which gave 5% transmissions; a 4-hr acquisition feed increased transmission to 41%. He reported a minimum inoculation access period of 7 min with the number of transmissions increasing to 29% when the transmission feeding period was 15 min and 59% after 60 min. He found that these aphids remained infective for only 3 hr and that the number of MV transmissions decreased sharply after the first hr.

In concurrent tests of two *Chaetosiphon* spp. as vectors of MV, Eulensen (1981) found that *C. thomasi* was a less efficient vector than *C. fragaefolii*. *C. thomasi* required a minimum acquisition feeding period of 30 min, which gave 4% transmission. The greatest percentage of transmissions, 21%, was reached after a 24-hr acquisition period. The minimum inoculation access period was 7 min. The percentage of transmissions increased to 13% with a 15-min transmission period, but did not significantly increase with longer periods. *C. thomasi* remained infective for only 1 hr. Mellor and Forbes (1960), however, reported that *C. fragaefolii* and *C. thomasi* were equally efficient in transmitting each of two variants of MV. Recent studies have shown that the progeny of a single aphid can be classified into both of these species (Crock and Shanks 1983), suggesting that the reported variations in vector efficiency for MV may be attributable to strain differences within a single aphid species.

### Properties of the Causal Agent

Kitajima et al. (1971) found aggregates of isometric, viruslike particles in thin sections of *F. vesca* leaves infected with several different isolates of MV (fig. 8). The electron-dense portions of these particles, presumably representing the nucleic acid core, were 17 to 22 nm in diameter. The center-to-center distance between the closest particles indicated that the entire nucleoprotein particle was 25 to 30 nm in diameter. Although most often found in aggregates in the phloem cells, the particles were also seen frequently in the plasmodesmal lumina of other leaf cells, including epidermal and parenchymatous tissue.

The virus has not been isolated and purified, and nothing more is known about its physical and chemical properties.

### Detection and Identification

MV in cultivars, whether alone or in combination with other viruses, can be detected and identified only by transmission to indicator plants. Frazier (1974) recommended three selected seedlings of *F. vesca* as indicators of MV. These were 'UC-4' (the best), 'UC-5', and 'UC-6'.

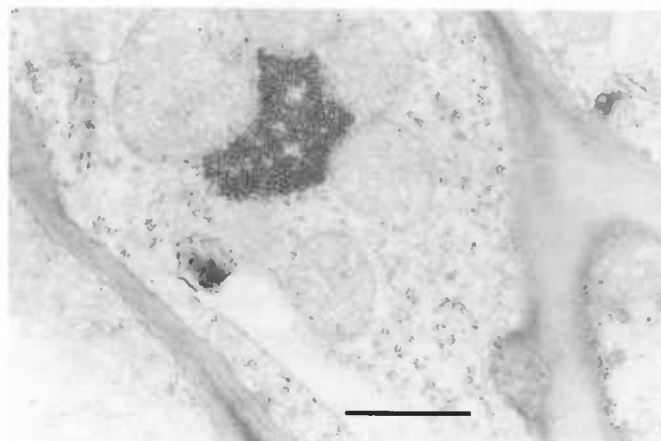


Figure 8.—Viruslike particles associated with strawberry mottle virus-infected leaves of *Fragaria vesca*. Bar represents 1000 nm.

(Courtesy E. W. Kitajima, University of Brasilia.)

Since field infections with MV are often accompanied by other viruses, graft transmission to indicator plants may cause a confusing symptom picture. MV can best be identified by aphid transmission. *Chaetosiphon fragaefolii* is usually the preferred test aphid. Given an acquisition feeding period of 4 to 6 hr, and two or three successive 1-hr transmission periods on indicator plants, the aphids will transmit only the semipersistent viruses. Of these, MV is more likely to be transmitted than vein banding or pseudo mild yellow-edge. Even if one of the latter two viruses were also transmitted, symptoms of MV could be recognized.

### Control Procedures

Control depends not only on the use of virus-indexed planting stock and control of aphids, but also on isolation from infected plants. Control of aphids will reduce the rate of virus spread, but since MV may be transmitted during very short feeding periods, insecticides do not provide complete protection against incoming viruliferous aphids. Krczal (1962) found that plants became infected when exposed to viruliferous aphids a short time after the plants were sprayed with the insecticides demeton-S-methyl or parathion. When plants were exposed to viruliferous aphids 5 hr after spraying, up to 95% of the plants became infected with MV (H. Krczal, unpublished data). Unless plants are isolated from sources of MV, they should be replaced periodically with new virus-indexed stock to maintain vigor and productivity.

**Therapy.** Mottle is one of the most heat-labile of the strawberry viruses and can usually be eradicated from infected plants by growing them at 37°C for 10 to 14 days; however, longer treatment at lower temperatures may be ineffective. Frazier et al. (1965) tested inactivation of several viruses in strawberry plants in the field. These were maintained for 4 mo or more in a naturally high-temperature environment in the field, where mean summer temperatures reached 32°C. Surprisingly, CV, which survives much longer than MV at 37°C in the growth chamber (see "Strawberry

Crinkle," p. 20), was eradicated from 41 of 50 plants, and MV was eradicated from only 1 of 32 plants in this field test.

### Remarks

The term "mottle" is used to include what appear to be numerous strains of a single virus. More than one virus may be involved, but this is not clearly indicated by present evidence. There is a very wide range of symptoms, and there are some differences in vector-virus-plant relationships. Studies of the properties of MV virions, including their serology, will be needed to determine their relationships.

### Strawberry Vein Banding

By N. W. Frazier and T. J. Morris

### Additional Common Names

Yellow vein banding (Frazier and Posnette 1958), chiloensis vein banding, and eastern vein banding (Frazier 1960a) are synonyms of strawberry vein banding virus (SVBV) (Frazier 1955a; Frazier and Converse 1980). Erdbeer-nekrosevirus (Schöniger 1958) may be a strain of vein banding virus, but this is uncertain. Leaf curl (virus 5) (Prentice 1952) is a disease complex formed by SVBV and crinkle virus. Schöniger noted the similarity of her isolate to leaf curl. Both Schöniger and Prentice used the East Malling clone of *Fragaria vesca* (EMC), which carries the latent-A strain of crinkle virus for their indicator plant. The complex of latent-A and the leaf curl strain of SVBV in EMC causes leaf curling on young leaves and premature discoloration, vein purpling, and necrosis of older senescing leaves (Frazier and Posnette 1958).

### History and Geographic Distribution

The leaf-curl strain of SVBV was first transmitted by Prentice (1952) from a 'Fairfax' plant imported from the United States into Great Britain where the disease does not occur in nature. It may occur in continental Europe if Nekrosevirus should prove to be a strain of SVBV (Schöniger 1958), or if the semipersistent virus described by Domes (1957) is related to it. Vein banding virus appears to be native to North America, where it occurs in many strains. It is common in clones of the beach strawberry, *Fragaria chiloensis* (L.) Duch., along the Pacific coast (Miller and Frazier 1970). It is presumed that the wild strawberries of Eastern United States: *F. vesca* L. subsp. *americana* (Porter) Staudt, the wood strawberry, and *F. virginiana* Duch., the Virginian strawberry, may harbor the virus. Eastern and western strains of SVBV differ. The eastern strains are of the leaf-curl type and are more difficult to detect on indicator plants than the western strains, which are of the vein-banding type.

SVBV also occurs in Australia, Brazil, and Japan, probably having been introduced in planting or breeding material.

### Economic Importance

The disease is now of minor importance because of low incidence in commercial strawberries. This is likely due to

the use of certified planting stock. SVBV reduces runner production, vigor, yield, and fruit quality in commercial cultivars and can be very severe in mixed infection with crinkle or latent-C viruses (Bolton 1974; Freeman and Mellor 1962; and Takai 1973).

### Symptoms on Natural and Experimental Hosts

Natural infection with SVBV is known only in species of *Fragaria*, all of which are probably susceptible. The garden burnet (*Sanguisorba minor* Scop.) was established as a symptomless experimental host by graft inoculation and by the dark strawberry aphid vector *Chaetosiphon jacobi* H.R.L. (Mullin et al. 1980).

### Symptoms on cultivars (of *Fragaria x ananassa* Duch.) and on *F. chiloensis*.

Few cultivars show dependably diagnostic symptoms although they may be adversely affected. The chlorotic pattern of western SVBV is clearly obvious on 'Marshall' in all of the disease complexes tested. Plant vigor can be severely reduced in infected plants. Western SVBV interacts strongly with crinkle virus and with decreasing severity with pallidosis, mottle, and mild yellow-edge disease agents. In contrast, infections of western SVBV in 'Hood' are detectable with difficulty in the acute stage but are symptomless in the chronic stage, while 'Tioga' is initially symptomless but old leaves develop a faint chlorosis of net veins during the chronic stage.

Symptoms of SVBV have not been detected in any infected clone of wild or ornamental *F. chiloensis*.

**Symptoms on indicator hosts.** The standard strawberry virus indicator clones are all sensitive to SVBV, but there is wide variation in sensitivity to different isolates of the virus. This variation is illustrated for two isolates on *Fragaria vesca* 'Alpine' (figs. 9 and 11). *F. virginiana* clone 'UC-12' (fig. 10) and *F. vesca* clone 'UC-6' are the most sensitive and diagnostic indicator clones for the virus (Frazier 1974b). Both are especially useful in diagnoses of eastern strains.

Three symptom types characterize the disease: leaf curl, vein banding, and necrosis.

1. The leaf curl symptom (epinasty of midribs and twisting of leaflets usually accompanied by epinasty and reduction of petioles) is most severe during the early phase of infection, particularly on *F. virginiana* indicator plants. It is most severe and is the dominant early symptom in mixed infections with crinkle virus (fig. 12). Leaf curl is more characteristic of eastern isolates than western isolates and is seldom more than mildly expressed during the chronic phase of the disease.

2. The vein banding symptom (chlorotic banding along main veins) is most intensely expressed in the first few leaves to develop after onset of symptoms (fig. 9A). In leaves developing later, the banding usually appears as discontinuous streaks or spots. The clarity of the vein banding

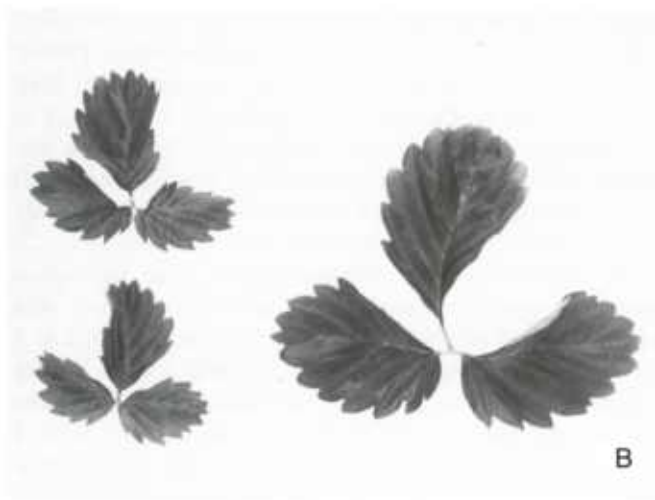


Figure 9. — A, Leaf of 'Alpine' (*Fragaria vesca* var. *semperflorens*) showing the chlorotic vein banding pattern of type strawberry vein banding virus; B, leaves of 'Alpine' infected with a very mild isolate of strawberry vein banding virus from North Carolina showing acute symptoms of faint vein clearing of net veins bordering main veins of basal leaflets of the two small leaves at the left and faint chronic banding symptoms in the large leaf at the right.



Figure 10. — *Fragaria virginiana* clone 'UC-12' infected with strawberry vein banding virus showing typical vein banding pattern.



Figure 11. — 'Alpine' plants: Left, healthy; and, right, infected with a severely stunting isolate of SVBV from Washington State.



Figure 12. — Leaves of *Fragaria vesca* L. var. *californica* (Cham. and Schlecht) Staudt, showing a severe leaf curl without vein banding due to a double infection of strawberry vein banding viruses and latent A crinkle virus.

pattern varies with the virus strain and host plant; the clarity is more striking in western than eastern isolates.

3. The necrosis symptom develops on mature leaves: Net veins may become darkened or necrotic and interveinal tissues may become discolored or necrotic. The leaves may become partly necrotic or die prematurely. The symptom is much more evident with eastern isolates than with western isolates. The symptom persists largely as a premature coloration of older leaves during chronic infections, and its severity is increased in combination with crinkle virus.

Symptoms of the leaf curl and necrosis types may be induced in strawberry by other causal agents, but the vein banding symptom is relatively diagnostic.

During the chronic stage of the disease, symptoms fluctuate in severity: A series of normal-appearing leaves may alternate with a series of leaves showing strong symptoms (fig. 13). The symptomless leaves seem to occur after transplanting or the application of fertilizer (Mellor and Fitzpatrick 1961), or may be produced in cycles independently of nutrient or temperature (Stingl and King 1965a).





Figure 13. — An excised crown of 'Alpine' with a chronic infection of type SVBV. The cluster of small leaves on short petioles show vein banding symptoms which are absent from the larger, normal-appearing leaves that developed 4 wk following a fertilizer application.

**Symptoms in complex with other diseases.** The symptoms of SVBV are additive in the presence of crinkle virus. In *F. vesca*, the vein banding pattern is masked or distorted and crinkle symptoms tend to dominate. This interaction is useful in the detection of subclinical strains of either virus (Frazier and Mellor 1970a). Similarly, the vein banding pattern is less evident in the presence of mottle virus, and the mottle symptoms tend to dominate. This interaction is also useful for detecting subclinical strains of either virus.

#### Natural and Experimental Transmission

SVBV has been transmitted by grafting, by dodder [*Cuscuta subinclusa* Dur. and Hilg. (Frazier 1955a)], and by the aphid vectors *Chaetosiphon fragaefolii* (Cock.) (Prentice 1952), *Amphorophora agathonica* Hottes [also known as *A. rubi* (Kltb.)], *Macrosiphum pelargonii* Kltb., *Myzus ornatus* (Laing) (Frazier 1955a), *Amphorophora idaei* Börn., *Chaetosiphon jacobii* (Frazier and Posnette 1958), *Aphis rubifolii* (Thomas), *Aulacorthum solani* (Kltb.), *Myzus* (*Nectarosiphon*) *ascalonicus* Donc., *Myzus persicae* (Sulz.), and *Chaetosiphon tetraerhodum* (Wlk.) [also known as *Pentatrachopus tetraerhodus* (Wlk.)] (Mellor and Forbes 1960).

Attempts to transmit the disease mechanically with preparations of purified virus (Morris et al. 1980) or isolated nucleic acid extracts (T. J. Morris, unpublished data) were not successful.

Strawberry aphids (*Chaetosiphon* sp.) are probably responsible for most of the field spread of the virus. Other less efficient vector species often occur in high populations on *Fragaria* and could also be important in SVBV epidemiology. The most efficient aphid vectors are *Chaetosiphon fragaefolii*, *C. jacobii*, and *C. thomasi*. These species, as well as *Myzus ornatus*, transmit some but not all strains of the virus, suggesting a subspecific virus-vector relationship (Frazier 1960a; Mellor and Forbes 1960). This is further supported by the fact that two eastern isolates of the virus were not transmitted by *C. jacobii* (N. W. Frazier, unpublished data).

SVBV is a virus of the semipersistent type that is acquired during a 30-min acquisition access feeding period (Frazier 1955a). Retention of the virus by feeding vectors has been reported to be 1 hr (Prentice 1952) for the leaf curl strain, 8 hr for the type strain (Frazier 1955a) and 24 hr for the Nekrosevirus strain (Schöniger 1958). A virus half-life of 10 hr was estimated for the type strain in fasted vectors (Frazier and Sylvester 1960), and the virus was not retained by *C. jacobii* through ecdysis (Frazier 1966a). The incubation period for symptom development in indicator plants was reported to be 3 to 5 wk for leaf-curl (Prentice 1952), 2 to 3 wk for vein banding (Frazier 1955a), and 3 to 4 wk for Nekrosevirus (Schöniger 1958). Betti and Costa (1980) have reported that plant leaves of intermediate maturity were a better source for virus acquisition by vector aphids than young or mature leaves.

#### Properties of the Causal Agent

SVBV is a member of the caulimovirus group (Kitajima et al. 1973; Frazier and Converse 1980). It has a limited but distinctive natural host range which distinguishes it from other caulimoviruses, and it is aphid transmitted in a semipersistent manner. Typical caulimoviruslike particles, 40 to 50 nm in diameter (fig. 14), have been isolated from infected strawberry plants, and they had a reported sedimentation coefficient of  $200 \pm 10 S$  (Morris et al. 1980). Cytoplasmic inclusion bodies (fig. 15) typical of other caulimoviruses have been reported in the vascular parenchyma and mesophyll cells of infected plants (Kaname 1975; Kitajima et al. 1973; Frazier and Converse 1980; Morris et al. 1980).

Purification of SVBV from infected leaves of *F. vesca* has been accomplished. The isolation of virions from viscous host extracts included clarification with organic solvents and fractionation on sucrose-CsCl step gradients and ECTEOLA cellulose columns (Morris et al. 1980). Yields of virus were invariably low. Attempts to purify inclusion bodies also resulted in poor yields (T. J. Morris and R. Mullin,

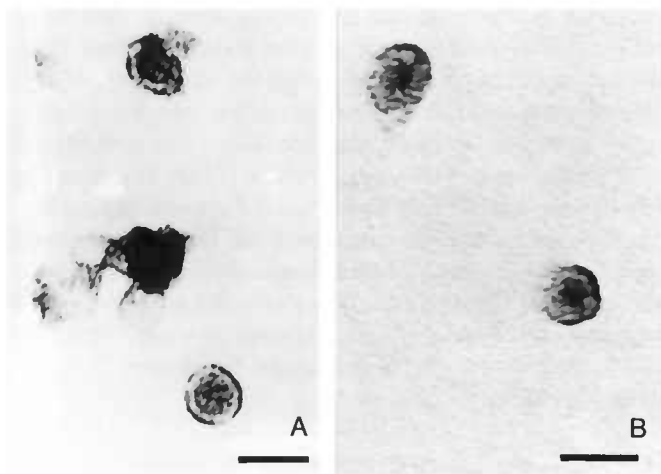


Figure 14. — Serologically specific electron microscopy of purified preparations of — A, Strawberry vein banding virus; and B, cauliflower mosaic virus after fixation in 1% formaldehyde. Both grids were precoated with cauliflower mosaic virus gamma globulin at 5 µg/ml, incubated with virus preparations for 2 hr and stained with uranyl acetate. The bar represents 50 nm.

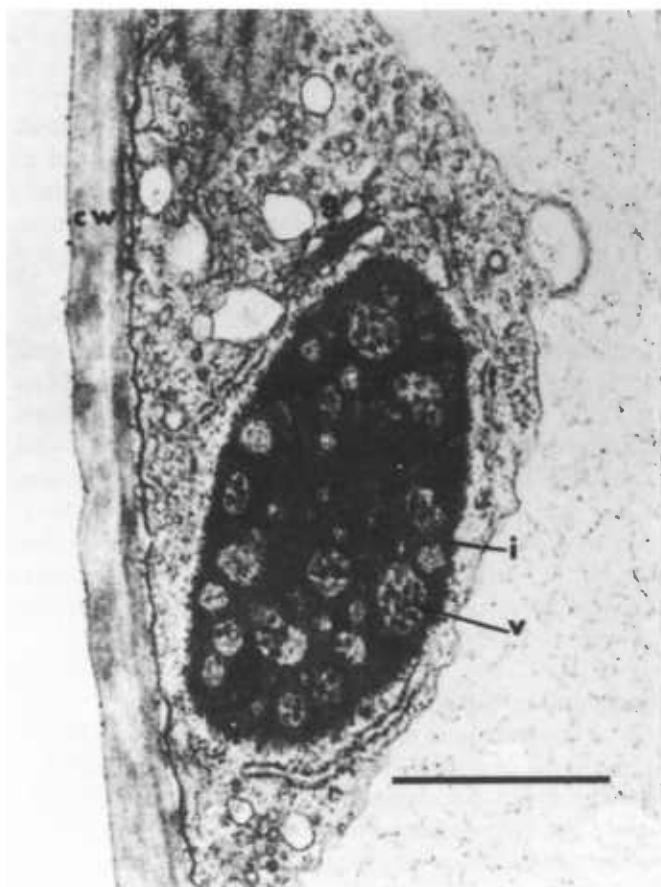


Figure 15. — An inclusion body in a strawberry leaf cell exhibiting vein banding symptoms. The virions (45 nm) are present throughout the inclusion; cw, cell wall; i, inclusion body; v, virions. Bar represents 1 micron.

unpublished data). These results, and the fact that a homogeneous virion population ( $200 \pm 10$  S) could only be identified in density gradients after fixation in 1% formaldehyde, probably reflect a problem of particle instability. A more complete characterization of virion proteins and nucleic acid remains to be accomplished.

Antiserum to SVBV has not been prepared, but a serological relationship to other caulimoviruses has been demonstrated (Morris et al. 1980). Purified SVBV reacted in immunodiffusion and ELISA tests with antiserum prepared to the cabbage-B strain of cauliflower mosaic virus. Similar cross reactions have been identified using dahlia mosaic virus antiserum but not with carnation etched ring virus antiserum (T. J. Morris, unpublished data). SVBV appears to be serologically distinct from other caulimoviruses tested to date, but a definitive assignment of serological relationships will require the production of an SVBV antiserum.

Many strains and/or variants of SVBV have been distinguished on the basis of symptoms, aphid vector specificity (Frazier and Posnette 1958; Frazier 1960a; Mellor and Forbes 1960), and in cross-protection tests. In cross-protection tests with seven isolates, there was complete or partial protection whether the virus was introduced by vectors or by grafting (Frazier and Converse 1980). The relative importance of strains is not known.

#### Detection and Identification

The severity of SVBV disease varies widely with host, isolate, and stage of infection. The virus is best diagnosed by graft or vector transmission to the indicator clones *F. virginiana* 'UC-12' or *F. vesca* 'UC-6' (Frazier 1974b), which show characteristic chlorotic vein banding. The virus can be separated from others by vector transmission. Chronically infected plants are often a poor source of virus for vectors (Prentice 1952), but availability can be restored by graft transmission to a new plant (Frazier and Posnette 1958).

A more rapid serological confirmation of SVBV can be accomplished in ELISA tests using cauliflower mosaic virus antiserum. Clarified plant extracts concentrated tenfold by precipitation with 8% polyethylene glycol (Morris et al. 1980) can give satisfactory results (T. J. Morris and R. Mullin, unpublished data). Routine serological detection however, will require the production of an SVBV-specific antiserum.

#### Control Procedures

The main control procedure is the use of commercially available planting stock certified to be free of the virus. The general incidence of SVBV is very low, and use of such certified stock is appropriate toward maintaining such a situation.

Elimination of the virus from mother plants by runner tip culture (Miller and Belkengren 1963) or by heat treatment for 10 days at 42°C (Bolton 1967) has been reported. The elimination of the type strain of SVBV from experimentally infected 'Hood' plants by meristem tip culture was 100% whether or not the plants were preheat treated for 6 wk at 37°C (R. Mullin, unpublished data). In these tests, the cultured plants were indexed and discarded after 6 mo. This could prove to be an insufficient incubation period for cloned meristems, as some dahlia plants similarly treated to eliminate dahlia mosaic virus remained symptomless for up to 10 mo (Mullin and Schlegel 1978).

### Remarks

Although SVBV is a relatively minor problem in commercial strawberry production, it is not a virus to be ignored. It can cause serious losses when associated with other viruses in disease complexes. In view of the long latent period in shoot apex propagated plants of the one other caulimovirus tested, SVBV could become a problem in the production of certified stock. The production of an antiserum and implementation of ELISA indexing to detect latent infections could be important steps in reducing this possibility.

## 245 Strawberry Crinkle

By N. W. Frazier, E. S. Sylvester, and J. Richardson

### Additional Common Names

The names "crinkle," "mild crinkle," "intermediate crinkle," and "severe crinkle" have been applied to diseases of cultivars caused by various strains of the crinkle virus alone or in combination with other strawberry virus diseases such as mottle, vein banding, and mild yellow-edge. In addition to strains that cause classical symptoms, there are milder strains that require sensitive indicator plants for detection. These are, in order of increasing severity: strawberry latent A virus, mild form (Rorie 1957); strawberry latent A and latent B viruses (Frazier 1953); strawberry lesion A and lesion B viruses (Frazier and Posnette 1958); and strawberry vein chlorosis virus (Prentice 1952; Frazier and Mellor 1970a).

### History and Geographic Distribution

Strawberry crinkle virus (SCV) was first reported in the 'Marshall' cultivar in Oregon by Zeller and Vaughan (1932). Vaughan (1933) demonstrated transmission of the virus by the strawberry aphid *Chaetosiphon fragaefolii* (Cock.) (previously known as *Capitophorus fragaefolii*, *Myzus fragaefolii*, and *Pentatrachopus fragaefolii*) to 'Marshall' test plants. Zeller (1933) reported an incubation period of 12 to 15 days in 'Marshall' plants and also recognized that crinkle was probably composed of two separable components — one causing a "mild crinkle" — but he did not indicate their symptom differences.

Crinkle was recorded in Great Britain by Ogilvie et al. (1934). Later Harris (1937a, b) transmitted the virus by

graft. He pointed out that symptoms were of two types (designated as mild and severe crinkle) and referred to an interaction of the two viruses. Prentice and Harris (1946) transmitted a virus that persisted in the strawberry aphid vector about 3 hr, and they concluded that it was probably the mild crinkle virus. This virus, Prentice (1948) designated as virus 1, and later (1952) he proposed the name "strawberry mottle," stating that he considered the form causing mild crinkle to be distinct. He also demonstrated that a form of severe crinkle could be caused by a complex of virus 1 with a long-persistent type of virus designated as virus 3 (Prentice 1949), and which he later named "strawberry crinkle" (Prentice 1952).

SCV occurs worldwide (North and South America, Britain, Europe, South Africa, New Zealand, Australia, and Japan) except in regions where strawberry aphids of the genus *Chaetosiphon* are not found on strawberry (Sylvester et al. 1976). The East Malling clone of *Fragaria vesca* L. 'EMC' was originally selected as a superior indicator because mottle virus caused more severe symptoms on this clone than any other. It was not until the discovery that 'EMC' was carrying latent A virus that this sensitivity was explained (Frazier 1953). In the meantime, the clone had been distributed throughout the world and many cultivars were inadvertently infected with latent A by stolon grafting to 'EMC'.

### Economic Importance

SCV is one of the most damaging of the virus diseases affecting strawberries. As a result of meristem culture to eliminate SCV from commercial cultivars, and certification programs to maintain clean planting stock, the losses from SCV have been minimized in recent years. Where SCV still occurs, severe strains reduce vigor and productivity and even mild strains, such as latent A, reduce vigor, runner production, yield, and fruit size of some varieties (Freeman and Mellor 1962; McGrew and Scott 1964; Barritt and Loo 1973). On the other hand, Takai (1973) found no significant differences between crinkle-infected and virus-free 'Kohyoku' lines. SCV most usually occurs in the field with mottle, vein banding, mild yellow-edge, and/or pallidosis. Each of these diseases exerts an important additive effect with SCV. Crinkle is a visual component of several degenerative disease complexes that have been a limiting factor in the production of strawberries in many areas.

### Symptoms on Natural and Experimental Hosts

SCV is known only in species of *Fragaria*, none of which is known to be immune. Strawberry cultivars vary widely in their sensitivity. More sensitive cultivars, such as 'Hood', show distinct symptoms with mild strains, while others, such as 'Shasta', may show no symptoms even when infected with severe strains. On sensitive cultivars, symptoms are characterized by chlorotic spots and deformation of leaves (fig. 16). Small, scattered, opaque, chlorotic to necrotic spots are associated with veins, and short lengths of cleared or yellowed veins often radiate from them. Leaflets are usually



Figure 16. — Cv. 'Marshall' plants infected, left to right, with strawberry crinkle; crinkle and strawberry

mild yellow-edge; crinkle, mild yellow-edge, and strawberry mottle viruses.



Figure 17. — Leaf of a cv. 'Marshall' plant exhibiting the "chlorotic sector" symptom associated with some strains of crinkle virus.

unequal in size, distorted, and crinkled. Sectors of leaflets may be yellowed (figs. 16 and 17), and petioles and leaves may be reduced in size.

**Symptoms on indicator hosts.** Clones of *F. vesca* show symptoms similar to those described above and vary in pattern and severity, depending on the virus strain and the indicator clone. Symptoms of mild strains are most evident during the early or shock stage of infection (fig. 18) and may consist only of a slight angular epinasty of a single leaflet on one or several successive leaves. Symptoms of more severe strains persist and include several components, not all of which are diagnostic.



Figure 18. — *Fragaria vesca* var. *semperflorens* cv. 'Alpine' infected with latent A crinkle by the aphid vector *Chaetosiphon jacobi* showing early symptoms of leaflet epinasty (11 and 3 o'clock) and abaxially curled leaflets (12 o'clock).

Although chlorotic spotting of the lamina is one of the most usual symptoms of SCV (fig 19), this symptom, by itself, is not reliable diagnostically for similar spots can be caused by mottle or mild yellow-edge infection (fig 20). The spots are irregularly distributed and associated with veins. At first, the spots are translucent, later becoming opaque and yellow, reddish, or necrotic. Crinkling of the lamina and uneven expansion of the leaflets can be caused by atrophy of the spots (fig. 21).

Angular epinasty of leaflets is a fairly reliable diagnostic early symptom of infection. Characteristically, the epinasty is sharply angular from some point (usually a spot or lesion)



Figure 19. — *Fragaria vesca* 'UC-1', left to right: Chronic symptoms of three isolates of strawberry crinkle virus inoculated by *Chaetosiphon jacobii*.



Figure 20. — Similar symptoms caused by three different diseases in *Fragaria vesca* var. *sempervlorens* cv. 'Alpine' infected by vector inoculations. Left to right: mild yellow-edge, crinkle, and mottle.

on a midrib and seldom affects more than one leaflet per leaf, and leaflets often become curled abaxially (fig. 18).

Lesions provide a reliable diagnostic symptom and occur on stolons, petioles (fig. 22), and leaves. Greenish, reddish, or necrotic areas or rings may be sunken or swollen and cause angular bending. Such lesions are most prominent and useful on *F. vesca*, occurring less frequently on *F. virginiana*, *F. chiloensis*, and on strawberry cultivars.

Chlorotic sectoring (fig. 17) is not a consistent symptom. Interveinal yellowing forms wedge-shaped sectors beginning at some point on a main vein and widening toward the leaflet margin. This symptom seems to be associated only with certain strains of crinkle.

Vein chlorosis usually is not conspicuous. Short lengths or small networks of veins from chlorotic spots can become finely cleared (fig. 21).

Petal streak is a highly diagnostic symptom of crinkle. It is particularly useful for the detection of mild infections and of the presence of crinkle in disease complexes. Short lengths of

Figure 21. — Leaf of *Fragaria vesca* 'UC-5' with crinkle virus symptoms of chlorotic spotting and vein chlorosis.

veins in petals become cleared or translucent permitting the background color — usually the green of a sepal — to show through, causing the translucent areas to appear darker than normal. The streaks may become necrotic, and affected petals may be deformed and dwarfed (fig. 23).

Symptoms in *F. virginiana* Duch. cvs. 'M1', 'UC-10', 'UC-11', and 'UC-12' are of the characteristically generalized, nondiagnostic syndrome typical of the *F. virginiana* reaction to infection with most graft-transmissible diseases. They consist of general chlorosis, vein clearing, abnormal development of new leaves, epinasty of leaflets and petioles, red or yellow old leaves, loss of vigor and stunting (Frazier 1974b). The 'M1' and 'UC-11' clones both have superior sensitivity to SCV, but 'UC-11' is a much more useful indicator over a wider range of diseases. The petal-streak symptom is much less useful in *F. virginiana* than in *F. vesca*.

*In complexes with other diseases in F. vesca.* Symptom severity of any complex in cultivars or in indicators depends on the severity of each disease component (fig. 24) (Frazier and Mellor 1970a).



SCV interacts very strongly with strawberry mottle and strawberry vein banding diseases. The interaction can be used to advantage in the detection of very mild strains of the diseases. In combination with mild yellow-edge, the symptoms of crinkle are partially masked and those of mild yellow-edge dominate.

#### Natural and Experimental Transmission

**Methods.** SCV is easily transmitted by grafting but not by sap inoculation and only very erratically by aphid vectors (Frazier and Mellor 1970a). The principal natural vector is *C. fragaefolii*. Although *C. jacobi* H. R. L. also is a vector, it is found only on wild *F. vesca* L. var. *californica* (Cham. and Schlecht.) Staudt, in the coastal mountains of central California (Mellor and Frazier 1970b). In recent years, this species has been used predominately at Berkeley because it is native to the locality on *F. vesca* and is an efficient vector of aphid-borne strawberry viruses. In addition, the dorsum of mature apterous aphids is uniquely dark-brown, and thus colony contamination with other species of *Chaetosiphon* can be readily detected. Takai (1973) reported *C. minor* (Forbes) not to be a vector of crinkle. Babović (1976) reported transmission of crinkle by the strawberry root aphid *Aphis forbesi* Weed — the first non-*Chaetosiphon* species to be so implicated.

#### Vector-Virus-Plant Relationships

The early detailed vector work on SCV (Prentice 1949; Prentice and Woolcombe 1951) established it as belonging to the group of viruses having a persistent vector-virus relationship, but with an exceptionally long (10 to 19 days) latent period in *C. fragaefolii*. The aphids remained infective for several days. Coupled with an incubation period of 4 to 8 wk in strawberry plants, a transmission cycle could take up to 2½ mo. Later tests with *C. jacobi* (Frazier 1968a) gave a mean (range) vector latent period of 32 (14 to 59) days, with lifelong retention of infectivity (recorded maximum of 71 days) with a maximum longevity of 106 days. There was



Figure 22. — Stolon tips and segments of petioles from *Fragaria vesca* 'UC-1' showing the lesion symptoms of strawberry crinkle virus.



Figure 23. — Flowers from *Fragaria vesca* var. *sempreflorens* cv. 'Alpine' plants: left, normal; right, petal-streak symptoms of strawberry crinkle virus.

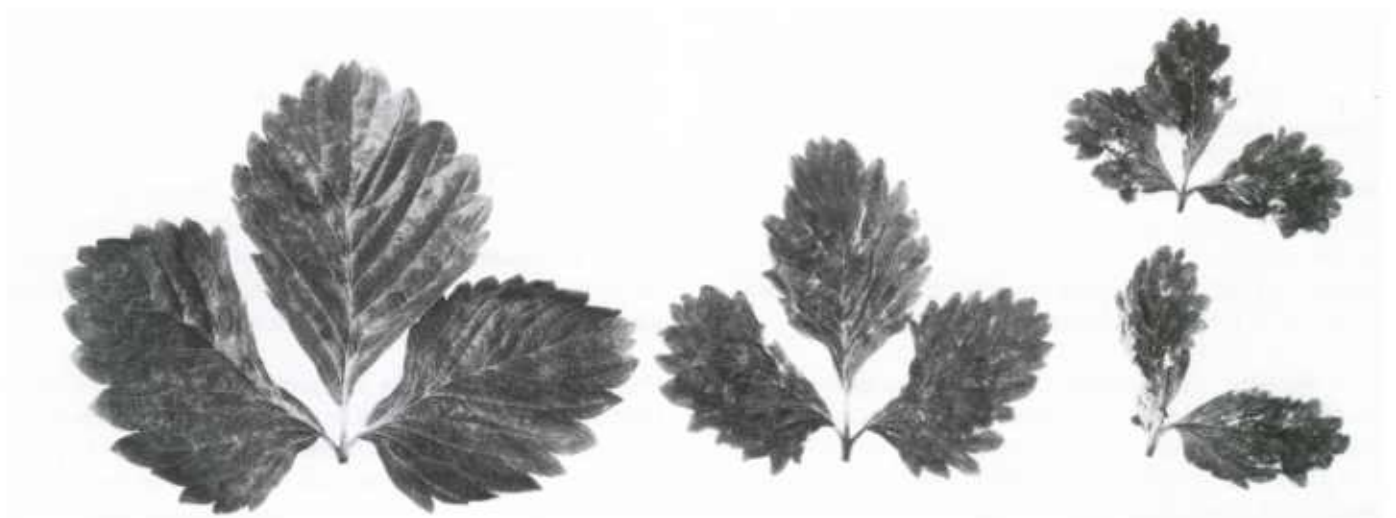


Figure 24. — Interactions of a mild strain of mottle virus with two mild strains of crinkle virus in *Fragaria*

*vesca* 'UC-1'. Left to right: mottle alone, with latent A, and with latent B.

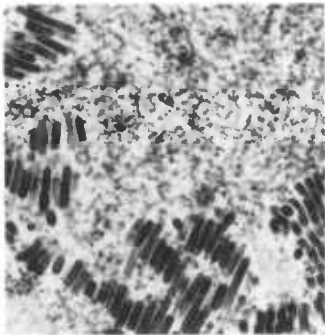


Figure 25. — Electron micrograph of nonenveloped strawberry crinkle virions in the cytoplasm of the salivary gland of the aphid vector *Chaetosiphon jacobi*. Magnification: x 20,900.

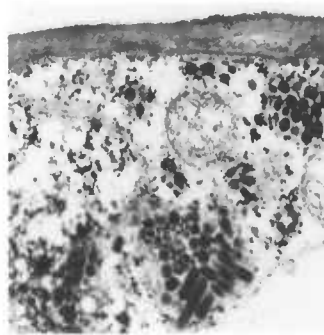


Figure 26. — Enveloped and nonenveloped strawberry crinkle virions in the petal of *Fragaria vesca*. Magnification: x 13,000.



Figure 27. — Negatively stained bullet-shaped virions of strawberry crinkle from the crushed head of the aphid vector *Chaetosiphon jacobi*. Magnification: x 63,000.

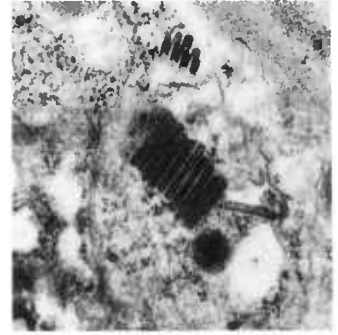


Figure 28. — Nonenveloped strawberry crinkle virions in the oesophageal wall of an inefficient aphid vector *Myzus ornatus*. Magnification: x 20,900.

evidence that vector efficiency, as well as values for these other parameters, varied with virus strains. The vector colony rearing, acquisition, and test feeding periods were carried out in a sheltered outdoor area (somewhat approximating field conditions) where the temperature ranged from 9.9° to 18.8°C during the several years of experimentation and was about 18.3°C in a glasshouse where inoculated plants were incubated.

More recently, (Sylvester et al. 1974) used serial passage via needle inoculation to establish that SCV multiplies in *C. jacobi* and is a propagative plant rhabdovirus. At 25°C, inoculated vectors had a median latent period of 6.2 days, and 10 to 12 days after injection they achieved a maximum rate of transmission of about 90%. The rate then declined to 12% by 11 to 24 days after injection, and ceased some 6 days later even though 50% of the insects were still alive. Injected insects lived for a maximum of 42 days after injection.

Crinkle virus also will multiply in other aphid species when injected, including *Hyperomyzus lactucae* (L.), a nonstrawberry-feeding aphid (Sylvester and Richardson 1981) and *Myzus ornatus* Laing, a polyphagous species that will colonize strawberry (E. S. Sylvester and J. Richardson, unpublished data). Injected *M. ornatus* occasionally will transmit virus to test plants, but with *H. lactucae*, host plant specificity precludes valid transmission tests being done.

### Properties of the Causal Agent

A variant of the C10 group of SCV (Frazier 1968a) examined in the electron microscope showed a bacilliform particle morphology, typical of a plant rhabdovirus (Richardson et al. 1972). SCV presumably belongs to the *Rhabdoviridae*.

Thin sections of infected *C. jacobi* vectors revealed enveloped and nonenveloped virions in the cytoplasm of most organs (fig. 25). The enveloped particles measured  $69 \pm 6 \times 190$  to 380 nm. Similar particles were found in thin sections of diseased leaves of *F. vesca* L. var. *semperflorens* (Duch. Ser. cv. 'Alpine' (Alpine strawberry) (fig. 26).

Bullet-shaped particles were exclusively seen in unfixed negatively stained preparations from infected aphids (fig. 27), and similar particles occasionally can be found in plant-dip preparations, especially from infected petals. Enveloped and nonenveloped particles also have been found in nonvector aphids *H. lactucae* (Sylvester and Richardson 1981) and *M. ornatus* (E. S. Sylvester and J. Richardson, unpublished data) infected by injection (fig. 28).

SCV has not been purified and there is no information on its physical or chemical properties or its serology; however, attempted cross-protection tests using *C. jacobi* and *H. lactucae* gave no evidence of interference between SCV and sowthistle yellow vein, another aphid-borne plant rhabdovirus (Sylvester and Richardson 1982).

### Detection and Identification

Some commercial cultivars show symptoms of severe strains of SCV especially when it is a component of a multidisease complex. Most, however, are symptomless carriers of mild strains. Detection depends on graft indexing to sensitive indicator cultivars. *F. virginiana* cvs. 'MI' and 'UC-11' are the most sensitive indicators, but the symptoms resulting are generalized rather than diagnostic. *F. vesca* cvs. 'UC-4', 'UC-5', and 'UC-6' are all very sensitive and show the diagnostic petal-streak and petiole-lesion symptoms.

### Control Procedures

Spread of SCV may be reduced by aphid control, isolation of plantings from infection sources, and a strawberry plant-free period in conjunction with an annual planting system where the local conditions and cultivars permit. The use of certified planting stock free of SCV is of major importance.

Plants free of SCV can be developed by several techniques. Heat treatment of infected plants with a constant temperature of 38°C or temperatures fluctuating daily from 35° to 41°C eliminates the virus in several months. Propagation of auxiliary buds from crowns of heat-treated plants reduces the required length of treatment to a few weeks (Posnette and Cropley 1958; Posnette and Jha 1960; Mellor and Fitzpatrick

1961; McGrew and Scott 1964). Apical meristems free of SCV may occasionally be removed from infected plants and grown on a culture medium, but success of the method is greatly improved when parent plants are given a short heat treatment (Belkengren and Miller 1962; Miller and Belkengren 1963; Vine 1968). SCV was inactivated in a high percentage of plants during a season of growth in a naturally high temperature climate where summer temperatures reached a mean of 32°C (Frazier et al. 1965).

## 245 Strawberry Mild Yellow-Edge //

By R. H. Converse, R. R. Martin, and S. Spiegel

### Additional Common Names

Strawberry virus 2 (Prentice 1948). Mild yellow-edge virus (MYEV) is a common but not essential component of the complex known in North America as yellows or xanthosis and in Great Britain as yellow-edge.

### History and Geographic Distribution

Yellows was the first virus disease of strawberry to be recognized. Horne (1922) described the disease in California. Plakidas (1926, 1927) showed that the disease was transmissible by the aphid *Chaetosiphon fragaefolii* (Cock.). Harris (1933) described a similar graft-transmissible disease in England, which he called "yellow-edge," and Massee (1935) showed that yellow-edge was transmitted by *C. fragaefolii*. Harris and King (1940) concluded that the two diseases were analogous, a conclusion supported by later work (Mellor and Fitzpatrick 1951; and Frazier and Posnette 1958).

MYEV is probably worldwide in distribution in strawberry cultivars. It is one of the most common viruses in cultivated strawberries in western North America, Europe, Israel (Leshem et al. 1962; Spiegel et al. 1981), South Africa (Engelbrecht 1967a), Australia (Greber 1979), New Zealand (Chamberlain 1934), and Japan (Goto and Nemoto 1974). MYEV and yellows have been reported from eastern North America, but are uncommon there (Plakidas 1964; Morgan 1965; and Frazier 1975b). *Chaetosiphon fragaefolii*, a vector of MYEV, has been found on *Fragaria chiloensis* (L.) Duch. in beach areas in southern Chile remote from cultivated strawberries (R. H. Converse, unpublished results).

The history of the yellows disease is given in detail by Plakidas (1964).

### Economic Importance

The yellows complex is undoubtedly one of the major diseases of strawberry in most parts of the world; however, because of the interaction of cultivars, viruses and virus strains, crop management, and environment, it is difficult to assess the importance of MYEV in the amount of economic loss that occurs. MYEV alone is not particularly damaging to most cultivars, but it seldom occurs alone. The complex of MYEV with other viruses, for example, mottle (MV), crinkle

(CV), vein banding (VBV), or pallidosis agent (PA), can cause severe loss of plant vigor, yield, and fruit quality.

In field studies of inoculated plants of cv. 'Hood', Barritt and Loo (1973) found that MYEV alone did not significantly reduce plant vigor, fruit yield, or fruit size, but the combination of MYEV and MV significantly reduced fruit size. Mullin et al. (1974) reported that meristem-derived 'Fresno' plants outyielded MYEV-infected 'Fresno' in field trials in California by 15 to 24%. In other field studies, Martin and Converse (1977) found that the vigor of 'Hood' plants that had been infected with the yellows complex one or more years previously was 17% below that of comparable healthy plants. In plants that had been infected during the current growing season, fruit percentage and weight were reduced 16 and 19%, respectively. Aerts (1980) reported that MYEV reduced yield of 'Gorella' in field tests in The Netherlands by 30%, mainly by reducing the number of fruit per infected plant.

In greenhouse studies, Mellor and Fitzpatrick (1961) found that MYEV further reduced the vigor of 'Marshall' already infected with MV and CV. Shanks and Crandall (1969) found that the yellows complex significantly reduced the yield of 'Columbia' but not of the more resistant 'Northwest'. Lawrence and Miller (1968) found that MYEV with MV and/or CV did not reduce vigor and yield of 'Northwest' but did reduce runner production in greenhouse studies.

### Symptoms on Natural and Experimental Hosts

**Symptoms on natural hosts.** In nature, the virus has only been found in *Fragaria*. The wild species *F. virginiana* Duchesne, *F. vesca* L., and some clones of *F. chiloensis* show symptoms; *F. ovalis* (Lehm.) Rydb. is a symptomless carrier.

Most strawberry cultivars are symptomless carriers of MYEV. At most, slight marginal chlorosis occurs on young leaves; however, cultivars vary greatly in the amount of symptom expression and crop loss caused by the yellows complex (Daubeney et al. 1972). No immune cultivars are known, and all are believed to suffer a decrease in vigor and yield when infected by yellows complex.

Experimentally, MYEV can be graft transmitted symptomlessly to *Sanguisorba minor* Scop. (Mullin et al. 1980).

**Symptoms on indicator hosts.** Symptoms of typical MYEV isolates are similar, whether the indicator is *F. vesca* 'EMC', 'UC-4', or 'Alpine' (fig. 29). Infected 'UC-6' is usually symptomless and is useful both for this "negative symptomatology" and to maintain MYEV isolates that might kill or severely weaken 'UC-4' or 'Alpine'.

Prentice (1948) described two symptom types for the MYEV that he separated from the yellow-edge complex: (1) typical symptoms, which included small, chlorotic flecks on the





Figure 29.—Left, symptoms in *Fragaria vesca* 'UC-4' of a mild strain of mild yellow-edge virus 45 days after grafting. Young leaves are mottled, and a few of the

older leaves show premature senescence. Right, normal 'UC-4' plant.



young leaves, chiefly on the smaller veins, with leaflets slightly cupped and slightly chlorotic, especially toward the margins, accompanied by gradual loss of vigor; and (2) much milder symptoms, consisting of slight but definite chlorotic mottling, some interveinal necrosis of older leaves, but very little cupping or marginal chlorosis. He also described somewhat similar but still milder symptoms of a persistent virus transmitted from 'Huxley's Giant', which on the 'East Malling' clone ('EMC') of *F. vesca* caused chlorosis and slight cupping, but not chlorotic spotting. Frazier and Posnette (1958), redescribing the same three isolates, added that the typical form also caused yellowing or reddening and the premature death of the older leaves. These isolates were all described on 'EMC'. Symptoms appeared 4 to 8 wk after inoculation by aphids.

Isolates of MYEV that occur along the Pacific coast of North America cause a similar range of symptoms. There appear to be intergrading strains, but experimental evidence is not sufficient to evaluate their relationships. Mellor and Fitzpatrick (1951), describing symptoms of the persistent component separated from the yellows complex in 'Marshall', considered that they most closely resembled symptoms described by Prentice for the persistent virus he isolated from 'Huxley's Giant'. A strain that commonly occurs on the Pacific coast resembles typical MYEV described by Prentice (1948), but symptoms appeared on aphid-inoculated *F. vesca* 8 to 15 wk after inoculation.

Timing and symptom intensity vary with the virus isolate, the indicator, and the season. In leaf-grafted indicators, the symptoms are chlorosis and some necrosis of the net-veins of one or two leaflets of the youngest leaf. On succeeding leaves, net-vein chlorosis and necrosis are general, and affected leaves soon die, so that the symptom picture about 2 mo after inoculation is distinctive. The first three or four leaves formed after inoculation appear normal, the next few

leaves are dead (fig. 30), and the youngest show epinasty, net-vein chlorosis and some small, scattered, necrotic spots and streaks (fig. 31). During later stages of infection, the youngest leaves may appear nearly normal, but older ones continue to die prematurely.

**Symptoms in complexes with other viruses.** MYEV in complex with the latent A strain of CV causes marginal chlorosis of the leaves, at least on the cvs. 'Royal Sovereign' and 'Marshall'. The symptoms of virus 2 on 'Royal Sovereign', described and illustrated by Prentice (1948), were probably those of MYEV with latent A.

The combination of MYEV with MV, CV, or both, sometimes with the addition of PA (see "Strawberry Pallidosis," p. 55, for a discussion of the role of PA in yellows), causes the disease known as xanthosis, yellows, or yellow-edge. Symptom severity varies with the number and severity of the component viruses and viruslike agents, length of infection, and susceptibility of the cultivar. Sensitive cvs., like 'Marshall', 'Hood', and 'Puget Beauty', are dwarfed and appressed to the ground. The older leaves are cupped and chlorotic at the margins. Petioles are abnormally short and stout. Young leaves are small, twisted, or cupped, with marginal or overall chlorosis. Yield is much reduced. Less sensitive cultivars exhibit similar but milder symptoms. Some cvs., like 'Totem', 'Tyee', and 'Northwest', are so tolerant of the yellows complex that infected plants show no symptoms (Daubeny et al. 1972). No immune cultivars are known, however, and all are believed to suffer a decrease in vigor and yield when infected.

#### Natural and Experimental Transmission

Natural transmission of MYEV is by several species of aphids, mostly in the genus *Chaetosiphon*, as a persistent or circulative virus. MYEV was separated from the yellows (yellow-edge) complex by serial transfers of infective aphids



Figure 30.—Typical stages of symptom production of mild yellow- edge virus on leaf-grafted *Fragaria vesca* 'UC-4': A, epinasty of young leaves; B, chlorotic flecking of young leaves; C, vein necrosis of maturing leaves; D, scorching of maturing leaves.

to a succession of *Fragaria* indicator plants by Prentice (1946, 1948) and by Mellor and Fitzpatrick (1951).

Vector aphids are *Chaetosiphon fragaefolii* (Cock.), *C. thomasi* H.R.L., *C. jacobii* H.R.L., *C. minor* (Forbes) (Frazier 1975b), and *Macrosiphum rosae* (L.) (Mellor and Forbes 1960). *Myzus persicae* (Sulz.) has been found to be a vector in recent greenhouse studies (R. R. Martin and R. H. Converse, unpublished results).

Studies of field transmission showed that spread of MYEV peaked in June in southwestern Washington State, but some spread occurred there even in winter (Shanks 1965). Field spread of the virus from infected plants appeared to occur at

random (Converse et al. 1979), although alate *Chaetosiphon* spp. may actively seek *Fragaria* as a host (Shanks 1965; Shanks and Finnigan 1970).

Experimental aphid transmission is sometimes erratic or unsuccessful. Some strains of aphids are inefficient vectors, and some strains of MYEV are less easily transmitted than others. Krczal (1979) reported that nymphs, apterae, and alatae of *C. fragaefolii* all transmitted the virus equally well, and that single aphids transmitted it to 16% of the test plants. He obtained 100% transmission by *C. fragaefolii* with an acquisition feeding period of 2 days and a transmission feeding period of 8 days. Frazier and Posnette (1958) found that *C. jacobii* acquired MYEV in 8 hr or less. Engelbrecht





Figure 31.—*Left*, typical chronic symptoms in *Fragaria vesca* 'UC-4' of a strain of mild yellow-edge virus from cultivated strawberry in western Oregon. Two mo after inoculation by leaf grafting, the youngest leaves show

chlorosis and necrosis of secondary veins, while many mature leaves are scorched and older leaves are dead. *Right*, comparably leaf-grafted *F. vesca* 'UC-6', which was infected but remained symptomless.



(1967b) reported a latent period for MYEV in *C. fragaefolii* of 24 to 40 hr after an 8 hr acquisition access period. Mellor and Frazier (1970a) reported that *C. jacobii* could retain the virus for 45 days, but efficiency of transmission declined after 28 days.

To test the effectiveness of inserting only one scion leaflet per indicator plant, Frazier (1974a) used *F. vesca* and defoliated the indicator plants at time of grafting. He found that one inserted leaflet was adequate for transmission of MYEV, and that the incubation period was 15 to 30 days, with a mean of 20 days.

MYEV can be graft transmitted to *Sanguisorba minor* Scop., but there are no symptoms (Mullin et al. 1980).

Mechanical transmission of MYEV was reported by Miller (1951) and Liu (1957), but their results have not been confirmed. Dodder transmission has not been reported. The virus is not known to be spread by seed or pollen.

#### Properties of the Causal Agent

Very little is yet known about the properties of MYEV. Somewhat distorted isometric particles 23 nm in diameter have been seen after partial purification of MYEV (fig. 32) (Martin and Converse, 1982b). No viruslike particles were observed in the electron microscope in thin-sectioned strawberry leaf tissue infected with MYEV (Greber 1979). Because of symptomatology and vector properties, MYEV is tentatively classified as a luteovirus (Matthews 1979).

#### Detection and Identification

It is seldom possible to detect and identify MYEV by its symptomatology in infected cultivars. Transmission to

sensitive clones of *F. vesca* is therefore necessary for detection and identification of this virus. On *F. vesca* indicator clones, MYEV symptoms usually appear within 3 wk after inoculation by aphid or by leaflet grafting. 'UC-4' is the most sensitive indicator. 'UC-6' remains symptomless when infected by most of the isolates of MYEV tested (Frazier 1974a,b; R. H. Converse and S. Spiegel, unpublished results). *F. vesca* var. *semperflorens* 'Alpine', *F. vesca* 'UC-5', and *F. virginiana* 'UC-10' and 'UC-11' are also useful indicators for MYEV.

On *F. vesca* indicators, physiological heat spot may be confused with early symptoms of MYEV. (See "Heat Spot of *Fragaria vesca*," p. 78.) Several leafhopper-borne disease agents, like aster yellows, green petal, tomato big bud, and



Figure 32.—Somewhat distorted, isometric particles 23 nm in diameter, from a partially purified preparation of strawberry mild yellow-edge virus. Bar represents 100 nm.

lethal decline (see strawberry leafhopper-borne disease chapters), may cause foliage symptoms similar to yellows on some strawberry cultivars, but these diseases also cause flower phyllody, sterility, or malformation. Tobacco necrosis virus (see "Tobacco Necrosis Virus in Strawberry," p. 64) appears to be associated with premature senescence of older leaves of some *Fragaria* indicator clones, mimicking MYEV symptomatology.

### Control Procedures

Many of the techniques that are useful for controlling MYEV also apply to other strawberry viruses and are discussed in the introductory chapter of the strawberry section of this handbook. (See "Detection and Elimination of Virus and Viruslike Diseases in Strawberry," p. 2.)

**Elimination of MYEV from strawberry cultivars.** Mellor and Fitzpatrick (1961) found that MYEV survived in strawberry cultivars exposed to a constant 38°C for 6 mo. Later work, however, showed that excision of the central growing point and almost complete defoliation of infected cultivars during heat treatment for 9 wk at 38°C stimulated the development of side crowns, which could then be excised and rooted in sand at normal greenhouse temperatures. Approximately 50% of the resulting plants were freed of MYEV. Nearly all axillary crowns excised after heat treatment of 12 wk or more were free of MYEV (F. Mellor, unpublished results).

Several cultivars were freed of viruses that are difficult to inactivate by thermotherapy by cutting crown disks 0.5 to 1 cm thick and propagating them in a peat-sand rooting medium (Posnette and Jha 1960). Plants of three cultivars were freed of MYEV by cutting stolon tips 0.5 to 1 mm or larger from plants held at greenhouse or at elevated temperatures and growing them out on culture medium and then transferring them to pots in the greenhouse (Miller and Belkengren 1963). The production of cultivars freed from MYEV and other viruses by a combination of heat treatment and tissue culture was described by Mullin et al. (1974, 1976).

Because MYEV usually occurs in complex with other viruses, no controls are unique for MYEV; those discussed for strawberry crinkle and strawberry mottle viruses are applicable to MYEV. (See "Strawberry Mottle," p. 10, and "Strawberry Crinkle," p. 20.)

### Remarks

There is a scarcity of published information on the MYEV particle. Once the antisera now prepared against it (Martin and Converse 1982b) have been improved to permit rapid detection of this virus in field samples, research on the ecology of MYEV will move forward from its present state. Despite advances in the development of virus-tested planting stocks and certification schemes, the strawberry yellows complex remains one of the major causes of economic loss to the strawberry industry. More thorough evaluation of the

total *Fragaria* gene pool from which cultivated strawberries are derived should provide clones with high levels of resistance to colonization by the aphid vectors of many strawberry viruses, including MYEV and high levels of tolerance to the individual viruses. These clones could then provide the basis for future cultivars.

245  
**Strawberry Latent C** //  
By J. R. McGrew

### Additional Common Names

Demaree and Marcus Type 2.

### History and Geographic Distribution

Typical symptoms of strawberry latent C disease (SLCD) were first described in *Fragaria vesca* L. by Harris and King (1942) from plants imported from the United States of 'Fairfax', 'Dorsett', and 'Premier'. SLCD will be used to refer both to the disease and its causal agent. Symptoms in the 'East Malling' clone of *F. vesca* ('EMC') were first illustrated by Demaree and Marcus (1951) who named the disease type 2. It was found in cultivars throughout the eastern United States.

McGrew (1958) showed that SLCD, with or without the latent A strain of strawberry crinkle virus, failed to produce symptoms in some seedlings of 'EMC', while other seedlings showed typical epinasty and subsequent dwarfing. SLCD was readily recovered from symptomless seedlings.

For several years, the geographic distribution of SLCD corresponded closely with that of 'Howard 17' (also known as 'Premier') (Bolton 1967; McGrew 1961), of which all true-to-name plants carried this disease agent (Bell 1955, Bolton 1964, Craig and Stultz 1964). Spread was detected occasionally in Arkansas (Fulton 1960), and Nova Scotia (Craig and Stultz 1964). Indexing at Beltsville, Md., (J. R. McGrew, unpublished data) detected spread of SLCD in the late 1970's into strawberry selections in Maryland, New Jersey, Iowa, Arkansas, and Minnesota. Limited indexing of selections and cultivars from North Carolina, Florida, Louisiana, California, and Wisconsin and of cultivars received from Japan, Taiwan, Germany, England, France, Poland, and Italy has not detected SLCD.

Natural spread appears limited to eastern North America and to areas where infected cultivars serve as a source of infection.

### Economic Importance

The detrimental effect of SLCD as a component of a complex in 'Catskill' (McGrew and Scott 1959) and alone and in complex in 'Jerseybelle' (Kender 1964) is significant. Miller (1960) found moderate to severe degeneration when SLCD was added to existing virus complexes in 10 selections and cultivars. However, several cvs., such as 'Premier' and 'Temple', infected with SLCD apparently were sufficiently tolerant to be commercially acceptable to growers.



Figure 33.—Strawberry latent C disease symptoms in *Fragaria vesca* 'EMK'. Left, 6 weeks after grafting; right, chronic symptoms, after several months.



### Symptoms on Natural and Experimental Hosts

Most strawberry cultivars appear symptomless when infected with SLCD. Some nondiagnostic loss of vigor may be found in others and severe leaf distortion in a few (Miller 1960) when SLCD is added to a complex which is itself symptomless.

Symptoms in sensitive clones of *F. vesca* (fig. 33) inoculated by leaf grafts include severe epinasty of young leaflets followed by moderate to severe dwarfing without epinasty, mottling, or distortion. In *F. virginiana* Duch. 'UC-10', a transitory mild yellowing is often seen, but it is not sufficiently distinct to be diagnostic for SLCD.

At Beltsville, Md., SLCD from many sources has produced rather uniform symptoms in sensitive indicator clones. The epinastic shock symptoms are severe and obvious. The later chronic dwarf symptoms range from severe to moderate, but still show obvious reduction in size. After the appearance of dwarfed but otherwise normal leaves, indicators are usually discarded. Whether some isolates might allow indicators to return to near normal size leaf production is not known.

N. W. Frazier (unpublished data) has found considerable variation in severity of symptoms from some sources of SLCD when the number or time that excised-leaf grafts remained in place were reduced (as in Frazier 1974a). He also found differences in symptom expression depending on age of leaf used from a recently infected source plant. Use of older leaves produced milder symptoms than younger leaves when grafted individually to indicator plants.

### Natural and Experimental Transmission

SLCD spreads in the field, and several aphid species have been implicated as vectors. Experimentally, it has been transmitted by runner and excised-leaf grafts and by dodder.

SLCD has been reported to be transmissible by *Chaetosiphon fragaefolii* (Cock.). Smith (1952) found that this species required more than 1 and fewer than 6 days to acquire SLCD, that infectivity persisted for at least 9 days, and that symptoms appeared in 23 or more days. Demaree and Marcus (1951) obtained transmission of SLCD to *F. vesca* by *Chaetosiphon minor* (Forbes) and *C. thomasi* H.R.L. (their "unnamed species") collected from plants in the field. Rorie (1957) suggested that *C. minor* rarely, if ever, transmits this virus. N. W. Frazier (unpublished data) was unable to transmit SLCD from three sources by use of *Chaetosiphon jacobii* H.R.L. and several species of leafhoppers, white flies, and thrips.

Natural spread of SLCD may be rapid under some conditions—six of nine plants exposed for one season in Michigan indexed positive (McGrew 1961), or movement may be rare (Fulton 1960).

Experimental transmission of SLCD is readily achieved by excised-leaf grafts. Days to symptoms on regular excised leaf grafts are 24 to 60, mean 34 (McGrew 1970b) and, when all leaves except grafts are removed, are 15 to 36, mean 21 (Frazier 1974).

Transmission by dodder has been successful using *Cuscuta subinclusa* Dur. and Hilg. (29 to 40 days) (Smith and Moore 1952) and with *C. campestris* Yunck. (35 or more days) (Fulton 1954).

### Properties of the Causal Agent

Cross inoculations or natural complexes indicate SLCD is distinct from other strawberry viruses, for example: crinkle, mottle, vein banding (J.R. McGrew, unpublished data) and pallidosis and mild yellow-edge (N. W. Frazier, unpublished data). Information is lacking on the nature of the causal agent and serological relationships of SLCD.

### Detection and Identification

Strawberry latent C cannot be identified in strawberry cultivars by symptoms. Transmission to sensitive indicator clones, as *Fragaria vesca* clone 'EMC' or 'UC-5', is necessary.

Further confirmation of suspected SLCD is to pass it by graft transmission through an SLCD nonsensitive *F. vesca* clone, such as 'Alpine', 'UC-1', 'UC-4' or 'UC-6', and to recover symptoms in a sensitive clone. Absence of symptoms in the SLCD nonsensitive clone eliminates the possibility of a complex with other graft-transmissible agents to which that clone is sensitive.

### Control Procedures

Control procedures include the use of certified planting stock free of known viruses and viruslike diseases, isolation of new plantings from infected sources, possibly the use of aphicides, and periodic replacement of planting stock.

Elimination of SLCD by heat treatment of entire plants was not successful at 38°C. However, axillary buds that developed on decapitated crowns and were removed after 3 days at 46.5°C or above were free of this disease (Bolton 1967).

A combination of heat treatment at 35°C followed by tip culture was partially successful in eliminating SLCD from a strawberry cultivar (McGrew 1965). Recent trials (J. R. McGrew, unpublished data) in which 14 explants from SLCD-infected sources were taken without prior heat treatment (10 of 10 between 0.4 and 0.8 mm, 1 of 2 at 1.0 mm and 1 of 2 at 1.8 mm) were free of the disease.

### Remarks

The frequency of detection of SLCD in the field appears directly related to the presence of nearby sources in the planting. The production of cultivar clones free of SLCD and moderate care in isolation of seedling, selection, and nursery blocks from known sources, followed by continued replacement of certified fruiting-field stocks, should result in the disappearance of this disease.

## Leafhopper-Borne Diseases

### 245 Aster Yellows in Strawberry //

By L. N. Chiykowski

(Chemistry and Biology Research Institute, Research Branch, Agriculture Canada, as C.B.R.I. Contribution No. 1295)

### Additional Common Names

Eastern (New York) aster yellows; western (California) aster yellows; August black root; spot dying; chlorotic phyllody.

### History and Geographic Distribution

The susceptibility of *Fragaria* species to aster yellows was first demonstrated by Frazier and Severin (1945) who experimentally transmitted a California or western strain to *F. vesca* L. var. *californica* (Cham. and Schlecht.) Staudt, using the aster leafhopper, *Macrostelus fascifrons* (Stål) (also known as *M. divisus* DeLong [nec] Uhler). In 1952, a disease characterized by phyllod flowers was reported as being common but not abundant in scattered commercial strawberry plants in central California (Frazier and Thomas 1953). The authors also stated that similar symptoms had been observed on various strawberry cultivars and in seedling test plants on rare occasions over the previous 20 yr. Western aster yellows was transmitted from such field-infected strawberry plants to aster and plantain (*Plantago major* L.) by means of the aster leafhopper. The same species was also used to transmit the disease from China aster to *F. vesca* L. var. *semperflorens* (Duch.) Ser. cv. 'Alpine' and return it to plantain.

In 1949, a disease causing phyllody was observed on strawberry in Louisiana by Plakidas (1951) and given the name "chlorotic phyllody." This disease is now generally believed to have been aster yellows. A severe outbreak of a disease in strawberry resembling aster yellows and relatively new to Arkansas was reported by Smith (1954). The cause was later verified by Fulton (1957b) who also showed that "August black root" or "spot dying" were the result of aster yellows infection.

Attempts by Kunkel (1926) to infect strawberry experimentally with eastern aster yellows using the aster leafhopper were unsuccessful. Chiykowski (1969), however, transmitted a disease from naturally infected strawberry cv. 'Sparkle' to aster by means of the aster leafhopper, and the symptoms produced were typical of eastern aster yellows. Transmission from an infected fleabane (*Erigeron* spp.) plant found adjacent to the strawberry planting gave similar results.

The geographical range of aster yellows in general covers the United States and Canada, but the distribution of the different strains within this range is not clearly understood. Although aster yellows has not been widely reported as a disease of



strawberry, it probably occurs in plantings throughout this range but is not normally detected because of its low incidence. Similar, possibly related, diseases occur in Europe, Japan, and Russia.

### Economic Importance

The incidence of aster yellows in strawberry is normally low, although it may occasionally reach as high as 20% (Smith 1954). Infected plants produce no marketable fruit and usually die within two mo after symptoms appear. Aster yellows infection may not always be recognized as such and instead may be attributed to other causes, as was the case with August black root and spot dying (Fulton 1957b). It could also be mistaken for other diseases, such as green petal (see "Strawberry Green Petal and Similar Diseases," p. 34) when both diseases occur in the same strawberry planting (Chiyskowski 1969). Thus, the real economic importance of aster yellows in strawberry may sometimes be underestimated.

### Symptoms on Natural and Experimental Hosts

The host range of aster yellows is extensive, consisting of approximately 300 plant species in at least 50 families. Included in this list of susceptible species are perennial, annual, monocotyledonous dicotyledonous, cultivated and wild plants.

**Symptoms on strawberry.** Foliage symptoms may appear at any time during the growing season. New leaves have shortened petioles, are reduced in size, chlorotic, and generally cupped (fig. 34). Older leaves may be reddened, be flat on the ground, and soon turn brown.

The type of flower and fruit symptoms expressed are dependent on time of infection in relation to initiation of flower buds. Plants infected late in the fruiting cycle may show normal petals and some degree of flower sterility. When infection occurs early in the flower initiation sequence, the following symptoms may be seen: virescent petals on partly or fully sterile flowers; virescent petals with small, green, foliaceous growths from the achenes; or entirely phylloid flowers. Several symptom stages may be present on a single plant of even a single inflorescence (fig. 35). Floral symptoms are important aids in the diagnosis of aster yellows infection because foliage symptoms alone are not reliably distinct from those of several other strawberry diseases; for example, verticillium wilt, yellows (xanthosis) virus complexes, or lethal decline. (See the Aphid-borne Diseases chapters, p. 10-31, and "Strawberry Lethal Decline," p. 38.)

Symptoms, although of the same general type, may differ in different cultivars (Frazier and Posnette 1958). In 'Lassen', the pathogen causes a severe "yellow-edge"-type symptom accompanied by phyllody and proliferation of flowers, but in 'Shasta', it causes scalding of leaves, cessation of growth, wilting, and rapid death of the plant; intermediate degrees have been observed on other cultivars. There is some



Figure 34.—*Fragaria vesca* 'Alpine' experimentally infected by leafhopper with western aster yellows disease. (Courtesy, N. W. Frazier, University of California.)

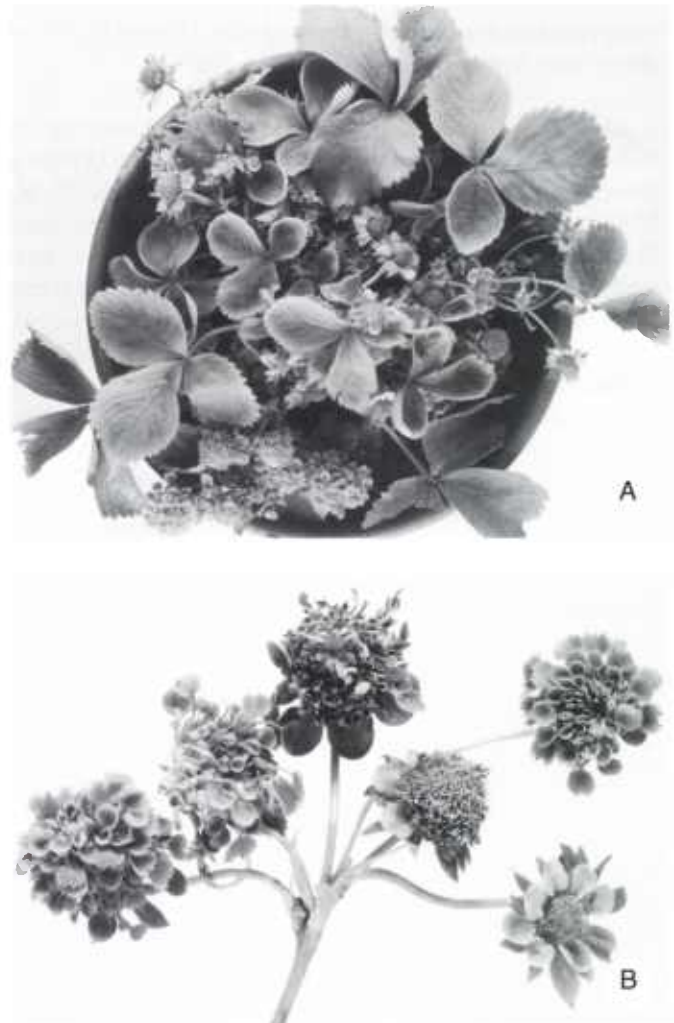


Figure 35.—Strawberry cv. 'Lassen' infected with aster yellows disease: A, Various types of floral abnormalities as well as dwarfed, cupped leaves; B, variation in symptoms in the flowers of one truss. (Courtesy, N. W. Frazier, University of California.)



Figure 36.—Aster yellows symptoms in China aster: A, Eastern strain causing numerous, spindly, chlorotic axillary shoots; B, western strain showing no axillary growth.

suggestion that pathogen strain may also influence symptom expression. The cv. ‘Sparkle’, naturally infected with an eastern strain of aster yellows, displayed reddish rather than virescent petals (Chiykowski 1969).

Although aster yellows has not been found occurring naturally in wild strawberry, it has been experimentally transmitted to several clones of *F. vesca* (Frazier and Thomas 1953). The first evidence of infection consists of a very mild veinclearing and a general chlorosis of the affected leaf. The disease rapidly increases in severity, causing development of adventitious crowns and chlorotic leaves. Petioles become progressively reduced in size until they become very minute shortly before the plants die. Leaflets exhibit upward or downward cupping, and some petioles are twisted or curved, tending to assume a horizontal position.

**Symptoms on other plant species.** Although symptoms on all plant species are not identical, some appear to be characteristic of aster yellows infection. Foliage symptoms in broad-leaved plants consist of veinclearing followed by chlorosis of the entire leaf and a reduction in leaf size. Infection often first appears on only one half of the leaf or plant, but gradually the entire plant becomes chlorotic and dwarfed. Adventitious growth, consisting of dwarfed, short-petioled leaves, from the crown of such hosts as carrot may result in a witches’-broom symptom. Pathogen strain

may affect the type of symptom produced on certain host species. In aster (*Callistephus chinensis* Nees), for example, the eastern strain produces numerous spindly, chlorotic axillary shoots while the western strain produces only a few short, fleshy, rosettelike shoots (fig. 36). Also, the western strain is considerably more severe and produces more stunting than does the eastern strain.

#### Natural and Experimental Transmission

**Leafhoppers.** Twenty-seven species of leafhoppers are known to transmit the western strain but only one, the aster leafhopper, is known to transmit the eastern strain (Chiykowski 1981b). However, the role played by the various species in the epidemiology of aster yellows in strawberry is largely unknown. The following three species have been shown capable of transmitting the pathogen to or from strawberry: (1) *M. fascifrons*, from strawberry to aster (Frazier and Thomas 1953; Chiykowski 1969) and from aster and celery (*Apium graveolens* L. var. *dulce* DC) to clones of *F. vesca* (Frazier and Thomas 1953); (2) *Colladonus montanus* (Van Duzee) from strawberry to plantain and celery (Frazier and Posnette 1958); and (3) *Colladonus geminatus* (Van Duzee), from strawberry to strawberry, plantain, and celery (Frazier and Posnette 1958). *Fragaria* spp., however, do not appear to be very favorable either as food or breeding hosts of these three leafhopper species, suggesting that their role as vectors of aster yellows in



strawberry is a minor one. Vector species such as *Scaphytopius acutus* (Say) (Chiykowski 1962c) and *Aphrodes bicincta* (Schränk) (Chiykowski 1977), which can feed and breed on strawberry, could play a role in areas where western strains of aster yellows are present.

**Grafting.** The pathogen can be transmitted from diseased to healthy strawberry plants by stolon grafts, stolon to petiole grafts, and excised leaflet grafts. Rapid death of diseased tissue often does not allow sufficient time for graft union to occur (Fulton 1957b). The pathogen has also been transmitted by excised leaf grafts from periwinkle (*Catharanthus roseus* (L.) G. Don) to strawberry (Braun and Keplinger 1962).

**Dodder.** *Cuscuta campestris* Yunck. has been used to transmit the pathogen from strawberry to periwinkle and back to strawberry (Fulton 1957).

### Properties of the Causal Agent

The causal agent, once thought to be a virus, is now considered to be a mycoplasma-like organism. The organism has been shown to be present in infected plants and leafhoppers (Maramorosch et al. 1968; Hirumi and Maramorosch 1969). Its general morphological characters are similar to those exhibited by the green petal organism (fig. 43). (See "Strawberry Green Petal and Similar Diseases," p. 34.)

### Detection and Identification

Aster yellows can be detected in strawberry by the symptoms produced on the foliage and on the flowers; however, in areas where green petal disease occurs (see following section), identification is more difficult because of general symptom similarities, overlapping host range, and some common vectors. Positive identification requires the use of symptomatology on specific hosts and transmission characteristics. (See "Strawberry Green Petal and Similar Diseases," table 3, p. 37.)

### Control Procedures

Because of the sporadic occurrence and generally low incidence of aster yellows in strawberry plants, no control procedures have been formulated for this crop. The disease appears to be self-eliminating in strawberry (Frazier and Thomas 1959), suggesting that incidence is dependent on disease sources bordering the plantings and the movement of infective leafhoppers from these sources into the crop. In other susceptible crops where the disease is of economic importance, various control methods have been used with varying degrees of success. These include insecticides for the reduction of leafhopper vectors, herbicides for the elimination of inoculum sources, antibiotics, resistant plants, and various cultural practices (Chapman 1973).

## 245 Strawberry Green Petal and Similar Diseases //

By A. F. Posnette and L. N. Chiykowski

(Contribution by East Malling Research Station and Chemistry and Biology Research Institute, Research Branch, Agriculture Canada, as C.B.R.I. Contribution No. M-1259)

### Additional Common Names

Clover phyllody.

### History and Geographic Distribution

Phyllody fruit on strawberry (defined as the reversion of floral organs such as sepals and bracts to leaves) was observed in Europe more than 300 yr ago and, although some of these may have been affected by green petal disease, they were thought to be genetical abnormalities. The infectious nature of a disease causing flower sterility and phyllody was recognized in Great Britain in 1951 and named green petal (Posnette 1953). Most observers agree that the disease had been present for many years prior to that date, but not recognized because of the similarity of symptoms to those caused by *Verticillium* spp. Frazier and Posnette (1956, 1957) showed that the causal agent of green petal was leafhopper transmitted and was also responsible for the phyllody disease of clover.

In North America, green petal was first reported by Gourley (1955) in Nova Scotia, although growers had observed strawberry plants with green petal symptoms and clover plants with phyllody some years earlier (Lachance 1952). The transmissibility by leafhoppers and relationship of the two diseases in Canada was demonstrated by Chiykowski (1962a, b).

Green petal in strawberry has been reported from North America, Britain, continental Europe, and Russia. In North America, the disease is recognized in eastern Canada from Montreal eastward through Quebec and the Maritime Provinces (Chiykowski et al. 1973). It probably occurs in the northeastern United States although its presence has not been recognized due, undoubtedly, to its similarity to aster yellows. (See "Aster Yellows in Strawberry," p. 31.)

Although green petal of strawberry and a clover phyllody disease have been shown in certain cases to be caused by the same pathogen, one cannot unequivocally state that all cases of phyllody in clover are in fact the same as green petal, since other diseases can also produce similar symptoms. Thus, in describing the geographical distribution of green petal one should only consider reports in which the clover phyllody has been shown to be the same as green petal.

### Economic Importance

The effect of green petal infection on plants is such that they will produce no marketable fruit. Incidence of the disease varies from year to year and appears to be dependent on climatic conditions and cultivars grown. In Great Britain, the annual incidence is usually between 2 and 5%, but in some years it can reach as high as 30%. In Prince Edward Island, Canada, the incidence in the two commonly grown cvs.

'Redcoat' and 'Sparkle' reached 28 and 60%, respectively, in 1966 (Willis and Thompson 1966). In 1967, green petal infection in fruiting fields ranged from 0.2 to 70% (avg. 34%) in Nova Scotia, 12 to 70% in Prince Edward Island, and 0.5 to 31% (avg. 8%) in New Brunswick (Stultz and MacNab 1970). By the early 1970's, the incidence had dropped dramatically. An extensive survey of first crop plantings through Quebec and the Maritime Provinces showed that the highest incidence observed was less than 3% (Chiykowski et al. 1973). Cutcliffe and Thompson (1977) reported an increase in the amount of green petal in Prince Edward Island in 1976 and suggested that cultivar selection might be responsible for the higher incidence.

### Symptoms on Natural and Experimental Hosts

**Natural hosts.** Strawberry ladino clover (*Trifolium repens* L.), red clover (*Trifolium pratense* L.), and alsike clover (*Trifolium hybridum* L.) appear to be the principal natural hosts of green petal. Although other plant species have been observed with similar symptoms, proof that the green petal pathogen is involved is lacking.

**Symptoms on strawberry.** The most obvious symptom and the one from which the name is derived is the appearance of flowers with green petals (fig. 37). Early flower symptoms are in the form of adherent virescent petals, some of which eventually become pink. Petal symptoms increase in intensity on later-developing flowers so that the petals are reduced in size and become dark green and leaflike (phyllody). Some flowers are sterile, while others produce a small, hard, green receptacle which remains dwarfed and does not ripen. The achenes stand out from the receptacle and appear unusually large. Although older foliage may remain normal in size and green or slightly darker green than normal, turning purple in some cultivars, new foliage is dwarfed, slightly asymmetrical, cupped, and pale green with chlorotic margins (figs. 38 and



Figure 38.—Mother and daughter strawberry plants infected with green petal disease showing green flowers and foliage with chlorotic margins.



Figure 39.—Foliage symptoms on strawberry infected with green petal disease. Leaf petioles are short and leaves are dwarfed, slightly asymmetrical, and have chlorotic margins.



Figure 37.—Green petal disease symptoms on strawberry showing green flower petals and affected fruit.

39). Leaf petioles are extremely short in contrast to normal foliage. The degree of cupping appears to vary with the cultivar. For example, cupping can be very pronounced in 'Redchief' but mild in 'Sparkle'. Few runners are formed, and these are stunted and produce only one or two plants. Often the runner terminates in a rosette or clump of small, short-petioled leaves (fig. 40). Most infected plants die after a few months.

**Symptoms on *Trifolium* spp.** The most characteristic symptom is phyllody. The degree of phyllody observed on an infected flower head is dependent on the stage of floral development at time of infection. In flowers which have begun development at time of infection, pedicels of individual flowers and calyx lobes elongate up to three times their normal length. In advanced infection, these calyx lobes become leaflike, complete with venation. The standard, keel, and wings of the flower, while remaining normal in color, are greatly reduced in size and in advanced infection may be completely absent. The flower then consists of an enlarged





Figure 40.—*Left*, Normal strawberry runner plant; *right*, Runner of plant infected with green petal disease showing a terminal rosette of small, short-petioled leaves.



Figure 41.—*Left*, Healthy inflorescence of Ladino clover; *right*, two inflorescences infected with green petal (clover phyllody) disease showing different degrees of phylloid development.



Figure 42.—Green petal (clover phyllody) disease symptoms in individual flowers of Ladino clover: *Left*, Healthy flower; rest, progressive development of phyllody showing reduction in petal size, absence of petals, enlargement of ovary, proliferation of ovary, replacement of ovary by a leaf, and enlargement of the calyx.

calyx and enlarged ovary. The ovary may become twice the normal size and develops a stipe. In advanced infection, the ovary may proliferate into a simple or trifoliate leaf, or the ovary may be replaced by a simple leaf (figs. 41 and 42). Foliage symptoms consist of chlorosis of new leaves with reduced leaf size and petiole length.

**Experimental hosts.** The pathogen has a relatively wide host range with at least 79 species in 22 families shown to be susceptible (Chiyskowski 1967, 1974). The host list includes annual and perennial weeds and cultivated species of monocotyledons and dicotyledons.

Foliar symptoms on these species generally consist of mild vein clearing, mild chlorosis, stunting as a result of a shortening of internodes, increased axillary growth, and reduced leaf size. Thickening of crowns in some species produces a witches'-broom effect. Many species produce considerably more flowers than do healthy plants. The most striking symptoms occur on the flowers. The calyx is greatly enlarged and leaflike with veins. The petals are enlarged, green, and take on leaflike characteristics. The ovary becomes pedicellate and may eventually split open, developing into a leaflike structure. Often the ovary is replaced by vegetative growth.

#### Natural and Experimental Transmission

**Leafhoppers.** Several leafhopper species have been shown capable of transmitting the green petal pathogen, but their roles in spreading the disease in the field have not been fully evaluated. Only *Aphrodes bicincta* (Schrank) has been shown to transmit the pathogen to and from strawberry, although species such as *Macrostes fascifrons* (Stål), *Euscelis lineolata* Brulle, and *Euscelis incisus* (Kbm.) (also known as *E. plebeja* [Fall.]) have been shown to transmit from strawberry to clover and to other hosts, but not back to strawberry. Other species, such as *Macrostes viridigriseus* (Edwards), *Macrostes cristatus* (Ribaut), *Anoscopus albifrons* (L.) (also known as *Aphrodes albifrons* [L.]), *Scaphytopius acutus* (Say), *Paraphlepsius irroratus* (Say), and *Speudotettix subfuscus* (Fall.), have been shown to transmit the pathogen from and to clover.

Experimental evidence suggests that *A. bicincta* is the principal vector responsible for infecting strawberry and that the other species are important in maintaining the disease in clover and weed hosts. Plants can become infected in the field from June to October, but transmission appears to reach a peak in August (Chiyskowski 1962a, Thompson 1968).

**Non-leafhopper transmission.** Green petal has been transmitted by the following species of dodder: *Cuscuta subinclusa* Dur. and Hilg., from clover to strawberry and to *Duchesnea indica* (Andr.) Focke; *C. campestris* Yunck., to strawberry cultivars and to *Fragaria vesca* L.; *C. arvensis* Beyr. and *C. europaea* L., from clover to *F. vesca*; and *C. gronovii* Willd., from periwinkle (*Catharanthus roseus* (L.) G. Don.) (also known as *Vinca rosea* L.) to periwinkle.

**Table 3.—Distinctions between green petal and aster yellows**

Point of comparison	Green petal	Eastern aster yellows	Western aster yellows
Latent period in vector	28-35 days at 25 °C	14-21 days at 25 °C	14-21 days at 25 °C.
Transmission by:			
<i>Macrostes fascifrons</i>	Higher by females	Sexes equal	Sexes equal.
<i>Aphrodes bicincta</i>	Yes	No	Yes.
<i>Scaphytopius acutus</i>	Yes	No	Yes.
Symptoms on:			
<i>Trifolium repens</i> petals	White and reduced or absent.	Reduced and green	(1)
<i>T. r.</i> calyx	Enlarged and leaflike	Near normal	(1)
<i>Callistephus chinensis</i> :			
Incubation period	23 days	16 days	14 days.
Head proliferation	Yes	No	No.
Chlorosis	Mild or absent	Severe	Severe.
Vein clearing	Mild	Severe	Severe.
Axillary growth	Slight and green	Profuse and chlorotic	None.
<i>Catharanthus roseus</i> :			
Axillary growth	Sparse or absent	Profuse	Absent.
Chlorosis	Mild or absent	Severe	Severe.
Petals	Dark green and leaflike	Light green, normal size and texture.	Light green, normal size and texture.
<i>Apium graveolens</i>	Resistant	Susceptible with long incubation period.	Highly susceptible.
<i>Nicotiana rustica</i> :			
Chlorosis	None	Severe	Severe.
Vein clearing	Mild	Obvious	Obvious.
Flowering	Profuse	Few	Few.

<sup>1</sup>No comparison made.

The disease has also been transmitted by grafting from strawberry to strawberry.

### Properties of the Causal Agent

The causal agent, once thought to be a virus, is now considered to be a mycoplasma-like organism (fig. 43). The organism has been shown to be present in infected plants and transmitting insects (Sinha and Paliwal 1969, 1970; Cousin et al. 1970; Beakbane et al. 1971).

### Detection and Identification

Green petal can be easily confused with some types of aster yellows disease. The two diseases have an overlapping plant host range, in some cases an overlapping geographical range, and some vector species in common. Positive identification requires the use of symptomatology on specific plant hosts and transmission characteristics (see table 3).

### Control Procedures

**Insecticides.** Studies on controlling green petal through control of vectors by chemical sprays have been limited. Collins and Morgan (1958) reported some control of spread in plots treated with malathion. Razvyazkina (1960) recommended weed-host eradication and general application of DDT, but gave no data to support these measures. Willis and Thompson (1966) reported that strawberry plantings transplanted with plants from nurseries treated the previous year with granular disulfoton applied as sidedressings had fewer infected plants than those treated with malathion sprays. Field experiments were conducted over a 2-yr period by Thompson et al. (1973) to determine the efficacy of several insecticides in reducing green petal disease. In the first year, disulfoton, soil-incorporated before planting, was most effective in reducing the incidence of infected plants. In the following

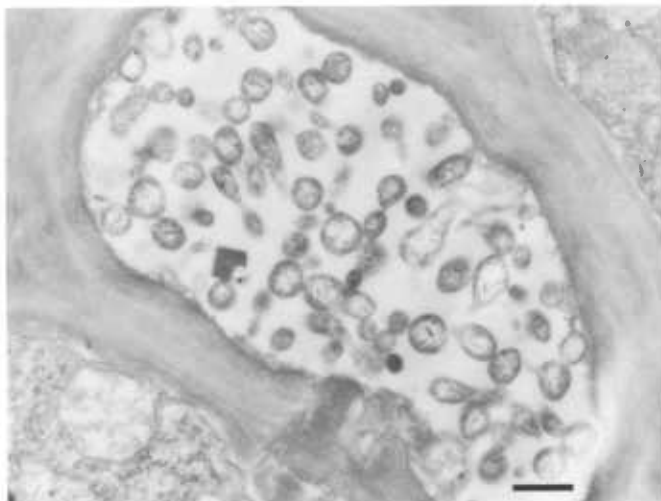


Figure 43.—Mycoplasma-like organism in phloem cell of Ladino clover infected with green petal (clover phyllody) disease. (Bar represents 500 nm.)

year, three foliar sprays of DPX-1410 (S-methyl 1-(dimethylcarbamoyl)-N-[(methylcarbamoyl) oxy] thioformate) (Du Pont of Canada), endosulfan, or oxydemeton-methyl gave the best results; however, fruit yields were not increased by any of the treatments.

**Antibiotics.** Demonstration that the mycoplasma causal agents of several yellows-type plant diseases are sensitive to tetracycline antibiotics (Ishii et al. 1967) offers another possible approach to controlling these diseases. Although antibiotics have not been used for control of green petal under field conditions, they have been experimentally tried in a greenhouse environment. Oxytetracycline, tetracycline, and doxycycline applied as root dips, foliage dips, or sprays to aster plants immediately after inoculation by leafhoppers delayed the development of symptoms, while nontetracycline antibiotics had no effect (Chiakowski 1972, 1973). Root treatment of infected asters with oxytetracycline resulted in remission of symptoms, although symptoms eventually reappeared some weeks later (Sinha and Peterson 1972).

**Cultural.** Field observations (Chiakowski et al. 1973; Collins and Morgan 1958; Gourley et al. 1971; Thompson and Cutcliffe 1972; Willis and Thompson 1966; Cutcliffe and Thompson 1977) and greenhouse experiments (Chiakowski and Craig 1975, 1978) suggest that strawberry cultivars differ in their reaction to green petal infection. While some of the differences are dependent on the cultivar-pathogen reaction, variation in disease incidence appears to depend on vector preference for certain cultivars. Therefore, selection of cultivars for resistance to both pathogen and vector species would be highly desirable for reducing green petal incidence.

The selection of growing site can also be important. Various clover species are known to be highly susceptible to green petal and also serve as breeding areas for such leafhopper

species as *A. bicincta* and *S. acutus*. Culture of strawberries near or adjacent to such crops should be avoided.

Origin of plants for new plantations should be carefully chosen. Stultz and MacNab (1970) found that amounts of green petal diseased plants observed in new plantations, regardless of their location, were characteristic of the nurseries from which the plants originated and were generally low when obtained from certified nurseries. In contrast, disease incidence in new plantations with plants taken from common field stock reached up to 50%.

**Heat therapy.** Green petal pathogen, originating from strawberry, was not eradicated from entire periwinkle plants treated at 40° to 42°C for 3 wk, but 4 of 10 cuttings from these plants were free of the pathogen. Another isolate originating from a *Helianthus* species was eradicated from one of three entire periwinkle plants treated at 40° to 42°C for 3 wk, and all of 10 cuttings were free of the pathogen (Posnette and Ellenberger 1963).

## 245 Strawberry Lethal Decline

By C. D. Schwartz, N. W. Frazier, and R. H. Converse

### Additional Common Names

Northwest disease. X-disease of stone fruits is possibly related to lethal decline disease (LDD) (Frazier and Jensen 1970).

### History and Geographic Distribution

LDD was first observed in western Washington about 1952 in 'Northwest' strawberry and was subsequently seen in other cultivars and unnamed strawberry seedlings. This disease was described by Schwartz and Frazier (1964). Since 1964, LDD has been reported in British Columbia and Oregon (Schwartz and Frazier 1970; Converse and Bartlett 1971).

### Economic Importance

LDD is a minor problem in strawberry fruit production, rarely affecting more than 2% of field plants, although 25% infection has been observed. This disease has been noted as a factor in survival of transplants. Late autumn infection in strawberry nurseries may produce no symptoms until after plants are dug, stored, and replanted. Transplant failure caused by LDD has ranged up to 8% (Schwartz and Frazier 1970).

### Symptoms on Natural and Experimental Hosts

LDD has been transmitted only to the genus *Fragaria*, but all cultivars and all species tested (*F. vesca* and *F. chiloensis*) are susceptible. The incidence of natural infection differs among cultivars.

**Symptoms in cultivars.** Symptoms first appear in mid-May and continue to appear for 2 mo. It is rare to find new infections from mid-July to mid-October, but new infections are visible after that into the autumn and winter. In the spring and in the greenhouse, infected plants develop dwarfed,



chlorotic, upward-cupped young leaves with unequal-sized leaflets. Mature leaves on infected plants may be dull, chlorotic, and somewhat bronzed above, rolled upward, and reddish or purplish beneath. This dull or “dirty” chlorosis is a characteristic symptom of LDD (fig. 44). As the disease progresses, more mature leaves exhibit these symptoms and die. Only a few, dwarfed, chlorotic, distorted young leaves remain before the plant dies.

In the autumn and winter, the premature reddening of the foliage of LDD-infected plants is often used as a diagnostic marker to eliminate infected mother plants and their daughter plant systems. At digging time, daughter plants of infected

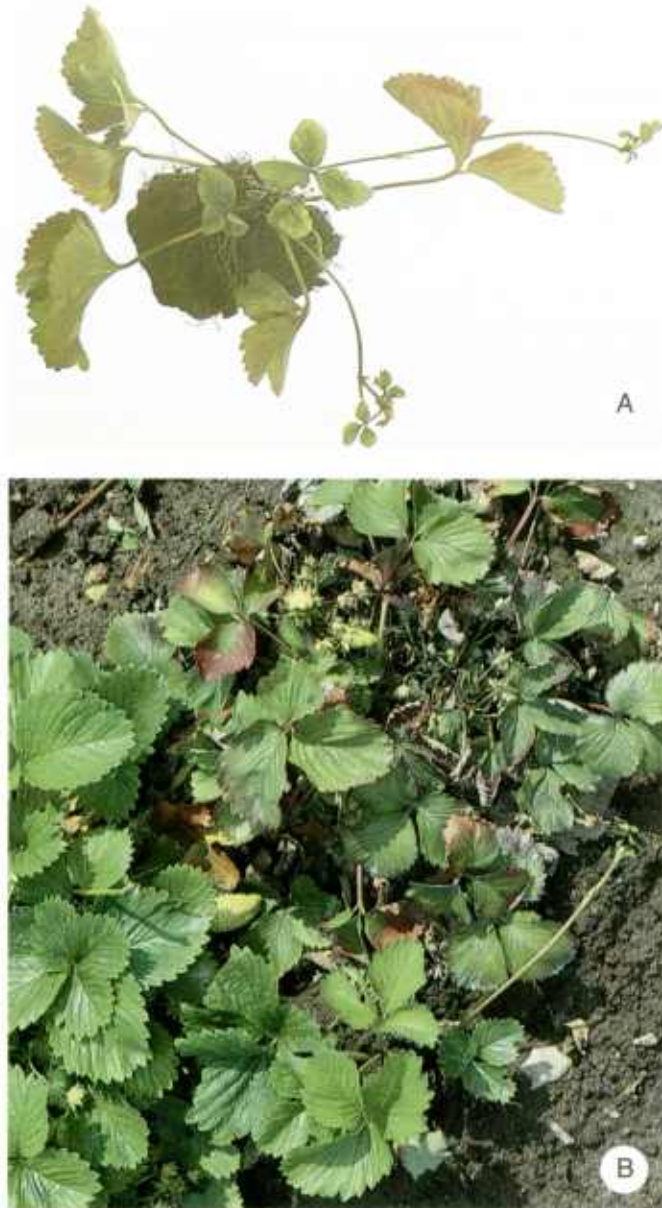


Figure 44.—A, ‘Hood’ strawberry infected with lethal decline disease, Elkton, Oreg., September 1970, showing bronzing of older leaves and cupping of younger ones; B, dwarfed, rosetted strawberry seedling infected with lethal decline disease (above) next to normal plant (below) in the same row.

mother plants are often conspicuously chlorotic and stunted, but some infected daughters may be symptomless. Apparently not all daughter plants in a stolon chain from an infected mother plant develop LDD, which suggests an uneven distribution of the pathogen within each plant. Infected daughter plants die within a month after transplanting as nursery stock.

In the field, plants that are infected with LDD usually die before producing flowers or fruit. Plants infected by grafting in the greenhouse usually have sterile blossoms without petals (but not virescent petals) on shortened pedicels. Receptacles may be elongated and necrotic and the calyx lobes reddened.

Cultivars graft-inoculated with LDD in the greenhouse develop bronzed, wilted leaves (fig. 45). These symptoms are preceded by degeneration of the older roots, which become dark brown to black throughout.

**Symptoms on *Fragaria vesca* indicators.** Symptoms of LDD after inoculation by leaflet grafting are about the same as in strawberry cultivars. Some variations in tolerance to LDD infection have been noticed among *F. vesca* clones, but all are eventually killed by the disease following graft inoculation.

The peach yellow leafroll-type of X-disease, caused by a mycoplasma-like organism (Nasu et al. 1970), was experimentally transmitted to *F. virginiana* cv. ‘UC-10’ and to ‘Hood’ strawberry by the leafhopper *Colladonus montanus* (Van Duzee). On these hosts, it produced symptoms resembling those of LDD (fig. 46) (Frazier and Jensen 1970). X-disease transmissions from *Prunus* species to *F. vesca* by dodder and by leaf graft have been reported in Eastern United States, resulting in mild or unspecified symptoms (Slack 1952; Braun and Keplinger 1962).



Figure 45.—Strawberry cultivar leaf-graft inoculated with lethal decline disease 4 mo previously, October 1963, Washington State University.



Figure 46.—Peach yellow leafroll strain of Western X-disease leaf grafted to *Fragaria virginiana* cv. 'UC-10', causing chlorotic discoloration of older leaves, severe dwarfing of younger leaves, crown necrosis, and runner death, soon becoming lethal to the entire plant.

### Natural and Experimental Transmission

**Natural transmission.** The causal agent of LDD was not transmitted from strawberry to strawberry by the aphid *Chaetosiphon jacobi* H.R.L. nor by five species of leafhoppers known to transmit aster yellows disease (see "Aster Yellows in Strawberry," p. 31): *Colladonus geminatus* (Van Duzee), *C. montanus* (Van Duzee), *Euscelidius variegatus* (Kirschbaum), *Fiebierella florii* (Stål), and *Macrosteles fascifrons* (Stål), or by a vector of Pierce's disease: *Graphocephala atropunctata* (Signoret) [also known as *Hordnia circellata* (Baker)] (Schwartz and Frazier 1964). *C. geminatus* transmitted the peach yellow leafroll agent (Western X-disease) to strawberry, causing symptoms similar to LDD, as already noted (Frazier and Jensen 1970). The leafhopper *Aphrodes bicincta* (Schrank) was able to acquire and transmit LDD from strawberry to strawberry in preliminary greenhouse tests (R. H. Converse, unpublished results).

LDD has spread in plants in fumigated soils. This observation is in keeping with a hypothesis that LDD is not nematode borne. Furthermore, mapping the locations of LDD-infected plants in a cultivated strawberry field suggested that most infections are widely scattered, with 39% clumping of LDD-infected plants in pairs or triplets (Converse and Bartlett 1971).

**Experimental transmission.** LDD has been transmitted by petiole insert leaflet grafting from *Fragaria* to *Fragaria*. The time lapse for appearance of symptoms following graft inoculation varies from 3 to 16 wk (mean, 8 wk). Because no

symptomless hosts of LDD are known and all susceptible hosts are severely weakened or killed after inoculation, sequential graft inoculation of LDD isolates becomes progressively more difficult. It has been found to be impossible to maintain isolates for long periods of time in the greenhouse.

Dodder transmission of a strain of X-disease from *Prunus* to *Fragaria* was claimed in Eastern United States, as previously noted (Slack 1952). Johnson (1969) graft inoculated *Nicotiana tabacum* L. with LDD-infected strawberry petioles. Using *Cuscuta subinclusa* Dur. & Hilg., he was then able to cause witches'-broom symptoms on one *Trifolium repens* L., which then gave rise to dwarfism, leaf distortion, and epinasty in one mechanically inoculated *Lycopersicum esculentum* Mill. plant. This work was not repeated and requires confirmation.

### Properties of the Causal Agent

Electron microscopy of sieve tubes of LDD-infected 'Northwest' strawberry revealed mycoplasma-like bodies averaging 173 nm in diameter (fig. 47) (R. H. Converse, unpublished data), thus supporting the possible relationship of LDD to Western X-disease suggested by Frazier and Jensen (1970) since it would be expected to be caused by a mycoplasma-like agent (Nasu et al. 1970; Granett and Gilmer 1971; Jones et al. 1974). Western X-disease and its leafhopper vectors were recently reviewed by Gold (1979). X-disease occurs in several "strains" in various parts of North America (Gilmer and Blodgett 1976).

Preliminary attempts to moderate LDD symptoms by heat therapy and by tetracycline therapy (Nienhaus and Sikora 1979; R. H. Converse, unpublished data) were unsuccessful.

### Detection and Identification

No method is superior to direct observation of characteristic symptoms in infected plants in the field. Transmission by leaflet graft to *F. vesca* can be done to confirm field diagnosis if it is necessary to distinguish between LDD and herbicide damage.

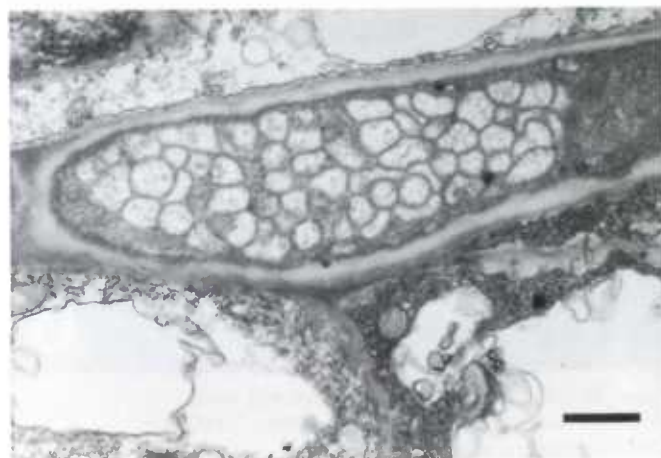


Figure 47.—Mycoplasma-like bodies in sieve tube of cv. 'Northwest' infected with lethal decline disease. (Bar represents 1 micron.)



## Control Procedures

Control is mainly by thorough roguing of infected mother-daughter plant systems in autumn and winter. No successful therapy of LDD has been reported.

## Remarks

The nature and identity of the causal agent of LDD are unknown. Until precise identification methods are developed for the causal agent, it will be difficult to determine the natural reservoirs and means of spread of this disease accurately.

## Strawberry Rickettsia Yellows and Mycoplasma Yellows

By R. S. Greber

### Additional Common Names

Lethal yellows for both diseases; little leaf or big bud for the disease associated with mycoplasma-like organisms (MLO). The etiological relationship, if any, with diseases such as "bronze leaf wilt" in Great Britain (McGrew and Posnette 1970) and "lethal decline" in the United States (Schwartz and Frazier 1964, 1970) is unknown. (See 'Other Leafhopper-Borne Diseases of Strawberry,' p. 45, and "Strawberry Lethal Decline," p. 38.). Strawberry rickettsia yellows and mycoplasma yellows diseases result in the development of small chlorotic leaves, decline, and death, usually without development of prominent green petal symptoms.

### History and Geographic Distribution

The first description of lethal yellows was in Australia by Stubbs (1968) and followed the production and use of planting material free of serious virus disease complexes such as severe yellow edge. The prevalence of virus diseases which produced similar symptoms had previously inhibited the recognition of the diseases associated with MLO and rickettsialike organisms (RLO) except for the distinctive green-petal disease. Shanmuganathan and Garrett (1976) described a little leaf disease in Victorian strawberries, and a similar disease in Queensland was referred to either as lethal yellows or "big bud." The latter name related to an etiological association with MLO from the tomato big bud disease. Neither of these names seemed particularly appropriate because the MLO disease of strawberries in the subtropics was frequently not lethal nor was it associated with flower bud phyllody or distortion. In Victoria, the MLO disease is invariably lethal (Shanmuganathan and Garrett 1976). The mycoplasma-associated strawberry lethal yellows, little leaf or big bud diseases are hereafter referred to as mycoplasma yellows or MLO disease, and the rickettsia-associated lethal yellows is referred to as rickettsia yellows or RLO disease.

Rickettsia yellows has been reported only from Queensland and was distinguished from mycoplasma yellows only following electron microscope thin section examination (Greber and Gowanlock 1979) of plants which seemed to have a slightly atypical yellows symptom. This disease was

frequently lethal under hot conditions. The disease has since been detected from other locations in Queensland, but is not easily distinguished from mycoplasma yellows because of symptom variation overlap between the two (Greber and Gowanlock 1979).

The Australian diseases are difficult to correlate with those of the little-leaf or lethal yellows types occurring elsewhere because of lack of data from electron microscopy of thin sections or chemotherapy evidence. Bronze leaf wilt (McGrew and Posnette 1970) probably falls into this group and perhaps also lethal decline (Schwartz and Frazier 1970) and witches'-broom (Mellor and Fitzpatrick 1961). (See chapters on these diseases in the Strawberry section.)

### Economic Importance

Both the MLO and RLO diseases occur sporadically and are of economic importance mainly because they invade runner production areas isolated from strawberry virus infection. Apart from losses of plants by death and roguing on the runner producing farms, it is difficult to avoid distribution of runners carrying latent infections. These cause more serious losses on fruit production farms because it is often too late to replant by the time symptoms are diagnosed. Serious outbreaks are infrequent and may become less frequent with present control measures.

### Symptoms on Natural and Experimental Hosts

**Mycoplasma yellows.** Older leaves develop some bronze or purple pigmentation and roll upward around the midvein. New leaves have small leaflets and shorter petioles. Leaf margins are yellow or chlorotic and the lamina becomes either chlorotic or bronzed (figs. 48, 49 A and B). Flower and fruit production is inhibited and plants often die, although under warmer conditions they often persist until rogued out. All runners attached to affected mother plants eventually develop symptoms. In graft-infected *Fragaria vesca* L. seedlings and cv. 'UC-1' indicators or in strawberry cultivars (fig. 49 B), symptoms are similar to those occurring in the field.



Figure 48. — Strawberry plant cv. 'Redlands Crimson' infected with mycoplasma yellows.



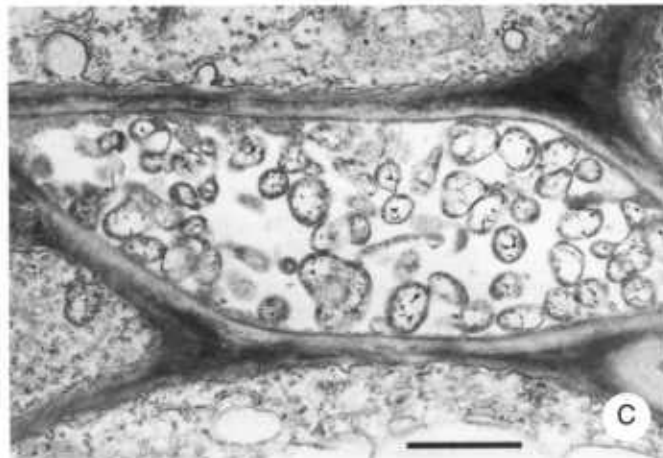
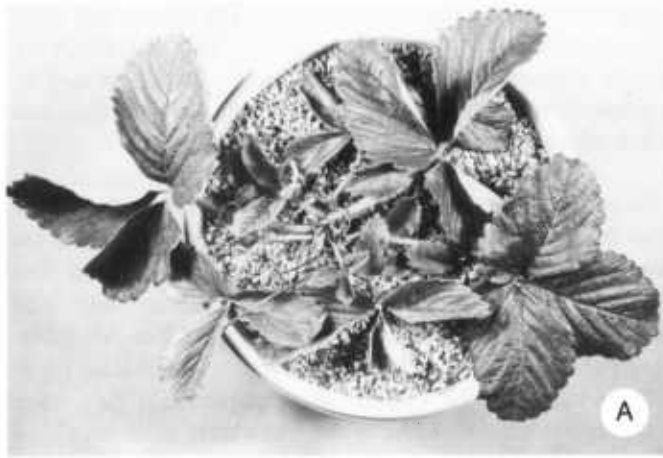


Figure 49. — A, Symptoms of mycoplasma yellows on strawberry cv. 'Earlisweet'; B, little leaf symptoms of mycoplasma yellows on strawberry cv. 'Redlands Crimson' following leaf graft transmission; C, mycoplasma-like bodies in sieve tube of strawberry cv. 'Redlands Crimson' infected with mycoplasma yellows. (Bar represents 1000 nm.); D, detail of mycoplasma-like bodies in sieve tube of mycoplasma yellows infected strawberry. (Bar represents 200 nm.)

The end result was invariably lethal with Victorian isolates (Stubbs 1968; Shanmuganathan and Garrett 1976). N. Shanmuganathan (personal communication) reports that flowers on affected plants in Victoria have small petals with a tinge of green. New leaves of these plants have shortened petioles and are reduced in size, chlorotic, and generally cupped. Affected plants wilt suddenly and die within a few weeks.

**Rickettsia yellows.** Bronze and purple pigments develop in older leaves. Younger leaves develop interveinal chlorosis and tend to bend back along the midvein (fig. 50 A). Leaves are usually more rigid, tear easily, and may be asymmetric (fig. 50 B). Under high temperatures (daily max.  $>30^{\circ}\text{C}$ ), some petioles and stolons become necrotic, and affected mother plants frequently wilt and die, leaving a cluster of chlorotic, infected runners. Flowers are not affected until plants wilt or become severely debilitated, when they

frequently abort. Often a mild vascular discoloration is evident when affected crowns are cut and examined.

Symptoms on strawberry cultivars and the indicator *F. vesca* cv. 'UC-4' resulting from leaf-graft infection follow a pattern similar to those in field plants (fig. 50 C). The severity of symptoms produced and frequency of death are increased by growing plants at high temperatures. When infected plants are maintained for several months at temperatures below  $25^{\circ}\text{C}$ , symptoms decrease and most plants eventually show normal growth, which usually develops following formation of a new crown from axillary buds.

#### Natural and Experimental Transmission

The disease symptoms for both the MLO and RLO diseases begin to develop on leaf-graft inoculated *F. vesca* and strawberry plants after 3 to 9 wk. Graft-inoculation from field plants with severe symptoms of either disease was efficient

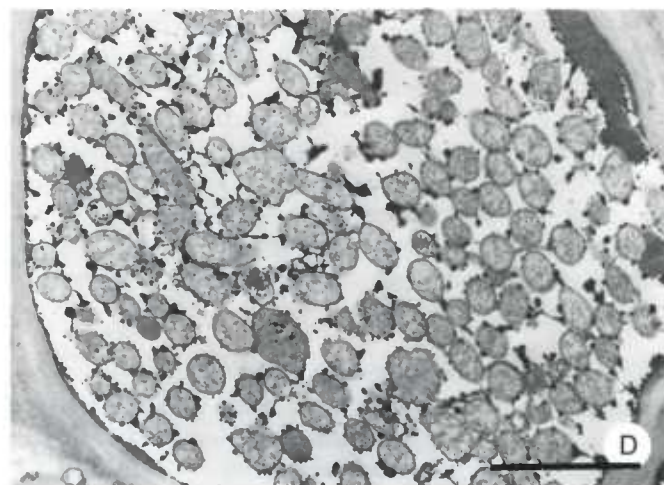
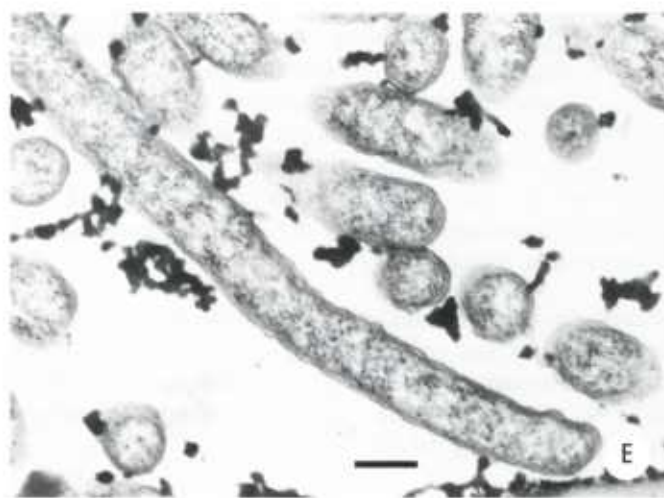


Figure 50.—A, 'Redlands Crimson' strawberry: Left, freed of rickettsia yellows by treatment with 1,000 ppm sodium penicillinate; right, infected with rickettsia yellows; B, 'Tiobelle' strawberry: Left, healthy leaf, right, leaf infected with rickettsia yellows; C, *Fragaria vesca* 'UC-4' infected with rickettsia yellows; D, closely-packed rickettsialike bodies, mostly in cross section, in a sieve tube of strawberry infected with rickettsia yellows. (Bar represents 1000 nm.) E, Detail of rickettsialike body in a sieve tube of 'Redlands Crimson' strawberry infected with rickettsia yellows. (Bar represents 200 nm.)

when two grafts were made to medium-age leaves on each test plant. Inoculation from chronic infections was less reliable, and RLO-infected plants which had been grown at less than 25°C were poor sources for leaf-graft transmission. This was correlated with difficulty in locating the organism in electron microscope thin sections of midribs and petioles.

Most reported vectors of RLO and MLO diseases are leafhoppers from the family *Cicadellidae*, and known leafhopper vectors of phloem-inhabiting RLO are from the subfamily *Agalliinae* (Nielsen 1979). Bronze leaf wilt has been shown to be transmitted by the leafhoppers *Euscelis* spp. and *Macrosteles* spp. (McGrew and Posnette 1970). Transmission of MLO diseases in Australia has been demonstrated only by *Orosius* spp. (Grylls 1979; Greber and Gowanlock 1979). The phloem RLO-associated disease, clover rugose leaf curl (Behncken and Gowanlock 1976), is



transmitted by *Austroagallia torrida* Evans (Grylls 1979). Attempts to transmit strawberry rickettsia and mycoplasma yellows by these vectors were unsuccessful (Greber and Gowanlock 1979), but the environmental conditions necessary for transmission to strawberry may not have been fulfilled, even though other plant species were infected in these tests. Since natural infection only occurs during high temperatures and the concentration of RLO in leaves at lower temperatures is poor (R. S. Greber and D. H. Gowanlock, unpublished data; Markham et al. 1975), it may be necessary

to conduct these vector tests at temperatures around 30°C to achieve transmission to strawberry.

Attempts to transmit clover phyllody organisms to strawberry in Victoria using *Orosius argentatus* (Evans), *Nesoclutha pallida* (Evans), and *Zygina zealandica* (Myers), were unsuccessful, but typical little leaf symptoms developed in strawberry after dodder (*Cuscuta campestris* Yunck.) transmissions from white clover with phyllody and tomato with big bud symptoms (N. Shanmuganathan, personal communication). Transmission from a clover green petal source and from a source which produced big bud disease of tomato to strawberry using dodder (*C. campestris*) was also reported by Helms (1962). Her transmission resulted in early death of *F. vesca* plants, and small green petals developed on most plants of strawberry cv. 'Climax', which then died after 2 to 4 mo.

The epidemiology of both mycoplasma and rickettsia yellows indicates that natural infection is highest following periods of hot, dry weather. At these times, natural vegetation is becoming dry and unattractive to leafhoppers, and the irrigated strawberry runner production plantings provide alternative hosts, which may not be preferred under normal conditions.

### Properties of the Causal Agent

There is a consistent association of MLO with mycoplasma yellows and RLO with rickettsia yellows. This has been shown by thin section electron microscopy of both naturally and experimentally infected plants. No such organisms were found in sections of healthy plants or those showing symptom remission following chemotherapy (Greber and Gowanlock 1979). This indicates a high probability of a causal association; however, the status of the organisms has not been demonstrated unequivocally by isolation to cell-free cultures and reinoculation to produce the diseases. The symptoms of both diseases show similarities and the organisms associated with the two diseases have similar phloem tissue locations.

Strawberry-infecting MLO have typical pleomorphic shapes in thin section and vary greatly in size and shape, but are usually in the range of 200 to 500 nm across (fig. 49 C). They have a single trilaminar membrane and characteristic radiating fibrils (fig. 49 D).

Strawberry-infecting RLO are found in sieve tubes and are accompanied by dark-staining granular material. In cells densely packed with the organism, they appear approximately circular in outline (fig. 50 D), but this could be due to their alignment, and more of the elongate forms are found in less densely packed cells (fig. 50 E). The elongate forms (up to 1µ) have ridged cell walls and an inner cell membrane. Ribosomelike and fibrillar structures are present within the RLO cells. Assignment of these organisms to systematic categories within Mycoplasmatales and Rickettsiales may also be premature until their characteristics have been

investigated in pure cultures; however because their morphologies closely resemble other members of these groups, the MLO and RLO terminology has been maintained in this chapter.

### Detection and Identification

Both diseases are readily detected in the field by the characteristic little-leaf-yellows symptoms, especially in the absence of virus diseases. They can be visually differentiated from fungal diseases, such as fusarium wilt, which cause necrosis of the older leaves rather than purple pigmentation and rolling of the laminae. This pigmentation and the chlorotic margins of younger leaves also distinguish the yellow diseases from bud nematodes infection (*Aphelenchoides besseyi* Christie). (See "Bud and Leaf Nematodes of Strawberry," p. 72.) Neither of the yellows diseases causes green-petal symptoms. (See "Strawberry Green Petal and Similar Diseases," p. 34.) Flowers may abort on plants infected with either strawberry rickettsia yellows or mycoplasma yellows, but flowering is more likely to continue with RLO infections.

Leaf graft transmission (allow for several weeks of latency), coupled with isolations to detect fungal parasites, can be used to confirm infection. However, it is difficult to distinguish between mycoplasma yellows and rickettsia yellows on symptoms alone, and etiology must be confirmed by examination of thin sections in the electron microscope.

### Control Procedures

Roguing of affected mother plants and all attached runners did not prevent distribution of latently infected runners when the infection period continued until near digging time. No severe infection, however, has occurred in Queensland after institution of a spray program on runner production farms, using 0.3 g dimethoate per liter (0.03%) every 5 days during summer. This program has not been in operation long enough to have proved its effectiveness under all conditions. Continuous maintenance of a high level of insecticide is practicable during this nonfruiting period.

In Victoria, control measures include insecticide spraying of uncultivated land adjoining runner crops, clean cultivation of a buffer strip 10 m wide around the crop, and biweekly spraying of insecticides on the runner crop (N. Shanmuganathan, personal communication).

Mycoplasma yellows usually terminates in death of the plant, but roguing is commonly practiced by fruit-growing farmers as soon as affected plants are recognized. Rickettsia yellows, on the other hand, can show natural symptom remission under cooler conditions, and plants will again become productive.

Treatment of rickettsia yellows-affected individual plants with penicillin drenches (1000 ppm sodium penicillinate) at 5-day intervals for 8 wk can effect a cure, especially if plants

are grown with daily temperature maxima below 30°C. In plants treated in this way, organisms or symptoms have not been detected at any time during the subsequent 2 yr of growth under a variety of environmental conditions. The procedure should enable infected clones to be freed of this disease.

### Remarks

The fact that the etiology of little leaf, yellows and similar diseases of strawberries in many countries has still not been investigated by thin-section electron microscopy causes difficulty in making comparisons between diseases which have been described from different locations. While the green-petal diseases have been well examined, there are few reports of other MLO-associated diseases of strawberry. There are also few reports of the effect of antibiotics on these diseases, and the vector transmission aspect has only been investigated by a few workers in recent years.

The characteristic yellows symptoms probably result from the effect on phloem function by the organisms, which are exclusively located in this tissue. Very similar symptoms are caused by these two quite unrelated organisms, which infect strawberry phloem tissue.

Most MLO are sensitive to tetracycline antibiotics, but the effectiveness of penicillin on an organism could imply that it has a true cell wall. The RLO which are associated with strawberry rickettsia yellows have been shown to have true cell walls. The presence of a cell wall as well as a cell membrane distinguishes RLO from MLO in thin sections. The RLO are also associated with a dark-stained granular material of unknown function in the sieve tubes, which is not present with MLO.

Now that the etiology of these diseases is understood, control measures can be instituted. All diseases of this type, which infect phloem tissue, are transmitted by cicadellid leafhoppers. None of this group of insects is known to breed on strawberries in Australia, and the inoculum and vectors probably originated from surrounding vegetation. There are many possible sources of MLO from other host species. RLO infect clover in Great Britain and Australia (Markham et al. 1975; Behncken and Gowanlock 1976), and the same or similar organisms may be the cause of corresponding diseases in strawberries.

### Other Leafhopper-Borne Diseases of Strawberry<sup>11</sup>

By L. N. Chiykowski and A. F. Posnette

Several leafhopper-borne diseases have been reported affecting or capable of affecting strawberry, but information on their economic importance, epidemiology, and relationship with other similar diseases is generally lacking. Their inclusion in this handbook will serve to alert the reader to their existence and possibly to stimulate further work to determine their effects on strawberry production.

### Bronze Leaf Wilt

(Clover witches'-broom). Frazier and Posnette (1957) distinguished two pathogens they transferred from clover to strawberry; one caused phyllody and green petal in clover and strawberry, respectively, and the other produced proliferation (witches'-broom) in clover and bronzing reddening and wilt in strawberry but no flower symptoms in either host.

The pathogen was transmitted by dodder, *Cuscuta campestris* Yunck., to *Fragaria* cultivars and by the leafhoppers, *Euscelis lineolatus* Brulle, *E. incisus* (Kbm.), and *Macrosteles* sp. from clover and strawberry to clover, carrot, celery, and tomato, but not to strawberry (Posnette and Ellenberger 1963). It was not transmitted by *Aphrodes bicincta* (Schrank), and the means by which strawberry plants become infected in the field is not known. The incidence of the disease in strawberry is difficult to assess because of its resemblance to green petal disease in the absence of flowers (see "Strawberry Green Petal and Similar Diseases," p. 34) and to *Verticillium* wilt. Bronze leaf wilt may be more prevalent than the lack of reports would indicate (McGrew and Posnette 1970).

The causal agent was assumed to be a virus of the yellows group because of its transmission by leafhoppers and dodder. Although no information is available on its morphology, the agent is now suspected of being a mycoplasma-like organism. The bronze leaf wilt pathogen appears to interfere with the transmission of green petal by *E. plebeja* (Fall.), but the green petal pathogen does not protect clover plants from infection by bronze leaf wilt. The latter pathogen retards the development of *E. plebeja* and apparently reduces the insect's longevity (Posnette and Ellenberger 1963).

### Delphinium Yellows

Using dodder, *Cuscuta campestris* Yunck., Posnette and Ellenberger (1963) transferred a pathogen from a garden *Delphinium* hybrid with phyllody to *Fragaria* cultivars and *Catharanthus roseus* (L.) G. Don. Symptoms in strawberry were leaf bronzing and rapid death, indistinguishable from those of bronze leaf wilt. The infected *C. roseus* plants became chlorotic and developed small flowers with green petals; some phyllody occurred but was less pronounced than with strawberry green petal disease.

The pathogen was transmitted by *Macrosteles sexnotatus* (Fall.) from *C. roseus* to *Trifolium repens* L. The affected plant died within 6 mo, after forming progressively smaller leaves on short petioles and without proliferation of axillary buds. The causal agent has not been identified.

### Clover Yellow Edge

Strawberry has been experimentally infected with a leafhopper-borne disease called clover yellow edge (Chiykowski 1976). The causal agent is a mycoplasma-like organism (Chiykowski 1981a) and is efficiently transmitted by



*Aphrodes bicincta*. The disease occurs naturally in clovers in eastern Ontario, Canada, and has been experimentally transmitted to 15 species of plants in six families, including the strawberry cvs. 'Cavalier' and 'Redcoat' and *Fragaria virginiana* Duch. Symptoms in strawberry include mild chlorosis and stunting of the whole plant, reduction in leaf size, chlorosis of leaf margins, asymmetrical leaflets, reduced runner development, and necrosis of runner tips. Flowers are reduced in size but never virescent or phylloid. Plants decline rapidly following the appearance of symptoms and die prematurely. The symptoms bear considerable resemblance to those described for lethal decline. (See "Strawberry Lethal Decline," p. 38.)

## Nematode-Borne Diseases

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### European Nepoviruses in Strawberry<sub>i</sub>/

By A. F. M<sub>i</sub>urant and R. M. L<sub>i</sub>ster

In Europe, four nepoviruses are important in strawberry, namely arabis mosaic virus (AMV), raspberry ringspot virus (RRV), strawberry latent ringspot virus (SLRV), and tomato black ring virus (TBRV). All four viruses are nematode-borne, have wide host ranges, and infect several kinds of small fruit. Their general properties and those of the diseases they cause are described in detail in the *Rubus* section of this handbook and only information that is specific to strawberry is presented here.

AMV and SLRV are transmitted by the same vector, *Xiphinema diversicaudatum* (Micoletzky), and therefore tend to occur together. Similarly, some strains of RRV and TBRV occur together because they have the same vector, *Longidorus elongatus* (de Man). For convenience in describing the diseases they cause, the viruses are discussed below in the pairs in which they occur naturally.

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### Raspberry Ringspot and Tomato Black Ring Viruses

#### Disease Names

No names have been applied to the disease caused by either of these viruses in strawberry.

#### History and Geographic Distribution

RRV was originally described by Cadman (1956) from raspberry affected by "leaf curl" disease in Scotland, but soon afterwards Lister (1958) reported it from strawberry growing in the same general localities. Harrison (1956, 1958a) showed that RRV was soil borne and was often accompanied in soils by a second soil-borne virus, at first called beet ringspot virus, but later shown (Harrison 1958c) to be related to TBRV (Smith 1946). Both viruses occurred in raspberry, sugarbeet, and many other crops (Harrison 1957) and also proved to be widespread in strawberry in Scotland (Lister 1960b, 1960c). The viruses are transmitted by nematodes of the genus *Longidorus*; strains of both viruses occurring in Scotland share the vector *L. elongatus* (Harrison et al. 1961; Taylor 1962) and therefore tend to occur together.

RRV and TBRV are now known to occur throughout Europe and the U.S.S.R. (see *Rubus* section). They apparently have not been found in strawberry outside the United Kingdom except in the U.S.S.R. (M. A. Keldysh, personal communication), although susceptible cultivars are widely grown elsewhere, as for example, in the United States (see table 4).

**Table 4.—Susceptibility of strawberry cultivars to British isolates of RRV and TBRV<sup>1</sup>**

Cultivar	RRV	TBRV
Auchincruive Climax	+	+
Blakemore	+	•
Cambridge Early	+	+
Cambridge Favourite	+	•
Cambridge Prizewinner	+	•
Cambridge Rearguard	+	+
Cambridge Rival	+	•
Cambridge Vigour	+	+
Catskill	+	•
Dixieland	+	•
General McMahon (= Dunbarton Castle)	•	+
Howard 17 (= Premier)	+	+
Huxley	+	–
Juspa	+	•
Madame Lefebvre	+	+
Marmion	•	+
Merton Princess	+	+
Missionary	+	•
Montrose	•	+
Pocahontas	+	•
Redgauntlet	+	+
Robinson	+	+
Royal Sovereign	+	+
Senga Sengana	+	+
Senga 54	•	+
Sparkle	+	•
Surecrop	+	•
Surprise des Halles	•	+
Talisman	+	+
Tennessee Beauty	+	+
Troubadour	•	+
Xenion	+	+
<i>Fragaria chiloensis</i> (L.) Duch.	+	+
<i>F. vesca</i> L. (various clones)	+	+

<sup>1</sup>From Lister (1970) with additional unpublished data of J. Chambers.

Note: + = susceptible, – = immune, • = unknown.

### Economic Importance

Though RRV and TBRV usually occur together, either alone can render crops of sensitive strawberry cultivars valueless within 1 or 2 yr of infection. With large outbreaks, the economic loss may therefore be considerable; however, because of their localized occurrence, the viruses are always of less general importance than others that are more widely distributed. Moreover, in Scotland, the introduction of

effective control measures has made diseases caused by these viruses much less common than formerly.

The viruses cause few or no symptoms in the early stages of infection and may therefore be inadvertently distributed in infected planting material. This can result not only in loss of the crop but also in the viruses becoming established in soils

already containing vector nematodes. It can also be a problem in the international exchange of planting material.

### Symptoms on Natural and Experimental Hosts

Both viruses infect a wide range of wild and cultivated plants. For a detailed description of the symptoms caused by RRV and TBRV in natural and experimental hosts, see the *Rubus* section of this handbook, p. 211.

**Symptoms on strawberry.** Table 4 lists the cultivars known to be susceptible to British isolates of RRV and TBRV. With the exception of cv. 'Huxley', which is almost certainly immune to TBRV, all cultivars that have been adequately tested are susceptible to both viruses. When exposed to infective nematode populations, however, some cultivars (for example, 'Redgauntlet') are more prone to infection and show more severe symptoms than others. In the field, most cultivars become infected more readily with RRV than with TBRV; however, because spread of the viruses is now effectively controlled in Britain, the field reaction of newer cultivars is unknown. The following description of the symptoms in long-established cultivars is based on the account given by Lister (1970). Similar symptoms may be caused by infection with either virus or by both viruses together.

Outbreaks of disease caused by RRV and TBRV occur in patches ranging from a few square meters to a few hectares in extent, reflecting the horizontal distribution of the vector in the soil (fig. 51). When infection arises from the use of infected planting material, however, infected plants are typically distributed randomly throughout the crop. Symptoms shown by affected plants vary greatly, depending on the cultivar and on the time of year. Lister (1970) distinguished the following broad symptom types, but emphasized that there was considerable overlap between them.

1. Cv. 'Talisman'. Clearly defined angular chlorotic spots and rings may be observed (fig. 52 A). Sometimes there are large areas of chlorosis with sharply defined borders (fig. 52 B), or the whole leaf may become chlorotic (fig. 52 C). Leaves produced later may show a streaky chlorosis (fig. 52 D), or may be entirely symptomless though they contain virus (that is "recovery"). The plants become progressively dwarfed and eventually die. Similar symptoms are seen in the cvs. 'Early Cambridge', 'Merton Princess', 'Cambridge Prizewinner', and 'Cambridge Rival'.

2. Cv. 'Auchincruive Climax'. Leaf chlorosis is sometimes localized, but tends to be generally distributed, appearing as streaks or as irregularly shaped chlorotic spots (fig. 53); leaves produced later are symptomless ("recovery"). The



Figure 51. — An outbreak of disease caused by raspberry ringspot virus and tomato black ring virus

in 'Talisman' strawberry in eastern Scotland. (Copy-right Scottish Crop Research Institute.)

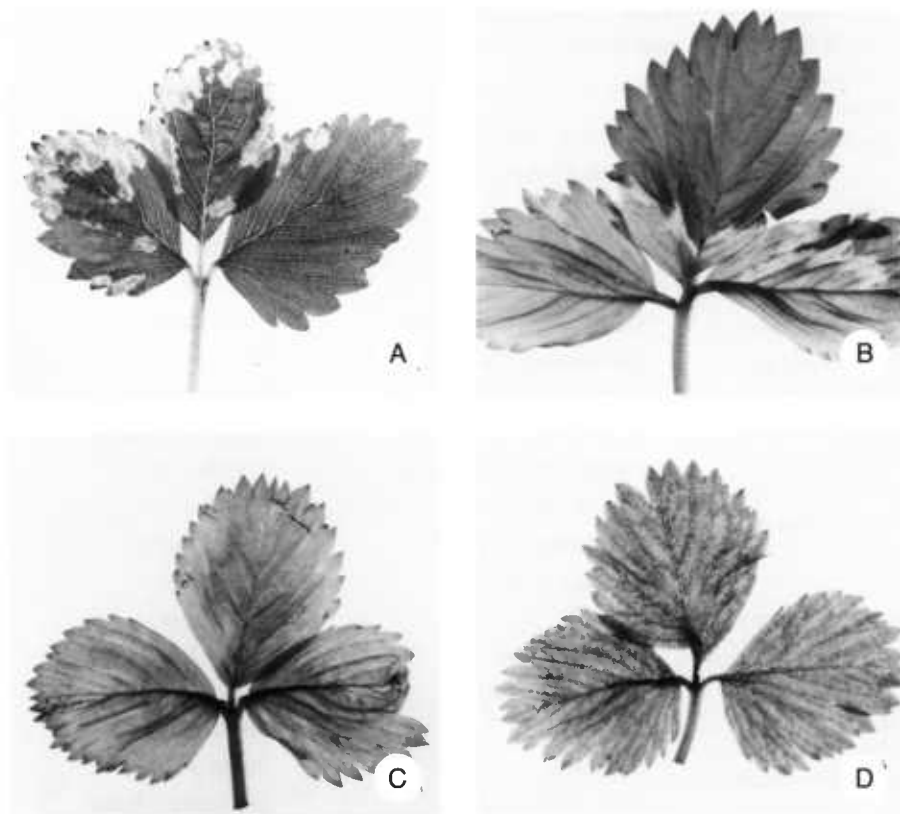


Figure 52. — Leaves of 'Talisman' strawberry collected in late spring showing the range of symptoms induced by tomato black ring virus: A, Angular chlorotic spots; B, large chlorotic areas with sharply defined borders; C, leaf completely chlorotic; and D, streaky chlorosis in a partially "recovered" leaf. (Copyright Scottish Crop Research Institute.)

plants are progressively stunted and eventually die. Similar symptoms are seen in the cvs. 'Cambridge Favourite', 'Cambridge Rearguard', 'Redgauntlet', and 'Madame Lefebvre'.

3. Cv. 'Huxley' (RRV only). Symptoms include clearly defined, irregularly shaped chlorotic blotches, often with a chlorotic center (fig. 54) but without line patterns or rings. Symptoms are less obvious on leaves produced in midsummer or in warm greenhouse temperatures, but totally symptomless leaves are not produced. The plants become progressively stunted and ultimately die.

4. Cv. 'Royal Sovereign'. This cultivar shows symptoms similar to those in 'Auchincruive Climax', but in addition develops prominent necrotic spots (fig. 55).

Symptoms in *Fragaria vesca* L. var. *semperflorens* (Duch.) Ser. cv. 'Alpine': Seedlings show yellow blotches in the first year of infection but are symptomless thereafter.

For information on "Natural and Experimental Transmission," "Properties of the Causal Agents," "Detection and Identification," and "Control Procedures," see the *Rubus* section of this handbook, p. 211.



Figure 53. — Leaf of 'Auchincruive Climax' strawberry showing irregularly shaped chlorotic spots and blotches induced by tomato black ring virus. (Copyright Scottish Crop Research Institute.)



Figure 54. — Leaf of 'Huxley' strawberry infected with raspberry ringspot virus showing chlorotic blotches and crinkling. (Copyright Scottish Crop Research Institute.)

## Arabis Mosaic and Strawberry Latent Ringspot Viruses

### Disease Names

Strawberry mosaic (Posnette 1956); strawberry yellow crinkle (Harris 1958). These names were applied to diseases of certain strawberry cultivars that proved later (Jha and Posnette 1959; Jha 1961) to be infected with AMV. The different symptoms were caused partly by the response of different cultivars and partly by virus strain variation. No name has been given to the disease caused by SLRV in strawberry

### History and Geographic Distribution

AMV was first described from *Arabis* (rockcress) by Smith and Markham (1944) and remained a laboratory curiosity until Cadman (1960) showed that it was the same as raspberry yellow dwarf virus, a sap-transmissible soil-borne virus isolated by Harrison (1958b) from raspberry, strawberry, blackberry, and several weed species. Lister (1958, 1960a, 1960b, 1960c) described the results of surveys, which showed that AMV was widespread in strawberry in England and also occurred in parts of Scotland and Northern Ireland. Lister (1964) found that AMV-infected plants of strawberry, raspberry, and other crops frequently contained a second



sap-transmissible soil-borne virus, which he called strawberry latent ringspot virus (SLRV). Both AMV and SLRV are transmitted by the same nematode, *Xiphinema diversicaudatum* (Harrison and Cadman 1959; Jha and Posnette 1959; Lister 1964), which explains why they commonly occur together in soils.

Both viruses have wide natural host ranges and are widely distributed in Europe; they have also been reported from several non-European countries. (See the *Rubus* section of this handbook.) Outside the United Kingdom, strawberry has been reported to be infected with AMV in Germany (Lister and Krczal 1962) and the Irish Republic (Staunton and Moore 1967), and with both AMV and SLRV in Hungary (Szilagyi 1980) and the U.S.S.R. (M. A. Keldysh, personal communication). Cultivars known to be susceptible are widely grown, for example, in the United States (see table 5).

### Economic Importance

The effects of AMV are severe enough in some strawberry cultivars to make the crop worthless within 1 or 2 yr of infection. SLRV usually occurs in mixed infections with AMV, but can probably have equally damaging effects on its own. The viruses are therefore of considerable economic importance in those areas of southern and southwestern Great Britain where they are prevalent; however, the adoption of effective control measures has now made the diseases induced by these viruses relatively uncommon in strawberry.

The introduction of adequate inspection and certification schemes for strawberry planting material is an important first step in eradicating AMV and SLRV, because newly infected plants may show no symptoms and the viruses can thus be inadvertently distributed. This can result not only in losses of crop but also in the introduction of the viruses into soils already containing vector nematodes. It is also a problem in the international exchange of plant material.

### Symptoms on Natural and Experimental Hosts

Both viruses infect a wide range of wild and cultivated plants. For a detailed description of the symptoms caused by AMV and SLRV in natural and experimental hosts, see the *Rubus* section of this handbook, p. 204.

**Symptoms on strawberry.** Table 5 lists the strawberry cultivars known to be susceptible to AMV; little information is available for SLRV, except that the cvs. 'Cambridge Favourite', 'Cambridge Vigour', and 'Tantallon' are susceptible.

As with RRV and TBRV, outbreaks of disease caused by AMV and SLRV occur as patches in the crop, reflecting the horizontal distribution of the nematode vector, except of course when infection results from the use of infected planting material. Strawberry plants show a very wide variation in response to infection with AMV, depending on the cultivar and on the strain of virus involved; however, the

**Table 5.—Strawberry cultivars and *Fragaria* species susceptible to AMV<sup>1</sup>**

Strawberry cultivars	
Black Prince	Lihama
Blakemore	Madame Lefebvre
Bradley Cross	Merton Princess
Cambridge 497	Midland
Cambridge Early Pine	Missionary
Cambridge Favourite	Northwest
Cambridge Prizewinner	Oberschlesien
Cambridge Profusion	Perle de Prague
Cambridge Rearguard	Pocahontas
Cambridge Rival	Prodige
Cambridge Vigour	Red Dragon
Captain Cook	Redgauntlet
Catskill	Robinson
Charles Lane	Royal Sovereign
Deutsche Evern	Sans Rivale
Dixieland	Senga 242
Dybdahl	Silver Jubilee
Early Cambridge	Sir Joseph Paxton
Glasnevin 'A'	Sparkle
Gorella	Surecrop
His Excellency	Surprise des Halles
Howard 17 (= Premier)	Talisman
Huxley	Tantallon
Indra	Tardive de Léopold
John Innes 580	Tennessee Beauty
Jucunda	Triomphe de Tihage
Juspa	Troubadour
King George	Xenion
<i>Fragaria</i> spp.	
<i>F. chiloensis</i> (L.) Duch.	<i>F. nipponica</i> Makino
<i>F. corymbosa</i> Los.	<i>F. nubicola</i> Lindl.
<i>F. cuneifolia</i> Nutt. <sup>2</sup>	<i>F. orientalis</i> Los.
<i>F. moschata</i> Duch.	<i>F. platypeltata</i> Rydb.
<i>F. moupinensis</i> (Franch.) Card.	<i>F. vesca</i> L.
<i>F. nilgerrensis</i> Schlecht.	<i>F. virginiana</i> Duch.
	<i>F. viridis</i> Duch.

<sup>1</sup>From Lister (1970) and Posnette and Manwell (1971), with additional unpublished data of J. Chambers.

<sup>2</sup>A natural hybrid of *chiloensis* x *virginiana*.



Figure 55. — Leaves of 'Royal Sovereign' strawberry infected with raspberry ringspot virus showing necrotic spots and chlorosis. (Copyright Scottish Crop Research Institute.)

plants are usually stunted and show chlorotic leaf mottle or mosaic symptoms. The reduction of growth may range from slight stunting to such extreme dwarfing (fig. 56) that the plants die within a year or two of infection. The leaves are often twisted, cupped, or crinkled. Chlorotic markings on the leaves range from a diffuse chlorotic mottle (fig. 57) to vivid yellow spots, blotches, and streaks (fig. 58). The prominent blotching symptom induced by AMV in the cv. 'Royal Sovereign' (fig. 58) was called strawberry mosaic by Posnette (1956), but the virus in these plants induced only diffuse mottle and necrotic spotting in graft-inoculated plants of cv. 'Cambridge Favourite' (Jha 1961). Leaf symptoms are most readily seen in late spring before the fruit is set, and again in autumn, but tend to disappear in midsummer, although the stunting is still evident.

The reactions of strawberry cultivars to infection with SLRV are largely unknown. The virus was found originally in plants of cv. 'Cambridge Vigour', which were stunted and had yellow blotches on the leaves; but AMV, which causes similar symptoms, was also present. Lister (1970) found that plants graft-inoculated with SLRV alone showed the same kind of symptom (fig. 59).

**Symptoms on *Fragaria vesca* 'Alpine'.** Some clones show chlorotic symptoms when infected with AMV, but most are symptomless except for loss of vigor. Plants infected with SLRV remained symptomless in greenhouse tests.

No immunity to AMV was detected among 13 species of *Fragaria* (table 5; Posnette and Manwell 1971), giving little hope that genes for immunity to this virus occur in *Fragaria*.

For information on Natural and Experimental Transmission, Properties of the Causal Agents, Detection and Identification, and Control Procedures, see the *Rubus* section of this handbook, p. 204.



Figure 56. — Plants of 'Cambridge Favourite' strawberry: Left, healthy; right, infected with arabis mosaic virus, showing extreme stunting. (Copyright Scottish Crop Research Institute.)



Figure 57. — Plant of 'Talisman' strawberry infected with arabis mosaic virus in the field and showing chlorosis and miniaturization. (Copyright Scottish Crop Research Institute.)



Figure 58. — "Strawberry mosaic" disease in cv. 'Royal Sovereign' infected with arabis mosaic virus. (Copyright East Malling Research Station.)



Figure 59. — Chlorotic blotches on leaf of 'Cambridge Vigour' strawberry graft-inoculated with strawberry latent ringspot virus. (Copyright Scottish Crop Research Institute.)

## Tomato Ringspot Virus in Strawberry

By R. H. Converse and R. Stace-Smith

### Additional Common Names

Peach yellow-bud mosaic in *Fragaria chiloensis* (L.) Duch. (Frazier et al. 1961) was later shown to be the same as tomato ringspot virus (TomRSV) by Cadman and Lister (1961, 1962). The disease in strawberries caused by TomRSV has been given no common name.

### History and Geographic Distribution

TomRSV was found in symptomless *F. chiloensis* along the coast of northern California (Frazier et al. 1961). Field infection of four strawberry cultivars by TomRSV was reported in western Oregon (Converse 1981); moreover, TomRSV from red raspberry has been successfully graft-inoculated to nine strawberry cultivars in the greenhouse and was lethal to eight of them (Mellor and Stace-Smith 1963).

TomRSV probably infects wild and cultivated strawberry wherever it occurs with its dagger nematode vector over a large part of the Western Hemisphere.

### Economic Importance

Since the natural infection of strawberry cultivars by TomRSV has only recently been reported (Converse 1981), surveys to determine its occurrence and damage to cultivated strawberries have not yet been made. TomRSV is one of the most common and severe virus diseases of red raspberry and blueberry in Oregon. (See "Tomato Ringspot Virus in *Rubus*," p. 223; and "Tomato Ringspot Virus in Blueberry," p. 117.) Therefore, crop loss to TomRSV in strawberry can be expected in Oregon and similar areas.

### Symptoms on Natural and Experimental Hosts

**Symptoms on natural hosts.** *Strawberry.* Wild *F. chiloensis* is symptomless when infected. Symptoms in cultivated strawberries range from none ('Lassen' and 'Sequoia'); to dwarfing, reduction in runner production, and occasionally mottled leaves in the spring ('Puget Beauty') (fig. 60), where symptoms usually resemble those caused by the common aphid-borne strawberry viruses like crinkle, mottle, and mild yellow-edge; and to death of whorls of outer leaves and subsequent death of plants, resembling *Verticillium* wilt disease symptoms, in 'Olympus' (fig. 61).

**Other hosts.** TomRSV has an extensive host range among woody and herbaceous plants, including many common shrubs and weeds, often seedborne and without symptoms. (See "Tomato Ringspot Virus in *Rubus*," p. 223, for a further discussion of natural hosts.)

**Symptoms on experimental *Fragaria* hosts.** *F. chiloensis* clones have been found in grafting tests to vary from immune, to symptomless, to weak with varying amounts of vein chlorosis (Frazier and Mellor 1970b). *F. vesca* indicator clones 'EMC', 'EMK', 'EM-1', 'UC-1', 'UC-3', 'UC-4', 'UC-5', and 'UC-6' all respond similarly to leaf-graft inoculation by TomRSV. Initial symptoms about 2 wk after grafting are epinasty, unequal leaflet development, a rather distinctive blotchy leaf mottling (fig. 62), and, sometimes, necrosis of leaf blade tissue. Symptoms do not follow a precise pattern and may resemble symptoms of other strawberry virus diseases. Chronic symptoms are usually mild, but fluctuate seasonally, both in severity and kind. Leaflets are neither



Figure 60.—Tomato ringspot virus causing mottling in leaf of 'Puget Beauty' strawberry.





Figure 61.—Dying 'Olympus' strawberry plants infected with tomato ringspot virus (24-mm-diameter coin included for size comparison).

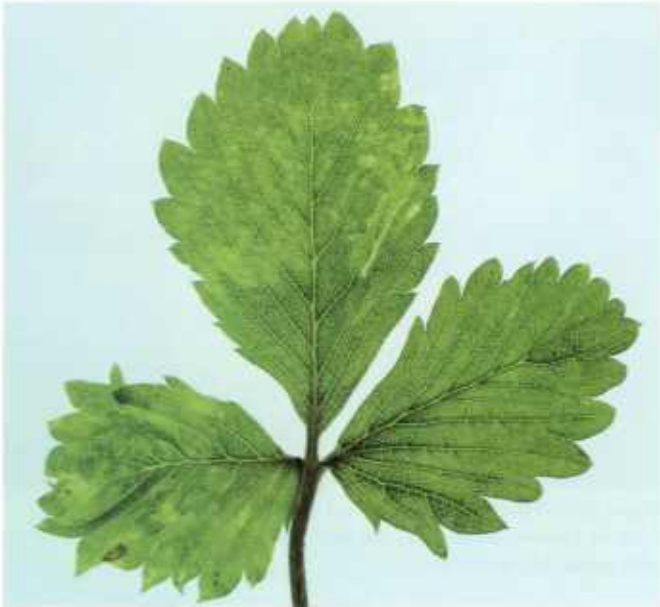


Figure 62.—Symptoms of tomato ringspot virus inoculated by leaf grafting to *Fragaria vesca* cv. 'UC-4'.

mottled nor distorted, but are slightly rounder and smaller than normal. The youngest petioles are redder, shorter, and more upright than normal, and are usually swollen. The abnormal thickening of petioles is a symptom diagnostic of TomRSV infection. Other seasonal symptoms include: premature yellowing and death of older leaves; transient epinasty of unfolding leaves and transient curling of runner tips; inflorescences on shortened, thickened stalks or peduncles, with an increased number of flowers having slightly phyllod sepals, an increased number of small, often virescent, petals; and distinct vein chlorosis, most pronounced on 'Alpine', which may only appear on a series of several leaves (Frazier and Mellor 1970b).



Figure 63.—Shock symptoms (dead leaf) of tomato ringspot virus inoculated by leaf grafting to *Fragaria virginiana* cv. 'UC-11'.

TomRSV is usually lethal in leaf-graft inoculated *F. virginiana* L. cvs. 'UC-10' and 'UC-11'. First, some younger leaves become necrotic (fig. 63), and, then the whole plant dies. Chronic symptoms in the 'M-1' clone of *F. virginiana* graft-inoculated with TomRSV differ from those in other members of the genus. Initial symptoms are similar to those in the more sensitive commercial cultivars: spreading necrosis appears at runner tips and as spots on young leaves, and soon kills all the leaves except those that were fully mature at the time of infection. Although crowns also appear to have been killed, after a few weeks numerous, miniature, adventitious shoots are produced. These bear dwarfed, deformed, chlorotic-mottled leaves which, 3 mo after infection, give the crowns of plants the appearance of rosettes (Frazier and Mellor 1970b).

**Symptoms in complex with other viruses.** In leaf graft tests on *F. vesca*, no interaction was detected between TomRSV and the latent A strain of strawberry mottle virus. Symptoms of each virus were additive in such complex infections (Frazier and Mellor 1970b).

#### Natural and Experimental Transmission

**Natural transmission.** TomRSV is transmitted to *F. chiloensis* and to strawberry cultivars by the American dagger nematode (*Xiphinema americanum* Cobb) (Frazier and Maggenti 1962; Converse 1981). *X. rivesi* Dalmasso, a closely related species, has recently been found associated with TomRSV transmission to other crops (Forer and Stouffer 1982). The field infection rate of cultivated strawberries by

nematode-transmitted TomRSV is slow—30% in 19 mo (Converse 1981). Single American dagger nematode adults and all four larval stages can acquire TomRSV after feeding on cucumber roots (*Cucumis sativus* L.) for 1 hr and can transmit it to test cucumbers within feeding periods of 1 hr (Téliz et al. 1966).

The following strawberry cultivars were infected by TomRSV transmitted by viruliferous American dagger nematodes in greenhouse tests: ‘Olympus’, ‘Puget Beauty’, ‘Rainier’, ‘Totem’, ‘Tyee’, and Oregon-USDA selections 4356, 4459, 4681, 4682, and 6108 (R. H. Converse, unpublished results).

TomRSV is seed transmitted in *F. vesca* and in cultivated strawberry, often without symptoms (Mellor and Stace-Smith 1963). We can expect TomRSV to occur in some strawberry runner plants used to establish new plantings, particularly those taken from fields with high American dagger nematode populations.

**Experimental transmission.** TomRSV can be transmitted by sap inoculation from young, infected strawberry leaves to several herbaceous test plants.

Test plant	Symptoms
<i>Chenopodium quinoa</i> Willd.	Local lesions on inoculated leaves followed by systemic shoot tip necrosis (fig. 64).
<i>Cucumis sativus</i> L. cv. ‘National Pickling’	Yellow local lesions on inoculated cotyledons followed by necrosis and withering of cotyledons, mosaic, distortion, necrosis, and stunting of the leaves (fig. 65).
<i>Nicotiana tabacum</i> L. cv. ‘Xanthi NC’	Local lesions on inoculated leaves followed by ring and line patterns on leaves and eventual appearance of symptomless leaves (fig. 66).

Several buffers can be used for these sap inoculations: 3% nicotine alkaloid + 0.01M  $K_2HPO_4$  + 0.01M cysteine hydrochloride, pH.9; and 2% polyvinylpyrrolidone (mol. wt. 10,000) in 0.05 M phosphate buffer, pH 7 (Brunt and Stace-Smith 1976; Converse 1979; Martin and Converse 1982a).

Pollen from TomRSV-infected strawberry plants is viruliferous, as determined by sap inoculation to herbaceous test plants and by serological tests; however, TomRSV has not been found to infect strawberry plants pollinated by viruliferous strawberry pollen (Frazier and Mellor 1970b).

### Properties of the Causal Agent

TomRSV is a nepovirus 28 nm in diameter. (See “Tomato Ringspot Virus in *Rubus*,” p. 223, for a detailed presentation of the properties of this virus.) Properties are also summarized in Commonwealth Mycological Institute and the Association of Applied Biologists Descriptions of Plant Viruses, No. 18 (Stace-Smith 1970). The virus is a good anti-



Figure 64.—Necrosis of *Chenopodium quinoa* that had been sap-inoculated with tomato ringspot virus.



Figure 65.—Yellow local lesions and systemic symptoms on *Cucumis sativus* that had been sap-inoculated with tomato ringspot virus.



Figure 66.—Local lesions on leaves of *Nicotiana tabacum* sap inoculated with tomato ringspot virus.

gen, and antiserum can be prepared following the standard procedures just referenced or can be purchased from organizations like the American Type Culture Collection, Rockville, Md. Antisera prepared against TomRSV isolates from a number of hosts have reacted well against TomRSV isolates from strawberry.

### Detection and Identification

TomRSV can seldom be identified by symptoms in infected strawberry cultivars. This virus can be readily detected and identified in leaves of suspect strawberry plants by preparing sap from them to use in enzyme-linked immunosorbent assay (ELISA) (Clark and Adams 1977; Converse 1981). Leaf grafting to *F. vesca* 'Alpine' or other *F. vesca* indicators can be used for detection and identification of this virus when the more sensitive and rapid ELISA procedures are not available. (See "Symptoms on Experimental Hosts" in this chapter.)

### Control Procedures

Because no *Fragaria* clones have been found that were completely infected by TomRSV, it has not been necessary to attempt to eradicate this virus from *Fragaria* clones. Heat inactivation of TomRSV in vivo was accomplished in peach after 21 days at 38°C (Nyland and Goheen 1969).

The principal control measures are: (1) control of the American dagger nematode by fumigation or avoidance of infested soils (Murant and Taylor 1965; Thomason and McKenry 1975); (2) use of certified nursery stock free from TomRSV and grown in fumigated soil; and (3) use of immune cultivars. The cvs. 'British Sovereign' and 'Sparta' were found to be immune to leaf-graft infection by TomRSV (Mellor and Stace-Smith 1963). Immunity to TomRSV may be fairly widespread in strawberry cultivars and can be purposely incorporated into advanced selections by plant breeders.

### Remarks

Serological tests to detect TomRSV should be included in future strawberry virus surveys in North America, as it is probable that this virus is widespread, damaging, and largely unrecognized in many strawberry growing areas where TomRSV is known to occur.

## Vectors Unknown

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### Strawberry Pallidosis

By J. P. Fulton

### Additional Common Names

None.

### History and Geographic Distribution

Although pallidosis was detected in the United States and in Australia in 1957 (Frazier and Stubbs 1969), the disease appears to be indigenous to North America. Its occurrence in Australia was associated with cultivars obtained from the U.S. Department of Agriculture at Beltsville, Md. (Frazier and Stubbs 1969). In addition to its occurrence in Eastern United States, it has been reported from Western United States (Frazier and Stubbs 1969; Mullin et al. 1975), from midwestern United States (Fulton and Moore 1982), and from eastern Canada (Craig 1981).

### Economic Importance

Comparative studies of pallidosis-free and pallidosis-infected 'Redcoat' and 'Midway' cultivars at Kentville, Nova Scotia (Craig 1981), indicated that pallidosis had no significant effect on total yield, marketable yield, or fruit size. Frazier and Stubbs (1969) indicated that certain strawberry cultivars developed very mild symptoms under greenhouse culture, whereas others remained symptomless. In Australia, mild symptoms and a lack of vigor were noted in field plantings of 'Midland', 'Klonmore', and 'Tennessee Beauty'. Mullin et al. (1975) and Craig (1981) suggested that the economic importance of pallidosis is as a result of the synergistic response when in combination with other viruses. Definitive studies of such synergistic effects have not been reported, however.

### Symptoms on Natural and Experimental Hosts

Pallidosis is latent in most commercial cultivars of strawberries, but very mild symptoms were observed in the cvs. 'Marshall', 'Lassen', 'Hood', and 'Northwest' (Frazier and Stubbs 1969). In standard indicator clones of *Fragaria vesca* L., pallidosis produces either no symptoms or very mild and transient symptoms. None of the *F. vesca* indicators are of value in detecting pallidosis. *F. virginiana* Duch. indicators, particularly the 'UC-10' and 'UC-11' clones, develop severe symptoms when inoculated with pallidosis (fig. 67). In these clones, the new leaves become recurved, reduced in size, and chlorotic (fig. 68). Runners are pale and shortened. When infected by severe isolates, some plants may die. Symptoms are most severe in greenhouse-grown plants during the winter. During the summer, symptoms fade unless plants are heavily shaded.





Figure 67. — *Fragaria virginiana* 'UC-10': Left, plant showing twisted, chlorotic leaves 46 days after infection with pallidosis disease by leaflet grafting; right, healthy plant.

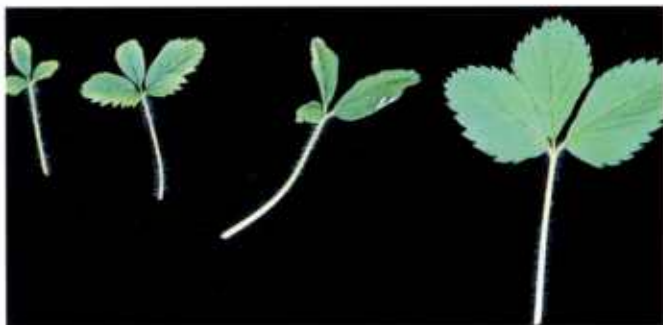


Figure 68. — *Fragaria virginiana* 'UC-10': Three leaves on the left are from a plant leaf-grafted with a leaflet infected with pallidosis disease. These leaves show dwarfing, distortion, and chlorosis 46 days after grafting; healthy leaf is on the right.

### Natural and Experimental Transmission

Pallidosis is readily transmitted by grafting. Symptoms generally appear between 18 and 30 days. There are conflicting reports regarding the spread of the pallidosis agent under greenhouse conditions. Frazier reported that pallidosis spread in his greenhouse in California (Frazier and Stubbs 1969). Movement of pallidosis has not been recorded in our greenhouses or screenhouses in Arkansas (J. P. Fulton, unpublished data). Natural spread in the field has been recorded in the United States and Canada, but the amount of spread appears to vary. Although field spread was not observed in California (Mullin et al. 1975), the disease has been detected in several cultivars widely grown on the west coast of the United States (Frazier and Stubbs 1969). Some spread was recorded in Nova Scotia (Craig 1981), but the rate of spread was slow. The rate of spread in Arkansas is apparently much greater (Fulton and Moore 1982). The pattern of spread is suggestive of an aerial vector although none has been detected (J. P. Fulton, unpublished data).

### Properties of the Causal Agent

Although pallidosis is usually referred to as a virus, the only indication that it is a virus is its transmission by grafting. Observations with the electron microscope have generally failed to demonstrate any viruslike particles or any ultrastructural changes suggestive of virus infection (J. P. Fulton, unpublished data).

### Detection and Identification

Pallidosis can be reliably detected only by grafts to *F. virginiana* ('UC-10', 'UC-11', and certain other *F. virginiana* clones) (Frazier 1974b). The symptoms in *F. virginiana*, however, are not diagnostic. At present, a graft-transmissible entity producing symptoms in the *F. virginiana* clones but failing to produce symptoms in *F. vesca* indicators is assumed to be pallidosis (see Remarks).

### Control Procedures

Contaminated clones are readily freed from pallidosis by shoot apex culture. Mullin et al. (1974, 1975) report almost 100% elimination of pallidosis by excising shoot apices 0.23 to 1.0 mm in length. The pallidosis agent was as readily eliminated without heat treatment as with such treatment. Observations in Arkansas indicate that pallidosis-free clones can be maintained in screenhouses without fear of recontamination. In California, this is not true. There is no indication of the degree of isolation required to maintain a field planting free of pallidosis.

### Remarks

Since the symptoms of pallidosis in *F. virginiana* indicators are not diagnostic, this entity is very poorly defined. Failure to produce symptoms in *F. vesca* associated with the production of symptoms in *F. virginiana* is the basis for designating this entity, but it is known that other viruses may act similarly. The latent A strain of strawberry crinkle virus is of this type. (See "Strawberry Crinkle," p. 20.) Perhaps mild strains of other viruses can evoke this response from these two species of indicators. More than one entity may currently be in the literature under the term "pallidosis."

Henriques and Schlegel (1975) observed long, flexuous viruslike particles 10 to 11 nm in diameter in phloem cells of a pallidosis-infected strawberry. Two types of inclusions resembling ultrastructural features associated with beet yellows infection in beet were also observed. Frazier (1975a) reported possible transmission of pallidosis by the leafhopper *Coelidia olitoria* (Say). Neither of these two reports has been confirmed, and additional studies will be required to determine their significance.

## **Tobacco Streak Virus in Strawberry**

By R. Stace-Smith, R. H. Converse, and H. A. Johnson

### **Additional Common Names**

Strawberry necrotic shock virus (Frazier et al. 1962).

### **History and Geographic Distribution**

Frazier et al. (1962) applied the name “necrotic shock” to a disease of strawberry in California that was characterized by being essentially symptomless on commercial cultivars, but inducing a severe necrotic reaction when grafted into *Fragaria vesca* L. indicator clones. Although the vector was not determined, evidence suggested that both the virus and the vector had host plants other than *Fragaria* spp. The virus was also detected in nursery plantings in Oregon and Washington and in commercial plantings in Oregon (Converse 1969). Stace-Smith and Frazier (1971) isolated tobacco streak virus (TSV) from necrotic-shock infected *F. vesca*. More recently, TSV has been isolated from commercial strawberry cultivars in Queensland (Greber 1979).

Although the recorded geographic distribution of TSV in strawberry is restricted to Western United States and Queensland, Australia, the virus is known to have a far wider geographic distribution (Fulton 1971).

### **Economic Importance**

Under field conditions, virus symptoms are rare in commercial cultivars. Experimentally infected cultivars show mild transient symptoms or a shock reaction followed by recovery. Tests in California showed TSV-infected strawberry plants to yield about 7500 kg less fruit per hectare and to develop one-quarter the number of runners as healthy plants (Johnson et al. 1983). When TSV is combined with other strawberry viruses, the effect of double infections on *F. vesca* indicator clones (Frazier et al. 1982) or the cultivar ‘Redlands Crimson’ (Greber 1979) may be mildly additive. Although TSV is often mild or symptomless in commercial cultivars, it contributes to a decline in productivity of field plantings. TSV also frequently invades nursery plantings of otherwise virus-free stock (Frazier et al. 1962; Converse 1969) being propagated in areas that are isolated from commercial strawberry plantings.

### **Symptoms on Natural and Experimental Hosts**

TSV has a wide natural host range, including both herbaceous and woody plants (Fulton 1948). The virus is known to occur naturally on such diverse crops as tobacco, cotton, dahlia, clover, tomato, pea, bean, grape, and raspberry, as well as weed species and native plants. The experimental host range is extensive; in one study where 169 species were inoculated, the virus was subsequently recovered from 87 species representing 21 families (Fulton 1948).

Several strains or variants, differing in host range and symptomatology, have been isolated. Combinations of

components from the different strains may yield infectious hybrid strains. Symptoms of TSV on natural and experimental hosts are diverse. Some species show mottling, ringspot, or necrotic symptoms as a shock reaction, but the plants usually recover and exhibit no chronic symptoms.

### **Symptoms on Strawberry**

TSV infections in commercial strawberry cultivars cannot be detected in field plantings. Symptoms on experimentally infected cultivars depend to a large extent on the isolate used as inoculum. In Australia, some cultivars show leaflet epinasty, petiole stunting, necrosis on the tips of expanding leaves, or etched rings and lines on the leaf surface (Greber 1979). In the United States, ‘Marshall’ strawberry leaf-grafted with Frazier’s-type isolate of TSV (A-8) developed foliar line patterns (R. H. Converse, unpublished data). Other cultivars show only transient symptoms on a single leaf (Frazier et al. 1962). Following shock symptoms, the infected strawberry plants recover and show no symptoms of infection.

Indicator clones of *F. vesca* are more sensitive to TSV infection than are strawberry cultivars. Plants that are inoculated by leaf grafting show symptoms as soon as 6 to 14 days or as long as 30 days under short photoperiod conditions (Frazier 1974a; Greber 1979; H. A. Johnson and J. I. Espejo, unpublished results). A California isolate of TSV induced a severe necrotic shock reaction in ‘Alpine’ (fig. 69 A), while a Canadian isolate caused chlorotic spots in grafted ‘UC-1’ (fig. 69 B). A Queensland, Australia, isolate does not induce necrosis but induces epinasty, petiole stunt, and chlorosis. As with the commercial cultivars, indicator clones of *F. vesca* that have been grafted or mechanically inoculated recover to produce new growth that appears entirely normal. Although no symptoms are apparent, recovered plants and the vegetative progeny from such plants generally remain infected with TSV.

### **Natural and Experimental Transmission**

Strawberry seed from crosses involving TSV-infected pollen or ovule parents are infected up to 35%, and TSV can readily be detected by ELISA in surface-sterilized, ungerminated seed lots. Mother plants pollinated with TSV-infected strawberry pollen did not become infected in these experiments, even when their seed was infected (Johnson et al. 1983). No information is available on the mechanism of natural infection in strawberry, although thrips are reported as vectors of TSV in several annual crops (Costa and Neto 1976; Kaiser et al. 1982). The fact that the virus can be recovered from isolated plantings established with virus-tested planting material suggests that the source of initial infections is a host other than strawberry.





Figure 69.—A, Necrosis of young leaves in *Fragaria vesca* var. *sempervlorens* cv. 'Alpine' 2 wk after leaf grafting from a California strawberry plant infected with tobacco streak virus. B, Yellow spots in leaf of *F. vesca* 'UC-1' after leaf grafting with an isolate of tobacco streak virus from strawberry in British Columbia, Canada.

Experimental transmission is achieved by grafting or by sap inoculation. The leaf-petiole graft is reliable; symptoms can be enhanced if indicator plants are pruned to one expanded leaf at the time of grafting (Frazier 1974a). Sap transmission from infected strawberry to herbaceous indicator hosts is more reliable when newly infected leaves of graft-inoculated plants are used as a source of inoculum. A buffer, consisting of 0.05 M phosphate plus 2% polyvinyl pyrrolidone (mol. wt. 10,000) pH 7.0, has been found to be satisfactory for mechanical transmission of TSV from strawberry to herbaceous plants (Martin and Converse 1982a). Sap transmission of TSV from herbaceous test plants into strawberry is more difficult, but it has been achieved using either infected cucumber sap or purified virus (Greber 1979).

### Properties of the Causal Agent

TSV is the type member of the ilarvirus group (Matthews 1979). The virus has four nucleoprotein components; each component contains mainly one RNA species. The three fastest-sedimenting components are required for viral replication. A mixture of the three largest RNA's is not infective, but can be activated by the viral coat protein or by the smallest TSV-RNA. The three largest RNA species have molecular weights of about  $1.1$ ,  $0.9$ , and  $0.7 \times 10^6$  daltons; the subgenomic RNA is mol. wt.  $0.3 \times 10^6$  daltons. All particles have the same density (in CsCl,  $1.36 \text{ g/cm}^3$ );  $s_{20,w}$  values lie within the range 80 to 110S. Particles are quasi-isometric; different components differ in diameter within a range of 26 to 35 nm. Protein subunit molecular weight is about 30,000 daltons. The virus is weakly to moderately immunogenic, and different strains show varying degrees of serological relatedness.

### Detection and Identification

Since TSV usually induces no obvious symptoms in strawberry cultivars, either graft or sap transmission or serological methods must be used to detect the virus. Frazier (1974b) evaluated several *Fragaria* indicator clones. All of those tested ('Alpine,' 'EMC', 'UC-1', 'UC-4', 'UC-5', 'UC-6', 'UC-11', and 'UC-12') showed a strong reaction following graft inoculation. *F. vesca* var. *sempervlorens* cv. 'Alpine' was the most satisfactory indicator. Symptoms induced on this host were sufficiently distinctive to distinguish the California isolate of TSV from most other strawberry viruses. Greber (1979) reported that the *F. vesca* clones were more useful than the *F. virginiana* clones. Of those tested, 'UC-4' showed the most obvious and distinctive symptoms after infection with the Queensland isolate of TSV.

Use of sap transmission techniques to detect TSV in strawberry plants is possible, but it is not as reliable as graft transmission. Unless ELISA techniques are used, however, the virus must be recovered from strawberry and transmitted to herbaceous hosts to identify it. Sap transmission may be achieved by macerating infected strawberry tissue in 0.05M phosphate buffer containing 2% polyvinyl pyrrolidone (mol.

wt. 10,000, pH 7.0) and applying the inoculum to herbaceous test plants. Use of this buffer has allowed sap transmission to TSV even from mature, infected strawberry leaves in the summer (Martin and Converse 1982a). The virus induces necrotic local lesions that appear within a few days after inoculation of indicator hosts such as cucumber (*Cucumis sativus* L.) (fig. 70 A), *Gomphrena globosa* L. (fig. 70 B), *Nicotiana tabacum* L. cv. 'Havana' (fig. 70 C, D), *Chenopodium quinoa* Willd., and *C. amaranticolor* Coste and Reyn. Symptoms induced on herbaceous indicator hosts are not adequate for positive identification of all strains of TSV. Cross-protection tests are of limited value because some strains fail to protect against other strains. Serological tests are more reliable, but since some strains fail to react or react weakly when tested with antisera prepared against other strains, it may be necessary to use more than one antiserum source. Globulin from an antiserum against a *Rubus* strain of TSV has given clear-cut results in ELISA tests against a number of U.S. Pacific coast strawberry TSV isolates (Johnson et al. 1983).

### Control Procedures

To date, no satisfactory control measures have been devised to prevent the introduction of TSV into plantings used for nursery stock propagation. Since these plantings are located in areas where there is no commercial strawberry production, infected native hosts may, apparently, serve as reservoirs for infection. The virus is known to be prevalent in the native trailing blackberry (*Rubus ursinus* Cham. and Schlecht.) (Brunt and Stace-Smith 1976; Converse and Bartlett 1979), and this, or other native plants, could be the virus sources. Eradication of naturally infected plants from the immediate vicinity of strawberry nursery plantings may be effective.

It is unlikely that TSV eradication from strawberry would ever be necessary since strawberry clones are not universally infected. TSV contamination could be a problem, however, in a strawberry breeding program if a promising seedling became infected prior to being multiplied. Should the necessity arise, the virus could be eradicated by the heat treatment-shoot tip culture technique. Greber (1979) found

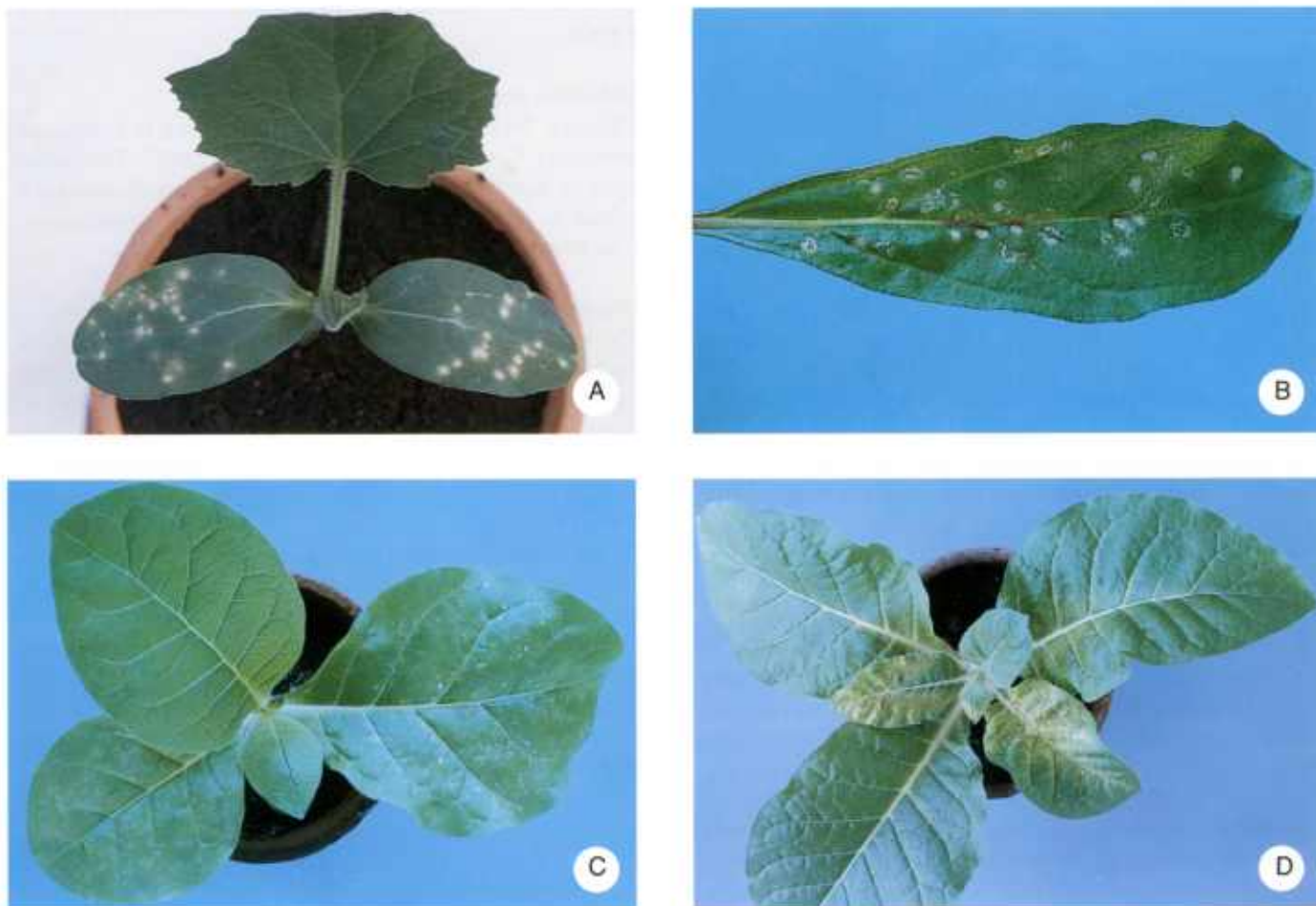


Figure 70.—Symptoms on herbaceous indicators after leaf sap inoculation from a strawberry infected with tobacco streak virus: A, Local lesions on inoculated *Cucumis sativus* cotyledons; B, local lesions on

inoculated *Gomphrena globosa* leaf; C, local lesions on *Nicotiana tabacum* cv. 'Havana' inoculated leaves; and D, systemic symptoms on inoculated 'Havana' plant.



that the Australian strawberry strain of TSV was intermediate between severe mottle virus and mild yellow-edge virus in ease of eradication. (See "Strawberry Mottle," p. 10, and "Strawberry Mild Yellow-edge," p. 25.) However, TSV was not eliminated in plants grown outdoors in the hot summer growing conditions of the Imperial Valley in California (Frazier et al. 1965).

#### Remarks

Major gaps in our knowledge are the mode of initial introduction of TSV into isolated plantings of virus-tested material and subsequent spread from strawberry to strawberry. Evidence suggests that the initial source of inoculum is not wild strawberry but species unrelated to *Fragaria*. Solutions to these problems have an important bearing on the nature of spread of TSV as well as on its control. Selective eradication of the inoculum source is more readily achieved than eradication of all potential sources of inoculum.

#### Strawberry Chlorotic Fleck

By J. P. Fulton

#### Additional Common Names

None.

#### History and Geographic Distribution

This virus disease was described by Horn and Carver (1962) from a strawberry cultivar grown at Tunica, La. It was later detected in other cultivars, including 'Dabreak', 'Headliner' (Horn 1965), 'Tangi', and a numbered selection (J. R. McGrew, personal communication), but only from Louisiana. There are no reports of its occurrence elsewhere.

#### Economic Importance

Chlorotic fleck disease significantly reduced both plant and fruit yields of the cv. 'Headliner' (Horn and Carver 1962). The cv. 'Tangi' produced 40 to 70% more plants when free of chlorotic fleck disease (J. R. McGrew, personal communication).

#### Symptoms on Natural and Experimental Hosts

Chlorotic fleck disease produces no overt symptoms in commercial cultivars. In *Fragaria vesca* L. indicators, young leaves are distorted and down-curved. Vein clearing followed by the appearance of small chlorotic spots is sometimes evident (fig. 71). On *F. virginiana* L. indicators chlorosis, down-curling and distortion of young leaves are observed. Symptoms in these indicators are generally evident 3 to 4 wk following grafting. There is a remission of symptoms with time.

#### Natural and Experimental Transmission

The disease has been successfully transmitted by grafting and by the aphid *Aphis gossypii* Glov. The type of vector relationship has not been determined (Horn 1965).



Figure 71.—Strawberry chlorotic fleck symptoms on the Beltsville strain of the East Malling (BEM) clone of *Fragaria vesca*. (Courtesy N. L. Horn, Louisiana State University.)

#### Properties of the Causal Agent

On the basis of aphid and graft transmission of the disease to indicators, chlorotic fleck disease is assumed to be caused by a virus.

#### Detection and Identification

Chlorotic fleck disease is detected by grafting to indicators, preferably *F. vesca* 'EMB' or *F. vesca* 'EMK'. The causal agent of this disease is very incompletely characterized and it cannot be distinguished from other viruses with confidence. (See Remarks.)

#### Control Procedures

The causal agent seems to be evenly distributed throughout the plant, including the meristem, and cannot be eliminated by tip culture alone. Heat treatment at 35°C for 40 days or longer may eliminate the virus (J. R. McGrew, unpublished data).

#### Remarks

Although chlorotic fleck disease is apparently distinct from others which have been described, not enough is known to unequivocally separate it from other viruses or mixtures. Neither symptomatology on *F. vesca* and *F. virginiana* nor elimination of causal agents by therapy permit unequivocal differentiation between chlorotic fleck and similar symptoms produced by mottle virus, latent A strain of crinkle virus, or vein banding virus, alone or in combination.

## Minor Diseases and Those Experimentally Transmitted Only

### 245 Strawberry Leafroll

By N. W. Frazier

#### Additional Common Names

None.

#### History and Geographic Distribution

Strawberry leafroll disease (SLRD) was first observed in Ontario, Canada, on a plant of 'Parson's Beauty' strawberry, and again on 'Premier' by Berkeley and Plakidas (1942) who observed the same disease in Geneva, N.Y., in 1940. Its occurrence in three plantings in Ontario and New Brunswick was recorded by Bolton (1964). The only report of the disease outside of northeastern North America was in the Alma-Ata district of Kazakstan, U.S.S.R., by Eliseeva et al. (1974).

#### Economic Importance

Because of its restricted distribution and sporadic, rare occurrence, SLRD must be considered to be of very minor importance economically. Bolton however, (1964) observed infections of 10 to 60% of plants in three plantings. The affected plants failed to yield any fruit and produced only a few, distorted runners. Intrinsically, the disease is very damaging to infected plants.

#### Symptoms on Natural and Experimental Hosts

The known natural hosts of SLRD are commercial cultivars of strawberry, *Fragaria X ananassa* Duch., all of which appear to be very susceptible. They can also be used as experimental hosts by graft transmission. Berkeley and Plakidas (1942) reported having transmitted SLRD to the scarlet strawberry *Fragaria virginiana* Duch. Frazier (1974b) reported the *F. virginiana* indicator clones 'UC-10', 'UC-11', and 'UC-12' to give strongly positive symptoms and the woods strawberry, *Fragaria vesca* L. indicator clone 'UC-5', to be uniquely sensitive, producing diagnostic symptoms. *F. vesca* clone 'UC-3', a parent of 'UC-5' (Frazier 1947b), and *F. virginiana* clone '2A17', selected at the University of California, Berkeley, but not distributed to other laboratories, also show excellent, typical symptoms of SLRD (see "Natural and Experimental Transmission").

Symptoms are basically the same on natural and experimental hosts. The diagnostic symptom is the downward rolling of leaflet margins, especially pronounced basally. In extreme examples, the leaflet may be tubular with the lamina narrowed and both margins rolled along their entire length, even overlapping. The leaves are chlorotic, rugose with cleared net veins, and petioles considerably shortened (fig. 72). Less severely rolled leaves have irregularly, sometimes deeply serrated margins. Affected leaves are reduced, and

symptoms may appear on only a portion of a leaflet. Often, diffusely chlorotic areas, usually banding main veins, may be detected. This symptom becomes clearly evident as a "peacock" pattern on clone '2A17' (fig. 73) when typical leafroll symptoms are absent.

#### Natural and Experimental Transmission

No natural vector is known. There probably is no single vector because SLRD appears to be caused by a complex of at least three different agents, each of which could have a different means of infection and spread.

N. W. Frazier (unpublished data) tested the ability of the dark strawberry aphid, *Chaetosiphon jacobi* H. R. L., to transmit SLRD over nonpersistent, semipersistent, and persistent acquisition and inoculation feeding periods. No apparent transmission of any disease resulted. Parallel tests of mechanical transmission using sap from leafroll diseased plants to commonly used herbaceous test plants were made. Tobacco mosaic virus infections were occasionally produced; however, Frazier did not clearly demonstrate that strawberry was the actual source of the tobacco mosaic virus infection.



Figure 72. — Symptoms of strawberry leafroll on *Fragaria vesca* clone 'UC-5'.

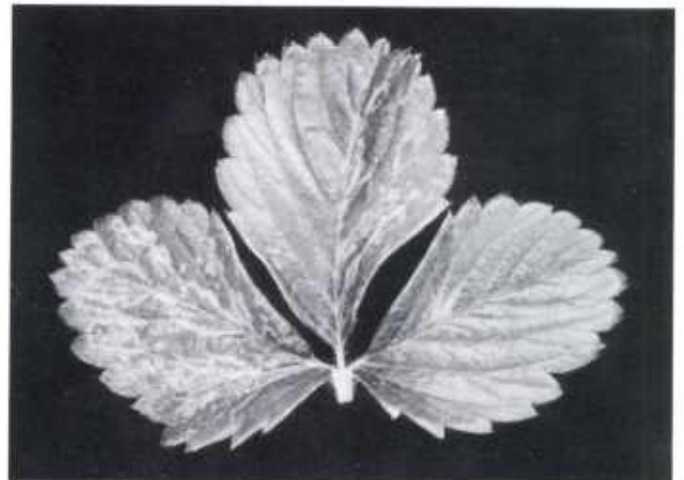


Figure 73. — Symptoms of peacock pattern associated with leafroll on *Fragaria virginiana* clone '2A17'.

**Strawberry Feather-Leaf** //

By N. W. Frazier

**Additional Common Names**

Sparkle virus disease.

**History and Geographic Distribution**

McGrew (1970a) reported that the disease was first detected in Arkansas in field-grown 'Midway'. It was also found in some plants of the first distribution of 'Sparkle' by the U.S. Department of Agriculture; in a single plant of 'Red Star' from Maryland; in 3 of 10 plants of 'StarKrimson' from Missouri; and in 2 of 8 'Paymaster' from Michigan.

**Economic Importance**

Unknown. Presumably very minor.

**Symptoms on Natural and Experimental Hosts**

The disease causes no symptoms in 'Sparkle' and only a slight mottle in 'Midland'.

In indicator clones, *Fragaria vesca* L. clones show the typical feather-leaf symptoms which include: dwarfing; leaves narrowed, straplike, somewhat rugose, with deeply serrated margins; and leaflets fused at the base (fig. 74). Although these symptoms are typical, they are not necessarily diagnostic unless accompanied by vein clearing or fasciation, translucent spots on young leaves (like heat spot disease; see "Heat spot of *Fragaria vesca*," p. 78), and often deeply and unequally serrated margins. Infections may be very obscure or very severe initially and often are first evident on one or more side crowns. Plants may develop a succession of diseased and normal-appearing leaves or normal-appearing crowns and diseased crowns on the same plant. When such diseased and normal-appearing crowns are propagated by runners (fig. 75) and leaves from both kinds of plants are used to inoculate healthy test plants, feather-leaf disease is transmitted as readily from the normal-appearing leaves as from the leaves with symptoms. 'Alpine' and 'UC-1' show the best symptoms of the indicators tested (Frazier 1974b).

*Fragaria virginiana* clones 'UC-10', 'UC-11', and 'UC-12' were susceptible to infection but showed only symptoms of pallidosis (see "Strawberry Pallidosis," p. 55). When such plants were indexed on *F. vesca* plants, typical feather-leaf symptoms developed.

**Natural and Experimental Transmission**

There is no information on natural transmission. Experimental transmission has been by grafting. Both McGrew (1970a) and Frazier (1974a) found transmission to require very extended incubation periods: McGrew, 35 to 240 days; Frazier, 86 to 268 days. In Frazier's work, nondiagnostic symptoms did appear more rapidly in some of the plants — indicative of the presence of more than a single disease-causing entity. Mechanical transmission was reported by Reed and Felix (1961), but their work has not been corroborated.

Infected plants of some clones, such as 'UC-3' and 'UC-5', irregularly produce flushes of normal-appearing leaves but have never been observed to grow out of SLRD. A high level of transmission by grafting was obtained from leaves showing leafroll symptoms, but when normal-appearing leaves were used for scions, transmission was very erratic. A double-crowned 'UC-5' plant with one crown in the chronic stage of SLRD infection and the other without symptoms yielded transmission from the crown with symptoms, but not from the symptomless crown.

Plants of clone '2A17' occasionally recovered from typical leafroll symptoms, leaving a peacock pattern (fig. 73) in evidence. Several plants then recovered from peacock symptoms and appeared as somewhat chlorotic normal plants. The remission process was never observed to revert. In grafting tests, SLRD could be transmitted only from leaves showing leafroll; peacock leaves transmitted either peacock pattern or pallidosis disease (see "Strawberry Pallidosis," p. 55); leaves from recovered plants showing neither leafroll nor peacock pattern transmitted only pallidosis disease.

'UC-11' plants graft-inoculated with leafroll-infected leaflets developed only severe symptoms of pallidosis disease, but not leafroll or peacock pattern. Neither leafroll nor peacock pattern could be recovered from the plants. Only pallidosis disease was transmitted, which cross-protected against type isolates of pallidosis disease and was strongly additive with isolates of mild yellow-edge, characteristics confirming a diagnosis of pallidosis for the transmitted disease.

Frazier (1974a) gave the incubation period for the development of leafroll symptoms as from 42 to 180 (mean 62) days — a relatively long period for strawberry virus diseases. Bolton (1970) reported the appearance of symptoms about 15 days after grafting — a period much closer to the mean of 22 days given for pallidosis disease by Frazier (1974a).

The work at Berkeley, Calif., was done with a single plant of an unknown cultivar which appeared to be infected with at least three distinct entities causing pallidosis, peacock symptoms, and SLRD. Whether the entities causing pallidosis disease or peacock symptoms are necessary for the causation of SLRD was not clear.

**Properties of the Causal Agent**

Not known.

**Detection and Identification**

The disease is visible in cultivars and can be identified by its unique symptoms on the cultivar or on 'UC-5' or other adequate indicator hosts by graft transmission.

**Control Procedures**

The disease is not inactivated at 41°C for 20 days. The fact that symptoms are exhibited by cultivars allows efficient control of SLRD by roguing out the diseased plants (Bolton 1970).





Figure 74. — Leaves of *Fragaria vesca* ('EMK', left; 'UC-1', right) with classic symptoms of feather-leaf disease. (Courtesy University of California Division of Agricultural Sciences.)



Figure 75. — *Fragaria vesca* 'UC-1' sister plants propagated by runners from a normal-appearing (left) and a feather-leaf diseased (right) crown on the same mother plant.

### Properties of the Causal Agent

No information.

### Detection and Identification

Both are dependent upon the production of characteristic symptoms on standard *F. vesca* indicators, particularly 'Alpine' or 'UC-1'. Incubation periods can be very long, over 8 mo. Symptoms in indicator plants are often difficult to detect with confidence. McGrew (1970a) stated that in combination with mottle, vein banding, or crinkle, there is no apparent effect by feather-leaf disease on the symptoms expressed by the other viruses.

### Control Procedures

There is little information, but as reported by McGrew (1970a), feather-leaf is susceptible to heat treatment at 38°C for 5 to 9 wk followed by culture of 28 to 300 mg excised buds.

### Remarks

The somewhat unreliable production of detectable symptoms and the sometimes very long period before they appear following graft inoculation, make feather-leaf disease one of the most difficult of the strawberry diseases to detect with confidence.

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### Strawberry Pseudo Mild Yellow-Edge

By N. W. Frazier

### Additional Common Names

None.

### History and Geographic Distribution

Strawberry pseudo mild yellow-edge disease (SPMYED) was described by Frazier (1966b, 1970a) who transmitted the causal agent from plants of the 'M-1' indicator clone of *Fragaria virginiana* Duch. This is the only record of the occurrence of this disease. The 'M-1' clone originated from a wild plant found in Minnesota by King and von Ruden (1962), which may have been infected prior to its collection.

### Economic Importance

Not known.

### Symptoms on Natural and Experimental Hosts

'M-1' is the only naturally infected host reported. Although yellow to reddish coloration of old leaves with necrotic areas on veins was commonly seen on 'M-1' plants (Frazier 1966), their association as symptoms of SPMYED was not demonstrated. During later work with three *F. virginiana* clones derived from 'M-1', similar symptoms were noted by Frazier (1974b) on 'UC-12' plants (fig. 76) but not 'UC-10' or 'UC-11'.



Figure 76. — Plants of *Fragaria virginiana* clone 'UC-12' that are (left) healthy (center) infected with mild yellow-edge disease; and (right) infected with pseudo mild yellow-edge disease. Note the more severe effect of the mild yellow-edge disease and the scalded, dead leaves with large necrotic spots associated with veins on the pseudo mild yellow-edge infected plant.

Experimental hosts are the *F. vesca* L. and *F. virginiana* indicator clones evaluated by Frazier (1974b). All of the *F. vesca* plants show very similar symptoms of SPMYED, which appear only on older leaves as mottled discolorations in shades of yellow and red followed by premature necrosis of the leaves. The mottling often clearly shows a vein-yellowing pattern, but typically appears as a stipple pattern of isolated or merged, irregular spots or areas. The pattern is least severe near the margin of the lamina (fig. 77). These symptoms on the older leaves are remarkably similar to those of mild yellow-edge disease. SPMYED can be distinguished from diseases caused by severe strains of mild yellow-edge virus (see "Strawberry Mild Yellow-edge," p. 25) by the absence of symptoms on young leaves and a less debilitating effect on plant growth (fig. 76), but is difficult to distinguish from symptoms caused by mild strains of mild yellow-edge virus.

The causal agent of SPMYED does not affect the symptom expression of pallidosis disease in 'UC-10' or 'UC-11' (see "Strawberry Pallidosis," p. 55.) (Frazier 1975b). Otherwise, symptoms in complexes with other diseases have not been determined.

#### Natural and Experimental Transmission

There is no information on natural transmission, but natural spread is probably by species of strawberry aphids — *Chaetosiphon* spp. *Chaetosiphon jacobi* H.R.L., the dark strawberry aphid, was the experimental vector, but does not occur in Minnesota, the only place where SPMYED is known to occur naturally.

Experimental transmission has been accomplished by leaflet grafting and by the vector *C. jacobi*. The disease has the semipersistent type of vector relationship: It can be acquired in less than 2 hr and retained for 6 but not 18 hr by the vector (Frazier 1966b). 'Alpine' test plants developed symptoms about 3 wk (15 to 27 days) following inoculation.

#### Properties of the Causal Agent

No information.

#### Detection and Identification

The disease can be detected by graft or vector transmission to *F. vesca* clones and to *F. virginiana* 'UC-12'. Identification can be accomplished by inoculating *F. vesca* plants, which will develop characteristic symptoms, and *F. virginiana* 'UC-10' or 'UC-11' plants, which will not develop symptoms. The 'UC-6' clone of *F. vesca* grafted with SPMYED develops the characteristic symptoms already discussed, but remains symptomless when grafted with most mild yellow-edge virus sources.

#### Control Procedures

No information: No field control is necessary, but it can be assumed that the disease could be eliminated by heat treatment and meristem culture.

#### Remarks

SPYMED appears not to be of economic importance.



Figure 77. — Stipple pattern of discoloration on an old leaf of 'Alpine' strawberry *Fragaria vesca* var. *sempreflorens*, characteristic of pseudo mild yellow-edge infection.

#### Tobacco Necrosis Virus in Strawberry

By R. H. Converse, R. R. Martin, E. Tanne, and S. Spiegel

#### Additional Common Names

Strawberry rosette necrosis virus (Faccioli 1974).

#### History and Geographic Distribution

Tobacco necrosis virus (TNV) was first reported in *Fragaria* by Fulton (1952) in Arkansas, and subsequently by Frazier (1955b) in California, primarily as a greenhouse disease of *F. vesca* indicator root systems and was thought to be of no economic importance. TNV occurs in cultivated strawberries in the field in Italy (Faccioli 1970, 1974), Bulgaria (Yankulova and Schmelzer 1974), and Japan (Kaname and Kishi 1973; Komuro et al. 1973). Satellite of tobacco necrosis virus (STNV) has also been reported to be associated with TNV in strawberry roots in Japan (Komuro et al. 1973).

#### Economic Importance

In Japan, TNV and STNV have been associated with a dwarfing disease (the Sukumi disease) of cultivated strawberry, but conclusive etiologic experiments are lacking (Kaname and Kishi 1973; Komuro et al. 1973). In Italy, a strain of TNV, called strawberry rosette necrosis virus, has been associated with dwarfing, leaf malformation and necrosis, and root necrosis of *F. vesca* and strawberry cultivars (Faccioli 1970). The occurrence of aphid-borne strawberry viruses, however, was not investigated in these experiments. In the United States and Israel, TNV has been found to occur in *Fragaria* spp. and cultivars in the greenhouse. In several standard *F. vesca* indicator clones like 'Alpine', TNV was associated with premature death of older

leaves and interfered with the use of this indicator to detect strawberry mild yellow-edge and pseudo mild yellow-edge diseases. (fig. 78)

TNV occurs commonly in roots of potted strawberries grown in the greenhouse. *F. vesca* roots are particularly prone to infection (fig. 78).

### Symptoms on Natural and Experimental Hosts

In strawberry cultivars, the presence of TNV and its fungal vector *Olpidium brassicae* (Wor.) Dang. in strawberry roots causes necrosis of small roots (Faccioli 1969). In Italy, TNV infection of *F. vesca* and cultivars was also linked with dwarfing, leaf malformation, necrosis, and mottling (Faccioli 1969, 1970). The possible association of TNV with the Sukumi disease of cultivated strawberry in Japan was previously mentioned under "Economic Importance."

TNV has a very wide natural and experimental host range among angiosperms (Price 1940). A number of herbaceous test plants develop necrotic local lesions when sap-inoculated with TNV. *Chenopodium quinoa* Willd. is a good experimental host for TNV by strawberry root sap inoculation. Small, necrotic, local lesions develop on *C. quinoa* leaves a few days after inoculation with sap from TNV-infected strawberry roots (fig. 79). Roots of mung bean (*Phaseolus aureus* Roxb.) are readily infected by *Olpidium* plus TNV and quickly develop characteristic chocolate-brown root lesions after such infections (fig. 80) (Teakle 1962).

### Natural and Experimental Transmission

TNV is spread naturally by zoospores of the obligately plant parasitic fungus *Olpidium brassicae*, which has a wide host range (Harrison 1977). Untreated greenhouse potting soils are frequently infested with TNV-carrying *Olpidium* resting spores, and plants grown in such soils are readily infected by *Olpidium* and TNV. Infection of strawberry roots by TNV has been obtained in soils air dried for 30 days (Komuro et al. 1973). Occurrence of TNV in small infected pieces of roots in the soil could explain these results since TNV fails to survive in air-dried soil even for 2 days (Smith et al. 1969).

TNV can be transmitted from infected strawberry roots by grinding them, using a mortar and pestle, with a suitable buffer like 3% nicotine alkaloid in water, or 0.05 M phosphate buffer + 2% polyvinyl pyrrolidone (mol. wt. 10,000), and inoculating the resulting sap to suitable indicator plants like *C. quinoa*, *C. amaranticolor* Coste and Reyn., and bean (*Phaseolus vulgaris* L.) (Fulton 1952; Frazier 1955b; Faccioli 1969). Local lesions (fig. 79) appear within 3 to 5 days on TNV-inoculated leaves.

### Properties of the Causal Agent

TNV and STNV have been studied in great detail. TNV is an isometric particle (26 nm in diameter) with a sedimentation coefficient ( $s_{200,w}$ ) of 118S. Its specific absorbance at 260 nm is 5.0 to 5.5. TNV usually occurs in very high concentrations



Figure 78. — Necrotic older leaves of *Fragaria vesca* 'Alpine' infected by tobacco necrosis virus.



Figure 79. — *Chenopodium quinoa*: Left, leaf infected with tobacco necrosis virus by sap inoculation from infected strawberry roots; right, healthy leaf.



Figure 80. — Mung bean seedlings showing large black root lesions after infection by *Olpidium brassicae* and tobacco necrosis virus in infested soil; two healthy seedlings on the right.

in infected roots (dilution end point of infected sap often reaches 1:1,000,000 in herbaceous hosts, but is usually low in *Fragaria*). TNV is quite heat stable in vivo (thermal inactivation point in infected sap is 85° to 95°C). TNV mol. wt. is  $7 \times 10^6$  daltons, with single-stranded RNA mol. wt. of  $1.4 \times 10^6$  daltons (Kassanis 1970b). STNV is a smaller isometric particle 17 nm in diameter. Its specific absorbance at 260 nm is 6.5;  $s_{(20^\circ, w)} = 50 S$ , and STNV mol. wt. is  $1.97 \times 10^6$  daltons, with RNA mol. wt. of  $0.28 \times 10^6$  daltons (Kassanis 1970a). Two major serotypes of TNV are known which cross-react poorly. STNV is serologically unrelated to TNV. When STNV is present with TNV, STNV may comprise the bulk of the virions present, so that serological detection may require sera containing antibodies against STNV as well as the proper TNV serotypes. TNV and STNV may occur in infected plants as free RNA as well as intact virions. Therefore, ELISA tests for TNV in infected strawberry root sap are often faint or even negative when bioassays on *C. quinoa* are positive.

### Detection and Identification

TNV with or without STNV can best be detected in strawberry by grinding roots of suspected plants (1:5, w:v) in 0.05 M phosphate buffer + 2% polyvinyl pyrrolidone (mol. wt. 10,000), with a mortar and pestle, and a little Celite (diatomaceous earth powder) and rubbing leaves of *Chenopodium quinoa*. TNV causes small local lesions on leaves 3 to 5 days after inoculation (fig. 79). TNV appears to be present more often at higher concentrations in infected *F. vesca* roots than in roots of infected strawberry cultivars. If only one or two local lesions develop on inoculated *C. quinoa*, they can usually be successfully subtransferred by mechanical inoculation to other *C. quinoa* leaves for confirming studies such as physical property determinations, agar gel, or other serological identification tests, including serologically specific electron microscopy.

### Control Procedures

Use of pasteurized, autoclaved, or fumigated soil for greenhouse experimental work with strawberries is necessary to prevent infection. The infected *Olpidium brassicae* zoospores carrying TNV and often STNV are able to move in films of water from infected to healthy plants, particularly on wooden greenhouse benches. Strawberry plants can be maintained free from TNV infection under such conditions by growing them on inverted clay pots or glass jars (Frazier 1955b). TNV is not known to be seed transmitted, and TNV-free 'Alpine' strawberry plants can be obtained by growing them from seed in *Olpidium*-free soil on greenhouse benches free from *Olpidium*-infected plants.

The need for, and the methods of control of, TNV in strawberry cultivars in the field have not been investigated.

No information is available on the elimination of TNV from infected strawberry clones by heat therapy.

### Remarks

The role of TNV in causing diseases of cultivated strawberries is not known. Feeder root necrosis has not been reported to occur in infected cultivars in the United States. There are now enough reports on the occurrence of TNV in field-grown strawberry cultivars throughout the world to warrant more serious study of this virus, of STNV, and of their vector, *Olpidium brassicae*, in causing strawberry diseases, alone or in combination with virus and other diseases of strawberry. The infection of strawberry plants by TNV in soils that have been air dried or pasteurized also requires additional study.

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## Strawberry Witches'-Broom and Multiplier Diseases //

By R. H. Converse

### Additional Names

For multiplier disease: multiplier plant, bushy plant, and spindly plant diseases. For strawberry witches'-broom: no other name.

### History and Geographic Distribution

Witches'-broom disease (WBD) was first reported in Oregon (Zeller 1927) and has been found in California (N. W. Frazier, unpublished data). A similar disorder was later described in The Netherlands (Kronenberg 1943). This disease is rare from all three reporting areas. Multiplier disease (MD) was first noted in Minnesota, Wisconsin, Illinois, and New York (Demaree and Marcus 1951) and has been subsequently reported and studied in Wisconsin (Sehgal and Boone 1963; Boone 1970).

### Economic Importance

Multiplier disease occurs rarely and is found mainly in the Great Lakes region of the United States and in British Columbia in Canada. Both diseases are readily detected, and infected plants can be readily rogued. Because of their rarity, neither is economically significant.

### Symptoms on Natural and Experimental Hosts

**On natural hosts.** Both diseases are only known to infect *Fragaria* spp. Witches'-broom diseased strawberry cultivars are dwarfed, very bushy in appearance, and have numerous branched crowns, with small leaves on spindly petioles (fig. 81). Runners when formed are very short, and severely broomed daughter plants become established close to infected mother plants. Plants of strawberry cultivars infected by MD are dwarfed and exhibit crown proliferation. Leaflets cup upwards on short, erect petioles. Runners are short and flowers are reduced in number and size, but fruit normally (fig. 82).

**On experimental hosts.** Witches'-broom diseased leaves grafted to *Fragaria vesca* var. *semperflorens* (Duch.) Ser. cv. 'Alpine' and *F. chiloensis* (L.) Duch. caused crown proliferation with numerous small leaves on thin petioles





Figure 81. — *Left*, strawberry witches'-broom disease (Marshall strain) on cv. 'Marshall'; *right*, healthy 'Marshall'. (Courtesy D. M. Boone, University of Wisconsin.)



Figure 82. — A, Multiplier disease on cv. 'Sparkle' B, Healthy cv. 'Sparkle'. (Courtesy D. M. Boone, University of Wisconsin.)

(Miller 1959; Frazier 1974). Leaf graft transmission from infected 'Puget Beauty' to 'Marshall' cultivars led to similar symptoms (Miller 1959). Typical crown proliferation and stunting were caused when MD was grafted into several strawberry cultivars and *F. vesca* clones, including 'EMC', 'UC-1' *F. vesca* var. *alba* (Duch.), *F. vesca* ssp. *bracteata* (Heller) Staudt, *F. chiloensis*, and *F. orientalis* Losink (Sehgal and Boone 1963).

### Natural and Experimental Transmission

The modes of natural transmission of WBD and MD are unknown, although WBD does spread slowly in the field (Zeller 1927). Experimentally, WBD was transmitted by leaf graft (Miller 1959, Frazier 1974) and stolon graft (Demaree and Marcus 1951). Zeller (1927) claimed to have transmitted WBD experimentally by *Chaetosiphon fragaefolii* (Cock.), although his data show only the development of short, epinastic leaflets on spindly petioles rather than crown proliferation. He may have mistakenly transmitted one of the aphid-borne viruses. Mellor and Forbes (1960) were unable to transmit WBD by either *C. fragaefolii* or *C. thomasi*. Sehgal and Boone (1963) transmitted MD by dodder (*Cuscuta subinclusa* Dur. and Hilg.) to *Fragaria vesca* 'EMC' and to strawberry but not to several herbaceous hosts. Transmission of MD was not achieved by means of the aphids *Aphis gossypii* Glov., *A. forbesi* Weed, several *Chaetosiphon* species, or the aster leafhopper *Macrostelus fascifrons* (Stål) (Sehgal and Boone 1963; N. W. Frazier, unpublished data).

### Properties of the Causal Agent

Strawberry WBD and MD both cause symptoms typical of yellows diseases that are commonly transmitted by leafhoppers and are caused by mycoplasma-like organisms (MLO) (Whitcomb and Davis 1970). As expected, temporary symptom remission of WBD was achieved by treatment of infected strawberry plants with oxytetracycline at 50 ppm in root mist culture (Huhtanen and Converse 1971). Structures resembling MLO were found in the phloem of WBD-infected strawberry petioles (fig. 83) (Doi and Okuda 1973; R. H. Converse, unpublished data). MD has not yet been similarly studied.

### Detection and Identification

So far, geography is the best criterion for identifying diseases associated with crown proliferation symptoms in strawberry



Figure 83. — Electron micrograph of mycoplasma-like bodies in sieve tubes of *F. vesca* cv. 'Alpine' infected with Miller isolate of witches'-broom disease. Bar represents 200 nm.



cultivars in North America. As previously noted, WBD has been recognized only in The Netherlands and on the Pacific coast of the United States and not in Canada. MD is known only from the Great Lakes region of the United States and from British Columbia. Crown proliferation; small leaflets borne on short, spindly petioles; and shortened runners are symptoms common to both diseases. No fruit are produced on WBD-infected cultivars, whereas normal-sized fruit are produced by a few, small flowers on MD-infected cultivars. *F. chiloensis* is said to be a good test plant for MD by leaflet grafting (Sehgal and Boone 1963), producing a tuft of small spindly leaves. There are no literature reports of grafts of WBD to *F. chiloensis*.

### Control Procedures

Both of these diseases cause pronounced and characteristic symptoms in infected strawberry cultivars. Both of them spread slowly in the field. Accordingly, the use of certified planting stock and roguing infected plants are adequate control measures. There is no published information on the elimination of either causal agent from infected plants. If they are caused by MLO, however, both should be readily heat inactivated in infected plants (Nyland and Goheen 1969).

### Remarks

The slight differences in symptomatology between WBD and MD that are reported in the literature could be ascribed to the environmental conditions and choice of cultivars in the areas where the work was done. The precise relationships between the causal agents of WBD and MD must await further etiologic studies of both diseases. Frazier believes that the WBD with which he worked in California was a complex of several undetermined causal agents (Frazier 1974).

### Minor or Little-Known Virus and Viruslike Diseases of Strawberry

By R. H. Converse

Several virus and viruslike diseases of strawberry, not discussed elsewhere in this section, have seldom been reported in the literature. Some of these diseases may have been important when reported, or may have been curiosities then and now, or may be of unrecognized importance in some strawberry growing areas at present. More data are required to establish their economic importance. Often, the relationships of these diseases and their causal agents to well-described diseases and pathogens remain to be determined. Key literature citations are given below. Many of these diseases were also discussed, sometimes in more detail than here, in Frazier (1970b).

Disease	Notes	Author and year
Strawberry necrosis (Nekrosevirus der Erdbeere)	Symptomless in cv. 'Herzberg's Triumph' and was mechanically transmitted to bean and a few other herbaceous test plants in Germany. Relationships unknown.	Maassen 1959, 1961
Strawberry band mosaic (Bandmosaik der Erdbeere)	Causes mosaic symptoms on leaves of cv. 'St. Anthony of Padua' in Hungary and is graft transmissible to cv. 'Muncheberg Early' but not to <i>F. vesca</i> . Relationships unknown.	Maassen and Nemeth 1961
Strawberry stunt	Infected cultivars were dwarfed with erect leaves, graft transmitted, and also claimed to be aphid transmitted. Reported in Minnesota (Skiles and King 1952) and possibly in Germany (Domes 1957), but the first report at least was probably multiplier plant disease in complex with other viruses (Boone 1970). (See "Strawberry Witches'-broom and Multiplier Diseases," p. 66.)	Zeller and Weaver 1941
Strawberry vein necrosis (called NEPO Virus No. 1 by R. M. Lister 1970)	Found in one strawberry plant (possibly cv. 'Champion') once in Minnesota and graft-transmitted to <i>F. vesca</i> , causing apical necrosis and recovery, and sap transmissible to several herbaceous hosts, similar to tobacco streak virus in many of its properties. (See "Tobacco streak virus in Strawberry," p. 57.)	Stingl and King 1965b
NEPO Virus No. 2	Graft transmitted to cv. 'Madame Moutout' and to <i>F. vesca</i> , causing leaf vein chlorosis, asymmetry, and followed by recovery; sap transmitted to many herbaceous plants, causing necrosis and tumors in beans. Relationships unknown. Reported once in northern Italy.	Canova and Tacconi 1965; Lister 1970

Tobacco mosaic virus	Loosely associated with Sukumi strawberry degeneration disease in Japan. See "Strawberry Leafroll," p. 61, for a report of its occasional association with this disease. Also isolated from <i>F. vesca</i> .	Cornuet and Morand 1960; Reed and Felix 1961; Kaname and Kishi 1973
Virus groups 1-6	Virus isolates from 39 strawberry cultivars indexed on <i>F. vesca</i> were cataloged into 6 groups, and previously reported viruses in strawberry were compared with them.	Schöniger and Bauer 1955

### ✓ Virus and Viruslike Diseases Experimentally Transmitted to Strawberry

By R. H. Converse

Strawberries, particularly *Fragaria vesca* L., have frequently been used as experimental host plants for viruses (hereafter used to include viruses and viruslike agents) from a number of other crops. Transmission has usually been by petiole insert leaf grafting, but aphids, dodder, and sap have also been used. It is possible to obtain virus transmissions to *Fragaria* from other Rosaceous genera, like *Rubus*. Table 6 lists literature reports of strawberry as a host of experimentally transmitted viruses not otherwise mentioned in this section.

## Nongraft Transmissible Diseases and Disorders

### 245 Strawberry June Yellows

By A. B. Wills

#### Additional Common Names

Leaf variegation, yellow leaf, gold leaf, spring yellows, transient yellows, 'Blakemore' yellows.

#### History and Geographic Distribution

Reports of variegation in strawberry species and cultivars in eighteenth and nineteenth century literature were noted by Stevens (1933) and in a comprehensive review of June Yellows by Darrow (1955). These reports are generally brief but June Yellows can be recognized with confidence from the description by Darwin (1896) of a variegated strawberry clone observed in 1859. Symptoms attributable to June Yellows have since been reported in cultivars or materials raised in the course of breeding or genetical studies. The earliest of such reports were by Richardson (1920, 1923) in England and Alderman (1926) in North America. The condition now occurs worldwide wherever strawberry breeding is done.

#### Economic Importance

Vigor and yield decline in affected clones as the disease progresses. In severely affected clones, plants are dwarfed and yields negligible. Serious economic losses can occur when a widely grown cultivar becomes affected and degenerates rapidly. A notable instance of this occurred in Britain when cv. 'Auchincruive Climax' became affected; symptoms were first seen in 1950, and by 1955 nearly all stocks had deteriorated so far as to be valueless (Wills 1962). The disease is a constant hazard in plant breeding, as significant numbers of progenies may have to be culled when it appears.

#### Symptoms

Symptoms are seen most clearly during the period of rapid spring growth; in most cultivars they disappear during the summer but may reappear in the autumn. Symptom expression is temperature sensitive and all but very severely affected plants become green when kept at a high temperature only to develop June Yellows again at ambient temperatures (Braak 1955).

Affected leaves are either uniformly pale-yellow when they unfold and become mottled green and yellow as they mature (fig. 84), or are mottled from the outset. The mottled areas are clearly delineated and tend to form sectorial patterns (fig. 85). The symptoms may become more conspicuous on successive leaves, but they usually disappear as the leaves age and may not occur on later-formed leaves. Permanent

**Table 6.—Viruses or viruslike agents experimentally transmitted to strawberry but not known to infect strawberry naturally**

Plant source	Virus or virus-like agents transmitted	<i>Fragaria</i> indicator	References
Apple and rose.	Apple mosaic (= rose mosaic).	<i>F. vesca</i> and cvs.	Cropley et al. 1960; Fulton 1952; Nyland and Engelbrecht 1958.
Apricot	Apricot ring pox.	<i>F. vesca</i> and cvs.	Nyland and Engelbrecht 1958.
Blackberry	Alpine mosaic agent	<i>F. vesca</i> 'Alpine'.	R. H. Converse, unpublished data.
Black raspberry.	Mild streak	<i>F. vesca</i> and cvs.	Braun and Keplinger 1962.
Chokecherry	X-disease	Strawberry cvs.	Braun and Keplinger 1962.
Peach	Prunus ringspot	<i>F. vesca</i> and cvs.	Nyland and Engelbrecht 1958.
	Tomato ringspot (= peach yellow bud mosaic).	<i>F. vesca</i> and cvs.	Nyland and Engelbrecht 1958.
	X-disease	<i>F. vesca</i> 'EMC'	Slack 1952.
Plum	Line pattern (= plum line pattern).	<i>F. vesca</i> and cvs.	Nyland and Engelbrecht 1958.
	White spot	<i>F. vesca</i> and cvs.	Nyland and Engelbrecht 1958.
Red raspberry	Senescence disorder	<i>F. vesca</i> 'Alpine'	R. H. Converse, unpublished data.
	Raspberry vein chlorosis	<i>F. vesca</i> 'Alpine'	Stace-Smith 1961.
	Green mosaic	<i>F. vesca</i> and cvs.	Braun and Keplinger 1962.
	Unknown disease, probably caused by tomato ringspot virus. (See "Tomato Ringspot Virus in Strawberry," p. 52.)	<i>F. vesca</i>	Vaughan and Wiedman 1955.
	Rubus yellow net	<i>F. vesca</i>	Stace-Smith and Mellor 1957.
	Various sources	<i>F. vesca</i> 'UC-1', 'UC-2'	Frazier 1963.
Sour cherry	Prune dwarf (= sour cherry yellows).	<i>F. vesca</i> and cvs.	Nyland and Engelbrecht 1958.
Sweet cherry	Rugose mosaic (= prunus ringspot).	<i>F. vesca</i> and cvs.	Nyland and Engelbrecht 1958.
	Big bud	<i>F. vesca</i> 'EMS-1', 'UC-1'	Helms 1962.
White clover	—	<i>F. vesca</i>	Krczal 1960.



Figure 84. — June Yellows showing variation in leaf symptoms on a single strawberry plant.



Figure 85. — June Yellows showing sectorial leaf mottling.

white streaks, usually accompanied by distortion of the leaf, appear spasmodically in some affected cultivars and have been observed unaccompanied by mottling symptoms in others.

The disease is progressive. Symptoms on affected plants become increasingly severe over a period of years; the plants become stunted and eventually die. The period elapsing before the disease first appears and the subsequent rate of progress vary greatly between cultivars.

#### Natural and Experimental Transmission

Natural transmission is known to occur only sexually, the disease being inherited through both pollen and seed parents. The proportion of affected seedlings in a progeny varies with the severity of symptoms in the parent plants and may even differ between progenies grown from seeds obtained from different flower trusses on one plant (Wills 1962). The disease can also occur in seedlings raised from symptomless plants of an affected cultivar or from nonaffected cultivars.

The relationship between mottling and white streak symptoms of leaves is still obscure because the two kinds of symptom can occur either together or separately.

#### Properties of the Causal Agent

Demaree and Darrow (1937) suggested that the cause might be a mutation making possible the appearance of a recessive character. Williams (1955) and Wills (1962) believed that an extra-nuclear plasmagene with a threshold for expression provided the best explanation of disease behavior, but both authors recognized an influence of plant genotype on disease development. More recently, Brown (1977) has postulated an interaction between a nuclear and a plasmid gene.

Attempts to transmit June Yellows by insects have been unsuccessful (Plakidas 1932; Morris and Afanasiev 1944; Guba 1933; Demaree and Darrow 1937; Berkeley 1931), as have sap inoculations (Plakidas 1932; Guba 1933) and grafts (Berkeley 1928; Plakidas 1929; Guba 1933; Demaree and Darrow 1937; Reid 1951; Williams 1955).

Thornberry et al. (1951) reported finding spherical particles in a variegated clone. Huhtanen and Converse (1971) observed partial and temporary remission of symptoms after applying a root spray of oxytetracycline and suggested that mycoplasma-like organisms might be involved in disease etiology. Recent attempts to associate viruses or viroids with June Yellows in extracts from affected plants have so far been unsuccessful (R. I. Hamilton and T. J. Morris, unpublished results).

#### Detection and Identification

No direct tests are known for latent June Yellows, but progeny testing by selfing has long been advocated as a means to detect carriers (Demaree and Darrow 1937). Bauer (1960) concluded that sib-crosses in  $S_1$  or back-crosses were more satisfactory because the plants obtained could be exploited in breeding nonsusceptible cultivars. He further advocated crossing  $S_1$  breeding materials to tetraploid *Fragaria vesca* L. var. *semperflorens* (Duch.) Ser. as a control test. Wills (1962) preferred a test cross to a cultivar known to have a genotype that permitted symptom development in susceptible offspring.

The disease is easily confused with other genetic variegations, some herbicide damage symptoms (see "Strawberry Herbicide Damage and Nutritional Imbalances," p. 79), or with the nonvariegated spring pallor shown by some genotypes. No other yellows condition shows both the seasonal variation and the sectorial symptom pattern characteristics of June Yellows.

#### Control Procedures

June Yellows showed no permanent response to prolonged growth at high temperatures (Wills 1962) or to heat treatment followed by meristem tip culture (East Malling Research Station 1969, 1970).



Propagation from nonaffected stock has proved effective in prolonging the useful life of some cultivars, for example, 'Blakemore' and 'Cambridge Favourite'. Careful inspection and roguing during spring are necessary for the production of certified planting material.

#### Remarks

Although June Yellows is not a wholly accurate name, it is the one most commonly used and it is desirable that it should be standardized.

#### Bud and Leaf Nematodes of Strawberry

By D. A. Slack and J. P. Fulton

#### Additional Common Names

Diseases associated with various species of bud and leaf nematodes have been termed spring dwarf, spring crimp, strawberry eelworm disease, red plant, summer dwarf, summer crimp, and Blätälchenkrankheit.

A more severe disease when nematodes are associated with a bacterium is termed cauliflower disease, strawberry bunch, and Blumenkohlkrankheit.

#### History and Geographic Distribution

Ritzema Bos (1890) first described a nematode from strawberry plants with cauliflower disease. Later, Crosse and Pitcher (1952) and Pitcher and Crosse (1958) demonstrated that this particular disease resulted from the interaction of a bacterium and nematode. Other studies in North America (Brooks 1931; Christie and Crossman 1936; Christie 1943) attributed milder disease symptoms to bud and leaf nematodes alone.

The cauliflower disease attributed to the nematode-bacterial association occurs in northern Europe only. Spring and summer dwarf are present in both northern and southern strawberry growing areas in the United States.

#### Economic Importance and Symptoms on Natural and Experimental Hosts

Although spring dwarf occurs more commonly in northern United States and summer dwarf more commonly in southern United States, the symptoms of these two diseases are similar. Initially the plant lies flat on the ground (fig. 86 A, B). Older leaves may take on a reddish coloration. New leaves are dwarfed with varying amounts of twisting, cupping, and crinkling (fig. 87). The main buds may die and secondary buds produce stunted, multiple-crowned plants. Fruit on affected plants are sometimes malformed (Slack et al. 1957).

The cauliflower disease is characterized by plants with crowns reduced to stunted, fleshy rosettes. The bulk of such a structure is composed of short, swollen leaf petioles, gall tissue, and rudimentary blossoms.



Figure 86. — A, Summer dwarf (dwarfed strawberry plants in center) caused by *Aphelenchoides besseyi*; B, left, spring dwarf caused by *Aphelenchoides fragariae*, right, normal strawberry plant.



Figure 87. — Spring dwarf of cultivated strawberry showing twisted young leaves.



### Natural and Experimental Transmission

Nematodes are spread to daughter plants produced from infected mother plants. Some dissemination of the nematodes occurs as a result of splashing rain and water movement. The nematodes persist for only short periods in the soil. Spread into new plantings is primarily the result of utilizing infested plants.

### Properties of Causal Agent

Three species in the genus *Aphelenchoides* are associated with the diseases described here. Spring dwarf is caused by *A. fragariae* (Ritzema Bos) Christie, while *A. besseyi* Christie is the cause of summer dwarf. In northern Europe, *A. fragariae* or *A. ritzemabosi* (Schwartz) Steiner and Buhrer are associated with the strawberry eelworm disease. Joint infections of either of these two nematodes and *Corynebacterium fascians* (Tilford) Downson cause the cauliflower disease. *A. besseyi* from rice does not infect strawberry and vice versa. Undoubtedly, a strain difference exists.

### Detection and Identification

Buds from suspected plants are dissected and macerated in water in a Petri dish or watchglass. After approximately 15 min, the material is observed at a low magnification for active nematodes. The characteristics of the several species and useful keys for their identification have been developed by Allen (1952).

### Control Procedures

Bud nematode problems are effectively controlled by careful attention to plant production. Certification programs should be cognizant of the nematode problems and allow no tolerance in inspection of certified plantings. When an occasional diseased plant is noted in fruit-producing fields, control can generally be effected by roguing.

### Cyclamen Mite Damage in Strawberry

By G. A. Schaefer

#### Additional Common Names

*Steneotarsonemus* (also known as *Tarsonemus*) *pallidus* (Banks), *Steneotarsonemus* (also known as *Tarsonemus*) *fragariae* (Zimmerman). Beer (1954) concluded that the European *S. fragariae* is synonymous with *S. pallidus*. This opinion is widely held although some authors consider them distinct species (Van Eyndhoven and Groenwold 1959), and the name *T. fragariae* is still frequently used in European literature. The common name approved by the Entomological Society of America is cyclamen mite. Other names used in the literature include strawberry mite, strawberry crown mite, and strawberry tarsonemid mite.

#### History and Geographic Distribution

The early history of this important pest of strawberry was reviewed by Smith and Goldsmith (1936). They noted that the mite had been observed on garden strawberries in Finland as early as 1892. In the United States, the pest was first

observed on strawberries by Darrow in 1928; however, it was long known as a pest of ornamental plants in greenhouses. The mite occurs throughout North America and Europe and has been reported from Africa, Australia, and Hawaii. As an outdoor pest, its distribution may be limited in areas of extremely high temperatures or low humidities. In the artificial environment of greenhouses, the mite could theoretically occur throughout the world.

### Economic Importance

Before suitable control methods were discovered, cyclamen mite was considered to be the most important pest of strawberries in California, the leading strawberry producing State in the United States. Reports on yield losses due to this pest are varied and range up to 70% (Savzdarg 1957). In Great Britain, detailed studies (Alford 1972) showed that severely injured plants exhibited 53% yield reduction, while moderately injured plants showed a 45% yield reduction. It was found that 63 mites per leaflet resulted in a 36% reduction in yield, compared to that obtained with 4 mites per leaflet. Although the mite is still considered a major pest on strawberry, with the development of modern pest management strategies devastating losses are no longer inevitable.

### Symptoms on Natural and Experimental Hosts

Cyclamen mite is primarily a greenhouse pest and under such conditions has a great range of host plants (Smith 1933). Outdoors, in addition to strawberry, they attack *Delphinium* and, more recently, the author has observed them on raspberry, although not in damaging numbers.

Because of the humidity requirements of the mite, its feeding activity is usually restricted to the protected areas within the folds of the youngest leaves. When injury is mild, the leaves continue to grow and unfold, but are reduced in size and have shortened petioles. The leaf may be dark green, and have a rugose, blistered appearance (fig. 88). The veins often assume a reddish brown color, and the plant itself appears dense as a result of the shortened petioles. Occasionally, the leaf edges have been observed to roll downward (fig. 89), thus suggesting leafroll virus symptoms. (See "Strawberry Leafroll," p. 61.) More severe symptoms result in a pale greenish-yellow color of the newly unfolding leaves (fig. 90). They are more severely reduced in size and distorted with the margins cupped upward. With high mite densities, even more severe symptoms may result. The petioles often fail to exceed 2.5 cm in length, and the new leaves do not unfold normally. They are much reduced in size, and present a silvery appearance because of the dense pubescence on their undersides. Later they die and turn brown, at which time they become quite brittle. In addition to foliar injury, the mites may also attack the blossoms causing them to darken around the base with resultant failure of fruit development.

### Properties of Causal Agent

Osterwalder (1928) described a "crinkle disease" of strawberry which he attributed to the mite's feeding activity. Because



Figure 88. — Rugose symptom on leaf caused by low-density cyclamen mite infestation.



Figure 89. — Leaf-rolling symptom caused by cyclamen mite.



Figure 90. — Severe crown symptoms due to high cyclamen mite density.

of the local symptoms of the “disease,” Harmson (1934) concluded that the injury resulted from feeding and was most likely not virus-induced. Massee (1933) considered the possibility that the mite was responsible for the presence of yellow-edge virus but was unable to confirm it. It is now concluded that the injury results when the mite penetrates the epidermis of the leaf and extracts the cell contents. Death of the leaf probably results under conditions of high mite density and reduced leaf growth, allowing for the release of high concentrations of destructive enzymes by the mite. Under conditions of low populations of mites and/or rapid leaf growth, the mites feeding activities appear to have a stimulatory effect on the epidermal cells resulting in their hypertrophy and proliferation (fig. 91 *A* and *B*). This reaction results in the distorted, blistered appearance of mildly infested leaves.

### Natural Spread

Because of the size of the adult mites, about 200 to 250 microns in length, as well as their dependence on a humid environment, they are unable to move any great distances under exposed conditions. Transmission probably occurs through the transfer of infested plant materials. The mite is generally introduced into a planting by means of infested



Figure 91. — Cyclamen mite damage: *A*, Cross section of a noninfested leaf. *B*, cross section of an infested leaf with proliferation of epidermal cells (enlarged).

nursery plants. Within-field spread then occurs through leaf contact and runner plants. Limited spread may occur via other agents, such as agricultural implements, clothing, bees, and other insects.

### Detection and Identification

Because of the small size of the mites, some degree of magnification is desirable when attempting to determine their presence. They may be most readily detected during periods of high population density. This occurs during fruit development, at which time vegetative growth is greatly curtailed. The different forms may be seen by opening the young, still-folded leaves. They may be seen clustering in large numbers (fig. 92) near the base of the leaflets. The adult female is oval in shape, has four pairs of legs, is pinkish or pale-amber to brown and has a glistening surface texture. The eggs that are generally present in greatest numbers are oval, transparent in color, and nearly one-half the size of the female. The presence of the mite may be more readily detected through recognition of the plant symptoms described above.

### Control Procedures

Cyclamen mite has been historically difficult to control, but a number of organic pesticides are now known to be effective (Schaefers 1963); however, these require the application of high gallonage drenches to the crown area. This may be most effectively accomplished during strawberry bed renovation, at which time the canopy foliage is removed and the crown growth is fully exposed. Granular systemic insecticides offer considerable promise for eradication in nursery stock. Organophosphate insecticides are particularly destructive to predatory mites. Several early studies suggest that differences in cultivar tolerance and/or resistance may exist, but, unfortunately, little research has been conducted on this aspect of cyclamen mite control.

### Remarks

The great variety of symptoms resulting from the feeding of this pest could mislead the observer to consider the possibility of viral causes.



Figure 92. — Cyclamen mites on underside of young leaflet (enlarged).

## Potato Leafhopper Injury in Strawberry //

By G. A. Schaefers

### Additional Common Names

None.

### History and Geographic Distribution

This insect pest earns its notoriety from the extensive damage, that is, "hopperburn," it causes on potato (Ball 1918). Poos and Wheeler (1943) were the first to rear the leafhopper from *Fragaria*. The economic significance of this association was reported by Campbell and Taylor (1962).

The potato leafhopper, *Empoasca fabae* (Harris), is not broadly distributed throughout the United States, occurring primarily in the East and Midwest. The insect overwinters in the South, migrating to the Northern States each year, usually starting around early May. On strawberry, they are most active during July and August.

### Economic Importance

The potato leafhopper for many years has been considered the most important economic species of the genus because of the damage it causes on potatoes, beans, clover, and alfalfa, (Delong 1931). Significant yield reductions have been attributed to the feeding of this pest on many crops. Leafhopper feeding on strawberry has been observed to reduce plant growth and inhibit runner production. To date, however, detailed studies have not been conducted to determine its effects on strawberry yield. Under defined conditions, and with certain cultivars, the insect requires control procedures, and thus may be classified as a major pest (Schaefers 1981).

### Symptoms on Natural and Experimental Hosts

The potato leafhopper has been reared from or collected on 138 host species (Poos and Wheeler 1943) and has been shown to produce a great variety of symptoms, depending on the host species as well as the time of the attack. These authors discuss the "diseaselike" injury caused on various hosts, such as "yellows" on alfalfa; "hopperburn," "leafroll," or "tipburn" on potato; "dwarfing" on peanuts; and "fruit spotting" on oranges.

In contrast to other *Empoasca* species, which are mesophyll feeders and cause a simple stippling of the leaf blade, *E. fabae* feeds in the vascular tissue, and causes a much more severe plant response. On commercial strawberry cultivars, feeding by both adults and nymphs produces a reduction in petiole length and leaf size (fig. 93). In greenhouse studies, as few as one or two nymphs were capable of nearly total inhibition of new growth. The leaves bend downward at right angles to the midvein and exhibit a general distortion. In more mature leaves, chlorosis begins at the edges and gradually moves down or towards the midvein (fig. 94). In no instance is the symptom referred to as "hopperburn" applicable to the symptoms caused by *E. fabae* feeding on



strawberry. Symptoms may be confused with those of early vein-banding disease (see "Strawberry Vein Banding," p.16) but distinct vein-banding symptoms are not evident.

### Properties of the Causal Agent

Plant symptoms resulting from leafhopper feeding have been attributed to one of three possible causes. These include inoculation of a pathogen, inoculation of a toxin, or mechanical blockage of the vascular system. While the vector hypothesis has been generally discounted, there remains good evidence for the support of the latter hypotheses. More recently (Medler 1941; Hollebone et al. 1966), it was proposed that saliva injected during feeding resulted in cell hypertrophy, which in turn interrupted translocation. This is in contrast to simple plugging of the vascular system with leafhopper stylet sheath material. In an expansion of the early "toxaemia" theory, entomologists have suggested that insect secretions are involved in the production of plant growth regulators, which may result in

various symptoms, including growth inhibition. It is now known that internal feeding by sucking insects is a complex process, and consequently each of the above may be an oversimplification of the facts.

The leafhopper cannot overwinter in the northern United States, and must migrate north each year from areas of Louisiana and Florida. The migrating forms move into the earliest available green crops, such as alfalfa, and from there into secondary crops as they become available. Eggs are deposited into the leaves and stems of strawberry, and the emerging nymphs feed on the undersides of the leaves. Young plants seem to suffer most severely.

### Detection and Identification

Upon observing the symptoms described above, one should examine the undersides of the leaves for the presence of the insect. The adults are smaller and lighter green. They may be distinguished from other insects occurring on strawberry by their habit of running sideways when disturbed. The presence of cast skins will indicate the past presence of the insect.

### Control Procedures

The leafhopper is easily controlled by a number of insecticides. Local recommendations should be consulted. Preliminary observations suggest that marked differences in tolerance exist among cultivars.



Figure 93. — Leafhopper injury on new growth.



Figure 94. — Leafhopper injury on mature leaf.

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### Miscellaneous Arthropod Damage in Strawberry //

By G. A. Schaefer

Diseaselike symptoms are commonly associated with feeding by members of the insect order *Hemiptera*. No less than 20 species are recognized as pests of strawberry. Members of this order feed by means of piercing and sucking type mouthparts. During this process, a complex of chemicals may be injected into the plant, which can have a profound influence on its physiological development. These chemicals include stylet sheath material, which is mainly protein and oxidizing enzymes, and watery saliva, which consists largely of hydrolyzing or digestive enzymes, amino acids, and, possibly, plant hormones (Miles 1972). The phytopathogenic effects of a few of the more important hemipterans and mites are reviewed here.

### Mealybug

Members of the family *Pseudococcidae*, or mealybugs, are among the most serious pests of plant life. Several species of the genus *Pseudococcus* occur on strawberry.

Hildebrand (1939) noted the similarities in symptoms between injury by mealybug feeding and crinkle virus. (See "Strawberry Crinkle," p. 20.) In both instances, small chlorotic spots may occur on the young unfolding leaves. In mealybug injury, however, the spots continue to enlarge. These eventually coalesce, and mosaiclike symptoms may



Figure 95. — Adult mealy bug on lower surface of a leaflet (enlarged).

result. Some dwarfing of the plant occurs with mealybug feeding, but the leaves do not show the rugosity that is characteristic of crinkle.

Mealybugs are small (2 mm), soft-bodied insects that appear on the stems and crown growth (fig. 95). They are characterized by flattened, elongate oval bodies covered by a white powdery wax, which extends from the sides in a series of short filaments with usually two longer ones at the posterior end.

Following chemical control, new growth will be symptomless.

### Shallot Aphid

While the strawberry aphid *Chaetosiphon fragaefolii* (Cock.) is important as a virus vector, even large numbers of these aphids free of viruses fail to produce any significant effect on plant development. In contrast, the shallot aphid, *Myzus (Nectarosiphon) ascalonicus* Doncaster, can have a severe effect on both the quality and quantity of strawberry yields (Alford 1976). Dicker (1950), upon first observing the plant injury caused by this aphid, noticed the resemblance to the symptoms of severe crinkle virus (see "Strawberry Crinkle," p. 20). Similarly, Mellor and Fitzpatrick (1961) pointed out the possibility of confusing these symptoms with those of crinkle virus or of certain virus complexes. Dicker noted that the petioles became shortened, and the laminae were small, puckered, twisted along the midribs, and curled. Some strawberry cultivars exhibit red or purplish flecks on the leaves. He concluded that the injury was purely mechanical and that symptoms disappeared with control of the aphid. The severity of the symptoms, however, would strongly suggest that salivary injections may be involved.

### Spittlebug

A spittlebug, *Philaenus leucophthalmus* L. attacks all portions of the strawberry plant and produces distortion or death of the stems and leaf blades (Mundinger 1946). Zeller (1933) reported that spittlebug injury resembles crinkle virus symptoms (see "Strawberry Crinkle," p. 20) in that the



Figure 96. — Deformed leaf caused by spittlebug feeding.

leaves become crinkled and dark green (fig. 96). Some fruit deformity results from the presence of spittlebug. While detailed studies have not been conducted, it appears that the deformity results from spittlemass interference with pollination rather than from some salivary secretion. Spittlebug injury may be readily distinguished from virus disease by the decided absence of systemic activity and the normality of the new growth. Fresh or dried spittle masses on the leaf surfaces provide evidence for the involvement of spittlebug. It is readily controlled with a number of insecticides.

### Tarnished Plant Bug

Several species of plant bugs (*Miridae*) are known to attack strawberries. These include *Lygus hesperus* Knight, *Lygus elisus* Van Duzee, and the tarnished plant bug, *Lygus lineolaris* (P. de B.). Allen and Gaede (1963) concluded that *L. hesperus* feeding during the blossom stage could cause the production of deformed fruit. The problem is a major one in many areas, and in a "normal" season, one nymph per blossom cluster can cause 30 percent injured fruit with about a 9 percent reduction in mean berry weight (Schaeffers 1980).

Injury by the tarnished plant bug is characterized by the presence of a number of well-developed achenes, which are closely located due to lack of development of that area of the receptacle (fig. 97). Upon dissection, the achenes will be found to be hollow. While detailed feeding studies have not been conducted, the bug may secrete a digestive enzyme during feeding, which functionally blocks the stimulation of receptacle growth by plant hormones. While a number of pathogens can result in the production of "seedy" fruit, tarnished plant bug injury is distinguished by the localized patches of seeds. Resistant cultivars have not yet been found,



but control can be readily obtained through the application of effective pesticides during blossom and early fruit development.

### Spider Mites

Spider mites are worldwide in distribution and are considered to be major economic pests on many field and orchard crops as well as ornamental plants. The twospotted spider mite, *Tetranychus urticae* Koch, and the strawberry spidermite, *Tetranychus turkestanii* Ugarov and Nikolski, are probably of greater concern to North American strawberry growers than any other arthropod pest on this crop.

Feeding injury resulting from low infestation levels of mites may result in temporary confusion with crinkle symptoms (Mellor and Fitzpatrick 1961). (See "Strawberry Crinkle," p. 20). Infected leaves are reduced in size and may present a speckled or stippled appearance (fig. 98). The mites feed on the lower surface of the older leaves. Feeding involves a

rasping of the leaf surface followed by a sucking of the cell contents. In contrast with the insects discussed previously, there is no indication that any phytopathogenic secretions are involved.

In higher population densities, symptoms on the upper leaf surface will be a bronzing and drying of the leaf tissues. Such feeding reduces plant vigor and yield and leads to stunting and possible death of the plants. Although some resistant cultivars have been identified (Kishaba et al. 1972), a number of acaricides may be effectively used in integrated pest management systems (Kennedy et al. 1976).

### Heat Spot of *Fragaria vesca*

By J. P. Fulton

Small chlorotic spots appearing on the leaves of *Fragaria vesca* L. indicators as a result of physiological stress can sometimes be mistaken for virus symptoms (fig. 99). These symptoms have been termed "heat spot" (Smeets and Wassenaar 1956) and are most commonly seen when plants are moved from a cool to a warm environment. Plants that are potbound or lacking in sufficient nutrients often respond with heat spot at temperatures above 24°C. Since this is a stress response, plants which have been inoculated with mild forms of certain viruses or viruslike agents such as mild yellow-edge virus or pallidosis agent (see "Strawberry Mild Yellow-edge," p. 25, and "Strawberry Pallidosis," p. 55) will also exhibit this type of spotting. To avoid the confusion this symptom may cause when indexing for viruses, utilize young, vigorously growing plants as indicators, add ungrafted controls, and hold plants at temperatures near 20°C.



Figure 97. — Tarnished plant bug injured berries.

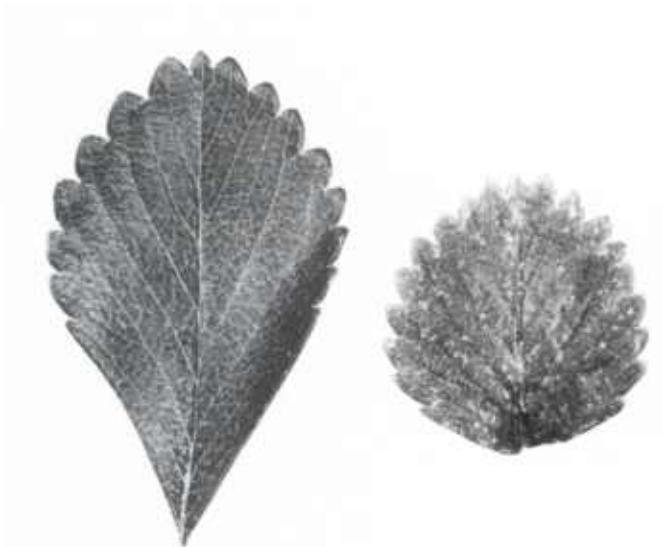


Figure 98. — Right, Spidermite injury on leaflet; left, normal leaflet.



Figure 99. — Heat spot symptoms on *Fragaria vesca* cv. 'EMK'.

## Strawberry Herbicide Damage and Nutritional Imbalances

By P. C. Crandall

Herbicide injury and the toxicity or deficiency symptoms that result from nutrient imbalances may occur in strawberry fields and are occasionally mistaken as symptoms of virus infection. Awareness of these possibilities and knowledge of symptomatology and conditions under which these situations may occur help to prevent mistaken diagnoses of field problems.

### Herbicide Injury

Herbicides that are registered for use on strawberries have been tested for crop tolerance over a wide range of conditions. Only after a number of years of such testing are they approved for use by growers. Recommended application rates are established to provide a good margin of safety below phytotoxic levels. The instructions on the product label include all precautions considered necessary for effective weed control and to prevent crop damage. In spite of this, strawberries are occasionally damaged by herbicides. Such phytotoxicity results from any one of a number of causes (Yarish 1980; Skroch and Sheets 1977).

**Overapplication.** Herbicides are plant killers. If too much is applied, crop injury can occur. Overapplication is a frequent cause of damage to strawberries. It can result from failure to follow the product label directions or miscalculation of the amount of herbicide put into the sprayer tank. The sprayer may not be adjusted to apply the correct amount of material per hectare. Since wettable powder herbicides are very abrasive, they cause the nozzle openings to erode rapidly, thus the application rate is increased. There is a tendency for the tractor to travel slower when going up a slope and at the ends of rows. Overapplication can also result from overlapping at the ends of the spray boom. (Lockerman et al. 1975). Sometimes, excessive herbicide application occurs when residual chemicals are applied to soil which already has a high chemical level as the result of application on a previous crop.

**Drift.** Injury from drift can occur at considerable distance from the place of application. The phenoxy herbicide, 2,4-D, is especially prone to drift either as minute droplets or as vapor. Such drift causes leaf and stem or fruit deformities on strawberries (figs. 100 and 101). The tendency to drift is increased by high temperature, windy conditions, and/or high nozzle pressures at the time of application.

**Moisture and soil texture.** The tolerance of strawberries to residual herbicides depends on the ability of the plants to endure low concentrations of the chemical and on the low solubility of the herbicide, which causes the active ingredients to be released slowly. Under normal soil and moisture conditions, an equilibrium is maintained that controls weeds in the surface layers of the soil with no



Figure 100. — Twisting of strawberry petioles caused by 2,4-D. (Courtesy W. A. Skroch, North Carolina State University, Raleigh.)



Figure 101. — Misshapen strawberry fruit resulting from 2,4-D application during time of blossom bud formation. (Courtesy W. A. Skroch, North Carolina State University, Raleigh.)

damage to the deeper-rooted strawberries. Damage can occur, however, if the roots are exposed, if the soil is not adequately settled around the plants, or if the soil has a coarse texture. Under loose soil conditions, especially if excessive moisture occurs soon after the herbicide is applied, the chemical is carried down into close proximity to the roots and causes injury. Danger from this source of injury is greater with the more soluble herbicides. Chemicals with long residual activity may accumulate to toxic levels as a result of repeated applications in soils high in organic matter or clay fractions.



**Stage of strawberry plant growth.** Susceptibility to phytotoxicity is related to the stage of plant growth, age of plants, and sensitivity of the cultivar, and it varies with the time of year and the chemical. Factors involved include rainfall, temperature, dormancy of the aboveground parts of the plants, activity of the root system and other actively growing sites, and the internal physiological processes of the plant.

Simazine, a residual herbicide, can be used safely in the Pacific Northwest of North America if applied to strawberries during late summer or early winter. When applied during the spring, it causes marginal chlorosis or necrosis of the leaves and considerable stunting of the strawberry plants (fig. 102). This chemical accumulates in soils that have high exchange capacities and, in perennial plants like strawberry, repeated use may cause toxicity. Simazine cannot be safely used on strawberries in Eastern United States where different growing conditions result in crop injury.

Napropamide, if applied during the time of runner development, prevents many of the runner plants from rooting.

The phenoxy herbicide 2,4-D can be safely applied to strawberries right after harvest or during the winter. Some leaf deformation may occur but is not serious. When applied in the autumn during blossom bud differentiation, it causes abnormally large, deformed fruits at harvesttime (fig. 101).

Dinoseb is satisfactory if applied to fully dormant plants; however, when applied to growing plants or semidormant plants it causes stunting and yield reduction.

**Cultivar tolerance.** Cultivars differ in their sensitivity to herbicides. This tolerance is related to concentration of the chemical, internal metabolism, growth habit, age, hairiness of the leaves, and other anatomical features. Most older cultivars have been evaluated for herbicide tolerance, but newly introduced cultivars should be carefully observed for herbicide sensitivity.

**Additional factors.** Other factors that influence phytotoxicity are chemical incompatibilities, temperature, humidity, plant vigor, and cultural practices.

**Symptomatology.** Visible symptoms of herbicide injury on strawberries vary widely. Some of the common symptoms are vein clearing (terbacil, fig. 103), marginal chlorosis or necrosis (chloroxuron, fig. 104), interveinal chlorosis or necrosis (simazine, fig. 102), leaf or fruit deformity and epinasty of stems, (2,4-D, figs. 100, 101), root damage and inhibition of root growth or stunting of plants (napropamide, fig. 105), or dying of plants. These symptoms may occur in definite patterns across the field or may affect plants in a random pattern.



Figure 102. --- Simazine injury on strawberry leaves.



Figure 103. — Vein clearing of strawberry leaves caused by terbacil. (Courtesy J. W. Braun, Washington State University, Vancouver.)



Figure 104. — Chloroxuron injury of strawberry leaves. (Courtesy R. S. Byther, Washington State University, Puyallup.)



Figure 105. — Typical strawberry leaf coloration that accompanies stunting. Stunting was caused by napropamide. (Courtesy J. W. Braun, Washington State University, Vancouver.)

**Diagnosing the problem.** Careful observation of injury patterns in the field sometimes helps to diagnose the problem. The injury may occur in streaks that are related to the spray boom length or to spray boom overlap. Injury may be worse at the ends of rows or when the sprayer is moving up a slope. Any of these injury patterns may indicate herbicide damage. Sometimes, it is possible to relate injury to soil texture or drainage. Often, the only way to determine the cause is to analyze the complete herbicide application schedule, including previous cropping history. Lockerman et al. (1975) list a number of pertinent questions to ask and field symptoms for which to look. Jennings and Nyvall (1977) and Skroch and Sheets (1977) emphasize the importance of “lookalike” symptoms that may be mistaken for herbicide injury.

### Nutrient Imbalances

Nutrient imbalances may cause visible symptoms of either deficiency or toxicity. Such symptoms indicate that a radically imbalanced situation has existed for some time. This imbalance results from either too much or too little of the nutrient in the soil, or from the application of another nutrient or material that produces an antagonistic effect on the absorption, translocation, or utilization of the nutrient. The chemistry of such imbalances has been studied extensively. The absorption and accumulation of each nutrient are influenced by the absorption of every other nutrient (Shear et al. 1948).

Deficiency and toxicity symptoms are visible responses to this imbalanced nutrition. These visible expressions of

extreme nutritional imbalances vary considerably among cultivars and may sometimes be complicated by the presence of more than one imbalance.

**Deficiency symptoms.** A description of nutrient deficiency symptoms of strawberries is helpful in diagnosing field problems. The following list includes composite descriptions derived from those published by Johanson (1963 and 1980), Johanson and Walker (1963), Lineberry and Burkhart (1943), Iwakiri and Scott (1951), Hoagland and Snyder (1933), Lott (1946), Davidson (1941), Boyce and Matlock (1966), Davis and Hill (1928), and Ulrich et al. (1980). More complete descriptions are included in the above publications.

### *Most Common Nutrient Deficiency Symptoms on Strawberries*

Deficient nutrient	Symptoms
Nitrogen	Young leaves small, pale, yellowish green on stiff, upright petioles. Plants stunted (fig. 106). Old leaves may have red serrations or be completely bright yellow or orange-red, later turning brown, with necrotic margins. Calyx on ripe fruit reddish.
Phosphorus	Dark, bluish purple to blackish upper leaf surfaces (fig. 107). Bottoms of leaves reddish purple, often blue in veins of older leaves. Leaves small, cupped downward, reduced number of flower buds.



Figure 106.—Nitrogen deficient strawberry plants on right showing small, light green leaves and stunted growth; normal plant on left. (Courtesy A. Ulrich, University of California, Berkeley.)





Figure 107. — Phosphorus deficiency. Bluish-purple coloration of upper strawberry leaf surfaces. (Courtesy F. D. Johanson.)

#### Potassium

Marginal chlorosis of mature leaves changing to reddish purple. Leaf margins scorch and turn upward. Areas between veins reddish brown except for a green triangle at base of leaflet (fig. 108). Lower midrib and short section of petiole darkens and becomes dry. Younger leaves unaffected or may show some interveinal chlorosis. Older leaves die forming a collar around base of plant. Some leaflets develop a red band across the middle of the underside.



Figure 108. — Potassium deficiency. Reddish-brown strawberry leaves with bright green triangle at base of leaflet. (Courtesy F. D. Johanson.)

#### Magnesium

Interveinal chlorosis of mature leaves beginning near upper margin, becoming reddish brown and necrotic. Serrations green during early stages. Interveinal areas develop necrotic patches giving a blotchy leaf pattern (fig. 109). Short petiole section

between blades and crown remains green. Young leaves remain green. Upward cupping of leaf margins.



Figure 109. — Magnesium deficiency. Marginal and interveinal chlorosis on older strawberry leaves, young leaves unaffected. (Courtesy A. Ulrich, University of California, Berkeley.)

#### Boron

Tipburn of early, unfolding leaves. Marginal and interveinal chlorosis of young leaves (fig. 110). Growing points die causing development of small, chlorotic, deformed, and cupped leaves near the center of plants. Short, brittle petioles and blasting of flowers with deformed fruits. Roots short and stubby with multiple branching.



Figure 110. — Boron deficiency. Tipburn and interveinal chlorosis on young strawberry leaves, older leaves with puckered, squared-off tips.

## Calcium

Newly emerged leaves develop tipburn and severe crimping of the tips of the leaflets (fig. 111). Brown lesions on leaf and fruit petioles with globules of sirupy sap exuding from large veins and petioles (fig. 112). Older leaves develop chlorotic areas or a purplish band across the center of the leaf blades, which later becomes necrotic. Fruit stunted with imperfect achene set.



Figure 111. — Calcium deficiency. Mature strawberry leaf with tipburn. (Courtesy A. Ulrich, University of California, Berkeley.)



Figure 112. — Calcium deficiency. Brown lesions with globules of sap on strawberry petioles. (Courtesy A. Ulrich, University of California, Berkeley.)

## Zinc

Young leaves pale green or yellow; serrations remain green. Leaf blades narrow, concave, and elongated. Larger veins remain green (fig. 113). Reddening between veins may occur in some cultivars. Leaves become stunted.



Figure 113. — Zinc deficiency. Young strawberry leaves pale green or yellow, serrations green. Leaf blades narrow and elongated, normal leaf on left. (Courtesy F. D. Johanson.)

## Manganese

Chlorosis of young leaves followed by fine, green-netted veining or discontinuous vein clearing (fig. 114). In later stages, the vein clearing may develop into purple stippling between larger veins. Leaf margins become necrotic and curl upward. Scorching develops inward from outer margins of leaflets (fig. 115).



Figure 114. — Manganese deficiency. Fine, green-netted veining or discontinuous vein clearing on strawberry leaflet (enlarged). (Courtesy A. Ulrich, University of California, Berkeley.)





Figure 115. — Manganese deficiency. Marginal scorching of strawberry leaves. (Courtesy A. Ulrich, University of California, Berkeley.)

#### Iron

Young leaves develop interveinal chlorosis and are light yellow to nearly white (fig. 116), followed by marginal necrosis. New leaves are stunted. Older leaves remain green for some time.



Figure 116. — Iron deficiency. Young strawberry leaves light yellow to nearly white. (Courtesy F. D. Johanson.)

**Nutrient toxicity.** Micronutrients also damage strawberry plants when present in excessive amounts. Among the most common are boron, copper, and manganese. Visual symptoms of such toxicities are not very specific. They tend to involve stunting, marginal necrosis, and death of the plants.

**Salt toxicity.** Salt toxicity results from a buildup of excess soluble salts in the soil, which causes severe stunting or leaf burn (Bernstein 1980). Stunting is related to the concentration of soluble salts in the soil solution and is often accompanied by typical chloride leaf burn (fig. 117). Such salt accumulations often result from the use of saline irrigation water. Buildups occur in heavier soils where leaching is slow, where not enough natural rainfall or irrigation water is available to leach salts out of the root zone, or in soils with a high water table. In high water table situations, the water evaporates from the soil surface leaving an accumulation of salts behind.

**Albinism.** Strawberries sometimes produce white or light-colored fruits that are normal in size but are insipid, mushy, and spoil rapidly after picking (fig. 118). This problem results from sugar levels that are inadequate for normal ripening (Ulrich et al. 1980). It often occurs during periods of cloudy weather on excessively vigorous plants. It can also occur on plants with a heavy set of fruit and poor leaf development. The latter is typical of plants that have received inadequate chilling before or after planting. Excessive levels of bromide ion in the soil following methyl bromide fumigation can also lead to the production of albino fruits.

**Diagnosing nutrient problems.** Specific plant and leaf symptoms are helpful for determining nutrient problems. Often, a careful observer can associate such symptoms with soil texture and drainage. Symptoms are usually not uniform from plant to plant or between areas of the field. When they appear to be related to changes in soil texture and drainage, nutritional problems should be suspected. Addition of deficient nutrients or correction of soil conditions to avoid deficiencies or toxicities should result in normal plant growth. Foliar symptoms can be confirmed by plant and soil chemical analyses. Such information helps to separate nutritional problems from virus or other disease problems. Nutritional symptoms related to nutritional imbalances may also be accentuated as a result of insect, disease, mechanical or freeze damage, or action of vertebrate pests on roots. These possibilities should always be considered.

**Importance of health virus indicator plants.** Since nutritional and other cultural problems can sometimes be mistaken for virus disease expression, it is important that plants to be used as indicator plants be healthy and normal in appearance. Such plants must be grown under adequate nutrition, light, and moisture conditions. Optimum levels vary with cultivar and must be determined from experience.



Figure 117. — Marginal necrosis of strawberry leaves typical of chloride (salt) injury. (Courtesy L. E. Francois, USDA Salinity Laboratory, Riverside, Calif.)



Figure 118. — Albino strawberry fruits caused by inadequate carbohydrate levels during ripening. (Courtesy R. D. Nelson, Driscoll Strawberry Associates.)

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## Section 2.

# 24 Virus and Viruslike Diseases of *Vaccinium* (Blueberry and Cranberry)

### Introduction

By D. C. Ramsdell, J. F. Hancock, and A. W. Stretch

### History of Blueberry and Cranberry Culture

Blueberries and cranberries belong to the genus *Vaccinium* in the heath family *Ericaceae*. There are several agriculturally important subgenera in *Vaccinium*, including *Cyanococcus* (true blueberries), *Oxycoccus* (cranberries), and *Euvaccinium* (bilberries and whortleberries). Members of all these subgenera grow wild in North America, whereas only *Oxycoccus* and *Euvaccinium* occur naturally on the European continent.

The berries of numerous *Vaccinium* species have been harvested from the wild by humans since early history, but only a few species have been extensively cultivated. The most important species, according to Camp's (1945) classification system, are highbush (*V. corymbosum* L. and *V. australe* Small), lowbush (*V. angustifolium* Ait.), rabbiteye (*V. ashei* Reade), and cranberry (*V. macrocarpon* Ait.). Others cultivated to a limited extent in North America or harvested from the wild are dryland (*V. altomontanum* Ashe and *V. pallidum* Ait.), evergreen (*V. ovatum* Pursh), mountain (*V. membranaceum* Dougl.), Canada (*V. myrtilloides* Michx.), and Constable's (*V. constablaei* A. Gray). In Europe, three additional species are harvested from the wild—bilberry (*V. myrtillus* L.), cowberry (*V. vitis idaea* L.), and European cranberry (*V. oxycoccus* L.).

The cranberry *V. macrocarpon* has been cultivated in North America since the early 19th century. Commercial production began in the Cape Cod region of Massachusetts, and it has since spread to other parts of Massachusetts, Wisconsin, New Jersey, Washington, and Oregon. Originally, wild selections made up most of the cranberry acreage, but improved cultivars now dominate most of the production regions. Massachusetts and Wisconsin produce the most cranberries.

The lowbush blueberry (*V. angustifolium*) has been cultivated in the Northeastern United States since the middle of the 19th century. The major lowbush blueberry regions are still in the Northeastern United States and Eastern Canada, although limited acreages are also in Wisconsin, Minnesota, West Virginia, and Michigan. Most of the lowbush production is based on native plants growing on their original location, however, lowbush blueberry cultivars have been planted on a very limited scale.

The highbush blueberry (primarily *V. corymbosum*) has only been widely cultivated for the last 50 yr. The first serious efforts were made in the early 1900's by F. W. Coville of the

U.S. Department of Agriculture and Elizabeth C. White at Whitesbog, N.J. Today, in 1982, the most important highbush blueberry regions in North America are located in Michigan and New Jersey. North Carolina, British Columbia, Oregon, and Washington also have significant acreages, and Arkansas has a small but growing industry. Most of the highbush blueberry acreage is now composed of hybrid selections, although approximately 10% of the cultivated blueberries in Michigan is the wild selection 'Rubel'.

Rabbiteye blueberries (*V. ashei*) have been cultivated for a shorter period of time than any of the other domesticated *Vaccinium* species. Until recently, cultivation was limited to plants transplanted from the wild in restricted areas of Florida, Georgia, and Alabama, but improved cultivars have been developed in the last 30 yr which have resulted in some expansion within the Southeastern United States and into other States, including Texas and Arkansas.

Outside of the United States and Canada, blueberry and cranberry cultivation has been limited. Highbush blueberries have been planted to some extent in Europe in the last 20 yrs, but the cranberry (*V. macrocarpon*) is still quite rare. Most of the *Vaccinium* berries eaten in Europe are shipped from North America or are harvested from wild populations of native species.

### Virus and Viruslike Diseases of Blueberry and Cranberry

A considerable amount of knowledge concerning *Vaccinium* virus and viruslike diseases (especially blueberry) and their etiology has been developed since the writing of "Virus Diseases of Small Fruits and Grapevines" (Frazier 1970). Further knowledge of virus-vector relationships has also been advanced during this period.

The viral etiology of blueberry shoestring has been well documented. The virus has been thoroughly characterized and an aphid vector identified (Lesney et al. 1978; Ramsdell 1979; Ramsdell and Stace-Smith 1979b).

Mycoplasmalike organisms (MLO) have been associated with blueberry stunt (Chen 1971; Hartmann et al. 1973), blueberry witches'-broom (Kegler et al. 1973; Blatný and Vaňa 1974; de Leeuw 1975), and cranberry false blossom (Chen 1971). Leafhopper vector(s) for each of these diseases have been strongly implicated or proved.

Three new virus diseases of highbush blueberry and their causes have been reported as having nematode vectors or putative nematode vectors since the 1970 book was published.



A new disease called blueberry leaf mottle has been described (Ramsdell and Stace-Smith 1979a), and the virus has been characterized (Ramsdell and Stace-Smith 1981). Although the physical and chemical properties of the virus indicate that it is a putative member of the nepovirus group (Harrison and Murrant 1977), nematode transmission has not been proven as of this writing (D. C. Ramsdell, unpublished data). Peach rosette mosaic virus has been shown under experimental field conditions to cause disease in two blueberry cultivars (Ramsdell and Gillett 1981). Tomato ringspot virus has been shown to be associated with a disease of highbush blueberry, showing symptoms somewhat similar to those of necrotic ringspot disease (Johnson 1972).

The causal agent and vector of blueberry mosaic remain unknown. Red ringspot disease has recently been shown to be associated with a large spherical virus embedded in inclusion bodies (Kim et al. 1981). The vector has not yet been identified for this virus. Similar virions and inclusion bodies have been associated with ringspot disease of cranberry, indicating that these two diseases may have a common causal agent (K. S. Kim, unpublished data).

Although rabbiteye blueberry is being planted in significant quantities in Southeastern United States, to date the only reports of virus or viruslike diseases occurring in this blueberry type show that blueberry stunt can be experimentally graft transmitted to it (Dale and Mainland 1981; R. M. Milholland, unpublished data).

Whereas in a previous review of this field, "Minor Virus Diseases of Ericaceae in Europe" and "Leaf-Spotting Diseases of Low-Bush Blueberry" were covered as subjects (Frazier 1970), these will not be covered in this handbook because of their relative lack of importance.

### Indexing and Detection Procedures

For detection and indexing of blueberry viruses for which antisera exist, enzyme-linked immunosorbent assay (ELISA) may be used successfully. ELISA works very well in the detection of blueberry shoestring virus, tobacco ringspot virus (necrotic ringspot disease), tomato ringspot virus, peach rosette mosaic virus, and blueberry leaf mottle virus. Alternatively, all of the previously listed viruses, with the exception of blueberry shoestring virus, are sap-transmissible to herbaceous indicator hosts, the two most useful being *Chenopodium quinoa* Willd. and cucumber (*Cucumis sativus* L.). A small amount of young terminal leaf tissue should be taken from several locations on a bush to be indexed. The tissue should be ground in a small amount (2-3 ml) of 0.05 M phosphate buffer (ph 7.2) containing 2 to 3% nicotine alkaloid, using a mortar and pestle. The resulting sap-buffer mixture is then rub-inoculated to leaves of the herbaceous plants that have been previously dusted with 300 to 600 mesh carborundum or diatomaceous earth. After 7 to 14 days, symptom-bearing leaves can be collected, sap expressed, and tested with agar gel double diffusion serology plates, using antisera to the previously listed viruses.

For blueberry mosaic, stunt disease, and witches'-broom, for which no antisera are available, and red ringspot virus [for which serological detection methods are in the process of being developed (D. C. Ramsdell, unpublished data)], budding or whip grafting techniques to sensitive blueberry cultivars (see below) offer the best method of detection. Alternatively, for detection of stunt and witches'-broom, electron microscopy of ultrathin sections could be used to detect the MLO in the sieve tubes of diseased leaf tissue although this is a slow, costly method.

For stunt and mosaic diseases, chip budding or whip grafting from dormant diseased or suspected diseased cultivars onto healthy 'Cabot' highbush blueberry is effective. A 2-yr observation period is necessary to allow symptoms to develop. For red ringspot disease, chip budding or whip grafting onto healthy 'Blue-ray', 'Cabot', or 'Darrow' is effective. Again, a 2-yr observation period is advisable (A. W. Stretch, unpublished data).

For detection of cranberry viruses and MLO, no serological methods are available. Because of their thin and wiry stems, cranberries are not suitable subjects for graft transmission. Until better detection techniques are developed, electron microscopy of suspected diseased tissue could be used for the detection of false blossom MLO in sieve tubes from ultrathin sections of leaf tissue. For detection of ringspot of cranberry, observation of symptoms on the crop plants is the only available method.

### Certification Programs

At present (1982), no program exists for growing virus-tested certified clean stock. Michigan and New Jersey have nursery inspection programs, but symptomless or latent infections are missed without the requisite indexing to assume freedom of infection from virus and viruslike entities. Arkansas with its fledgling blueberry industry is promulgating a mother block system of growing inspected stock (J. P. Fulton, personal communication). North Carolina has developed a system of combating stunt by indexing their cultivars on 'Cabot' and keeping their clean "nuclear" stock in screenhouses for a source of expansion stock to be grown in the field without a screenhouse for 1 yr. This is then sold as "registered" stock (R. M. Milholland, personal communication).

The recent expansion of research on *Vaccinium* virus diseases by USDA, ARS, at Corvallis, Oreg., has given impetus to develop a stringent thermotherapy, indexing, and certification program, involving most commonly used blueberry cultivars. This program will be a joint effort between the USDA, the North American Blueberry Council, and research and regulatory personnel from Michigan, New Jersey, Oregon, and Washington (R. H. Converse, personnel communication). This program should result in the orderly distribution of clean, true-to-name cultivars of blueberry stock, which can then be expanded by nurseries in the various States. Thereafter, it is expected that appropriate personnel in those States will ensure that such stock remains clean.

### Blueberry Shoestring

By D. C. Ramsdell

#### Additional Common Names

None

#### History and Geographic Distribution

The disease in highbush blueberry (*Vaccinium corymbosum* L.) was described by Hutchinson (1950) and later was shown by Varney (1957) to be of virus or viruslike etiology. The disease is most prevalent in Michigan and New Jersey and has been found in Washington (P. Bristow, and D. C. Ramsdell, unpublished data) and North Carolina (R. M. Milholland, unpublished data). A recent survey for the disease in Oregon failed to show its occurrence there (Converse and Ramsdell 1982). The disease has been reported in Nova Scotia (Lockhart and Hall 1962) in lowbush blueberry (*Vaccinium angustifolium* Ait.). There have been no reports of shoestring in blueberries from other parts of the world.

#### Economic Importance

Shoestring virus in Michigan has infected about 145,000 plants on 10,000 acres and has caused a loss of approximately \$3 million (D. C. Ramsdell, unpublished data). Economic loss in New Jersey is not as great as in Michigan, but it is substantial.

#### Symptoms on Natural and Experimental Hosts

On highbush blueberry, there are several symptoms. The most prominent symptom consists of elongated (0.2 × 1.2 cm) reddish streaks on current year and 1-yr-old stems (fig. 119), especially on the side exposed to the sun. At blossom time, some petals will exhibit red streaks (fig. 120). Affected leaves are straplike (shoestring symptom, fig. 121), curled, or crescent shaped. Many leaves on a bush may be found with this symptom, or it may show on only as few as one or two shoots near the crown. A few leaves may show red vein banding or reddish streaking along the midrib and, on occasion, oak leaf patterns. Immature berries on infected bushes may develop a premature reddish-purple cast (fig. 122). Shoestring disease has been observed in highbush cvs. 'Blue-ray', 'Burlington', 'Coville', 'Earliblue', 'Jersey', 'June', 'Rancocas', 'Rubel', and 'Weymouth'. Cvs. 'Blue-crop' and 'Atlantic' possess field immunity. Yield of infected bushes is greatly decreased. Bushes become progressively diseased along a row. Missing bushes in such a pattern are typical of shoestring infection (fig. 123). Healthy bushes replanted in a field with disease show symptoms after about 4 yrs.

There are no known herbaceous hosts (Lesney et al. 1978).



Figure 119.—A current-year stem of cv. 'Jersey' showing elongated reddish streaks typically caused by shoestring disease.



Figure 120.—Blossom streaking symptoms (arrow) often associated with shoestring disease.





Figure 121.—Leaf-strapping symptom on cv. 'Jersey' caused by shoestring disease.



Figure 122.—Red or purple fruit coloration symptom associated with shoestring infection.

### Natural and Experimental Transmission

**Natural transmission:** Natural transmission in highbush blueberry is by the blueberry aphid, *Illinoia pepperi* (MacGillivray) (D. C. Ramsdell, unpublished data). Spread in the field is from bush to bush and is not a random phenomenon according to mathematical analysis of spread (Lesney et al. 1978). Nematode vectors (*Xiphinema* spp.) are not associated with the disease, and pollen does not contain the virus (D.C. Ramsdell, unpublished data; Lesney et al. 1978). The initial mode of spread is through infected, vegetatively propagated planting stock. After the stock is planted, transmission from infected bushes is mediated by the blueberry aphid.

**Experimental transmission:** Only blueberry seedlings and young, lush, vegetatively propagated woody cuttings will become infected when rub-inoculated with purified virus. Rub inoculation of lush blueberry seedlings or rooted cuttings of a susceptible cultivar, for example, 'Jersey', with purified virus will result in transmission and disease symptoms within 5 or 6 mo (Lesney et al. 1978). Chip budding and whip grafting from diseased plants to healthy small 'Jersey' bushes will produce symptoms within 1 yr (Varney 1957).

### Properties of the Causal Agent

Blueberry shoestring virus (BBSSV) is not a member of any recognized virus group. It is a single component, isometric virus with a diameter of 27 nm (Ramsdell and Stace-Smith 1979b; Ramsdell 1979). The virus sediments at 120 S and contains single-stranded RNA that makes up 20% of the molecular weight of the virion. Molecular weights of the RNA and protein subunit are  $1.45 \times 10^6$  and 30,000 daltons, respectively. In ultrathin sections of infected leaf tissue, viruslike particles were found in epidermal, palisade, and mesophyll cells (Hartmann et al. 1973). Particles were found in xylem, but not in phloem cells. Epidermal leaf cells and root xylem cells contained crystalline arrays of particles.

### Detection and Identification

Although BBSSV is a good immunogen and antisera are available, agar gel diffusion tests of triturated, infected blueberry tissue do not detect the virus. However, enzyme-linked immunosorbent assay (ELISA) (Ramsdell and Stace-Smith 1979b) will readily detect the virus in symptomless tissue. Blossom tissue possesses the highest virus titer for the purpose of detection, but young leaf tissue works well also. Bushes should be sampled thoroughly, that is, a half-dozen samples taken from various locations on a bush. The virus is unequally distributed in infected bushes (D. C. Ramsdell, unpublished data). The time of year for sampling is not critical, as long as sampling is thorough.

### Control Procedures

Roguing by itself has not proven to be a successful means of control. A combination of thorough roguing of symptom-bearing bushes, followed by a rigorous insecticide-based aphid control program using an airblast ground sprayer,



Figure 123.—Typical pattern of shoestring spread in the field. The disease progresses from bush to bush along the row. (Note missing bushes below arrow).

appears to be the best method to halt significant further spread in a field showing disease. If a field is sufficiently diseased so as to be uneconomical, complete removal of bushes followed by replanting with an immune cultivar, for example, 'Bluecrop', would be the best strategy; however, if susceptible cultivars must be grown, then a thorough aphid vector control program would be necessary.

No information has been developed for thermotherapy.



## Leafhopper-Borne Diseases

### Blueberry Stunt<sup>245</sup>

By D. C. Ramsdell and A. W. Stretch

#### Additional Common Names

None

#### History and Geographic Distribution

The viruslike nature of stunt was first described by Wilcox (1942). Stunt was first observed in New Jersey, but is now known to exist in eastern Canada, Maine, Massachusetts, New Hampshire, New York, Michigan, North Carolina, Pennsylvania, Maryland, Virginia, and most recently in Arkansas (Gocio and Dale 1982).

#### Economic Importance

Stunt can cause severe yield reduction in the most susceptible cultivars. In northern areas with cold winters, for example, Michigan, the disease is not present in epidemic proportions.

The disease is relatively more serious in New Jersey. In these two States, roguing diseased bushes and insecticidal control of the sharp-nosed leafhopper *Scaphytopius magdalensis* (Prov.) have been effective control strategies.

#### Symptoms on Natural and Experimental Hosts

All cultivars of highbush blueberries are susceptible. 'Rancocas' is the only cultivar with a high degree of resistance. Stunt occurs naturally in *Vaccinium vacillans* Torr., *V. atrococcum* Helbr., *V. stamineum* L., and *V. myrtilloides* Michx. Symptoms have been observed on graft-inoculated *V. amoenum* Ait., *V. altomontanum* Ashe, and *V. elliotii* Chap. (Hutchinson et al. 1960), and *V. ashei* Reade (Dale and Mainland 1981; R. M. Milholland, unpublished data).

One plant of *V. darrowi* Camp has been successfully infected using dodder (M. T. Hutchinson, unpublished data), and periwinkle (*Catharanthus roseus* (L.) G. Don) has also been infected by means of dodder.

**Symptoms on highbush blueberry.** Overall dwarfing of the bush is a primary symptom, hence, the name stunt (figs. 124 and 125). Downward leaf cupping and puckering is a



Fig. 124.—Overall stunting effect due to stunt disease on a bush of 'Wolcott' cv. The branch in the right center showing twiggy growth is infected. The branch above it

in the upper center is healthy. (Courtesy R. D. Milholland, North Carolina State University.)



characteristic symptom along with a reduction in leaf size (fig. 126). Leaves on infected bushes are often chlorotic, with chlorosis most pronounced along the leaf margins and between lateral veins. Midribs and lateral veins usually retain normal green coloration. Chlorotic areas often turn a brilliant red in the late summer. Stem internodes become shortened, and growth of normally dormant buds causes twiggy branching.

**Symptoms on rabbiteye blueberry.** On the cv. 'Garden Blue', the disease in later stages of development is characterized by a slight reduction in leaf size and internode length and marginal chlorosis, but no leaf cupping (Dale and Mainland 1981).

**Symptoms on wild *Vaccinium* spp.** Symptoms on wild species of *Vaccinium* are generally like those on highbush cultivars.

**Symptoms on *Catharanthus*.** Leaves of infected *C. roseus* are pale green, marked with patches or transverse bands of dark green. Leaf size is not markedly reduced and virescence does not occur, but flowers are smaller and fewer than on healthy plants.

#### Natural and Experimental Transmissions

**Natural transmission:** To date, the only known vector is the sharp-nosed leafhopper *Scaphytopius magdalensis* (Hutchinson 1955; Maramorosch 1955). Apparently, the vector is present in all blueberry growing areas where stunt is present. It has recently been found in the relatively new Arkansas growing area (Dale and Moore 1978).

**Experimental transmission:** The causal organism is easily graft transmitted. Plants grafted at bud break may show symptoms in 2 to 3 mo. If grafts are made later in the season, symptoms may be delayed until the following year. Kunkel in 1947 (Tomlinson et al. 1950) was the first to use dodder (probably *Cuscuta campestris* Yunck.) to transmit the causal organism to *C. roseus*. *Cuscuta subinclusa* Dur. and Hilg. has also been used successfully.

#### Properties of the Causal Agent

Ultrathin sections made from stunt-infected cv. 'Jersey' bud and leaf tissue revealed mycoplasma-like organisms (MLO) in sieve elements (Chen 1971). The MLO ranged from spherical to oval or had irregular morphology. The diameter ranged from 160 to 700 nm (fig. 127). No MLO were found in healthy tissue. Ultrathin sections of diseased tissue from 'Collins' and 'Concord' cultivars revealed similar MLO pleomorphic bodies and what appeared to be crystalline inclusion bodies (Hartmann et al. 1972).

#### Detection and Identification

The most characteristic symptoms of the disease consist of chlorotic leaf margins and interveinal areas of the leaf. Leaf area is reduced and leaves are cupped downward. General stunting of infected bushes and twiggyiness (a proliferation of



Fig. 125.—Stunting effect and “twiggyiness” (stem proliferation) on a stunt-infected cv. 'Jersey' bush.



Fig. 126.—Leaf yellowing and cupping of leaves typical of stunt infection.

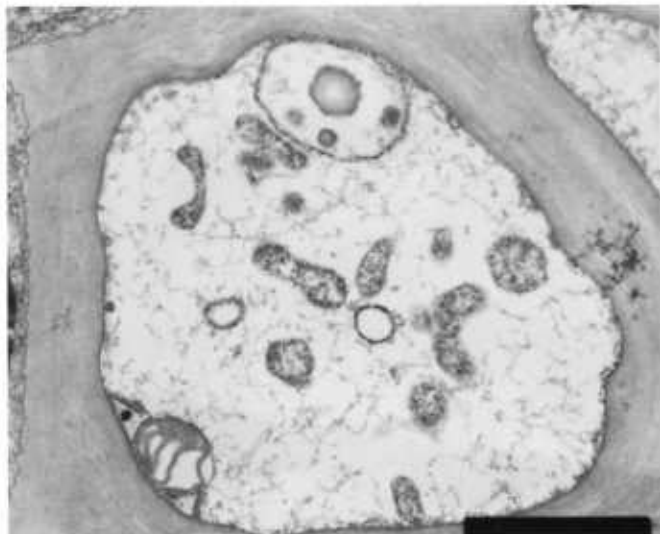


Fig. 127.—Mycoplasma-like organism in phloem sieve element from infected blueberry petiole tissue. Bar represents 1000 nm. (Courtesy J. X. Hartmann.)

twigs) are all part of the symptomatology. If symptoms are not definite, grafting onto cvs. 'Cabot' or 'Jersey' may result in stronger symptoms. Alternatively, aniline blue stained freehand sections viewed under UV-fluorescence light microscopy have been reported as a possible method of detection (Gocio and Dale 1982). One-way ELISA tests with an antiserum made to *Spiroplasma citri* failed to detect the causal organism in stunt infected blueberry leaf sap (Converse and Ramsdell 1982).

### Control Procedures

In Michigan and New Jersey, a combination of field inspections, roguing of infected plants and a diligent insecticidal spray program to control leafhoppers has been effective in controlling stunt. In warmer climates, such as in North Carolina and Arkansas, control of disease spread may be more difficult. Strict inspection of source bushes is necessary before any propagating wood is taken. Thermotherapy would probably be effective because of the MLO etiology of stunt.

### Witches'-Broom of *Vaccinium*<sub>d</sub>

by G. T. N. de Leeuw

### Additional Common Names

Blueberry little leaf, heksenbezemziekte van de bosbes; Hexenbesenkrankheit der Heidelbeere; Kleinblattrigkeit der Heidelbeere; metlovitost borůvky, malolitost borůvky.

### History and Geographic Distribution

Blattný and Starý (1940) described witches'-broom in *Vaccinium myrtillus* L. as a virus disease. More recent work, however, strongly indicates that some diseases of *Vaccinium* spp., formerly ascribed to virus infections, are actually caused by mycoplasma-like organisms (MLO).

Witches'-broom has been found in The Netherlands, Germany, Czechoslovakia, Yugoslavia, Scotland, and France. The disease occurs in lowlands and on slopes of hills and mountains, most frequently, in dry locations in Scotch pine forests. The distribution depends on a suitable environment for the leafhopper vectors.

### Economic Importance

According to Blattný and Blattný (1970), Witches'-broom disease is of great economic importance in Czechoslovakia. Many thousands of tons of *V. myrtillus* berries are harvested annually, with a large proportion for the export market. In some areas losses may exceed 15% of the crop. Losses in *V. vitis-idaea* L., *V. uliginosum* L., and *Vaccinium oxycoccus* L. are negligible.

### Symptoms in Natural and Experimental Hosts

Infected *V. myrtillus* plants show a very dense, bushy growth. This is due to the erect position of the excessively formed new branches. The excessive ramification of the plants is associated with a striking reduction in size of branches and leaves. The leaves may have a length of 4 mm or even less, instead of the usual 15 to 20 mm (fig. 128). Branches of plants affected in a later stage of growth have only an erect position instead of the plagiotropic position on normal, healthy plants. Plants affected earlier in their development remain small, are heavily branched and have smaller leaves. Young plants at the edge of a diseased group of plants remain extremely small (about 5 cm), have very small leaves, and do not form subterranean suckers (fig. 129). Diseased plants drop their leaves later in autumn than healthy plants, which may lead to frost damage. Sometimes the leaves show some reddening due to an increased anthocyanin formation. Diseased plants do not flower at all. (Bos 1960).

Blattný and Blattný (1970) distinguished two types of witches'-broom in *V. myrtillus*. Type A symptoms include severe vertical branching and brooming; severe stunting, with plants reaching only 2 cm in height, leaves only 1.5 mm long rather than the normal 15 to 20 mm; frequent reddening of the leaves; partial drying and dieback; and sterility, with the exception of a few small flowers and berries on mildly affected plants. Type B symptoms include less vertical branching; less stunting, with plants reaching 10 cm; leaves 6 mm long; light-green leaves because of paler intercostal tissues; a light-pink color rather than a reddening of the leaves; little drying up and no death of plants; and a few flowers and berries smaller than normal.

In localities where type A symptoms occur, plants are not affected by the disease characterized by type B symptoms, and the reverse holds true where type B is found. Type A symptoms are prevalent in Czechoslovakia, West Germany, The Netherlands, and East Germany. Type B occurs in Czechoslovakia, East Germany, and Yugoslavia. *V. vitis-idaea*, *V. uliginosum*, and *V. oxycoccus* show similar symptoms, that is, upright growth of shoots, brooming, stunting, reduced leaf size, leaf reddening, and sterility.





Fig. 128.—Twigs of a healthy (left) and witches'-broom diseased *V. myrtillus* plant (right).



Fig. 129.—Extremely small plants from the edge of a group of *V. myrtillus* plants affected by witches'-broom.

### Natural and Experimental Transmission

Blattný (1963) proved that the leafhopper *Idiodonus cruentatus* Panz. can transmit the disease to *V. myrtillus*; however, the disease also occurs abundantly where *I. cruentatus* has not been found. Probably other leafhoppers are vectors as well. Experiments have indicated that the leafhoppers *Empoasca solani* (Curt.), *Neophilaenus exclamationis* Thumb., *Aphrodes bicincta* (Schrank), *Euscelis* ssp., and *Macropsis fuscus* (Zett.) do not transmit the disease.

Warm periods in summer, autumn, and in dry springs are favorable for leafhoppers. The disease increases after such weather conditions. Excessive tree felling in the forest also supports the occurrence of leafhoppers and causes an increase in the number of diseased plants.

Witches'-broom in *V. myrtillus* may also be transmitted by grafting (Bos 1960; Uschdraweit 1961; Blattný and Blattný 1970), or by implantation of bast (phloem) tissue into stems (Blattný and Stařý 1940). The incubation times vary according to the season in which grafts are made and to environmental conditions.

Attempts to transmit the disease by means of *Cuscuta campestris* Yuncker, *C. epithymum* Murray, and *C. subinclusa* Dur. and Hilg. were unsuccessful. Up to now, 1981, no disease has been found in seedlings obtained from the very few viable seeds from lightly diseased plants.

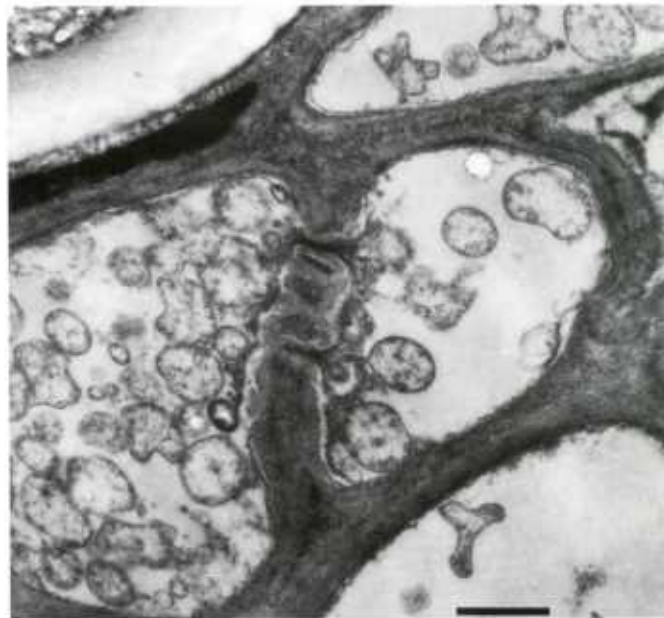


Fig. 130.—Electron micrograph of mycoplasma-like organisms in sieve tubes of witches'-broom diseased *V. myrtillus* plants. Bar represents 500 nm.

### Properties of the Causal Agent

Kegler et al. (1973), Blattný and Vána (1974), and de Leeuw (1975) detected mycoplasma-like organisms (MLO) in the sieve tubes of *V. myrtillus* plants affected by witches'-broom (fig. 130). The consistent association of MLO with witches'-broom symptoms and the absence of these organisms in the sieve tubes of healthy plants make it very likely that MLO are the causal agents of this disease. Whether these organisms are related to the etiologic agents of blueberry stunt and cranberry false blossom has yet to be investigated. (See "Blueberry Stunt," p. 106, and "Cranberry False Blossom," p. 110.)

### Detection and Identification

The disease may be detected by graft or vector transmission to *V. myrtillus*, *V. vitis-idaea*, *V. uliginosum*, or *V. oxycoccus*. The presence of MLO in the sieve tubes of suspected plants can be detected with the aid of fluorescence microscopy or electron microscopy.

### Control Procedures

The eradication of diseased plants as soon as they are recognized is desirable. Prevention of leafhopper migration may reduce the extent and distribution of the disease. Control of vectors is particularly important where it is necessary to prevent the spread of the disease from wild hosts to plantations of cultivated *V. corymbosum* L. There are no studies on the therapy of this disease.

### Remarks

Early evidence of witches'-broom in *V. myrtillus* was found in Czechoslovakia in herbarium species dated 1925 and 1926. *V. myrtillus* f. *parvifolium* Domin (f. *microphyllum* auct.), characterized by very minute leaves; as well as *V.*



*myrtillus* f. *erectum* Otruba with erect twigs; and *V. myrtillus* f. *pygmaeum* Ostenf. are probably affected by witches'-broom (Blattný and Blattný 1970). When grown under unsuitable conditions, *V. myrtillus* and *V. vitis-idaea* can be dwarfed and infertile. By the absence of the vertical growth and brooming, such plants can be distinguished from plants with witches'-broom disease.

Witches'-broom, blueberry stunt, and cranberry false blossom cause similar symptoms in their respective hosts, but are transmitted by different leafhoppers. A strain relationship between these MLO is possible but has not yet been demonstrated.

## **Cranberry False Blossom //**

By A. W. Stretch

### **Additional Common Names**

Wisconsin false blossom.

### **History and Geographic Distribution**

Shear (1908) first described and named the disease. Its transmissibility and mode of transmission were established by Dobrosky (1929). Chen (1971) determined that false blossom was associated with mycoplasma-like organisms (MLO) rather than virus-like particles as originally surmised. False blossom appears to be indigenous to Wisconsin. The disease was probably distributed from Wisconsin to other U. S. cranberry growing areas in diseased vines (Stevens 1931). The disease is now found in Massachusetts, New Jersey, New York (including Long Island), Oregon, Washington, and Nova Scotia.

### **Economic Importance**

In the early 1900's, this disease caused serious losses which reached a peak in the 1920's and early 1930's. Since that period, control of the vector has reduced the spread to a point where losses are small. Large-scale planting of resistant cultivars has reduced its economic impact.

### **Symptoms on Natural and Experimental Hosts**

False blossom disease is known to occur only on American cranberry (*Vaccinium macrocarpon* Ait.) and European cranberry (*Vaccinium oxycoccus* L.). Kunkel (1945) was able to transmit false blossom through dodder (*Cuscuta campestris* Yunck.) to 28 species of plants in 10 different families. In nature, the host range appears limited by vector feeding preferences and natural resistance.

**Symptoms in cranberry.** False blossom is most easily recognized at bloom when the flowers on infected plants assume an upright position because the pedicels are straight (fig. 131), rather than arched, as on a normal plant (fig. 131). The calyx lobes of diseased flowers become enlarged, the petals are short and streaked with red and green, and the stamens and pistils are abnormal, usually resulting in a sterile

flower. Normally latent axillary buds are also stimulated and give rise to branches with a witches'-broom effect (fig. 132). The leaves on these branches are closely appressed to the stem. In autumn, they take on a reddish hue before normal autumn coloration develops. Terminal flower buds are enlarged, in an advanced stage of development, and very susceptible to spring frost injury, since they are protected by only one layer of scale leaves as compared with four layers in a normal flower bud. Symptoms in complex with ringspot virus have not been determined. (See "Ringspot of Cranberry" p. 123).

**Symptoms on experimental hosts.** The false blossom MLO caused all of the 28 species artificially inoculated through the use of dodder (Kunkel 1945) to become chlorotic, to assume a more upright habit of growth than is normal, and to produce marked effects of flowering and fruiting organs. At present, no information is available on suitable indicator hosts.

### **Natural and Experimental Transmission**

**Natural Transmission:** Dobrosky (1929) proved that the blunt-nosed leafhopper *Scleroacus vaccinii* (Van Duzee), also known as *Euscelis striatulus* Dobrosky (nee Fallén), was a vector of false blossom disease. No other vectors have been implicated. The shortest time from feeding of the leafhopper vector to symptom development on cranberry was 30 days, and the longest was a year or more (Dobrosky 1931).

**Experimental Transmission:** False blossom has been transmitted from cranberry to periwinkle (*Catharanthus roseus* (L.) G. Don, tomato (*Lycopersicum esculentum* Mill.), and other plant species, using dodder (*Cuscuta campestris*) (Kunkel 1945). Graft transmission from cranberry to cranberry has not proven useful because of the difficulty of grafting thin-stemmed cranberry vines. Kunkel (1945) was able to graft transmit the MLO into periwinkle, tomato, and other herbaceous plants.

### **Properties of the Causal Agent**

The MLO described by Chen (1971) have not been grown in pure culture and then reintroduced into cranberry to prove a positive causal association with diseased plants. The MLO were found in the sieve-tube elements, and their shapes ranged from spherical to oval to irregular. Each body was surrounded by a single unit membrane. The size range of these MLO was 80 to 300 nm (fig. 133).

### **Detection and Identification**

The disease is detected most successfully at the bloom stage when the normal arching of the flower pedicel is replaced by an upright habit of growth. Witches'-brooming and upright growth above the level of normal vines make diseased plants stand out. Identification is based on symptoms in cranberry. Electron microscopy of diseased tissue can detect MLO, but cannot distinguish among them. Development of a cultural method for the MLO and production of specific antisera should provide a means for positive serological identification of the false blossom causal organism.



Fig. 131. — Cranberry false blossom symptoms on cranberry. Diseased uprights with abnormal flowers and straight pedicels (right). Healthy uprights with normal flowers and arched pedicels (left). (Courtesy D. M. Boone.)

### Control procedures

Chemical control of the leafhopper with parathion applied at the dangle stage before bloom is the primary method of control. Followup sprays 10 to 14 days and 4 wk after midbloom with parathion or azinphosmethyl are also needed for leafhopper control. The growing of cultivars that are less attractive as food plants than cultivars like 'Howes' has been very important in control. 'Shaw's Success' is the most resistant cultivar, followed in descending order by 'McFarlin' and 'Early Black'. 'Howes' is the most susceptible. Flooding a bog in June — just before the flower buds open and after the leafhopper eggs have hatched — has proved effective, but timing is critical and vine damage is a distinct possibility. No information is available on therapy of this disease.



Fig. 132. — Witches'-brooming associated with cranberry false blossom (right) compared with normal upright production (left). (Courtesy D. M. Boone.)

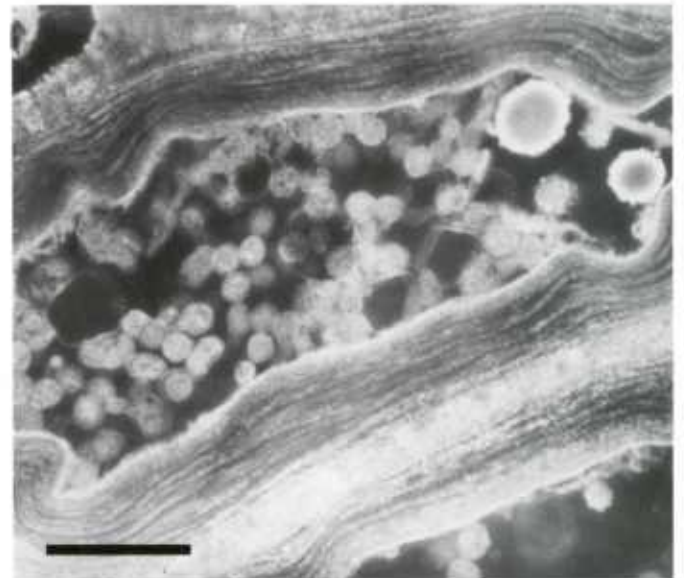


Fig. 133. — Electron micrograph of mycoplasma-like organisms in sieve tubes of false blossom diseased cranberry leaves. Bar represents 500 nm. (Courtesy T. A. Chen.)



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## Blueberry Leaf Mottle

By D. C. Ramsdell

### Additional Common Names

None.

### History and Geographic Distribution

The disease was observed for the first time in 1977 in a 4-ha planting of mature cv. 'Rubel' bushes near Hartford, Mich. The bushes exhibited a general decline and dieback condition and leaf mottling. The disease has been found in cv. 'Jersey' bushes in a few other plantations. Symptoms consisted of some bush stunting and smaller leaves, but leaf deformation and pronounced mottling were absent. The symptomatology of this disease is different from that of other virus-caused diseases of blueberry. As of this writing, the disease has been diagnosed and serologically confirmed in a total of five fields in southwestern and western central Michigan. It has not been reported from other blueberry growing areas. A serologically distantly related virus, grapevine Bulgarian latent virus, has been isolated from *Vitis vinifera* L. grapevines near Pleven, Bulgaria (Martelli et al. 1977, 1978; Ramsdell and Stace-Smith 1979a). A virus serologically closely related to BBLMV (but reported as a strain of grapevine Bulgarian latent virus) (Uyemoto et al. 1977) has also been isolated from a single *Vitis labrusca* L. 'Concord' grapevine in New York State.

### Economic Importance

Bushes infected with blueberry leaf mottle virus (BBLMV) are stunted and very unproductive. Bushes that have been infected for several years die, probably due to winter injury as a result of their weakened condition.

### Symptoms on Natural and Experimental Hosts

BBLMV has a fairly narrow host range (Ramsdell and Stace-Smith 1979a). Mostly herbaceous indicators in the genus *Chenopodium* show symptoms. *Cucumis sativus* L. 'Straight Eight' may show chlorotic local lesions on inoculated cotyledons. *Nicotiana clevelandii* Gray shows systemic pinpoint necrotic local lesions 1 to 2 mm in diameter in new leaves.

**Symptoms on highbush blueberry.** Cv. 'Rubel' shows the most striking symptoms. Bushes that have been infected for several years develop a severe dieback of older stems, leaving stunted, deformed new growth coming from the crown area (fig. 134). Leaves show a mottling pattern and sometimes chlorotic roughly circular "windows" (fig. 135). In the most severe cases, leaf malformations such as leaf strapping and curling can occur. Productivity is nil. Healthy



Figure 134. — A cv. 'Rubel' blueberry bush showing effect of stem dieback and stunted regrowth as a result of blueberry leaf mottle disease.

seedlings of the cv. 'Rubel', if rub-inoculated with purified virus, will show a leaf mottling within several months after inoculation (fig. 136) (Ramsdell and Stace-Smith 1979a). Cv. 'Jersey' exhibits milder symptoms. Stem dieback is not very prevalent. There is some stunting and growth reduction present. Leaves are somewhat smaller, and slight leaf mottling of lower leaves occurs in the crown area. Productivity is greatly reduced.

The pattern of spread appears to be random in affected fields. Attempts to transmit the virus using *Xiphinema americanum* Cobb have given negative results (T. C. Vrain, unpublished data, and J. M. McGuire, unpublished data). Pollen grains were extremely high in virus content in 13 out of 15 symptom-bearing bushes sampled, according to ELISA tests (D. C. Ramsdell, unpublished data). Disease spread is probably mediated by honey bees, which are an integral part of highbush blueberry culture.

### Symptoms on Indicator Hosts

The following herbaceous indicators are useful:

*Chenopodium quinoa* Willd.

Chlorotic local lesions, mottle, and apical death within 7 to 10 days.



Figure 135. — Leaf mottling and chlorotic “windows” in leaves from a cv. ‘Rubel’ blueberry bush with blueberry leaf mottle disease.

*Chenopodium amaranticolor* Coste and Reyn.

Systemic mottle within 10 to 14 days.

*Nicotiana clevelandii* Gray

Pinpoint necrotic local lesions, 1 to 2 mm in diameter, on noninoculated leaves within 14 to 21 days.

#### Natural and Experimental Transmission

Natural Transmission: Unknown. Although BBLMV possesses physical and chemical properties of a nepovirus, similar to tomato ringspot and cherry leaf roll viruses (Harrison and Murrant 1979; Ramsdell and Stace-Smith 1981), the suspected nematode vector, *Xiphinema americanum*, is not associated with the disease (D. C. Ramsdell, unpublished data). The pattern of spread in the field is random rather than circular, the latter being typical of a nematode-mediated pattern of spread. The fact that pollen has been found to contain high levels of virus indicates that natural spread may be by pollen. Although not yet demonstrated, the disease is no doubt spread by infected propagating material.

Experimental Transmission: Bud and graft transmission tests have not been done. Sap transmission from infected



Figure 136. — A cv. ‘Rubel’ leaf showing mottling symptoms a few months after a healthy seedling was rub-inoculated with purified blueberry leaf mottle virus.

blueberry to herbaceous host plants is easily done by grinding young terminal leaf tissue in 0.05 M Tris-HCl buffer pH 7.0, with 2% (v/v) nicotine alkaloid added. A mortar and pestle works well for grinding the tissue.

#### Properties of the Causal Agent

BBLMV is a putative member of the nepovirus group (Ramsdell and Stace-Smith 1981). It has three types of isometric particles with sedimentation coefficients of 53, 120, and 128 S. The virions contain two pieces of single-stranded RNA with molecular weights of 2.15 and  $2.35 \times 10^6$  daltons, respectively. The protein coat subunit has a molecular weight of 54,000 daltons. BBLMV is serologically distantly related to grapevine Bulgarian latent virus. The virus has a narrow host range, causing known diseases in blueberry and grape only.

#### Detection and Identification

Visual inspection will give a strong indication that the disease is caused by BBLMV, but sap transmission to the aforementioned herbaceous indicators is useful for detection of BBLMV. Final confirmatory results using such serological tests as agar gel double diffusion are necessary. BBLMV is a



good immunogen. Antisera with titers of 1:1024 are easily obtained. Instead of using herbaceous indicators, ELISA tests made directly with young infected blueberry leaf tissue or blossoms works well (D. C. Ramsdell, unpublished data).

### Control Procedures

Until the mode of spread is proved, all that can be done is to inspect fields visually for infected bushes and then rogue them. If the disease is indeed pollen spread, the effect of honey bees upon spread will need to be scrutinized carefully. No information is available on thermotherapy.

### Necrotic Ringspot of Blueberry

By D. C. Ramsdell

### Additional Common Names

Pemberton disease.

### History and Geographic Distribution

The disease was first discovered in a commercial field in New Jersey and was brought to the attention of researchers in 1955. Varney and Raniere (1960) demonstrated its virus or viruslike etiology. Lister et al. (1963) demonstrated the association between tobacco ringspot virus (TRSV) and necrotic ringspot disease. The disease occurs in Connecticut, Illinois, Michigan, and New Jersey. It has recently been reported in Arkansas (McGuire and Wickizer 1979) and in Oregon (Converse and Ramsdell 1982).

### Economic Importance

Necrotic ringspot disease causes a slow, but steady decline in bush productivity in susceptible cultivars, for example, 'Pemberton', 'Stanley', 'Rubel', 'Concord', and 'Collins'. In some cases, bush death occurs, especially in Northern States such as Michigan which have extremely cold winters. Until recently, cv. 'Jersey' was thought to be resistant to necrotic ringspot, but a severe strain of TRSV was found to be associated with a decline disease of 'Jersey' (Ramsdell 1978).

### Symptoms on Natural and Experimental Hosts

TRSV, the causal agent, has a broad host range, causing diseases in both herbaceous and woody plants (Stace-Smith 1970b). TRSV isolates causing necrotic ringspot, found by Lister et al. (1963), infected the following herbaceous hosts as a result of mechanical inoculation: *Nicotiana tabacum* L., *N. rustica* L., *N. clevelandii* Gray, *Petunia hybrida* Vilm., *Datura stramonium* L., *Phaseolus vulgaris* L., *Cucumis sativus* L., *Chenopodium amaranticolor* Coste and Reyn., and *C. quinoa* Willd. The TRSV isolate associated with the decline of cv. 'Jersey' was found to cause more severe and rapid necrosis in herbaceous hosts than standard necrotic ringspot isolates of TRSV (Ramsdell 1978).

**Symptoms on highbush blueberry.** Cultivars that are susceptible, for example 'Pemberton', exhibit stem dieback



Figure 137. — Cv. 'Pemberton' showing leaf deformation symptoms (arrow) due to necrotic ringspot disease caused by tobacco ringspot virus.

and stunting. Leaves are deformed and somewhat thickened (fig. 137). Leaves become chlorotic and show necrotic spots. Some of these may drop out giving a shot-hole or tattered effect (fig. 138). On other susceptible cultivars, for example, 'Concord' and 'Stanley', symptoms are expressed as short internodes and small straplike leaves (fig. 139).

### Symptoms on Indicator Hosts

The following herbaceous indicator hosts and their reactions to TRSV are useful for preliminary identification of the causal virus, but serological confirmatory tests are necessary: *Chenopodium quinoa* Willd.

Necrotic lesions on inoculated leaves; apical dieback within 6 to 7 days.

*Cucumis sativus* L. cv. 'National Pickling'

Chlorotic lesions 1 to 3 mm in diameter on inoculated cotyledons, followed by systemic chlorosis and necrotic lesions on new leaves.

*Nicotiana tabacum* L. cv. 'Burley'

Necrotic ringspot on inoculated leaves, mosaic symptoms on new leaves.



Figure 138. — Cv. 'Pemberton' showing leaf necrosis and shot-hole effect (arrow) due to necrotic ringspot disease caused by tobacco ringspot virus.

### Natural and Experimental Transmission

**Natural transmission:** Natural spread is thought to be by *Xiphinema americanum* Cobb. This nematode has been shown to be most consistently associated with the disease in blueberry (Griffin et al. 1963; Tjepkema et al. 1967; Raniere 1964). McGuire (1964) has shown that single nematodes can transmit TRSV from herbaceous to herbaceous hosts. The disease spreads slowly in a roughly circular manner in the field. The disease is also spread through vegetatively propagated hardwood cuttings.

**Experimental transmission:** The disease can be bud or graft transmitted to 'Pemberton' or other susceptible cultivars. TRSV can be successfully sap transmitted from young leaf tissue from terminals of stems or from suckers coming from the crown. Dormant buds are also a good source of tissue for successful transmission to herbaceous indicators (D. C. Ramsdell, unpublished data). Phosphate buffer (0.05 M, pH 7.2) containing 2% (v/v) nicotine alkaloid is a satisfactory buffer for grinding leaf tissue with a mortar and pestle for sap inoculation to the aforementioned herbaceous indicators.



Figure 139. — Cv. 'Stanley' showing shortened terminal growth and small straplike leaves (arrow) due to necrotic ringspot disease caused by tobacco ringspot virus.

### Properties of the Causal Agent

TRSV is a member of the nepovirus group (Harrison and Murant 1977). It is a multicomponent virus with three particles about 28 nm in diameter, sedimenting at 53, 91 and 126 S (Stace-Smith 1970a). The single-stranded RNA is composed of two different molecular weight species,  $1.4 \times 10^6$  and  $2.4 \times 10^6$  daltons, and both are necessary for infection. The protein coat is composed of 42 subunits, each having a molecular weight of about 55,000 daltons. The virus causes disease primarily in woody or semiwoody plants, but also causes some diseases in herbaceous ornamentals and agricultural crop plants. Natural transmission is by the nematode *Xiphinema americanum*.

### Detection and Identification

Susceptible cultivars exhibit disease symptoms that are fairly characteristic. Visual inspection in commercial plantings will give a primary indication that necrotic ringspot disease is present. For more definitive diagnosis, however, sap inoculation to herbaceous indicators and confirmation by serology as previously outlined is the best method for detection. Alternatively, enzyme-linked immunosorbent assay (ELISA) works very well (D C. Ramsdell, unpublished data; Converse and Ramsdell 1982). Bud, blossom, and leaf tissue from shoot terminals is the best for both herbaceous plant and ELISA indexing. Alternatively, bud or graft indexing may be done to detect symptomless infection by budding or grafting onto healthy cultivars which will readily show symptoms, for example, 'Cabot', 'Concord', 'Pemberton', or 'Stanley'.

### Control Procedures

Roguing of diseased bushes, several bushes beyond those that are showing symptoms, is required to remove symptomlessly infected bushes. Soil fumigation with high rates of nematicides 1 yr after bush removal is necessary to halt



further spread of the disease. For new plantings, disease-free stock planted into nematode vector-free soil will prevent the disease.

No information is available concerning thermotherapy of blueberry tissue to rid it of TRSV.

## **Peach Rosette Mosaic Virus in Blueberry //** By D. C. Ramsdell

**Additional Common Names**  
None.

### **History and Geographic Distribution**

This virus occurs only in southwestern Michigan and southeastern Ontario, Canada. The disease has only been observed in blueberries in an experimental planting where blueberries were planted in infested soil in a vineyard site (Ramsdell and Gillett 1981).

**Economic Importance**  
No information.

### **Symptoms on Natural and Experimental Hosts**

Peach rosette mosaic virus (PRMV) has a very narrow experimental and natural host range (Dias 1975). In nature, the virus occurs in woody plants (grape and peach) and in some weed species (Ramsdell and Myers 1978). Sap transmission of the virus by rub inoculation is readily done using *Chenopodium quinoa* Willd.

**Symptoms on blueberry.** Symptoms have been observed only on highbush cvs. 'Jersey' and 'Berkeley'. In cv. 'Jersey', leaves become strap shaped (fig. 140 A) and/or deformed into a crescent shape (fig. 140 B). In cv. 'Berkeley', the leaves are spoon shaped and smaller than normal (fig. 140 C). Symptoms on diseased leaves are not equally distributed over an infected bush. No necrotic or chlorotic lesions have been observed on leaves. These latter two symptoms are peculiar to tobacco and tomato ringspot virus induced diseases, respectively (see these two disease chapters). No twig or fruit symptoms have been observed as a result of PRMV infection.

### **Symptoms on Indicator Hosts**

The only two reliable herbaceous indicator hosts are:  
*Chenopodium quinoa* Willd.

Faint chlorotic local lesions occur in inoculated leaves within 4 to 10 days followed by epinasty and abscission. Uninoculated terminal leaves become mottled and twisted. Death of the terminal growing point usually occurs within about 2 wks.

*Chenopodium amaranticolor* Coste and Reyn.

Faint chlorotic local lesions may or may not occur in inoculated leaves within 4 to 10 days. Uninoculated terminal leaves show mottling, and often death of the terminal growing point will occur after about 2 wks.

### **Natural and Experimental Transmission**

**Natural transmission:** Natural transmission of PRMV is by *Xiphinema americanum* Cobb. Healthy peaches and grapes become infected when planted in infested soils (Cation 1942, 1951). Steam or chlordane treatment of infested soil from peach orchards prevented transmission (Fulton and Cation 1959). Large populations of *X. americanum* are found associated with soils around PRMV-diseased grapevines (Ramsdell and Myers 1974). Healthy *Vitis labrusca* L. cv. 'Concord' and some French hybrid grapevines became infected within 3 yr when planted in soil infested with *X. americanum* (D. C. Ramsdell, unpublished data).

**Experimental transmission:** Hand-picked *X. americanum* transmitted PRMV from *Chenopodium quinoa* to *C. quinoa*, but not to healthy grapes. The percentage of transmission was low and erratic (Dias 1975).

### **Properties of the Causal Agent**

PRMV is a member of the nepovirus group (Harrison and Murant 1977). The following physical and chemical properties have been reported (Dias 1975; Dias and Cation 1976). The virus has three types of isometric particles about 28 nm in diameter, sedimenting at 52, 115, and 135 S. The middle and bottom components have estimated percent RNA values of 37 and 44%, respectively. The single coat polypeptide has a molecular weight of about 55,000 daltons. The virus has a narrow natural host range, being restricted to peach, grape, and some weed species. Shortened internodes and leaf malformation (rosetting in peach) are the main symptoms caused on these woody hosts. Natural transmission is by the nematode vector *X. americanum*.

### **Detection and Identification**

The symptoms described for PRMV-caused disease of blueberry should not be considered diagnostic. Any bushes suspected of having the disease should be tested by grinding a small amount of young terminal leaf tissue in 0.05 M phosphate buffer pH 7.2 containing 2% (v/v) of nicotine alkaloid, and rub-inoculating carborundum-dusted *C. quinoa* and *C. amaranticolor* plants. Symptom-bearing herbaceous tissue should be tested serologically to confirm the presence of PRMV. Alternatively, ELISA tests can be run directly on suspected infected blueberry tissue (Ramsdell and Gillett 1981; Ramsdell et al. 1979).

### **Control Procedures**

If PRMV is found infecting a blueberry field, suspect bushes should be tested by the previously mentioned methods to determine the extent of infection. Infected bushes should then be removed. Root pieces should be thoroughly removed also. After a season has passed and the soil has been well worked, preplant soil fumigation should be done using nematicides at high rates to kill vector nematodes thoroughly. After sufficient aeration time, new plants free of PRMV could be replanted in the area. No information is available on thermotherapy of PRMV.



Figure 140.—Symptoms of peach rosette mosaic virus infection in highbush blueberry: A, Leaf strapping; B, malformation in cv. 'Jersey'; and C, spoon-shaped leaves on terminal growth of cv. 'Jersey'.



## Tomato Ringspot Virus in Blueberry //

By D. C. Ramsdell

### Additional Common Names

None.

### History and Geographic Distribution

The disease with tomato ringspot virus (TomRSV) was reported for the first time in 1972 in a field near Mossyrock, Wash. (Johnson 1972). It was more recently reported from Oregon (Converse and Ramsdell 1982). There has been no other report of TomRSV-associated disease in blueberry.

### Economic Importance

No quantitative data exist for deleterious growth or yield effects due to TomRSV infection of blueberries.

### Symptoms on Natural and Experimental Hosts

TomRSV has a wide experimental and natural host range; species in more than 35 dicotyledonous and monocotyledonous families are susceptible (Stace-Smith 1970b).

**Symptoms in blueberry.** The blueberry bushes reported infected by Johnson (1972) in Washington exhibited the following symptoms: Leaves were in some cases malformed and exhibited roughly circular chlorotic spots (2 to 5 mm in diameter); in addition, stems, twigs and branches exhibited circular, brownish necrotic spots of similar size (figs. 141 and 142). Younger terminal leaves exhibited a tendency toward leaf-strapping and a mottle pattern (fig. 143) (observed by the author; not part of Johnson's 1972 description). Bud grafts made in summer from infected blueberries to plants of the red raspberry cv. 'Puyallup' produced ringspot and oak leaf patterns in the foliage the following spring (Johnson 1972).

### Symptoms on Indicator Hosts

See the listing of useful herbaceous indicator hosts in the Rubus disease section, "Tomato Ringspot Virus in *Rubus*," p. 223.

### Natural and Experimental Transmission

**Natural transmission:** Natural transmission is thought to be via the dagger nematode *Xiphinema americanum* Cobb. High populations were associated with two of the three blueberry fields where TomRSV was found associated with the disease in Oregon (Converse and Ramsdell 1982). Infected planting stock is also undoubtedly another source of spread.

**Experimental transmission:** The red raspberry cv. 'Puyallup' was successfully bud-inoculated from a TomRSV-diseased source bush. The following season the leaves showed ringspots and oak leaf patterns (Johnson 1972). The virus was also successfully sap transmitted from young diseased blueberry leaves to *Nicotiana tabacum* L., *Chenopodium quinoa* Willd., and *Cucumis sativus* L. (Johnson





Figure 141.—Cv. 'Earliblue' infected with tomato ringspot virus. Note circular chlorotic lesions on the leaves and necrotic circular lesions on stems.



Figure 142.—Leaves of cv. 'Earliblue' showing a closeup view of chlorotic circular lesions associated with tomato ringspot virus infection.

1972). The causal virus has been successfully transmitted using herbaceous plants (Téliz et al. 1966), but it has not been transmitted using blueberry plants.

#### Properties of the Causal Agent

See the *Rubus* disease section, "Tomato Ringspot Virus in *Rubus*," p. 223.

#### Detection and Identification

The most practical and quickest way to detect TomRSV from blueberry is to perform enzyme-linked immunosorbent assay (ELISA) tests on suspected diseased buds or young leaf tissue (Converse and Ramsdell 1982). Alternately, diseased bud or young leaf tissue can be ground in a small amount of 0.05 M phosphate buffer containing 2% nicotine alkaloid (v/v) and rub-inoculated onto carborundum-dusted *Chenopodium quinoa*, *Cucumis sativus* cv. 'National Pickling', and *Nicotiana tabacum* cv. 'Burley' indicator plants. Sap expressed from symptom-bearing tissue can then be tested against TomRSV antiserum in agar gel diffusion plates for confirmation that TomRSV is present.

#### Control Procedures

Roguing of diseased bushes several bushes beyond those that are showing symptoms is a necessary measure to remove symptomlessly infected bushes. Soil fumigation with high rates of nematicides 1 yr after bush removal is necessary to halt further spread of the disease. For new plantings, disease-free stock planted into nematode vector-free soil will prevent the disease. No information is available concerning thermotherapy of blueberry tissue to rid it of TomRSV.



Figure 143.—Young leaves of cv. 'Earliblue' showing leaf-rolling and mottling symptom associated with tomato ringspot infection.

## Vectors Unknown

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### Blueberry Mosaic

By D. C. Ramsdell and A. W. Stretch

### Additional Common Names

Variegation.

### History and Geographic Distribution

The disease was recognized as a variegation and thought to be of genetic origin before it was shown to be of viruslike etiology (Varney 1957). It has been observed in plantings of cultivated blueberries throughout Eastern United States, Michigan, Indiana, Oregon, and in British Columbia, Canada.

The disease has been observed on older cultivars of highbush blueberry, for example, 'Cabot', 'Concord', 'Earliblue', 'Pioneer', 'Rubel', and 'Stanley'. Another different-appearing type of mosaic disease on cv. 'Coville' may be of genetic origin. The disease has been observed occasionally on *Vaccinium vacillans* Torr., which is a lowbush dryland type.

### Economic Importance

No quantitative data are available. Diseased bushes have a noticeable reduction in yield — the fruit is of poor quality and such fruit may ripen late.

### Symptoms on Natural and Experimental Hosts

Symptoms include mild to brilliant mottle and mosaic patterns of chrome yellow, yellow and yellow green. Sometimes, the leaves will also have areas of pink (fig. 144). The distribution of symptoms on a bush is spotty. Symptoms may show on the major portion of a bush or on only one or two stems. Symptoms may be ephemeral, showing in a given year, not showing the next year, and then reappearing a year later. On cv. 'Coville', the mosaic pattern is less brilliant. Rather than a bright yellow mosaic, the pattern is a light green alternating with a deep green (figs. 145 A and B) and may be a genetic disorder.

### Natural and Experimental Transmission

Cvs. 'Herbert', 'Stanley', and 'Burlington' graft-inoculated at bud break showed symptoms on 27, 31, and 51 days, respectively (Raniere 1960). Ten additional cultivars inoculated at the same time failed to develop symptoms during the course of the experiment. Mosaic is not sap or dodder transmissible to herbaceous hosts. There is no known vector, but the disease does spread slowly in the field.

### Properties of the Causal Agent

Attempts at purification of virus particles by several methods using symptomatic blueberry leaf tissue have failed to yield





Figure 144.—Chrome yellow, yellow, and green mosaic patterns on leaves of cv. 'Rubel' blueberry infected with blueberry mosaic.



Figure 145. — A and B, Light- and dark-green mosaic symptoms on leaves of cv. 'Coville', which probably has a genetic disorder that can be confused with regular blueberry mosaic.

any detectable virus particles (D. C. Ramsdell, unpublished data). Electron microscopic examination of ultrathin sections made from symptomatic leaf tissue, petioles, and roots did not yield any definitive virions. Packets of roughly spherical viruslike particles, 28 to 30 nm in diameter, were found, but these could have been ribosomal structures altered as a result of the disease (none were formed in similarly treated healthy tissue) (K. K. Baker and D. C. Ramsdell, unpublished data).

#### Detection and Identification

Symptoms are generally distinct and diagnostic. Confirmatory diagnosis in plants with mild symptoms may be done by graft inoculating 'Stanley', 'Cabot', or a similar indicator cultivar.

#### Control Procedures

Since blueberry mosaic is not known to be latent in blueberry cultivars, prompt removal of visibly infected bushes may be worthwhile. No information is available on thermotherapy.

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## Red Ringspot of Blueberry

By D. C. Ramsdell, K. S. Kim, and J. P. Fulton

### Additional Common Names

Ringspot (Hutchinson and Varney 1954).

### History and Geographic Distribution

The symptoms of this disease were first described by Hutchinson (1950), and its virus or viruslike nature was determined by Hutchinson and Varney (1954). The disease is most important from an economic standpoint in New Jersey, and it is widespread in recent plantings in Arkansas (Kim et al. 1981). The disease has also been reported from Michigan, Connecticut, Massachusetts, New York, North Carolina, and most recently in Oregon (Converse and Ramsdell 1982), but it is not of much economic importance in these States. Paulechova (1972) has reported the occurrence of red ringspot in Czechoslovakia on wild *Vaccinium myrtillus* L.; however, she did not establish that the disease was caused by the virus by graft transmission, but only showed that a fungal pathogen was not present.

### Economic Importance

No actual bush loss or crop loss data are available for this disease, from the standpoint of growth reduction, yield loss, or fruit damage.

### Symptoms on Natural and Experimental Hosts

The cultivars most commonly observed showing symptoms include 'Blueray', 'Bluetta', 'Burlington', 'Cabot', 'Coville', 'Darrow', 'Earliblue', and 'Rubel'. The cv. 'Jersey' exhibits apparent immunity, and 'Bluecrop' has shown excellent field resistance. The disease has been observed in wild blueberry plants in New Jersey. These plants belong to the *V. australe* Small and *V. corymbosum* L. group. Red ringspot disease of blueberry and cranberry ringspot may be caused by the same virus. The disease symptoms are similar, and similar inclusion bodies are found in ultrathin sections from diseased cranberry (K. S. Kim and A. W. Stretch, unpublished data); however, cross-graft experiments have not yet been conducted to establish this relationship.

Attempts to rub transmit purified red ringspot virus (RRSV) to herbaceous hosts have been unsuccessful (Kim et al. 1981).

On highbush blueberry, the disease causes the following symptoms: Stems 1 yr old and older often exhibit red ring spots (fig. 146) or red blotches that are roughly circular, but not ringlike (fig. 147). Reddish-brown circular spots, 2 to 6 mm in diameter, develop on older leaves in mid- to late summer (fig. 148). Younger leaves usually do not show the red spots. Sometimes spots, if sufficiently numerous, coalesce. These circular spots often have a green center. These spots are most prominent only on the upper surface of the leaf. Powdery mildew *Microspheera alni* DC ex Wint. var. *vaccinii* (Schw.) Salm.) can cause similar symptoms on



Figure 146. — Stem of a red ringspot infected cv. 'Blueray' bush exhibiting typical well-defined ringspots (arrow).

the leaves; however, the leaf spots caused by this disease are prominent on both sides of the leaf. The cv. 'Rancocas' may show fruit symptoms as part of the disease syndrome, consisting of circular light areas of blotching on the fruit. The cv. 'Bluetta' sometimes shows a red ringspotlike disorder typified by red leaf spotting, which is probably caused by a genetic disorder. There are no ring spots on the stems associated with the genetic disorder.

There are no known experimental herbaceous or woody experimental hosts (other than blueberry).

### Natural and Experimental Transmission

**Natural transmission:** The disease appears to spread actively in New Jersey; however, in Michigan the disease does not spread at all in the field. Mapping of spread in New Jersey plantings indicates that spread is generally from bush to bush within the row (A. W. Stretch, unpublished data). Since spread does not occur in Michigan in fields with populations of the blueberry aphid (*Illinoia pepperii* MacGillivray) present, it is unlikely that this aphid is the vector. In New Jersey, where active spread occurs in the field, mealybug (probably *Dysmicoccus* sp.) is the suspected vector (A. W. Stretch, personal communication).





Figure 147. — Stem of a red ringspot infected cv. 'Blueray' bush exhibiting red blotches that are not ringspots (arrow).

Experimental transmission: The only experimental transmission is by chip budding or whip grafting to healthy susceptible highbush blueberry cultivars.

### Properties of the Causal Agent

Red ringspot virus (RRSV) is an isometric virus 42 to 46 nm in diameter (Kim et al. 1981). The virions are found embedded in inclusion bodies (fig. 149) in ultrathin sections made from symptom-bearing infected leaf tissue. The circular inclusion bodies are found in both the cytoplasm and in the nucleus. There are two centrifugal components in ultracentrifuged linear-log sucrose density gradients (Kim et al. 1981). In ultracentrifuged cesium chloride step gradients, the virus forms two distinct bands of densities 1.3 and 1.4 g/cm<sup>3</sup>, whereas in a sibling tube of cauliflower mosaic virus (CaMV) a single band was formed at 1.35 gm/cm<sup>3</sup>. In one-way tests, purified RRSV did not react with CaMV antiserum in agar gel double diffusion tests nor in enzyme-linked immunosorbent assays (ELISA) (Kim et al. 1981). In two-way agar gel tests with RRSV and CaMV antiserum, no serological relationship was shown between the two viruses (D. C. Ramsdell, unpublished data).



Figure 148. — Red ringspot infected leaves of cv. 'Blueray' showing typical reddish-brown circular leaf spots, which appear on older leaves in the late summer. Younger leaves will usually not exhibit these spots.

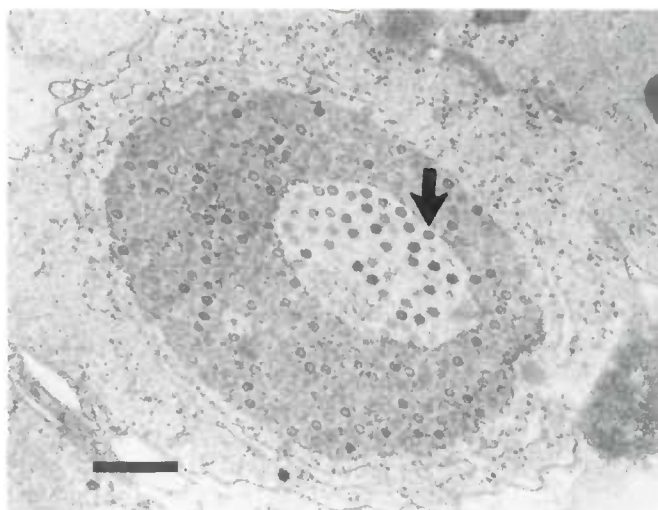


Figure 149. — An inclusion body from a red ringspot infected blueberry leaf showing embedded virions (arrow). Bar represents 500 nm.

### Detection and Identification

Symptomatology is a useful means of detecting red ringspot, provided that one is cognizant of the possible confusion with the symptoms caused by powdery mildew infection of leaves. Symptoms seen in cv. 'Bluetta' must be confirmed by further tests to eliminate the possibility that they are the result of a genetic disorder. If one is testing plants for a clean stock program, tests beyond visual inspection are necessary to detect symptomless infection. Budding or whip grafting from stems from suspected diseased bushes to healthy very susceptible cultivars, for example, 'Blueray', 'Burlington', 'Cabot', or 'Darrow' should be done in the spring just before bud break. Symptoms may develop within 3 mo, but longer observation is advisable. Methods of direct detection using ELISA are being developed (D. C. Ramsdell, unpublished data).

## Control Procedures

The use of disease-free plants and roguing of diseased bushes from the field are the only currently recommended measures for control, until the vector is determined. Stretch and Scott (1977) have published a method for producing red ringspot-free softwood cuttings based upon extensive indexing. They have also shown that heat treatment of red ringspot diseased plants at 37°C for 8 wk did not eliminate RRSV from propagants taken from resulting new shoot growth.

## Ringspot of Cranberry

By A. W. Stretch

## Additional Common Names

None.

## History and Geographic Distribution

The disease was first reported to occur on cranberry in New Jersey in 1962 by Stretch (1964). It was observed in Wisconsin in 1963 by Boone (1966). No other published reports of occurrence have been made, but there is a strong possibility that the disease could be found in areas where vines originating in New Jersey were grown.

## Economic Importance

Ringspot causes malformation and necrosis of berries of the cv. 'Searles', and adversely affects the keeping quality of fresh berries (Boone 1967). Distribution of the disease is so limited, however, particularly in 'Searles', that the total economic loss is negligible.

## Symptoms on Natural and Experimental Hosts

Cranberry fruits from affected plants show pale, circular patches or whitish rings. The rings on the cv. 'Howes' (fig. 150) are usually larger and more distinctive than those on 'Searles'. Where the berries are only slightly colored, the area within the rings is a much deeper red than the area outside. Affected berries of 'Searles' are often malformed (fig. 151), and many of them show necrosis at the blossom end (fig. 151). In extreme cases, entire berries are necrotic. Ringspot symptoms are also produced on the leaves (fig. 152). These become apparent when the leaves assume their reddish autumn color; the rings stay green, while the remainder of the leaf turns red. No information on symptoms in experimental hosts is available.

## Symptoms on Indicator Hosts

At present, no information is available.

## Natural and Experimental Transmission

At present, no information is available.

## Properties of the Causal Agent

The causal agent has not been positively identified. K. S. Kim (unpublished data) has observed viruslike particles and



Figure 150. — Ringspot on the fruit of the cranberry cv. 'Howes'. (Courtesy D. M. Boone.)

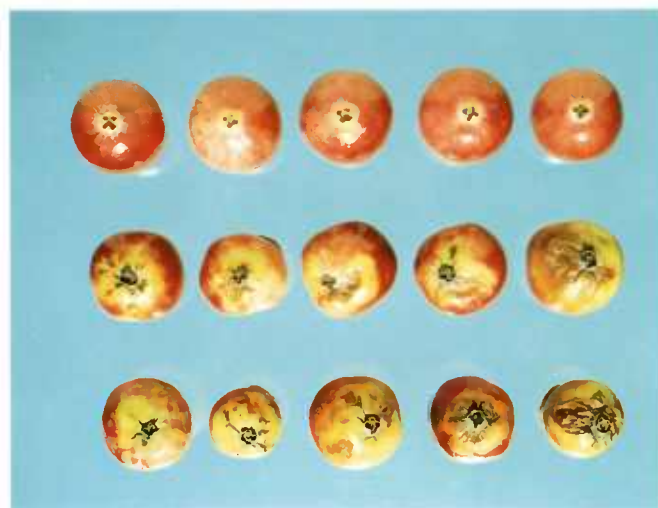


Figure 151. — Ringspot symptoms on the cranberry cv. 'Searles' showing necrosis (bottom two rows) compared with healthy fruit (upper row). (Courtesy D. M. Boone.)

associated inclusion bodies in diseased cranberry leaf tissue. The observed bodies are similar to the caulimovirus type recently reported associated with blueberry red ringspot disease (Kim et al. 1981).

## Detection and Identification

Ring symptoms produced on the fruit and leaves are good indicators. The rings are particularly prominent on the cv. 'Howes'.

## Control Procedures

The use of diseased vines should be avoided in planting new cranberry beds. No information is available on thermotherapy of this disease.





Figure 152. — Leaf symptoms of ringspot of cranberry.  
(Courtesy D. M. Boone.)

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### Section 3.

## Virus and Viruslike Diseases of *Ribes* (Gooseberry and Black and Red Currant)

#### Introduction

By R. Casper

During the last 10 yrs, no new major virus diseases of *Ribes* crops have been detected. Some of the diseases of *Ribes* crops are still restricted to certain areas or even single fields. Others are widespread and common, but most of them do not cause heavy losses in yield.

Black currant reversion is certainly the major disease of this crop in Europe and is found in most areas growing black currants. Many investigations on the cause of the disease have been undertaken, but the causal agent is still not defined. Reports about potato virus Y as the cause of black currant reversion have not been confirmed despite careful investigations in other laboratories. These reports are probably based on an unnoticed virus transmission by aphids in the greenhouse. Despite other assumptions published during recent years, the causal agent of black currant reversion remains unknown.

Since berry crops have received increasing attention in recent years, interest in some of the poorly understood *Ribes* diseases caused by viruses or viruslike agents may grow and result in further useful research.

#### Detection of Virus and Viruslike Diseases in *Ribes*

By A. N. Adams and J. M. Thresh

Many of the viruses infecting *Ribes* crops cause latent infection or slight symptoms that may be restricted to only a small part of the plant. For detection and diagnosis, one should either use serological techniques where possible or inoculate indicator plants mechanically by vectors or by grafts.

**Graft transmission.** Techniques that do not involve buds are recommended to avoid transferring eriophyid mites (see chapter in black currant section on "Viruslike Disorders," p. 142), which damage growth and cause viruslike symptoms. Good results have been obtained by using patches of bark in June, July, and August. The bark can be held in place with polythene tape. Earlier in the season, or when working with young seedlings, graft-transmissible diseases can be transferred in "chips" from unhardened green stem tissue. The chips may be held in place with self-adhesive bandages.

**Mechanical transmission.** Several viruses infecting *Ribes* can be inoculated to herbaceous plants, but extracts made with buffer are usually only slightly infectious. The addition to extraction buffers of either nicotine (1-2% v/v),

polyethylene glycol (mol. wt. 4000 or 6000), or polyvinyl pyrrolidone (mol. wt. 24,000 or 44,000, 1-2% w/v) greatly increases infectivity.

*Ribes* plants are very difficult to infect by mechanical inoculation. Best results are obtained by using very young seedlings grown from seeds extracted at harvest, sown in trays of soil, and stored at 1°C. After several weeks, the trays can be withdrawn as required and moved to higher temperatures to provide a succession of seedlings.

**Serology.** Until recently, *Ribes* viruses could be diagnosed reliably by serology only after transfer to herbaceous hosts. Cucumber mosaic and arabis mosaic viruses, however, can be detected in bud or leaf extracts of infected black currant by enzyme-linked immunosorbent assay (ELISA). ELISA testing of extracts from *Ribes* plants would probably be successful with other viruses for which good quality antisera are available. Such tests are likely to be at least as sensitive as inoculation to herbaceous plants. Extracts from *Ribes* for ELISA tests are best made with polyvinyl pyrrolidone, mol. wt. 24,000 or 44,000 at 2% w/v in the buffer (Clark et al. 1976).

Before ELISA, or any other test, can be reliably used for indexing, a detailed study should be made of the seasonal distribution of the causal agent in the host. The sampling date and tissue type can then be selected to maximize the chances of collecting samples from infected bushes that contain detectable amounts of this agent.

**Indicator plants.** These are shown in table 7.

Table 7.—Detection of virus and viruslike diseases of *Ribes* crops

Vectors and viruses or viruslike agents	<i>Ribes</i> host plant infected		Indicator plants	
	Naturally	Experimentally	Herbaceous <sup>1</sup>	Woody
VECTORS: APHIDS				
Cucumber mosaic <sup>2</sup>	Black currant Red currant <i>Ribes aureum</i>	Gooseberry	<i>C. amaranticolor</i> (L) <i>C. quinoa</i> (L) <i>N. tabacum</i> (S)	Black currant ‘Amos Black’
Vein banding	Black currant Gooseberry Red currant	<i>R. aureum</i>		Black currant ‘Amos Black’ Gooseberry seedlings ‘Leveller’ Red currant seedlings ‘Jonkheer van Tets’
VECTOR: ERIOPHYID MITE				
Reversion	Black currant Red currant <i>R. alpinum</i> <i>R. bracteosum</i> <i>R. ‘carrierei’</i> <i>R. rubrum</i> var. <i>pubescens</i> <i>R. spicatum</i>	<i>R. aureum</i> <i>R. sanguineum</i>		Black currant ‘Baldwin’ ‘Öjebyn’
VECTORS: NEMATODES				
Arabis mosaic <sup>2</sup>	Black currant Red currant Gooseberry	<i>R. aureum</i> <i>R. sanguineum</i>	<i>C. amaranticolor</i> (S) <i>C. quinoa</i> (S) <i>N. tabacum</i> (S)	Black currant ‘Amos Black’
Raspberry ringspot <sup>2</sup>	Black currant Red currant Gooseberry <i>R. sanguineum</i>		<i>C. amaranticolor</i> (L) <i>C. quinoa</i> (S) <i>N. tabacum</i> (S)	Black currant ‘Amos Black’ Red currant ‘Clemenceau’
Strawberry latent ringspot <sup>2</sup>	Black currant Red currant		<i>C. amaranticolor</i> (S) <i>C. quinoa</i> (S) <i>N. tabacum</i> (S)	
Tomato ringspot <sup>2</sup> (American currant mosaic)	Black currant Red currant		<i>C. amaranticolor</i> (S) <i>C. quinoa</i> (S) <i>N. tabacum</i> (S)	
Tobacco rattle <sup>2</sup>	Red currant		<i>N. tabacum</i> (S) <i>C. quinoa</i> (L)	
VECTOR: UNKNOWN				
Black currant yellows	Black currant			Black currant ‘Amos Black’
Gooseberry mosaic	Gooseberry	Black currant Red currant <i>R. divaricatum</i> <i>R. tenue</i> <i>R. triste</i>		Gooseberry ‘Whitesmith’ Black currant ‘Blacksmith’ ‘Öjebyn’
Interveinal white mosaic	Red currant	Black currant <i>R. sanguineum</i>	<i>C. quinoa</i> (S) <i>N. rustica</i> (S)	
Yellow leaf spot (European currant mosaic)	Red currant			Red currant ‘Laxton’s No. 1’ ‘Fay’s Prolific’

<sup>1</sup>Herbaceous plants susceptible to sap-inoculation become infected locally (L) or systemically (S). *C.* = species of *Chenopodium*, *N.* = species of *Nicotiana*.

<sup>2</sup>Antisera available.

## Virus and Viruslike Diseases of Gooseberry Aphid-Borne Diseases

### Gooseberry Vein Banding

By A. N. Adams and A. F. Posnette

#### Additional Common Names

Adernbänderung der Stachelbeere; gooseberry mosaic; žilko-  
va mosaiká angrestu.

#### History and Geographic Distribution

What seems to have been gooseberry vein banding disease was first noted in Czechoslovakia (Blatný 1930), but it was not shown to be graft and aphid transmissible until much later (Posnette 1952). Infection is widespread in Europe but is not reported from elsewhere. Tasmania is one of the few countries where gooseberries have been grown extensively without infection, probably because the original introductions were healthy and several of the known aphid vectors are absent (Posnette 1970).

#### Economic Importance

The disease is obviously severe in cultivars such as 'Leveller' but is usually mild in others such as 'Careless', which is the most widely grown cultivar in England. Commercial plantations of the older cultivars are almost totally infected in England, and the disease is common in Europe (Kleinhempel 1968; Putz 1972; Thomsen 1970).

A meristem clone of cv. 'Careless' outyielded otherwise comparable bushes infected with gooseberry vein banding

disease (GVBD) by approximately 15%, and cuttings from uninfected bushes grew faster and survived better than those from infected bushes (Adams 1979). The economic effects in different regions will depend on the sensitivity of the predominant cultivars.

#### Symptoms on Natural and Experimental Hosts

GVBD has been transmitted only within the genus *Ribes* despite attempts to infect herbaceous plants, including alternate hosts of the vectors (East Malling Research Station 1970).

All cultivars of gooseberry, black currant, and red currant appear to be susceptible. Many other *Ribes* species or hybrids develop typical symptoms when inoculated with GVBD including: *Ribes x holosericeum* Otto & Dietr. (*R. petraeum* Wulf x *R. rubrum* L.), *R. Koehneanum* Jancz. (*R. multiflorum* Kit. ex R. & S. x *R. sativum* Syme), *R. multiflorum* Kit., *R. leptanthum* A. Gray, *R. longeracemosum* Franch., *R. x robustum* Jancz. (*R. niveum* Lindl. x *R. inerme* Rydb.), *R. x rusticum* Jancz. (*R. uva-crispa* L. x *R. hirtellum* Michx.), *R. sativum* Syme, and *R. Watsonianum* Koehne. Symptoms have not been seen in *R. divaricatum* Dougl. or in an F<sub>1</sub> hybrid between *R. sanguineum* Pursh and *R. grossularia* L. inoculated with GVBD. Some trial selections with *R. divaricatum* or *R. sanguineum* ancestors have remained symptomless for several years after inoculation, but it is not known whether this is due to resistance, tolerance, or immunity (Knight and Manwell 1980; Knight 1981).



Figure 153. — Vein banding in gooseberry cv. 'Leveller'.



Symptoms in gooseberry: The main veins are banded with translucent, pale-yellow areas. In the first leaves to expand in the spring, the whole vein reticulum may be banded; however, in leaves developing on extension growth, only single veins or short lengths of the main veins may be affected. Leaves with vein banding are often chlorotic and distorted asymmetrically (fig. 153). Seedlings with severe leaf symptoms are stunted; the rooting and vigor of cuttings are depressed by this disease.

Symptoms in black currant: The first leaves to expand in the spring show pale-yellow vein banding, usually restricted to one side of the lamina. Later leaves develop a clearing and yellow banding of the main veins which in some cultivars is restricted to individual lobes. The graft-transmitted vein banding is more precisely delineated than the more diffuse mottle caused by aphid toxins. The two symptoms may also be distinguished seasonally, since the former symptom appears before large infestations of aphids.

Symptoms in red currant: Leaves formed early in the spring show yellow banding of the main veins. Later, leaves produced on extension shoots may have the vein reticulum cleared or narrowly banded with translucent tissue.

Indicator hosts: Seedlings of sensitive gooseberry cultivars, such as 'Leveller', can be used as indicators, but they are slow growing, possess spines, and are prone to infection by powdery mildew, *Sphaerotheca mors-uvae* (Schw.) Berk. & Curt. Black currant cv. 'Amos Black' is without spines, but it is susceptible to powdery mildew; the conspicuous symptoms are transient. Two second backcross derivatives of *R. sanguineum* x gooseberry East Malling Research Station selection numbers (1385/81 and 1385/90) with reduced spines react with distinct symptoms lasting for several weeks and may prove to be useful indicators, although they are susceptible to powdery mildew (Knight 1981).

#### Natural and Experimental Transmission

GVBD is readily transmitted by chip budding or patch grafting to other gooseberries and to black currant. This disease has only occasionally been transmitted from gooseberry to red currant (Karl and Kleinhempel 1969).

The causal agent has not been transmitted by sap inoculation to *Ribes* test plants or herbaceous species.

The causal agent has been transmitted from gooseberry plants by *Aphis grossulariae* Kalt., *Aphis schneideri* (Börn.), *Hyperomyzus pallidus* H.R.L., *Nasonovia ribisnigri* (Mosley) (Posnette 1952, 1964), *Cryptomyzus ribis* (L.), *Hyperomyzus lactucae* (L.), and *Myzus persicae* (Sulz.) (Karl and Kleinhempel 1969). Transmission was semipersistent, aphids requiring an acquisition access period of at least 30 min, the infection rates increasing with longer feeding periods. Aphids were not inoculative after 3 hr of test feeding (Posnette 1964).

Slow natural spread occurs in gooseberry in England. In a field trial over a 5-yr period at East Malling, only 2 out of 50 gooseberry seedlings became infected (Posnette 1964). Reinfection of bushes of a meristem-derived clone of cv. 'Careless', at seven sites in Kent and Wisbech, Cambridgeshire, was also slow. Only 5 of 496 bushes became infected in 6 yr, although they were adjacent to infected commercial material at six of the seven sites (Adams 1979). This contrasts with the almost total infection of most commercial cultivars. Many of these are very old, as they were raised in private gardens in the north of England for exhibition at gooseberry-growing societies, which were popular in the 18th and 19th centuries (Rake 1958). Slow spread combined with indiscriminate vegetative propagation may have resulted in the present situation.

Factors limiting spread are migratory habits of the most prevalent aphid vectors and nonpersistence of the virus in the vector. Alatae of only two vectors feed on *Ribes* during the summer, those of the other species migrating to herbaceous host plants in the Compositae.

#### Properties of the Causal Agent

Nothing is known of the morphology of the causal agent nor of its properties *in vitro*.

#### Detection and Identification

The leaf symptoms of infection with GVBD are distinctive and are unlikely to be confused with any other disorder except the effects of aphid toxicity. Aphids, particularly *Hyperomyzus lactucae*, produce a yellow, diffuse vein banding, usually accompanied by interveinal mottling, in contrast to the translucent clearing along the veins caused by the graft-transmissible disease. Correct diagnosis is difficult when bushes are infested with aphids and for some time after aphids have flown away or been killed by sprays; consequently, early spring is the best time for inspection.

#### Control Procedures

Gooseberry plants are heat sensitive, and the usual procedure of propagating tip scions from heat-treated plants has been unsuccessful. Plants of 'Careless' gooseberry free of gooseberry vein banding disease were raised by meristem culture (Jones and Vine 1968). Recent improvements to the technique (Hedtrich and Feucht 1981) should facilitate further application.

The very slow reinfection of plants with gooseberry vein banding disease indicates that healthy plants will remain virus-free with little isolation. The prospects of controlling this disease by the issue of healthy planting material are therefore good.

## Nematode-Borne Diseases

### 245 Gooseberry Deterioration //

By F. A. van der Meer

#### Additional Common Names

None, but it is caused by raspberry ringspot virus.

#### History and Geographic Distribution

Reported from The Netherlands by Houtman (1951). Van der Meer (1960, 1965a) isolated raspberry ringspot virus (RRV) from affected bushes, whereas this virus could not be detected in healthy-looking bushes. The disease has not been reported from other countries.

#### Economic Importance

Very little.

#### Symptoms on Natural and Experimental Hosts

Natural infection with RRV has been found in the gooseberry cvs. 'Whitesmith' and 'Whynham's Industry', which are the only ones grown in the area where the disease occurs. Infected bushes of cv. 'Whynham's Industry' show an indistinct mosaic of the leaves. Berries are small and misshapen and ripen very late in summer (fig. 154). Affected bushes usually die within a few years. RRV-infected bushes of cv. 'Whitesmith' do not show symptoms.

Experimentally infected plants of *Chenopodium quinoa* Willd. develop necrotic local lesions and systemic necrosis, whereas *C. amaranticolor* Coste and Reyn. develops only necrotic local lesions. The virus causes symptoms in several other herbaceous hosts.

#### Natural and Experimental Transmission

The virus is assumed to be transmitted by *Longidorus elongatus* (de Man.). This nematode is common in the area where the disease has been found and is known to infect red currant with RRV. In comparison with red currant, gooseberries are rather resistant to natural infection.

Experimentally, the virus can be transmitted to herbaceous hosts by sap inoculation. Red currant seedlings inoculated with sap from infected tobacco developed definite symptoms of spoon leaf (van der Meer 1965a). (See "Spoon Leaf of Red Currant," p. 146.)

#### Properties of the Causal Agent

Because RRV occurs only in deteriorating plants of cv. 'Whynham's Industry' and not in healthy plants of this cultivar, RRV is believed to be the cause of gooseberry deterioration. RRV isolates from red currant and gooseberry are indistinguishable serologically from each other and from



Figure 154. — Gooseberry deterioration: Top, healthy shoot; below, shoot of deteriorated 'Whynham's Industry'.

isolates obtained from other hosts in Scotland (Maat 1965). (For additional information on properties of RRV, see the *Rubus* section, p. 214-219.)

#### Detection and Identification

Infection can be detected by sap inoculation to *C. quinoa* and other herbaceous hosts. Identification is only possible by serological tests.

#### Control Procedures

Tests on *C. quinoa* should be made when selecting healthy gooseberry stocks. Sensitive cultivars like 'Whynham's Industry' should not be grown on RRV-infested land. There is no information on therapy of infected plants.

### 245 Latent Infection of Gooseberry with Arabis Mosaic Virus //

By F. A. van der Meer

#### Additional Common Names

None.

#### History and Geographic Distribution

Latent infection of gooseberry with arabis mosaic virus (AMV) has only been reported from East Germany (Kleinhempel 1970, 1972).

#### Economic Importance

Very little.

### Symptoms on Natural and Experimental Hosts

Naturally infected gooseberries (Kleinhempel 1972) and red currants (Kleinhempel 1972; Thresh 1967) do not show symptoms. On naturally infected black currants, AMV causes chlorotic blotches of the first leaves. This is followed by mottle and ringspot symptoms in May. Later developing leaves are virtually symptomless (Thresh 1966b).

Experimentally infected *Chenopodium quinoa* Willd. and *C. amaranticolor* Coste and Reyn. show many local lesions and develop systemic mosaic and stunting. (For details on further hosts and experimental hosts, see "European Nepoviruses in Strawberry," p. 46.)

### Natural and Experimental Transmission

AMV is transmitted by the nematode *Xiphinema diversicaudatum* (Micoletsky) to several crop hosts.

Experimentally, the virus can be transmitted from gooseberry to herbaceous host by sap inoculation.

### Properties of the Causal Agent

No details are reported about precise serological relationships between AMV isolates from gooseberry and those obtained from other hosts. (See the *Rubus* section, p. 204, for further details on properties of AMV.)

### Detection and Identification

Detectable by sap inoculation to *C. quinoa*. Identification is only possible by serological tests.

### Control Procedures

Tests on *C. quinoa* should be made when selecting healthy gooseberry stocks. There is no information on therapy of AMV-infected gooseberry.

## Viruslike Disorders

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### ✓ Gooseberry Mosaic and Leaf Malformation of Gooseberry By A. N. Adams

#### Gooseberry Mosaic

A single gooseberry plant, cv. 'Lady Delamare,' was found in Poland with a bright yellow mottle and vein yellowing that was distinct from the normal symptoms of gooseberry vein banding (Basak and Maskiewicz 1980). The disease was graft-transmitted to gooseberry cvs. 'Whitesmith' and 'Resistenta', which showed similar symptoms to those on 'Lady Delamare', and to black currant cvs. 'Blacksmith', 'Öjebyn', and 'Roodknop', which reacted with symptoms of localized, patchy yellowing that gradually spread to most of the leaf area to resemble black currant yellows (Posnette 1952). Inoculated plants of *Ribes divaricatum* Dougl., *R. nigrum* L. var. *europaeum* Jancz., *R. tenue* Jancz., and *R. triste* Pall. also showed symptoms but not *R. Gordonianum* Lem. (*R. aureum* Pursh. x *R. sanguineum* Pursh), *R. x nigrolaria* (*R. nigrum* L. x *R. uva-crispa* L.), and *R. prostratum* L'Her. The disease was apparently latent in red currant cvs. 'Jonkheer van Tets' and 'Large Red Dutch'.

The host range and symptoms of the disease suggest that it differs from gooseberry vein banding.

#### Leaf Malformation of Gooseberry

The 'claw leaf' or 'hawthorn leaf' condition in England mentioned by Thresh (1970) was not graft-transmitted. Other reports of similar malformations and of dieback in Scotland (Gray 1949; and Gray and Everett 1956) could be records of spoon leaf. (See "Spoon Leaf of Red Currant," p. 146.)

## Virus and Viruslike Diseases of Black Currant

### Mite-Borne Diseases

#### Reversion of Black Currant "

By A. N. Adams and J. M. Thresh

#### Additional Common Names

Atavismus; zvrátcerného rybízu.

#### History and Geographic Distribution

Reversion disease was first described in The Netherlands (Ritzema Bos 1904) and later in England (Amos and Hatton 1927) where it was already common in gardens and plantations, usually associated with infestations of the black currant gall mite (*Cecidophyopsis ribis* (Westw.)), which had long been known as a serious pest of black currant. The direct damage caused by the mite was at first confused with the effects of the disease agent that it transmits. This is because reversion and its vector have a complex and unique relationship with their black currant host (Thresh 1964a).

Reversion has been recorded in virtually all the countries where black currants are grown. Infection is rare in New Zealand and Australia, but widespread in most European countries.

#### Economic Importance

Reversion is of major importance in Europe, and its effect on crop yield is severe (Krczal 1976; Cropley et al. 1964). The disease is less common in Great Britain now, compared with 10 to 15 yr ago because certified bushes have been planted extensively and endosulfan has been widely used as an effective acaricide (Thresh and Blandy 1979).

#### Symptoms on Natural and Experimental Hosts

Black currant is the usual natural host of reversion, but it has also been recovered from naturally infected *Ribes bracteosum* Dougl. ex Hook., *R. rubrum* L. var. *pubescens* Swartz and *R. X Carrierei* Schneid. (*R. glutinosum* Benth. x *R. nigrum* L.) (Thresh 1970), *R. alpinum* L., *R. spicatum* Robson (a species of red currant growing wild in Finland), and some commercial cultivars of red currant (Bremer and Heikinheimo 1980, Rakús 1973). Several other species and hybrids have been infected by graft inoculation, but none has given more conspicuous symptoms than black currant.

Symptoms in black currant. All the main commercial black currant cultivars in Western Europe are susceptible although there are differences in tolerance to infection. Some Russian cultivars, derived mostly from *R. nigrum* L. var. *sibiricum* (E. Wolf) or *R. dikuscha* Fisch. ex Turcz., are reported as resistant to infection (Saumjan 1964, Potapov and Grinenko 1968), and some may be immune (Knight and Manwell 1980).

Symptoms in black currant do not appear until the year after inoculation, and they are at first restricted to one or a few shoots. One-third to one-half of the bush is affected in the second year, and infection is fully systemic by the third or fourth year. The habit of growth is affected, together with specific effects on the flowers and leaves.

Infection decreases the number and size of the primary leaves that subtend flowers, and leaves produced during the blossom period are chlorotic. There is little difference in the amount of color of the later extension growth, although the shoots of healthy bushes tend to be fewer and longer than those of reverted bushes. These differences are not sufficiently great or constant for routine diagnosis.

The common strains of black currant reversion agent occurring in Britain and many other European countries decrease the hairiness of the sepals, so that the flower buds are almost glabrous and appear brightly colored, compared with the gray, downy appearance of normal buds (fig. 155). The difference is readily apparent unless the bushes are wet or infected with an unusually avirulent strain (Thresh 1971).



Figure 155. — Left, healthy flower buds; right, glabrous flower buds of black currant with reversion.

In the U.S.S.R., Scandinavia, and some other areas, the flowers of reverted bushes are often glabrous and severely malformed, with the style elongated, stamens absent, and the petals sepallike in appearance (fig. 156). The "double" flowers seem to be caused by a particular strain of reversion that is uncommon in Great Britain and Western Europe below the Scandanavian Peninsula.

Infected bushes develop leaves that are flatter than usual and have a smaller basal sinus. Infection also decreases the number of main veins and marginal serrations (fig. 157). After some experience, these differences can be used for accurate diagnosis in May, June, or July, when attention should be given only to leaves on undamaged shoots of the





Figure 156. — A, malformed flowers of black currant cv. 'Rus' infected with reversion from Eastern Europe. B, malformed flowers of black currant infected with

reversion in Finland (left). Uninfected flowers on the right are at the fruit swelling stage. (Courtesy O. Heikinheimo.)

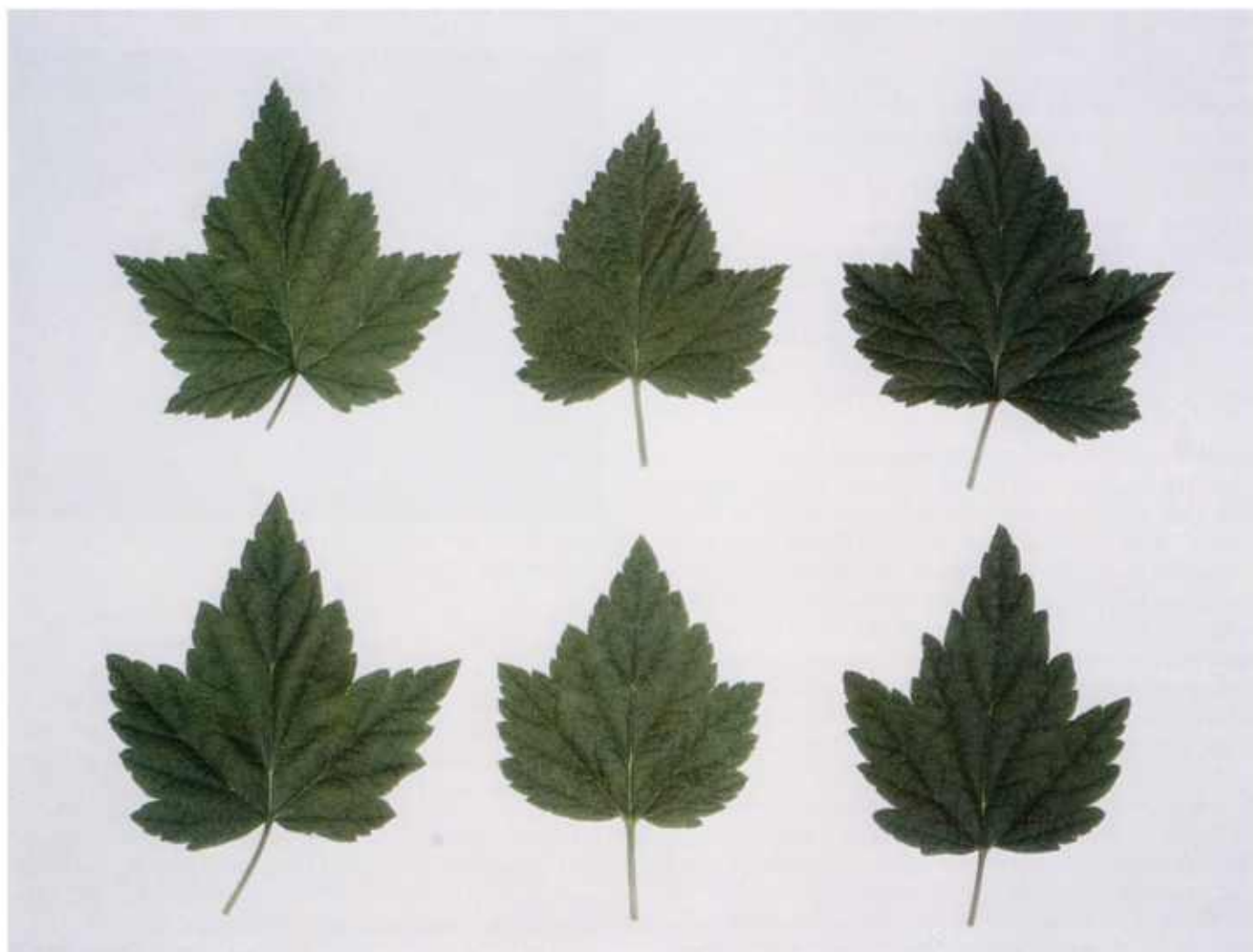


Figure 157. — Top row, left to right, healthy leaves of black currant cvs. 'Baldwin', 'Ben Lomond' and 'Ben Nevis'; bottom row, leaves of the same cultivars infected with reversion.

extension growth. Forked shoots and those damaged mechanically or by mites, capsid bugs (*Lygocoris pabulinus* (L.)), or other insects develop atypical leaves. A further complication is the inherent differences between the leaves of certain cultivars.

Leaves that are invaded by some strains of the reversion agent develop a nonrecurring chlorotic vein pattern (fig. 158). This was originally thought to be a separate disease (Posnette 1952). This reversion is of limited diagnostic value, as symptoms are often slight and restricted to a few leaves that may be concealed by later growth. Jacob (1976a) reported an enhancement of chlorotic symptoms by holding plants under continuous light at 23°C.

Symptoms of infectious variegation are particularly severe when bushes are also affected by reversion.

Symptoms in red currant. Faint flower and leaf symptoms are associated with infection in commercial cultivars of red currants in Finland (Bremer and Heikinheimo 1980). Leaf malformation and the transient vein pattern typical of the disease in black currant were observed by Thresh (East Malling Research Station 1968) in red currant cultivars inoculated with reversion from black currant.

Symptoms on indicator hosts. Numerous cultivars are probably suitable as indicators, but 'Baldwin' (Thresh 1970) and 'Öjebyn' (Bremer and Heikinheimo 1980) consistently react with foliar symptoms the year after inoculation.

### Natural and Experimental Transmission

Reversion is readily transmitted by various grafting methods, but some are unsuitable because the mite vector could be transferred in buds taken from the infected source. This risk is avoided by using patch or chip grafts. Reversion has not been transmitted by pollen or seed, and a report of sap transmission (Jacob 1976a) has not been confirmed. (See "Properties of the Causal Agent," p. 135-136.)

Reversion is transmitted by the gall mite *Cecidophyes ribis* (Westw.). The disease is transmitted efficiently during the late spring and early summer when mites disperse from old galls to the buds of new growth. There is no further spread until the following year, and mites remain within infested buds, which become rounded galls and fail to develop flowers or leaves (Thresh 1965). Disease symptoms do not develop until the year after infestation. There is generally a close correlation between the incidence of reversion disease infection and the distribution of galls during the previous winter.

There are up to 35,000 mites in a single gall, and several hundred galls may occur on a heavily infested bush, from which mites may crawl, leap, or be blown during the



Figure 158. — Black currant leaves showing the chlorotic vein pattern of reversion. (Courtesy F. A. van der Meer.)

dispersal period. Insects, said to act as carriers of the mites (Massee 1928), may spread the disease to plants growing considerable distances from large sources of infection (Thresh 1966a). A feature of crucial importance in epidemiology is that bushes infected with reversion are much more susceptible to infestation by mites than healthy ones (Thresh 1964a).

Some cultivars of black currant derived from *R. nigrum* var. *sibiricum* are resistant to gall mites (Pavlova 1964, Anderson 1971) and react to infestation by forming necrotic tissues that support few or no mites. Gooseberry has been used as a donor for mite resistance in black currant, and progenies with the single gene *Ce* for resistance appear to be virtually immune (Knight et al. 1974), although a small proportion of plants become reverted when exposed to very heavy infestations (Knight 1981). A nongall-forming strain of *C. ribis* has been found in England on *Ribes*, including gooseberry and gooseberry x black currant hybrids. The mite occurs in relatively low numbers and is not known to transmit reversion (Easterbrook 1980).

There is little information on the transmission process owing to the difficulty of transferring mites and their usual requirement to feed within galls or buds. The disease has been transmitted by single mites that were eradicated by endrin 4 h after transfer from galls to healthy seedlings (Thresh 1970). A minimum acquisition access time of 3 h and retention in the mite for up to 25 days were reported by Jacob (1976b).

### Properties of the Causal Agent

Reversion has been associated with: mycoplasma-like organisms (Zirka et al. 1977), bacteria (Silvere and Remeikis 1973), and potato virus Y (Jacob 1976a), but evidence to confirm that any of these agents cause the disease is lacking. Although Jacob (1976a) appeared to have fulfilled Koch's postulates for potato virus Y, this virus could not be detected

in other German reverted material either by ELISA or by immunosorbent electron microscopy by R. Casper, H. Krczal, and D. Lesemann (unpublished results). Furthermore, potato virus Y was not detected by sap inoculation or by ELISA in reverted bushes in England (Thresh et al. 1978).

### Detection and Identification

Diagnosis depends on the ability to recognize the characteristic effects of reversion on flowers or leaves. If growth is damaged or malformed and the symptoms are indistinct, suspect material should be graft-inoculated to 1-yr-old bushes of an indicator such as cv. 'Baldwin'.

### Control Procedures

Stringent quarantine measures should be enforced to avoid disseminating mites, additional infection, or further strains of the disease to countries not already affected.

In countries where reversion occurs, new plantations should be established with bushes or cuttings from stocks certified as free from infection. Planting should be upwind, at least 90 m from any contaminated holding, and routine acaricides should be used (Ministry of Agriculture 1979). Plantations should be inspected just before flowering begins, and again later in the summer for leaf symptoms, to diagnose and remove infected bushes. These measures are to a large extent interdependent, and all must be adopted for full effectiveness.

Healthy clones are normally obtainable by selecting disease-free plants; however, reversion-free plants can be obtained from diseased bushes by heat therapy and top grafting to seedlings (Campbell 1965). Mites can be eliminated from cuttings by warm water treatment (Savzdarg 1957, Thresh 1964b).

Although there are sources of resistance both to reversion and to its vector, these characters have not yet been bred into cultivars that are commercially viable in Western Europe (Keep 1975). Considerable progress, however, has been made towards the integration of agronomic qualities, disease resistance, and gall mite resistance (Keep et al. 1982).

## Aphid-Borne Diseases

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### Green Mottle of Black Currant<sup>11</sup>

By A. N. [Adams and J. M. [Thresh

#### Additional Common Names

None, but the disease is caused by cucumber mosaic virus.

#### History and Geographic Distribution

Green mottle of black currant is caused by cucumber mosaic virus (CMV), a cosmopolitan pathogen with an unusually wide host range. Infection in black currant was reported in a few bushes in each of several plantations or nurseries in England and Wales (Thresh 1966b), in a single bush in Germany (Kleinhempel 1970), and in cv. 'Primorskij Champion' in the Soviet Far East (Gordejchuk et al. 1977).

#### Economic Importance

Infected bushes are stunted and bear little crop. Infection does not seem to spread rapidly between black currant bushes, and the disease is not sufficiently widespread to be of economic importance.

#### Symptoms on Natural and Experimental Hosts

All the main British commercial cultivars of black currant are susceptible and develop similar symptoms, but there are differences in tolerance. The cultivar 'Amos Black' is particularly sensitive and is recommended as an indicator.

CMV causes green mottle disease of red currant and an "arc mosaic" of the golden currant (*Ribes aureum* Pursh) in Germany (Schmelzer 1963). Numerous other weed and cultivated plants are susceptible, including many standard indicator plants.

Small, rapidly growing black currant seedlings develop symptoms 3 or 4 wk after graft inoculation. Established bushes do not produce symptoms until the following year, when they are often restricted to the inoculated shoots and those nearby. Symptoms are very variable. They are seen best as the leaves become fully expanded and tend to be inconspicuous in young and senescing leaves. Large sectors of some leaves become pale green (fig. 159A). At other times, discoloration is restricted to broad bands along certain main veins, sometimes giving a "watermark" effect that is best seen by transmitted light (fig. 159B).

CMV is the only sap-transmissible virus of *Ribes* crops that infects tobacco systemically and causes only local lesions in both *Chenopodium amaranticolor* Coste & Reyn. and *C. quinoa* Willd. The latter hosts are particularly sensitive to sap inoculation and develop necrotic local lesions from which the virus may be transmitted to other herbaceous host plants.

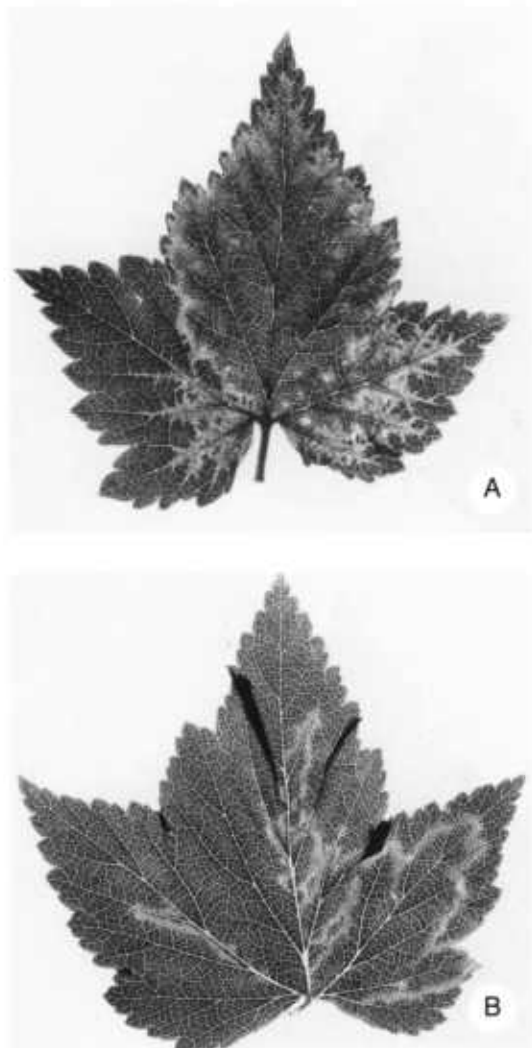


Figure 159. — Green mottle of black currant: A, in April; B, in June.

### Natural and Experimental Transmission

Green mottle disease is readily transmitted by grafting. It is easily transmitted by sap inoculation to herbaceous hosts but only occasionally to black currant seedlings. Extracts of leaves, flowers, roots, or dormant buds in buffer with nicotine (10 g/L) or polyethylene glycol (mol. wt. 4000 or 6000) or polyvinyl pyrrolidone (mol. wt. 24,000, 10 g/L) are highly infectious to *C. quinoa* (Manwell et al. 1979).

CMV is transmitted in the non-persistent manner by the 60 or more aphid species known to be vectors (Kennedy et al. 1962). An isolate of CMV from black currant has been transmitted by *Myzus persicae* (Sulz.) and by five species that spend all or part of their life cycles on *Ribes* crops: *Aphis grossulariae* Kalt., *A. schneideri* (Börn.), *Cryptomyzus ribis* (L.), *Hyperomyzus lactucae* (L.), and *Nasonovia ribisnigri* (Mosley) (Thresh 1970). The virus is readily transmitted between herbaceous hosts and from black currant to herbaceous hosts. Black currant seedlings are infrequently infected either by aphids from black currant or from herbaceous hosts.

There is little natural spread within plantations, and infection seems to spread into or within the crop from weeds or cultivated hosts, perhaps by aphids that visit black currant temporarily.

### Properties of the Causal Agent

Cucumber mosaic virus has been studied in detail. (See "Cucumber Mosaic Virus in Raspberry," p. 191, in the *Rubus* section of this handbook for some properties of CMV.) The authoritative review by Francki et al. (1979) should also be consulted.

### Detection and Identification

Suspect bushes can be indexed by sap transmission to *Chenopodium quinoa* and other herbaceous hosts or by graft inoculation to cv. 'Amos Black'. Tests with buds provide an opportunity to check the health of dormant bushes (Thresh 1970). Extracts from infected black currant leaves made in June reacted strongly in ELISA tests using an antiserum to CMV of unknown origin (A. N. Adams, unpublished data).

### Control Procedures

Control measures are unnecessary, but care should be taken to eliminate infected bushes from nursery stocks and those used for propagation.

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### Vein Clearing and Vein Net Disease of Black Currant

By A. N. Adams and J. M. Thresh

#### Additional Common Names

None.

#### History and Geographic Distribution

Vein clearing and vein net disease was described by Thresh (1966b) and attributed to infection with gooseberry vein banding virus. Although this disease is widespread in gooseberry and red currant, infected black currant bushes are rare in Britain (Thresh 1966b) and in continental Europe (Baumann 1974; Kleinhempel 1972; Putz 1972).

#### Economic Importance

Infection does not seem to be sufficiently widespread to cause serious losses. Infected bushes are slightly stunted and carry almost a full crop.

#### Symptoms on Natural and Experimental Hosts

The disease has only been transmitted within the genus *Ribes*.

Gooseberry and red currant develop symptoms typical of vein banding (Karl and Kleinhempel 1969; Thresh 1970).

All the principal commercial cultivars of black currant in Great Britain are susceptible to graft inoculation, but they differ greatly in their tolerance to infection. 'Baldwin' and 'Wellington XXX' rarely develop symptoms in the field, but



'Wellington XXX' has been used as an indicator in Germany (Baumann 1974). 'Amos Black' and 'Westwick Triumph' are particularly sensitive, but the latter is not recommended for use as an indicator because it grows slowly.

Sensitive black currant cultivars develop a broad yellow banding and occasional clearing of the main veins of the first-formed leaves that subtend flowers. Later leaves develop a clearing and narrow yellow banding of the main veins (fig. 160A). Entire leaves of cv. 'Mendip Cross' are affected in May and June with a vein net pattern (fig. 160B). Symptoms in other cultivars, such as 'Amos Black', are often restricted to individual lobes of occasional leaves that become slightly distorted and asymmetrical (fig. 160C).

#### Natural and Experimental Transmission

The causal agent is readily transmitted by graft inoculation but not by sap. Black currant seedlings have been infected by aphids, *Nasonovia ribisnigri* (Mosley) transferred from gooseberry with vein banding (Posnette 1964). The causal agent has been transmitted between seedlings of the following species by aphids (Karl and Kleinhempel 1969): from gooseberry to black currant (*Hyperomyzus lactucae* (L.)), from black currant to gooseberry and black currant to black currant (*Myzus persicae* (Sulz.)), and from black currant to red currant (*Cryptomyzus ribis* (L.) and *H. lactucae*).

Infection in black currant nurseries and plantations spreads slowly, presumably by aphids.

#### Properties of the Causal Agent

There is no information on the morphology or properties in vitro of the causal agent.

#### Detection and Identification

Infection in sensitive cultivars can be diagnosed in April, May, or June, provided that aphids have not been allowed to distort the growth and cause phytotoxicity. In tolerant cultivars or late in the season, suspect bushes should be graft inoculated to the sensitive cv. 'Amos Black'.

#### Control Procedures

Infected bushes should be removed during routine inspection. Spread is likely to be slow if healthy stocks are planted and aphids are controlled. No information is available on therapy of infected cultivars.



Figure 160. — Vein clearing and vein net disease of black currant: A, in early leaf; B, in cv. 'Mendip Cross' in midsummer; C, in cv. 'Amos Black' in midsummer.

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## Yellow Mottle of Black Currant<sup>1/</sup>

By A. N. Adams and J. M. Thresh

### Additional Common Names

None, caused by Arabis mosaic virus.

### History and Geographic Distribution

Yellow mottle of black currant is caused by Arabis mosaic virus (AMV). Yellow mottle has been reported in only one English nursery and in a few plantations established with bushes distributed from it (Thresh 1966b). The virus has been isolated, once from black currant in France (Putz and Stocky 1971) and once from black currant in the Soviet Far East (Gordejchuk et al. 1977).

### Economic Importance

Infected bushes produce little fruit and are less vigorous than healthy ones, but infection does not seem to be sufficiently widespread to cause serious economic losses.

### Symptoms on Natural and Experimental Hosts

AMV has a wide host range in weeds, herbaceous test plants, and cultivated crops, including gooseberry, red currant, raspberry, and strawberry.

Isolates from black currant have been graft transmitted to *Ribes aureum* Pursh, *R. sanguineum* Pursh, and all the black and red currant cultivars tested. The black currant cultivars reacted similarly with no great differences in sensitivity.

Black currant bushes graft-infected in July or August develop symptoms the following year, when the first-formed leaves show a conspicuous yellow mottle (fig. 161A). This may be irregularly distributed or may form yellow spots and rings. Symptoms are less conspicuous on leaves of the extension growth, and by midsummer the slight specks or flecks on the leaves are barely detectable, so that diagnosis must be confirmed by indexing (fig. 161B).

*R. sanguineum* reacts similarly to black currant, whereas red currant cultivars become infected without showing symptoms.

In herbaceous hosts, isolates from black currant behave like the virulent strains obtained from raspberry. *Chenopodium amaranticolor* Coste and Reyn. and *C. quinoa* Willd. are particularly sensitive to infection by sap inoculation, and within 2 wk develop local and systemic symptoms that closely resemble those caused by strawberry latent ringspot virus, that is, chlorotic local lesions and systemic chlorosis and necrosis, particularly in the tip leaves. (See "Raspberry

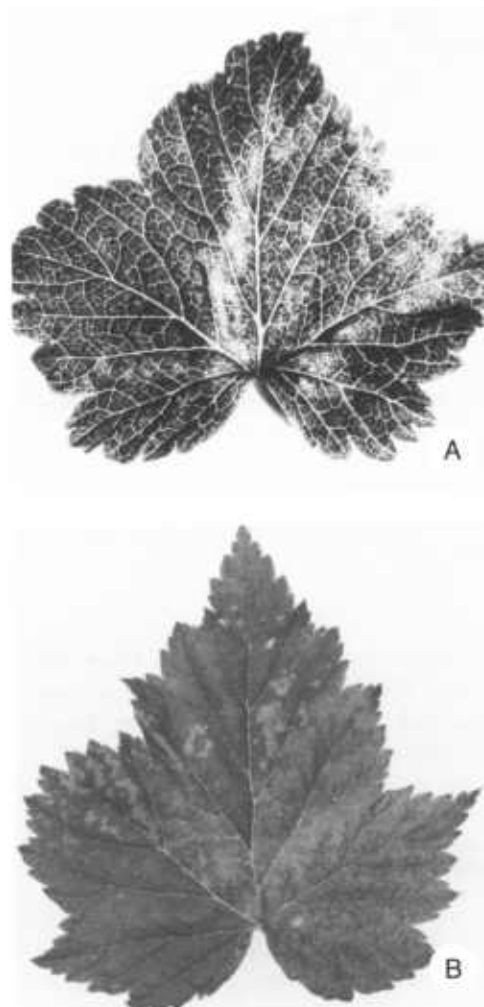


Figure 161. — Leaves of black currant with yellow mottle caused by arabis mosaic virus: A, in an early leaf; B, in midsummer.

Yellow Dwarf and Associated Diseases of *Rubus* Caused by Arabis Mosaic and Strawberry Latent Ringspot Viruses," p. 204.)

### Natural and Experimental Transmission

AMV is readily transmitted between woody hosts by graft inoculation. Extracts of dormant buds or leaves made with nicotine, polyethylene glycol, or polyvinyl pyrrolidone included in the buffer are highly infectious to herbaceous hosts from which the virus is transmissible by sap inoculation to black currant seedlings.

In *Ribes*, there have been no transmission experiments with *Xiphinema diversicaudatum* (Micoletsky), the nematode vector of AMV in other crops. *X. diversicaudatum* was present in the soil of an infected nursery in England. It is assumed to be the natural vector, although spread is also caused by the indiscriminate propagation and sale of infected cuttings (Thresh 1970).

### Properties of the Causal Agent

See "Raspberry Yellow Dwarf and Associated Diseases of *Rubus* Caused by Arabic Mosaic and Strawberry Latent Ringspot Viruses," p. 104, for some properties of AMV. The authoritative review of Murant (1970) should also be consulted.

### Detection and Identification

Black currant cv. 'Amos Black' is a suitable indicator for graft-inoculation tests.

Infection is readily detected by sap inoculation to *Chenopodium quinoa*, but further tests are needed to distinguish AMV from other nepoviruses. (See detailed discussion of nepoviruses in the *Rubus* section, p. 204-228.)

AMV is easily detectable by ELISA in extracts of leaves or buds (Clark et al. 1976).

### Control Procedures

Black currant stock should not be placed at sites where virus and vector are present. Special stocks destined for propagation should be checked to ensure that they are free of infection. No information is available on the therapy of infected black currant cultivars.

### Other Nepoviruses Isolated From Black Currant

By A. N. Adams and J. M. Thresh

Strawberry latent ringspot virus (SLRV) was isolated from a single black currant bush cv. 'Baldwin' growing in Scotland. The bush was also infected with reversion, and it was not determined whether SLRV caused specific symptoms in black currant (Lister 1964).

Two other nepoviruses were detected in black currant in the Soviet Far East (Gordejchuk et al. 1977). Raspberry ringspot virus was isolated from the leaves of cvs. 'Primorskij Champion', 'Likernaya', and 'Buraya'; infection was associated with yellow ringspots in the early spring, followed by a yellow mottle. Tomato ringspot virus was isolated from the leaves of black currant plants cv. 'Primorskij Champion' that showed yellow spots and rings.

Tomato black ring virus was isolated from several bushes of black currant cvs. 'Öjebyn', 'Sunderby', and 'Brödorp' at one locality in Finland and was identified by serology (K. Bremer, unpublished data).

### Vectors Unknown

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### Black Currant Yellows<sup>1/</sup>

By J. M. Thresh

### Additional Common Names

None.

### History and Geographic Distribution

Black currant yellows was found in one English nursery (Posnette 1952) and subsequently in several plantations established with bushes distributed from it. The disease has not been reported in other countries, although the term "yellows" has been applied indiscriminately to other symptoms not necessarily caused by infection by a graft-transmissible agent.

### Economic Importance

Infected bushes are stunted and their crop is greatly reduced (Cropley et al. 1964), but infection is not sufficiently widespread to cause serious economic losses.

### Symptoms on Natural and Experimental Hosts

The disease has only been transmitted between black currants, and the different cultivars react similarly to infection.

Bushes graft infected in July or August develop symptoms the following year. Slight, indistinct chlorotic flecks are produced in April and May, followed in June and July by a more distinct olive-green mosaic affecting large sectors of leaves (fig. 162). There is a similar sequence in subsequent years, and the most conspicuous symptoms follow periods of warm, sunny weather.

### Natural and Experimental Transmission

Slow spread in a field experiment at East Malling in England suggests that there is a rare or inefficient natural vector although none has been found, and tests with the aphids *Hyperomyzus lactucae* (L.), *H. pallidus* H. R. L., *Nasonovia ribisnigri* (Mosley), *Aphis grossulariae* Kalt., and *A. schneideri* (Börn.) were unsuccessful (Cropley et al. 1964).

Infection is readily transmitted by graft inoculation but not by sap inoculation.

### Properties of the Causal Agent

No information.

### Detection and Identification

Yellows may be more widespread than present evidence suggests because the symptoms are easily overlooked or attributed to soil or nutritional disorders. Conspicuous



Figure 162. — Symptoms of black currant yellows.

symptoms are produced some weeks after those caused by other virus and viruslike diseases of black currants, and necessitate an additional late inspection at a time when damage by the leaf spot fungus (*Drepanopezzia ribis* (Kleb.) von Hohnel) may be prevalent. Preliminary diagnosis can be confirmed by graft transmission to 'Amos Black' or other black currant cultivars.

#### Control Procedures

No information.

#### Infectious Variegation

By A. N. Adams and J. M. Thresh

#### Additional Common Names

Gold dust (Campbell and Adam 1968).

#### History and Geographic Distribution

The symptoms of infectious variegation were described (Posnette 1952) several years before transmission of the disease was reported (Ellenberger 1962). Infection occurs throughout some little-grown British cultivars. A similar condition has been noticed in other European countries (Kristensen et al. 1962; Putz 1972).

#### Economic Importance

No information is available.

#### Symptoms on Natural and Experimental Hosts

Black currant is the only known host and cvs. 'Daniel's September' and 'Laxton's Nigger' develop a bright chrome or pale-yellow mosaic of the early leaves (fig. 163A). This is followed in summer by a broad yellow banding of the main veins, forming a vein net pattern (fig. 163B). Symptoms differ greatly in severity between years.

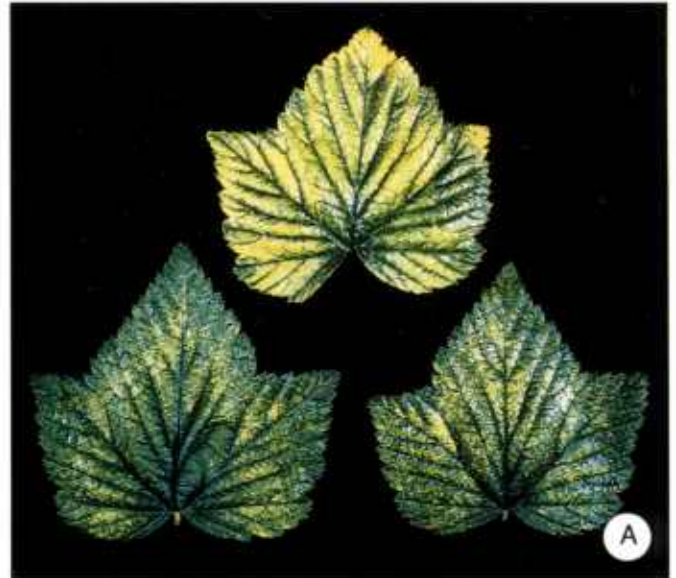


Figure 163. — Infectious variegation in black currant: A, In early leaf; B, in midsummer.

The common cvs. 'Baldwin' and 'Wellington XXX' develop only very slight symptoms in occasional seasons.

#### Natural and Experimental Transmission

The disease was reported to be graft transmitted by Ellenberger (1962) and by Kristensen et al. (1962) but confirmatory evidence is lacking. Similar diseases were not graft transmitted by Posnette (1952), Campbell and Adam (1968), or Putz (1972). Sap inoculation to herbaceous hosts failed (Ellenberger 1962), and no experiments on insect transmission have been reported.

#### Properties of the Causal Agent

No information



### Detection and Identification

Seedlings grown at East Malling in England and Wageningen in The Netherlands from certain black currant crosses have developed symptoms exactly resembling those of infectious variegation (Thresh 1970). Campbell and Adam (1968) reported that gold dust symptoms appeared to a variable extent in different cultivars and that this was reflected in the seedling progeny; they concluded that the disorder was due to an inherited factor. Graft transmission tests to seedlings must therefore be suspect and, if the disease is transmissible, a clonal indicator is essential.

### Control Procedures

No information.

### Remarks

Gold dust may be a nontransmissible syndrome similar to but distinct from infectious variegation (Campbell and Adam 1968). In the absence of consistent evidence for a graft-transmissible pathogen, however, these two apparently identical syndromes are considered to be synonymous.

### Viruslike Disorders

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#### Viruslike Disorders

By A. N. Adams and J. M. Thresh

#### Spring Vein Banding

In early spring, the first-formed leaves of fruiting bushes sometimes develop a yellow banding of the main veins that is very conspicuous in certain years. Affected leaves usually subtend flowers and soon abscise. Symptoms rarely develop on nursery bushes or on the leaves of the extension growth and are missed unless the bushes are inspected as flowering begins.

The symptoms have been seen in many cultivars. These symptoms are distinct from those caused by gooseberry vein banding virus, which are mild or absent on the first-formed leaves of black currant and occur on leaves of extension growth.

Transmission experiments have been hampered by the lack of suitable indicators; young seedlings must be kept for at least 2 yr for flowers to develop. No symptoms occurred in graft-transmission tests to red currant and gooseberry seedlings (J. M. Thresh, unpublished data).

#### Aphid Damage

Several species of aphids that feed on black currant cause downcurling of leaves and stunting. In addition, the feeding of *Hyperomyzus lactucae* (L.) causes a yellow mottling and vein banding (fig. 164), which may occur on uninfested leaves. These symptoms tend to be confused with those of gooseberry vein banding virus; however, the vein banding caused by aphids is usually broader and more opaque than that caused by virus. Other evidence of aphid colonization, such as leaf curling and cast skins, is also likely to be present.

#### Damage of Leaves by Eriophyid Mites

*Cecidophyopsis ribis* (Westw.), the black currant gall-mite vector of reversion, normally inhabits buds, which develop into rounded galls and fail to produce flowers or leaves. The leaves subtending infested axillary buds develop normally, but leaves produced after an apical bud has become infested are severely malformed. Ultimately, almost trifoliate leaves appear (fig. 165).

Reverted bushes (that is, those infected with reversion disease) are much more susceptible to mites than healthy ones; and malformation tends to be associated with reversion infection. This explains why distorted trifoliate leaves were long considered the ultimate stage of reversion; however, reversion disease affects the shape and venation of leaves without affecting their bilateral symmetry.



Figure 164. — Vein banding in black currant caused by *Hyperomyzus lactucae*.



Figure 165. — Leaf malformation in black currant caused by the eriophyid mite, *Cecidophyopsis ribis*.

### Sectorial Chimera

Occasionally, black currant bushes develop single leaves or a sequence of leaves with abnormally distributed chlorophyll that is entirely absent from certain lobes or restricted to the palisade or mesophyll cells. There is no evidence that this condition is transmissible, and it is assumed to be a sectorial chimera. It is of no economic significance, although affected shoots should be removed from nurseries to avoid perpetuating the disorder.

## Virus and Viruslike Diseases of Red Currant

### Aphid-Borne Diseases

#### 245 Red Currant Vein Banding

By F. A. van der Meer

#### Additional Common Names

Adernbänderung; Nerfbandmozaiek; Nervová mosaika.

#### History and Geographic Distribution

Symptoms of vein banding in red currants were first described in Czechoslovakia (Blatný 1930) and Germany (Winter 1940). Transmissibility of the disease was shown first in The Netherlands (van der Meer 1961). Later on, the disease was reported from Great Britain (Thresh 1967), East and West Germany (Kleinhempel 1968; Baumann 1974), France (Putz 1972), and Romania (Ghena and Botar 1974). Red currant vein banding probably occurs wherever red currants are grown.

#### Economic Importance

There is little exact information about the effect of vein banding on growth and cropping of red currants. Growth reduction of about 50%, probably partly due to shock effects, has been reported for infected red currant seedlings (Kleinhempel 1970). Cuttings of infected 'Jonkheer van Tets' showed 28% growth reduction in their first growing season, and vein banding-free bushes of 'Jonkheer van Tets' consistently outyielded infected bushes of the same cultivar (Adams 1979). Vein banding reduces the number of cuttings produced in stool beds of red currant (van der Meer 1980). Economically, vein banding is the most important virus disease of red currant because of its general occurrence and because symptoms are easily overlooked during field inspections.

#### Symptoms on Natural and Experimental Hosts

In most red currant cultivars, symptoms are vein banding and vein clearing, which are often restricted to parts of the leaves. This uneven distribution of symptoms in leaves is very characteristic and enables one to recognize the disease when mild vein clearing is the only symptom, as happens quite often (fig. 166). Symptoms are more pronounced by transmitted than by reflected light (fig. 167). They are most conspicuous in May and June and may disappear during July and August. In sunny years, infected plants may not show any symptoms, whereas symptoms may be very pronounced when cloudy weather prevails during April and May. Infected plants are somewhat stunted in comparison with healthy ones.



Figure 166.—Vein banding and vein clearing in red currant, cv. 'Rovada'.

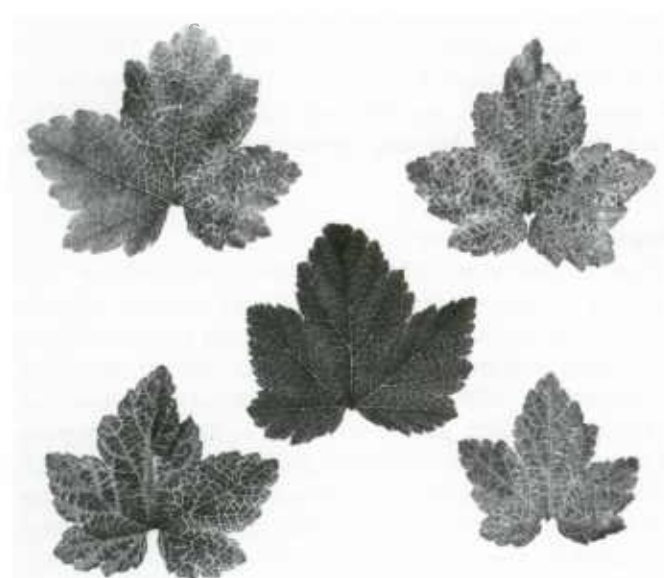


Figure 167.—Vein banding and vein clearing in red currant cv. 'Fay's Prolific'; healthy leaf is in the center.

Experimentally infected black currants show vein clearing in the first-formed leaves of the fruiting wood and in some leaves of the extension shoots. Symptoms are less conspicuous in black than in red currants. Very young seedlings of red currants and gooseberry infected by aphids show severe vein banding and stunting, which is sometimes accompanied by small necrotic lesions in the leaves. Seedlings of cv. 'Jonkheer van Tets', used as indicators, may show a similar shock reaction in May when kept in an unheated greenhouse or when graft inoculation has been done in August of the year before. Symptoms in black currant and gooseberry seedlings resemble those caused by gooseberry vein banding virus.

#### Natural and Experimental Transmission

Red currant vein banding has been transmitted by aphids as well as by patch grafting.

The following aphid species have been established as vectors: *Aphis schneideri* (Börn.), *A. triglochinis* Theob., *Cryptomyzus galeopsides* (Kalt.) subsp. *citrinus* H.R.L., *C. ribis* (L.), *Hyperomyzus lactucae* (L.), and *Nasonovia ribisnigri* (Mosley) (van der Meer 1965b, 1965c). These aphids hibernate on currants, but most species migrate to summer hosts in June. (See above references for details on the seasonal host ranges of the aphids; also, Hille Ris Lambers (1949, 1953.) In experiments of Kleinhempel (1968), *Myzus persicae* (Sulz.) also transmitted red currant vein banding. Transmission rates with all aphid vectors have been low.

Virus-vector relationships have been studied by F. A. van der Meer (unpublished data) and Karl and Kleinhempel (1969). Aphids can acquire the virus in 30 min, but the number of infected plants is increased after an acquisition time of 24 hr. The virus is transmitted less readily after an inoculation access period of 30 min than after a longer period. Vectors were observed to be infected 4 hr after leaving the infected source, but not after 8 hr.

Natural spread of the disease has not been studied extensively. The ease with which vein banding-free plants can be found in very old plantations indicates that natural spread in cropping bushes is very slow. Observations of Adams (1979), however, show that natural spread in cv. 'Jonkheer van Tets' can be rather fast, although his experiments were on a small scale. Natural spread has been noticed in stool beds of red currant (van der Meer 1980).

Despite numerous attempts, no virus has ever been transmitted in sap inoculations from diseased currants to herbaceous plants. In experiments with several thousands of red currant seedlings, seed transmission has never been observed.

#### Properties of the Causal Agent

The assumption that red currant vein banding is caused by a virus is based on its type of symptoms and on the observation that it is transmitted by aphids and by grafting; however, virus particles have not been detected so far.

#### Detection and Identification

This disease is detectable by its symptoms in the field, although symptoms can be very inconspicuous in some years. Symptom expression is promoted by growing potted plants in an unheated and shaded greenhouse. Under such circumstances, infected plants of most cultivars show clear symptoms, which offer the opportunity to select healthy stock material. 'Rondom' seldom shows symptoms, and infected plants can only be detected with certainty by indexing them on seedlings of cv. 'Jonkheer van Tets' (van der Meer 1965d).

#### Control Procedures

To ensure a good start of new plantations, planting material should be free of vein banding. Such material can be produced by using cuttings from isolated stool beds that have



been started with healthy material. Experience in The Netherlands, however, has taught that even in isolated propagation fields some cultivars, for instance cvs. 'Stanza' and 'Rondom', may become reinfected to a rather high degree within 8 to 10 yr. Propagation fields, therefore, have to be renewed periodically. For this purpose, small amounts of nuclear stock material have to be kept in insect-proof screenhouses continually (van der Meer 1980).

Heat treatment experiments with red currant have almost completely failed so far. Plants are rather sensitive to temperatures of 35° to 37°C. They produce very little extension growth during treatment, and they usually die within 4 to 6 wk. With the exception of one tip of cv. 'Prince Albert', all tips of many treated plants grafted to seedlings, or rooted in a mixture of sand and peat, appeared still infected.

### Remarks

In black currant and gooseberry seedlings, red currant vein banding causes symptoms that resemble those of gooseberry vein banding virus. However, attempts to induce vein banding in red currants by grafting them with infected gooseberry either failed (F. A. van der Meer, unpublished data) or were partly successful (Karl and Kleinhempel 1969). Both red currant and gooseberry vein banding are transmitted by the same aphid species and show the same virus-vector relationships. Therefore, it seems reasonable, pending further research, to consider them to be related strains.

### Green Mottle of Red Currant

By F. A. van der Meer

### Additional Common Names

None, but the disease is caused by cucumber mosaic virus.

### History and Geographic Distribution

Green mottle of red currant has been noticed in The Netherlands (van der Meer 1962) and in England (Thresh 1967) as a very uncommon disease of red currant.

### Economic Importance

Very little.

### Symptoms on Natural and Experimental Hosts

In naturally infected bushes of cv. 'Maarses Prominent', the virus causes a severe yellowing especially in the center part of the leaf near the petiole (fig. 168). Affected leaves may be deformed, and branches sometimes die back. In cv. 'Jonkheer van Tets', the virus causes green mottle, as well as line patterns, which can be very clear in the spring (fig. 169). The virus usually remains localized in the lower parts of only some branches.



Figure 168.—Cv. 'Maarse's Prominent' with symptoms of green mottle caused by cucumber mosaic virus.

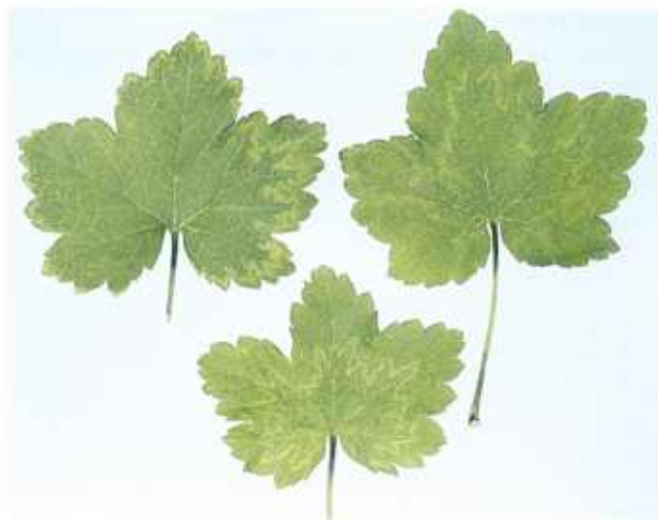


Figure 169.—Leaf pattern in cv. 'Jonkheer van Tets' infected with green mottle caused by cucumber mosaic virus.

In *Chenopodium quinoa* Willd., necrotic local lesions appear about 3 or 4 days after infection. *Nicotiana tabacum* L. cv. 'White Burley', *N. glutinosa* L., and *N. rustica* L. react with a mild systemic mottle, whereas cucumber develops a severe mosaic.

### Natural and Experimental Transmission

Natural spread in currants appears to be rare.

Experimentally, the virus can be transmitted mechanically to many herbaceous hosts and can be transmitted between red currant bushes by grafting. In greenhouse trials, the aphid *Myzus persicae* (Sulz.) transmitted the virus from tobacco to tobacco.



### Properties of the Causal Agent

The virus has been identified serologically as a strain of cucumber mosaic virus (CMV). An antiserum has been made to one red currant isolate (Maat 1966). In agar-gel diffusion tests, all six red currant isolates found in The Netherlands reacted similarly. (See "Cucumber Mosaic Virus in Raspberry," p. 191, for details of particle morphology and properties in vitro.)

CMV can be transmitted by many aphid species, including some that live on *Ribes* species (Kennedy et al. 1962), and the virus occurs commonly in many weed species. In view of the nonpersistent character of the virus, red currant can probably be infected by any aphid vector of CMV that feeds temporarily or by chance on currant. A high resistance to infection and the nonsystemic behavior of CMV in red currants may be the reasons why infection occurs rarely.

### Detection and Identification

The symptoms of green mottle may be confused with those of other diseases. Preliminary diagnosis should be checked by sap inoculation to *C. quinoa* and cucumber. The identification must be confirmed by serological tests.

### Control Procedures

The virus can easily be controlled by selection and propagation of healthy planting material.

### Remarks

CMV has also been found in black currants (Thresh 1966) (see p. 136) and in *Ribes aureum* Pursh (Schmelzer 1963).

## Nematode-Borne Diseases

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**Spoon Leaf of Red Currant //**  
By F. A. van der Meer

### Additional Common Names

Lepelblad; Löffelblättrigkeit.

### History and Geographic Distribution

Spoon leaf disease was first reported from The Netherlands by Maarse (1926, 1938b) who mentioned that the disease occurred in patches and suggested that it could be caused either by a virus or by soil conditions.

The infection was later found to be caused by a soil-borne virus (van der Meer 1960, 1965a), subsequently shown to be a strain of raspberry ringspot virus (RRV) (Harrison 1961; Maat et al. 1962). The early symptoms of infection are similar to those previously attributed to red currant ringspot virus (Klessner 1951), now considered to be synonymous with RRV.

Infection in red currant has been reported from The Netherlands and from East (Richter et al. 1966) and West Germany (Schuch 1963).

### Economic Importance

The Bangert in The Netherlands is the only known area where the disease is epidemic in red currants. Plants becoming naturally infected in the first years after planting often exhibit strong growth reduction. Cuttings from infected bushes give rise to very weak young plants (Maarse 1938a). Kleinhempel (1970) mentions a crop reduction of 32% for artificially infected bushes of cv. 'Rote Spätlese'.

### Symptoms on Natural and Experimental Hosts

Naturally infected red currants often show a bright yellow mosaic and ringspots as shock symptoms in the first 1 or 2 yr after infection (figs. 170 to 172). In later years, infected plants show leaf deformation; whereas mosaic and ringspot do not appear. Depending on the cultivar and the virus strain involved, leaf deformations may differ very much in severity. Leaves of some infected bushes only show a slightly reduced dentation, whereas leaves of other infected bushes become almost round with very little dentation (figs. 173 and 174). Margins of the leaves commonly curl down or up, creating a spoon-shaped appearance.

Experimentally infected *Chenopodium quinoa* Willd. develops necrotic local lesions and systemic necrosis. *C. amaranticolor* Coste & Reyn. shows only local lesions. The virus causes a bright systemic yellowing in tomato and in petunia. In this respect, RRV from red currant differs from

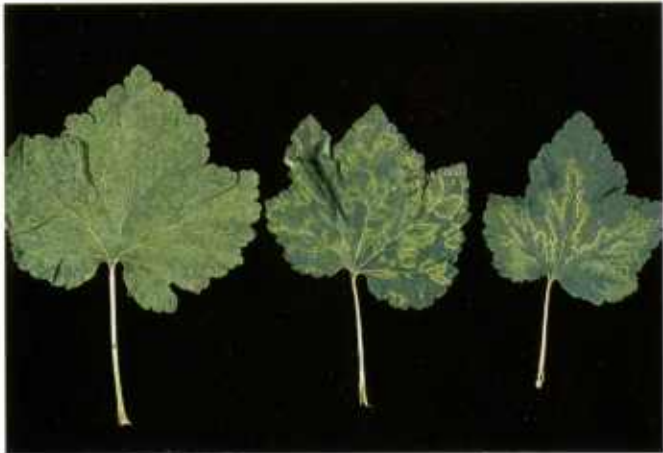


Figure 170.—Various shock symptoms of spoon leaf in cv. 'Fay's Prolific'; that is, mosaic, rings, and leaf pattern.

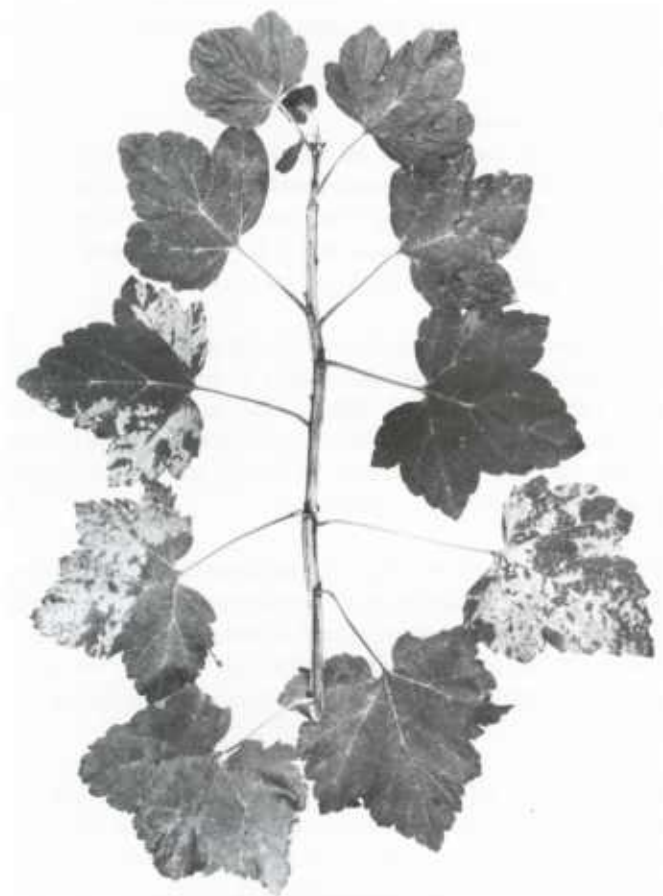


Figure 172.—Sequence of shock symptoms and chronic symptoms of spoon leaf in shoot of cv. 'Fay's Prolific' after natural infection.

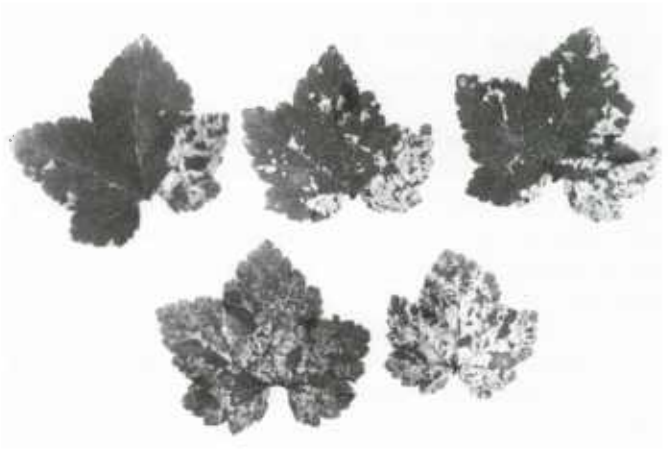


Figure 171.—Type of mosaic that is most common as a shock symptom of spoon leaf in cv. 'Fay's Prolific'.

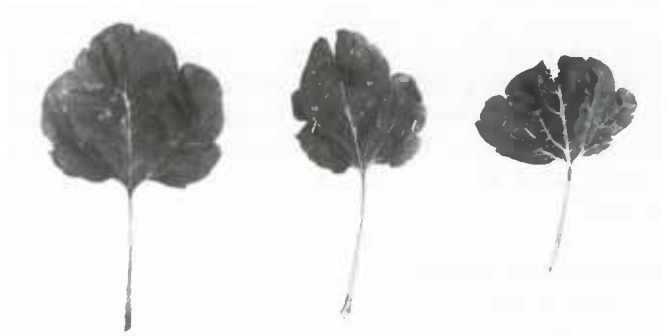


Figure 173.—Typical symptoms of spoon leaf in cv. 'Fay's Prolific'. Leaves are almost round, with little dentation.

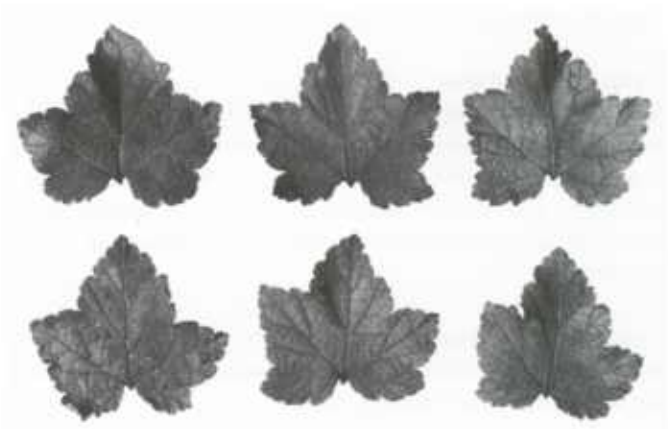


Figure 174.—Leaves of healthy 'Fay's Prolific'.

Scottish RRV strain (Harrison 1961; van der Meer 1965a). (See the *Rubus* section, p. 211, for further details on symptoms in other herbaceous hosts.)

### Natural and Experimental Transmission

The virus is transmitted naturally by *Longidorus elongatus* (de Man.) (van der Meer 1965a). This nematode transmits the virus to red currants and to several weed species. Gooseberries and black currants are rather resistant to natural infection.

Experimentally, the virus can be transmitted to gooseberry and black currant by grafting. By means of sap inoculation, the virus can be transmitted from red currant to many herbaceous hosts.

### Properties of the Causal Agent

Red currant spoon leaf is caused by a strain of RRV that is serologically indistinguishable from Scottish isolates obtained from raspberry. The longevity of currant isolates in vitro is 3 to 4 wk at 18°C., and the thermal inactivation point is 70° to 75°C. (See "Raspberry Ringspot and Associated Diseases of *Rubus* Caused by Raspberry Ringspot and Tomato Black Ring Viruses," p. 211, for further properties of RRV.)

### Detection and Identification

The early symptoms of infection may resemble those of other viruses and viruslike diseases, and the spoon leaf condition may be difficult to distinguish from reversion or other leaf abnormalities. Consequently, detection should be done by sap transmission to herbaceous hosts, whereas identification must be confirmed by serological tests.

### Control Procedures

Stocks for propagation should be tested to ensure that they are free of infection. Preplant treatment with dichloropropane-dichloropropene, and possibly other nematicides, provides a good control of the disease when red currants have to be planted in soil where the virus and its nematode vector are present.

There is no information on therapy of infected plants; however, there is as yet no need for therapy since no cultivars are known to be universally infected.

### Remarks

In testing procedures, transmission of the virus by sap inoculation from currant to herbaceous hosts is greatly facilitated by adding 2% nicotine solution to the inoculum.

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## Infection of Red Currant With Arabis Mosaic and Strawberry Latent Ringspot Viruses<sub>1/</sub>

By F. A. van der Meer

### Additional Common Names

No common names are justified, according to present information on symptomatology.

### History and Geographic Distribution

Arabis mosaic virus (AMV) and strawberry latent ringspot virus (SLRV) were first isolated from red currant while testing an English collection of cultivars by sap inoculation to herbaceous hosts (Thresh 1967). The same viruses were not detected in many similar tests done over a period of 6 yr in The Netherlands. AMV was isolated from material of French bushes sent in by B. Lantin (F.A. van der Meer, unpublished data). The infected bushes were part of a cultivar collection at Angers and had been imported recently from elsewhere. Both AMV and SLRV have been detected in red currant in East Germany (Kleinhempel 1968).

### Economic Importance

According to the present information, infection seems to occur only incidentally. Kleinhempel (1970) found little or no effect of SLRV on cropping of red currant cv. 'Rote Spatlese'. There is no further information on the effect of AMV and SLRV on growth and cropping of red currants.

### Symptoms on Natural and Experimental Hosts

Naturally infected red currants usually do not show symptoms with either virus (Thresh 1967). One out of two SLRV-infected bushes found by Kleinhempel (1968) showed bright yellow on only a few leaves, whereas 16 AMV-infected bushes did not show any symptoms.

Young red currant seedlings, experimentally infected with AMV by sap inoculation from herbaceous hosts, developed slightly discolored leaves. Graft-inoculated black currant bushes of the principal commercial cultivars reacted with a yellow mottle of the leaves that becomes inconspicuous by midsummer.

Experimentally infected *Chenopodium quinoa* Willd. and *C. amaranticolor* Coste & Reyn. developed faint chlorotic local lesions, followed by systemic mosaic and stunting, often accompanied by collapse and death of the apex.

### Natural and Experimental Transmissions

Both viruses are naturally transmitted, presumably by the nematode *Xiphinema diversicaudatum* (Micoletsky), which is known as a vector in other crops.

Experimentally, the virus can be transmitted from currant to currant by grafting and from currant to herbaceous hosts by sap inoculation. Addition of 2% nicotine solution to the inoculum facilitates transmission from currants to herbaceous plants.

### Properties of the Causal Agent

AMV and SLRV isolates from red currant closely resemble those obtained from strawberry. (See the *Rubus* section, p. 204, for further details on properties of AMV and SLRV.)

### Detection and Identification

The disease is detectable by sap inoculation to *C. quinoa*. Identification is only possible by serological tests.

### Control Procedures

Selection, indexing, and propagation of healthy planting material should be done in places where the nematode vector does not occur.

There is no information on therapy of infected plants.

### Tomato Ringspot Virus in Red Currant

By H. E. Williams, Q. L. Holdemann, and R. Casper

#### Additional Common Names

In red currant, the following names have been used: currant mosaic (Hildebrand 1939); American currant mosaic (Hildebrand 1942); and tomato ringspot (Hildebrand 1942). Strains of tomato ringspot virus (TomRSV) infecting various hosts cause diseases known as tomato ringspot, peach yellow-bud mosaic, and grape yellow vein.

#### History and Geographic Distribution

Naturally infected red currant is known in the United States—where the disease has been reported from New York, New Jersey (Hildebrand 1939, 1942), and California (H. E. Williams and G. Nyland, unpublished data)—and the U.S.S.R. (Eastern Siberia) (Gordejchuk et al. 1977). TomRSV may have been disseminated in infected planting material to other parts of the world. TomRSV has been found in Yugoslavia in American cultivars of red raspberry ('Geneva' and 'Hilton') (Jordović et al. 1972).

#### Economic Importance

Hildebrand (1939) reported reduction in bearing and occasional killing of plants.

#### Symptoms on Natural and Experimental Hosts

In red currant: At temperatures below 21°C., TomRSV causes symptoms consisting of varying degrees of chlorotic spotting and vein banding (fig. 175). Chlorosis may vary from a few spots or blotches, to a mild vein banding in a leaf, or to a chlorotic leaf with only a few green spots. The chlorotic areas may die and turn brown. On a single plant, the amount of chlorosis may vary from mild symptoms on a single leaf to conspicuous yellowing of the entire plant. In a critical temperature range (21° to 27°C.), the symptoms on young leaves are limited to chlorotic spots and rings. No symptoms appear on new growth formed at temperatures above 27°C.

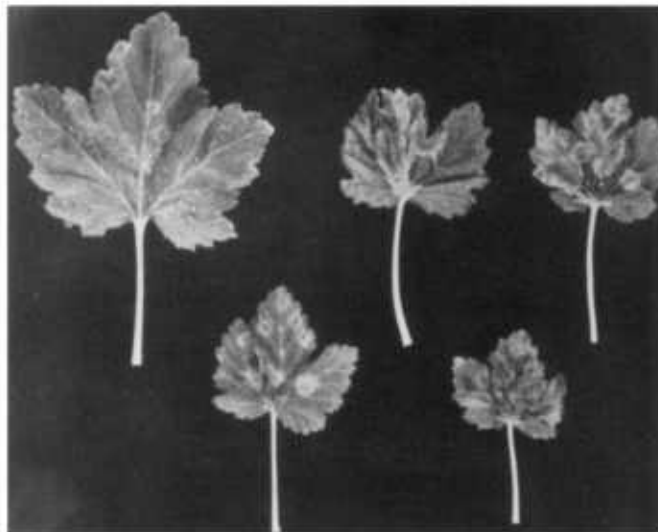


Figure 175.—Tomato ringspot virus in red currant. (Courtesy of E. M. Hildebrand and The Botanical Society of America.)

In herbaceous hosts: Experimental host range is wide; species in more than 35 dicotyledonous and monocotyledonous families are susceptible. In crops, the virus occurs mostly in ornamentals and woody or semiwoody plants, but also occurs naturally in many annual weed species. TomRSV is transmissible by sap inoculation, readily to herbaceous hosts but with difficulty to woody hosts (Stace-Smith 1970).

The host ranges of some TomRSV isolates differ, including differences between the two red currant isolates.

#### Diagnostic species

*Chenopodium amaranticolor* Coste & Reyn. and *C. quinoa* Willd. Small chlorotic local lesions; systemic apical necrosis.

*Cucumis sativus* L. (cucumber). Local chlorotic spots; systemic chlorosis and mottle.

*Phaseolus vulgaris* L. (bean). Chlorotic local lesions; systemic rugosity and necrosis of tip leaves.

*Nicotiana tabacum* L. (tobacco). Necrotic local spots or rings; systemic etched ring and line patterns. Leaves produced later are symptomless but contain virus.

*Petunia hybrida* Vilm. (petunia). Local lesions; necrotic collapse of young leaves.

*Lycopersicon esculentum* Mill. (tomato). Local necrotic flecks; systemic mottle and necrosis.

*Vigna unguiculata* L. (Walp.) (cowpea). Chlorotic or necrotic local lesions; most isolates cause systemic tip necrosis.



## Propagation species

*Nicotiana tabacum* or woody plants such as raspberry or currant are suitable for maintaining cultures. Cucumber or petunia are good sources of virus for purification.

## Assay species

*Vigna unguiculata*, *Nicotiana tabacum*, *Chenopodium amaranticolor*, and *C. quinoa* are useful local lesion hosts. Cucumber is useful as a source and bait plant for nematode transmission experiments (Téliz et al. 1966).

## Natural and Experimental Transmission

No information is available on natural spread in currant. The nematode *Xiphinema americanum* Cobb (and probably related species), a known vector of TomRSV, has been observed colonizing field soil under a monoculture of currant. Presumably, TomRSV could be introduced into a new area in infected plants, cuttings, scions, corms, and seed of cultivars and weeds. If a nematode vector were present, the virus could become established and perpetuated in weeds and other plants.

TomRSV has been transmitted from red currant to herbaceous hosts by sap inoculation, but from currant to currant only by grafting. Sap inoculation from herbaceous hosts back to red currant has not been achieved. The incubation period in currant is unknown but in herbaceous hosts, it is 3 to 7 days. There is no evidence of transmission through the seed of red currant, although this has been indicated or demonstrated for TomRSV in other hosts (Price 1936; Kahn 1956; Mellor and Stace-Smith 1963).

## Properties of the Causal Agent

The disease is caused by TomRSV (Price 1936). For details, see "Tomato Ringspot Virus in *Rubus*," p. 223, and Stace-Smith (1970). The New York red currant isolate was demonstrated to be related to Price's original TomRSV isolate by cross-protection tests (Hildebrand 1942). The California isolate is assumed to be the same as, or related to, TomRSV.

## Detection and Identification

Detection of TomRSV is made by sap inoculation from young tips of currants to young, vigorous plants of several herbaceous hosts, for example, cowpea, bean, cucumber, *Chenopodium amaranticolor*, and tobacco. Identification is made serologically with antisera to the type strain of TomRSV by agar gel test or ELISA.

## Control Procedures

Plants for propagation should be grown only from indexed, certified, virus-tested sources. The results of Hildebrand and Weber (1944) suggest the possibility of exploiting cultivar resistance. No information is available on therapy of this virus in red currant.

## Remarks

Hildebrand's original hypothesis (1942) that TomRSV in red currant and currant mosaic virus (CurMV) (Hildebrand 1939) are distinct viruses was based on the following: (1) TomRSV was not detected in two collections of plants showing currant mosaic symptoms, and (2) symptoms attributed to TomRSV in red currant were different from those attributed to CurMV. In later unpublished works, however, E. M., Hildebrand (personal communication) determined that only currant mosaic symptoms appeared on plants at temperatures below 21°C. The two sets of symptoms could be induced sequentially in a given plant by altering the temperature at which the plant is grown. Based on this evidence, he concluded that American currant mosaic is caused by TomRSV.

Until the problem of mechanical transmission of TomRSV to currant is resolved, we assume that the two distinct sets of symptoms described by Hildebrand (1939, 1942) are caused by TomRSV.

TomRSV has never been recovered from plants not showing symptoms of currant mosaic, either in natural infections (Hildebrand 1942; H. E. Williams and G. Nyland, unpublished data) or in graft-infected plants (Hildebrand and Weber 1944).

No relationship is known between American currant mosaic caused by TomRSV and European currant mosaic. (See "Yellow Leaf Spot of Red Currant," p. 152.)

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## Leaf Pattern of Red Currant// By F. A. van der Meer

## Additional Common Names

None, but the disease is caused by tobacco rattle virus (TRV).

## History and Geographic Distribution

The disease was first found in a single bush of cv. 'Fay's Prolific' in The Netherlands. The virus isolate from this bush, obtained by sap inoculation to herbaceous hosts, did not react with antisera against known viruses of *Ribes* (van der Meer 1970), and no viruslike particles could be detected. Later on, the disease was found in two bushes of cv. 'Jonkheer van Tets'. Isolates, made from these bushes, contained long and short particles, typical for TRV, and reacted positively with TRV antisera (F. A. van der Meer, unpublished data).

## Economic Importance

Because of its rare occurrence, the disease is of little economic importance.

## Symptoms on Natural and Experimental Hosts

Naturally infected red currants show sharply defined oak leaf patterns on leaves of some branches (fig. 176). Symptoms may remain restricted to certain branches for several years



Figure 176.—Symptoms of leaf pattern in cv. 'Fay's Prolific', caused by tobacco rattle virus.



Figure 177.—Rings in leaf of *Ribes sanguineum* cv. 'King Edward VII', caused by tobacco rattle virus.

and often are only present in some of the first-formed leaves. Oak leaf patterns are sometimes accompanied by light-green mosaic (fig. 177).

Experimentally infected *Chenopodium quinoa* Willd. always reacts with sharply defined lesions after 2 or 3 days. The isolate obtained from cv. 'Fay's Prolific' caused necrotic local lesions in *Nicotiana tabacum* L. and no systemic symptoms. Isolates obtained from cv. 'Jonkheer van Tets' caused local and systemic necrosis in *N. tabacum* L. (fig. 178).

#### Natural and Experimental Transmission

TRV is known to be naturally transmitted by nematodes of the genus *Trichodorus* to several crop hosts and many weeds (Harrison 1970). Presumably, red currants are infected in the same way.

Experimentally, the virus can be transmitted from red currant to herbaceous hosts by sap inoculation. Such transmissions



Figure 178.—Necrotic lesions in leaf of tobacco, caused by an incomplete strain of tobacco rattle virus from red currant.

must be done in April and May, and the inoculum should be prepared by macerating leaves in a 2% nicotine solution.

#### Properties of the Causal Agent

Because TRV could only be detected in leaves showing symptoms, we believe that TRV causes red currant leaf pattern.

Complete TRV isolates produce long (about 190 nm) and short (45-115 nm) particles. Long particles contain the genetic information for the production of infectious RNA, whereas the short particles contain the genetic information for the production of the coat protein of the particles. Incomplete isolates produce only infectious RNA. They can be obtained from complete isolates by using inocula that contain only long particles and have also been found in naturally infected plants (Harrison 1970). Incomplete isolates are less stable than complete isolates and often remain local in infected plants. The isolate obtained from cv. 'Fay's Prolific' was probably incomplete (fig. 178). Similar isolates have been obtained from lilac (van der Meer 1976a). Two of these have been converted to complete isolates by inoculating them together with the short particles of a complete red currant isolate (Huttinga 1976).

#### Detection and Identification

Because symptoms of leaf pattern disease of red currant are easily confused with symptoms of other diseases, detection is

only possible by sap inoculation to herbaceous hosts. Serological tests are necessary for identification of the virus; however, incomplete isolates (short particles) do not respond to such tests because they consist only of RNA without a protein coat.

### Control Procedures

Healthy stock material should be tested by sap inoculation on *C. quinoa* and should not be propagated in soil where both virus and vector occur.

There is no information on therapy of infected plants; however, for practical purposes, therapy is not necessary because very probably no cultivars are universally infected.

### Remarks

Incomplete TRV isolates have been obtained from *Ribes sanguineum* Pursh showing ringspot and leaf pattern (Schmelzer 1970). Symptoms in *R. sanguineum* are often restricted to a few leaves. In experiments of van der Meer (1976b), TRV could be isolated only from leaves with symptoms, which indicates that TRV indeed is the cause of ringspot and leaf pattern in *R. sanguineum* (fig. 177).

## Vectors Unknown

### 245 Yellow Leaf Spot of Red Currant //

By F. A. van der Meer

### Additional Common Names

Aucuba mosaic; European currant mosaic.

### History and Geographic Distribution

Yellow leaf spot of red currant has been described and referred to as aucuba mosaic in Czechoslovakia (Blatný 1930) and The Netherlands (Houtman 1951; van Katwijk 1953; van der Meer 1961), and as European currant mosaic in Great Britain (Thresh 1967). Similar symptoms have also been found in West Germany (Schuch 1957), East Germany (Kleinhempel 1968), and France (Lantin 1968).

### Economic Importance

The disease is of minor importance because it is quite uncommon, and severe effects on growth and cropping have not been reported.

### Symptoms on Natural and Experimental Hosts

Naturally infected red currants cv. 'Fay's Prolific' and several unnamed old Dutch cultivars develop small light-green or white spots that are scattered over the whole leaf (fig. 179). Symptoms may vary in severity between years. The outer margin of the leaves occasionally becomes light yellow. Infected plants appear somewhat stunted.

Experimentally infected bushes of cvs. 'Laxton's No. 1' (Thresh 1967) and 'Fay's Prolific' (Kleinhempel 1968) may show pale-yellow patches or bands along the main veins. Seedlings from cv. 'Jonkheer van Tets' show rather mild symptoms, whereas infected black currant seedlings of cv. 'Baldwin' remained symptomless.

### Natural and Experimental Transmission

There is no information on the way and rate of natural spread in red currants. At one locality in The Netherlands, however, a patch of diseased bushes occurred across the boundary between two farms where bushes of different origins had been planted on each farm, thus indicating natural spread of the disease.

Experimentally, the disease can be transmitted between currants by grafting.

### Properties of the Causal Agent

Symptoms and graft transmissibility indicate that yellow leaf spot of red currant is caused by a virus. Negative results of sap inoculation experiments, reported by some authors (van der Meer 1966; Thresh 1967; Kleinhempel 1968), suggest

## Interveinal White Mosaic

By F. A. van der Meer

### Additional Common Names

Tussenervig wit mozaïek. Alfalfa mosaic virus (in Great Britain) and an unidentified virus (in The Netherlands) have been associated with this disease.

### History and Geographic Distribution

The disease was first described in The Netherlands (van der Meer 1961), where it had been found in five separate bushes of cv. 'Fay's Prolific', each of which was situated in a different area. In Great Britain, a similar disease has been found in only one bush of 'Laxton's No. 1' (Thresh 1967).

### Economic Importance

Infected bushes are somewhat stunted, but infection is not sufficiently widespread to cause important economic losses.

### Symptoms on Natural and Experimental Hosts

Naturally infected bushes of cv. 'Fay's Prolific' show light-yellow or white patches that are often situated in the angles between the main and secondary veins. Symptoms of interveinal white mosaic in red currant seedlings are shown in figures 180 and 181. Often, first-formed leaves are also chlorotic. Later-formed leaves often show only a bright-yellow or white margin. The late symptom is of limited diagnostic value, for it may also occur in yellow leaf spot-infected bushes. (See 'Yellow Leaf Spot of Red Currant,' p. 152.) Moreover, white leaf margins occasionally occur also in bushes that do not show other viruslike symptoms and from which no sap-transmissible viruses can be isolated.

Black currant and *Ribes sanguineum* Pursh graft inoculated with Dutch sources develop symptoms that resemble those in red currant. Black currant cultivars graft inoculated with the English source, developed very mild symptoms that can be easily overlooked.

Sap inoculations from 'Fay's Prolific' caused mosaic and chlorosis on china aster (*Callistephus chinensis* (L.) Nees) (fig. 182). On *Nicotiana rustica* L., symptoms may vary greatly in severity. During winter and spring, local yellowing and small gray rings may appear 7 to 10 days after inoculation. These symptoms become systemic, and infected plants are stunted. In summer, the symptoms are inconspicuous and may be hardly visible. No symptoms were obtained on *Phaseolus vulgaris* L. A virus isolate obtained from the English 'Laxton's No.1' source induced necrotic local lesions and systemic mosaic in *C. quinoa* and was identified serologically as a strain of alfalfa mosaic virus (ALMV).

### Natural and Experimental Transmission

Natural spread has not been observed, and infection has only been found in incidental bushes. With the Dutch isolates, no experiments have been done with aphids or other potential

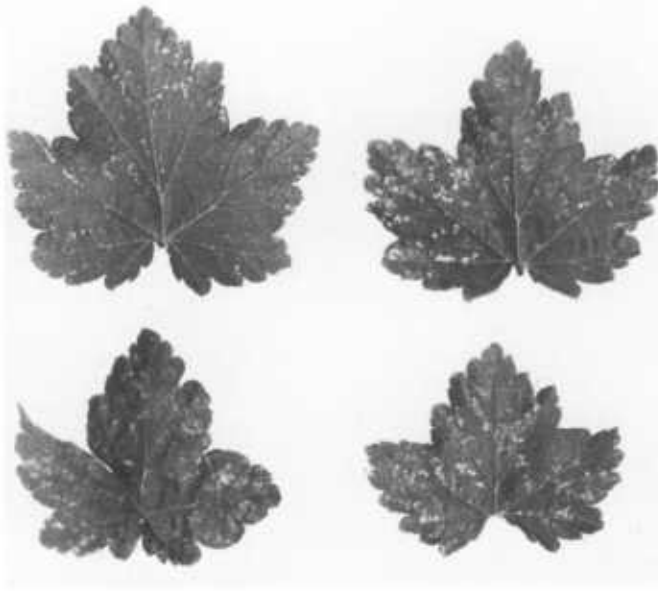


Figure 179.—Yellow leaf spot in 'Fay's Prolific' red currant.

that the causal virus is not mechanically transmissible. By sap inoculation to *Nicotiana glutinosa* L., however, Jacob (1976a) isolated potato virus (PVY) from five red currant cultivars that showed yellow leaf spot. This virus occurred latently in several other red and white currant cultivars (See "Remarks," p. 153.)

### Detection and Identification

This disease is detectable by its symptoms and by graft transmission to the sensitive cvs. 'Laxton No. 1' and 'Fay's Prolific'. Sap transmission to *Chenopodium quinoa* Willd. should be attempted to check for the presence of interveinal white mosaic, which can be confused with yellow leaf spot in certain seasons. (See "Interveinal White Mosaic," p. 153.) Sap transmission to *N. glutinosa* or *Solanum demissum* Lindl. cv. 'A6' should be attempted to check for the possible presence of PVY (De Bokx and Huttinga 1981).

### Control Procedures

The disease has never been observed in plantations that had been started with certified planting material. This suggests that selection and propagation of healthy stocks give adequate control.

### Remarks

Jacob (1976a) also isolated PVY from reversion-infected black currants and concluded from his further experiments that PVY is the cause of black currant reversion. Other workers, however, have not been able to confirm the presence of PVY in reverted black currants (R. Casper, H. Krczal, and D. Lesemann, unpublished results; Thresh et al. 1978). (See also "Reversion of Black Currant," p. 133.)



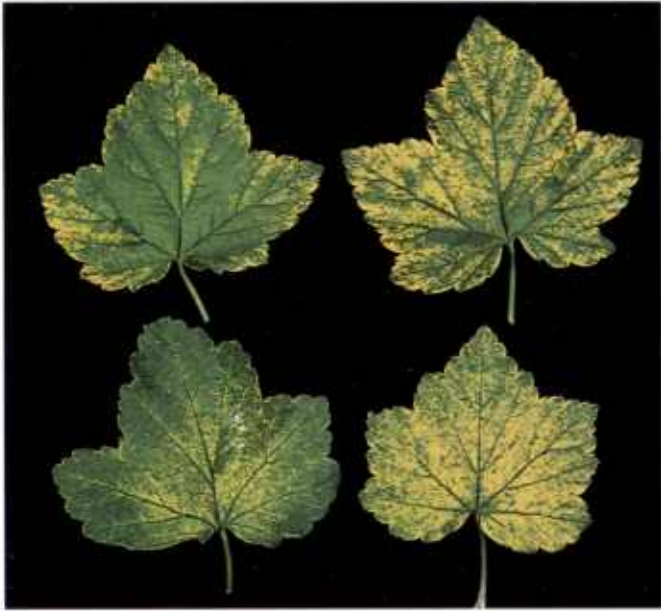


Figure 180.—Severe symptoms of interveinal white mosaic on first leaves of a red currant seedling in the spring.

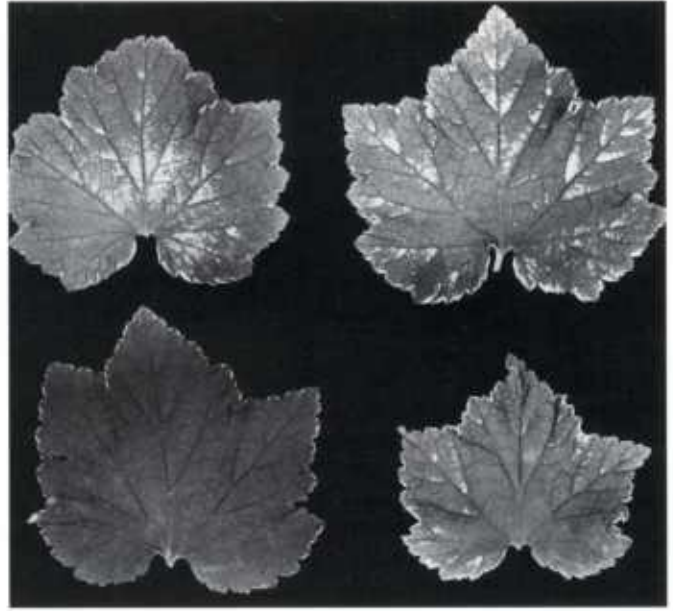


Figure 181.—Symptoms of interveinal white mosaic in a red currant seedling.

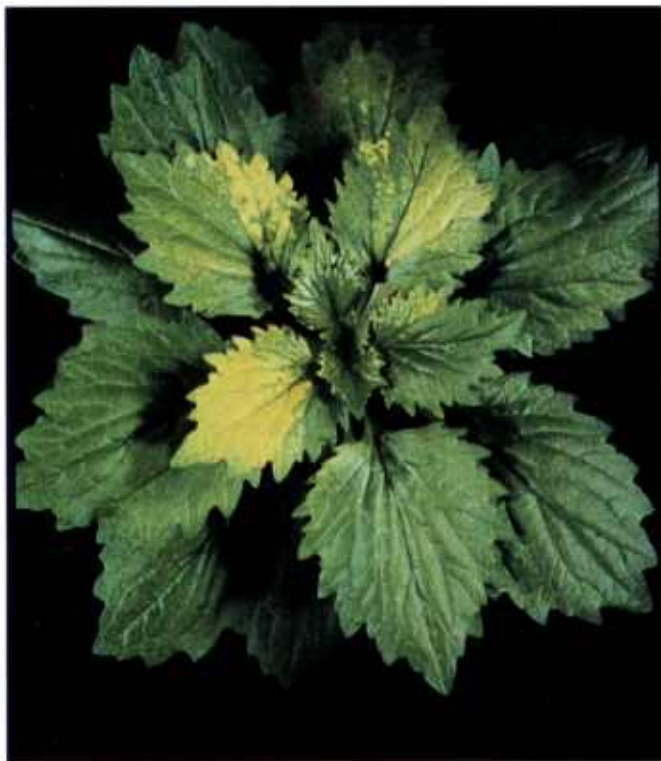


Figure 182.—Mosaic and chlorosis in *Callistephus chinensis* sap-inoculated from 'Fay's Prolific' showing interveinal white mosaic.

vectors. ALMV is known to be transmitted by many aphid species (Jaspars and Bos 1980) and occurs in many wild and cultivated plant species, so the English source of interveinal white mosaic may have resulted from aphid transmission.

Experimentally, the disease can be transmitted between currants by grafting. Both ALMV and an unidentified virus

found associated with this disease in The Netherlands can be transmitted from currants to herbaceous hosts by sap inoculation.

#### Properties of the Causal Agent

Two of the five sources found in The Netherlands were also infected with raspberry ringspot virus and have not been investigated further. Isolates from two other sources have been studied more intensively. Serologically, they were not related to nepoviruses or to ALMV, and no particles could be found in crude sap by using negative staining methods. Both isolates had a thermal inactivation point of about 45°C, and tobacco sap remained infective for 8 h, but not for 24 h at 20°C (F. A. van der Meer, unpublished data).

The ALMV isolate from the English source of interveinal white mosaic has not been studied intensively. ALMV has bacilliform particles. The thermal inactivation point of the virus ranges between 50° and 70°C and crude sap remains infective from 1 to 4 days at 20°C. (For further information on properties of ALMV, see Jaspars and Bos 1980.)

Circumstantial evidence obtained from extensive field observations and indexing suggests that the Dutch isolates that are not fully identified, as well as the English ALMV isolate, are able to cause interveinal white mosaic in red currant. This, however, has not been confirmed by retransmission experiments from herbaceous hosts to red currants.

#### Detection and Identification

Interveinal white mosaic can be detected by the obvious chlorosis of the first leaves in spring and by the light-yellow or white patches in later-developing leaves. Subsequent tests on *C. quinoa* distinguish interveinal white mosaic from the

somewhat similar symptoms of yellow leaf, which is not associated with a sap-transmissible virus causing symptoms in *C. quinoa*. Viruses found associated with interveinal white mosaic induce local and systemic symptoms in *C. quinoa*, which distinguishes these viruses from cucumber mosaic virus and tobacco rattle virus found also in *Ribes* but causing only local symptoms in *C. quinoa*. (See chapters on these respective viruses in *Ribes*, p. 145 and 150.) Virus isolates should be further characterized by testing with antisera against ALMV and nepoviruses known from *Ribes*.

### Control Procedures

The limited occurrence suggests that the selection and propagation of healthy stocks will give adequate control. There is no information on therapy of infected plants.

### Remarks

In host-range studies, Schmelzer (1963) transmitted ALMV mechanically from *Petunia* to gooseberry. Infected plants showed a yellowish mosaic. Red currant inoculated in the same way in Schmelzer's experiments remained uninfected.

### Full Blossom of Red Currant //

F. A. van der Meer

### Additional Common Names

Plnokvetosti ribezle.

### History and Geographic Distribution

Described by Rakús (1971) as a disease of red currant cv. 'Houghton Castle' in Czechoslovakia. The disease has not been reported from other countries.

### Economic Importance

According to Rakús (1978), full blossom is an economically important disease in Czechoslovakia. It has been found in several red currant cultivars all over the country. Diseased bushes are reduced in size and produce sparse crops of small berries (Rakús 1975).

### Symptoms on Natural and Experimental Hosts

Flower malformations are the most typical symptoms of affected bushes (figs. 183 to 188). Stamens are often absent, whereas several styles per flower may be present instead of one. Ovaries on red currants infected with full-blossom disease are often half-inferior (fig. 187) or superior (fig. 188), while those on normal plants are regularly inferior (fig. 186). Petals may become sepal-like in appearance, and petals as well as sepals may enlarge and become leaf-like (fig. 189). Misshapen berries may take on a cauliflower-like structure (figs. 190-191) (Rakús 1971). These flower malformations suggest a relationship with yellows or witches'-broom diseases of plants, many of which are believed to be caused by mycoplasma-like organisms (MLO). Affected red currants, however, do not show witches'-broom symptoms or extreme yellowing.

Symptoms of full blossom have been found in naturally infected plants of the red currant cvs. 'Houghton Castle', 'Jonkheer van Tets', 'Erstling aus Vierlanden', and several unnamed cultivars. Graft-inoculated plants of red currant cv. 'Heinemann's Rote Spätlese' and black currant cv. 'Baldwin' became infected latently.

### Natural and Experimental Transmission

There is no information on the way and degree of natural spread. Experimentally, the disease can be transmitted by patch grafting.

### Properties of the Causal Agent

Full blossom has been found associated with MLO (Rakús et al. 1974). Since MLO could not be detected in healthy red currants, we believe these organisms cause full blossom; however, like so many other so-called MLO diseases of plants, Koch's postulates have not yet been fulfilled for this disease agent.

### Detection and Identification

The disease can be detected by the typical flower malformations. Nuclear stock material should be indexed by graft inoculation to cv. 'Houghton Castle.'

### Control Procedures

New plantations should be started with planting material derived from disease-free stock.

There is no information on therapy of infected material. Since hot water treatment is known to be effective for several MLO diseases of woody hosts (Nyland and Goheen 1969, van der Meer 1975), it would be interesting to see if full blossom can be cured in this way.

### Remarks

Full blossom of red currant shows some similarity to descriptions of severely malformed blossoms of black currant



Figure 183.—Racemes with symptoms of full blossom disease; healthy raceme (cv. 'Houghton Castle') at right.



Figure 184.—Normal red currant flower, x 29.  
(Courtesy of D. Rakús.)



Figure 185.—Red currant flower affected by the full blossom disease, x 14. (Courtesy of D. Rakús.)

(Ritzema Bos 1905; Hatton and Amos 1917). According to Adams and Thresh (see "Reversion of Black Currant," p. 133), this condition is not common in Great Britain and Western Europe. During surveys in The Netherlands, it was found very seldom and only in reverted black currant bushes. Such malformations are generally assumed to be part of the black currant reversion syndrome. Results of Rakús et al. (1974), however, indicate that full blossom of red currant is not related to reversion of black currant.

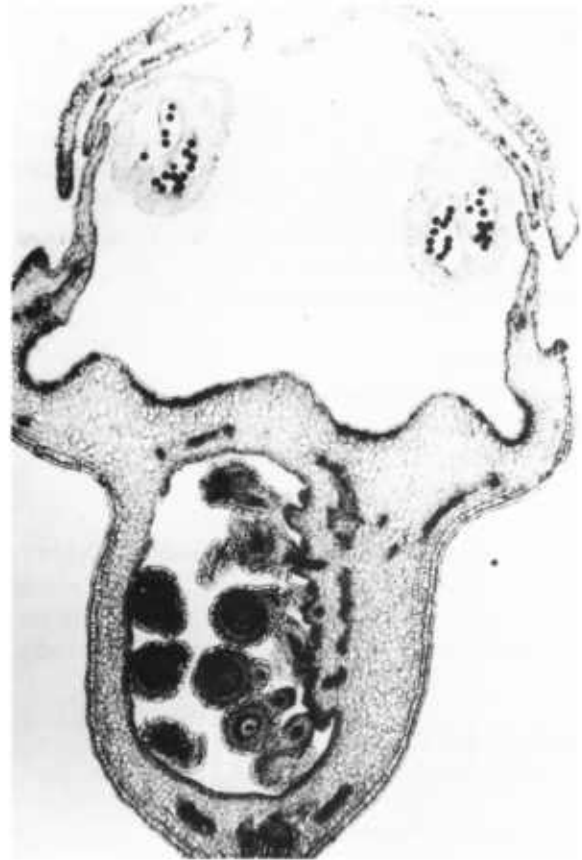


Figure 186.—Longitudinal section of normal red currant flower with inferior ovary, x 39. (Courtesy of D. Rakús.)

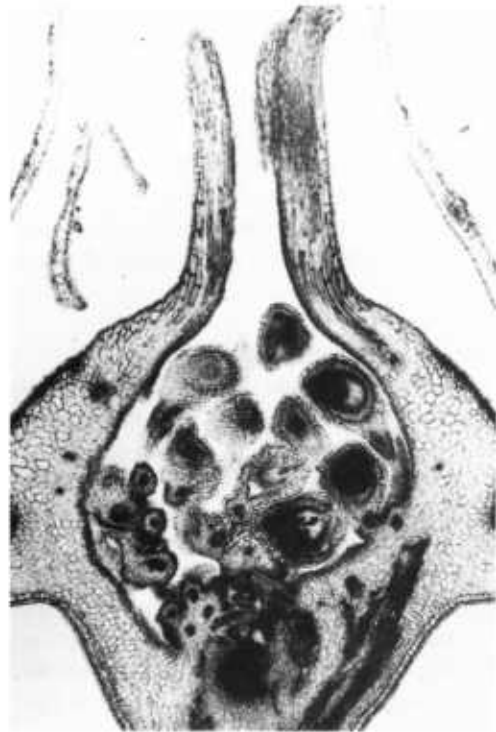


Figure 187.—Longitudinal section of abnormal flower with half inferior ovary from full blossom-diseased red currant, x 32. (Courtesy of D. Rakús.)



Figure 188.—Longitudinal section of abnormal flower with superior ovary from full blossom-diseased red currant, x 38. (Courtesy of D. Rakús.)



Figure 189.—Longitudinal section of abnormal flower from a full-blossom diseased red currant with "leaf-like" structures in the ovary, x 38. (Courtesy of D. Rakús.)

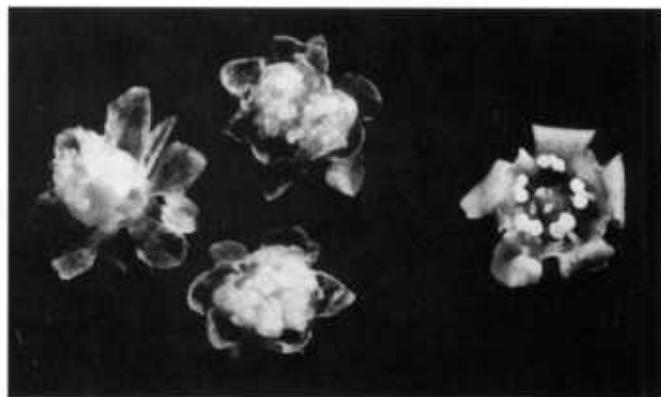


Figure 190.—Abnormal flowers with cauliflowerlike structures from full blossom-diseased red currant; normal flower at right. (Courtesy of D. Rakús.)

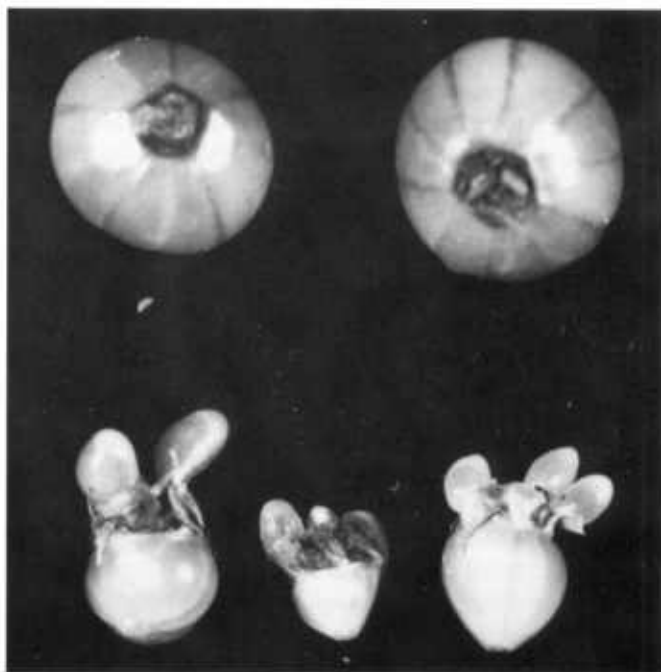


Figure 191.—Extrusion of seeds from berries from a full-blossom diseased red currant; normal berries at top. (Courtesy of D. Rakús.)



## Viruslike Disorders

### Leaf Malformation of Red Currant„

By F. A. van der Meer

#### Additional Common Names

Reversion of red currant.

#### History and Geographic Distribution

Red currant bushes with abnormally shaped leaves have been reported from Germany (Winter 1940; Schuch 1957; Behrens 1964), The Netherlands (Dijksterhuis 1950; van Katwijk 1953), Czechoslovakia (Blatný and Paulechova 1964), Great Britain (Thresh 1967), and France (Putz 1972). The condition is sometimes referred to as reversion of red currant; however, transmissibility of the disease has seldom been reported, and few attempts have been made to infect black currant.

#### Economic Importance

Natural spread of leaf malformation in red currants and serious effects on cropping have been reported from certain localities in The Netherlands, East Germany, Czechoslovakia. Elsewhere, certain cultivars have a tendency to produce malformed leaves, but there is no evidence of spread, and yields are little affected except in cvs. 'Fay's Prolific' and 'Rondom'.

#### Symptoms on Natural and Experimental Hosts

Naturally affected plants may show several apparently distinct leaf abnormalities. For example, bushes of certain Czech cultivars derived from *Ribes petraeum* Wulf. develop leaves that closely resemble those of black currant with reversion. The lobes and marginal serrations are fewer and more rounded than usual, although the bilateral symmetry of the leaves is unaffected.

At places in The Netherlands and East Germany where the disease was reported to be epidemic, affected bushes developed asymmetrical leaves with reduced numbers of lobes and marginal serrations. In black currant, such symptoms are known to be caused by gall mites. (See "Reversion of Black Currant," p. 133.)

Bushes of cv. 'Rondom' are particularly liable to develop abnormal leaves, and several different types occur. In the most extreme instances, the leaves are rugose, distorted, and asymmetrical (figs. 192 to 194).

A common leaf abnormality of 'Fay's Prolific' (fig. 195) may resemble symptoms of spoon leaf, but is associated with an unusual branching habit of this cultivar (See "Budlessness of Red Currant," p. 160.)



Figure 192. — Leaf malformation of red currant cv. 'Rondom'.



Figure 193. — Leaf malformation together with reduced cropping of cv. 'Rondom'.

#### Natural and Experimental Transmission

There is no information on the method of natural spread of the various types of red currant leaf malformation.

Experimentally, black currant reversion has been graft-transmitted from a naturally affected red currant clone to black currant. Black currant reversion, however, could not be detected in various other suspected sources in Great Britain (Thresh 1967) nor in suspected bushes of cvs. 'Herons' and 'Rondom' in West Germany (Baumann 1974).



Figure 194. — Healthy red currant cv. 'Rondom'.

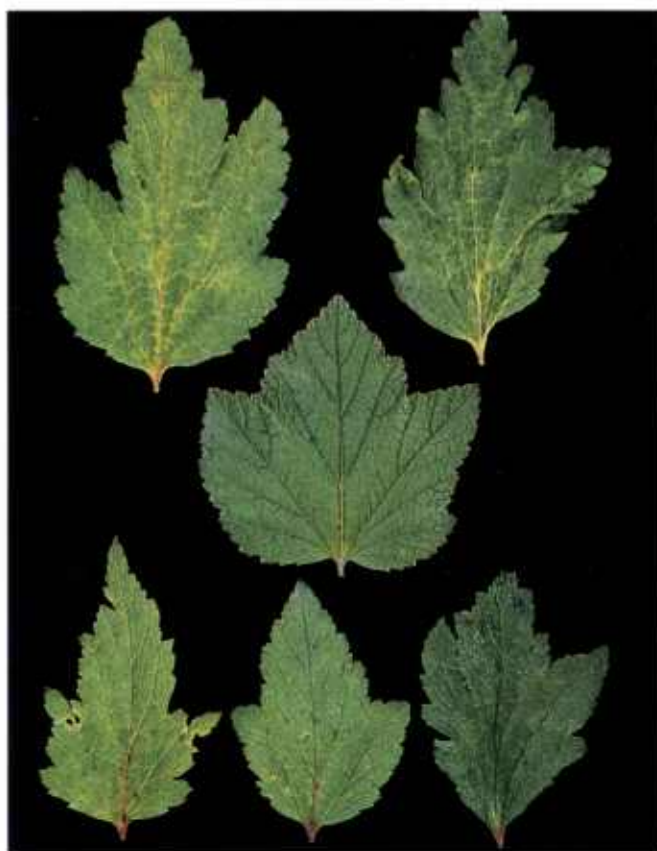


Figure 195. — Leaf malformation of red currant cv. 'Fay's Prolific'. Normal leaf in center.

None of the various types of leaf malformation in cv. 'Rondom' could be transmitted by grafting (van der Meer 1965b; Putz 1972).

#### Properties of the Causal Agent

Several apparently distinct causes of the various leaf abnormalities have been reported as follows:

1. Black currant reversion has been detected in a clone of *Ribes rubrum* L. that was virtually sterile and showed reversionlike symptoms. Black currant reversion is transmitted by the black currant gall mite *Cecidophyopsis ribis* (Westw.). The disease is caused by a graft-transmissible agent; however, properties of this agent are as yet unknown.

2. In East Germany and in The Netherlands, spread of leaf malformation was found to be associated with *C. selachodon* van Eyndhoven, an eriophyid mite that closely resembles the black currant gall mite, yet is a morphologically distinct species (van Eyndhoven 1967) that does not infest black currant (van de Vrie 1967). It is not known whether the leaf malformations involved were caused solely by mites or whether a virus or viruslike agent was also involved.

3. The instability of cv. 'Rondom' has been attributed to its labile genetic structure and complex origin from an interspecific cross (Rietsema 1955). Seedlings of cv. 'Rondom' are heterogeneous and often exhibit the same leaf abnormalities as the parent.

4. The common malformation in cv. 'Fay's Prolific' is associated with a peculiar branching habit. Neither mites nor graft-transmissible diseases have been implicated, and the condition seems to be an inherent characteristic of the cultivar.

#### Detection and Identification

The disease is only detectable by visual selection. To distinguish between reversion infection and genetical malformations, graft inoculations should be done on sensitive black currant cultivars.

#### Control Procedures

Stocks used for propagation should be carefully inspected to avoid perpetuating the gross leaf abnormalities affecting cultivars such as 'Fay's Prolific' and 'Rondom'.

Malformations caused by eriophyid mites can be avoided by the use of an appropriate acaricide; however, additional measures will be necessary if a graft-transmissible disease agent is also involved. Further research will be necessary before final recommendations can be made for localities where a leaf malformation is spreading.

No information is available on therapy of affected plants.

## Budlessness of Red Currant

By F. A. van der Meer

### Additional Common Names

Blind bud; knoploosheid.

### History and Geographic Distribution

Typical symptoms were first described in The Netherlands (Maarse 1936). They have since been reported from Germany (Winter 1940), England (Thresh 1967), and France (Lantin 1968; Putz 1972), and probably occur wherever sensitive cultivars are grown.

### Economic Importance

Considerable losses occur in nurseries and plantations where sensitive cultivars such as 'Fay's Prolific' are grown. The yield of cropping bushes is strongly reduced because of weak growth and a reduced number of flower-producing buds. Affected planting material is not suitable for sale because growth is unsatisfactory.

### Symptoms on Natural and Experimental Hosts

The condition is known only in red currant, especially in cultivars derived from *Ribes sativum* Syme (also known as *R. vulgare* Lam.). 'Fay's Prolific' is by far the most sensitive, although cvs. 'Versailles' and 'Rondom' are sometimes affected.

Shoot extension growth of affected bushes stops in early summer, often after leaves have been formed that are abnormally dark green and rounded, with reduced dentation (fig. 196). Axillary buds fail to develop or appear at some distance from the nodes (fig. 197). This condition has been termed "concaulescence" (Reinders 1957) and occurs also in other plants such as *Symphytum officinale* L. (Boraginaceae). Buds of affected growth are often small and may produce weak laterals with terminal and axillary buds that die or fail to develop (fig. 198).

In a survey during each of two winters, all observed bushes of cv. 'Fay's Prolific' were affected to some extent. Some were almost normal; whereas others were severely stunted and almost entirely affected.

There are indications that the budless condition is less severe in bushes that are not infected with red currant vein banding virus.

### Natural and Experimental Transmission

Natural and experimental transmission in 'Fay's Prolific' could never be established because no unaffected stocks of this cultivar have been located.

Seedlings of cv. 'Jonkheer van Tets' grew normally after grafting to affected shoots of cv. 'Fay's Prolific'. No transmission has been obtained in grafting experiments with

several cultivars (Putz 1972). Seedlings of cv. 'Fay's Prolific' are unstable indicators because they often develop budlessness by the time flowering commences.

### Properties of the Causal Agent

The budless condition is assumed to be genetical.

### Detection and Identification

In summer, diseased bushes can be recognized by their unusual leaves and dwarfed appearance. The malformed leaves alone are not sufficiently reliable for diagnosis because they may resemble those affected by spoon leaf disease. (See "Spoon Leaf of Red Currant," p. 146.) In winter, the very short and budless shoots are diagnostic for this disorder.

### Control Procedures

Maarse (1936) suggested that cuttings should be taken only from stocks that are little affected. Any abnormal bushes that appear in the nursery or in the early years of a bearing plantation should be removed. Affected shoots that appear subsequently should be pruned back to a normal lateral shoot or bud.

Several unsuccessful attempts were made to produce normal plants by growing bushes for several weeks at 37°C.

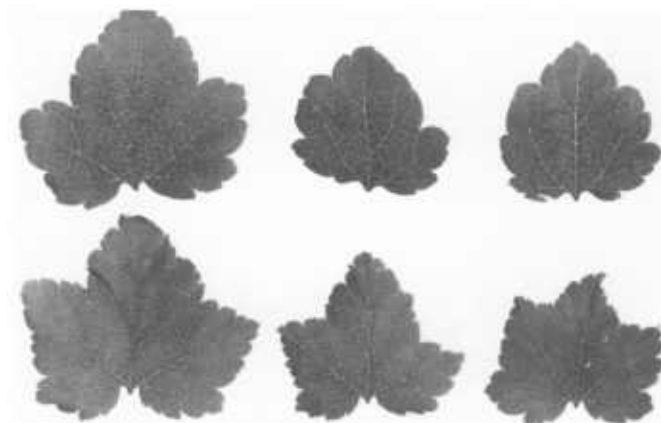


Figure 196. — Leaves of red currant affected with the budlessness disorder and showing abnormal rounding and reduced dentation, top row, compared with normal leaves, bottom row.



Figure 197. — Shoot of 'Fay's Prolific' red currant affected with the budlessness disorder with buds situated at some distance above the nodes.



Figure 198. — Two-year-old branch of 'Fay's Prolific' red currant with one-year-old short, budless laterals.



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## Section 4. Virus and Viruslike Diseases of *Rubus* (Raspberry and Blackberry)

### Introduction

By R. Stace-Smith

In "Virus Diseases of Small Fruits and Grapevines," Converse (1970c) discussed the occurrence, detection, and control of *Rubus* viruses. Our understanding and knowledge of these aspects of *Rubus* virus diseases has not altered appreciably during the intervening years since the first edition appeared. I will not attempt to update this introductory chapter, although I will comment on a few significant advancements. A marked improvement has been made in virus detection with the introduction and widespread use of the enzyme-linked immunosorbent assay (ELISA) technique (Clark and Adams 1977). For those *Rubus* viruses for which antiserum is available, this serological procedure has become an important means of virus detection. Unfortunately, its use is restricted to those viruses that can be purified in sufficient quantity to inject into a laboratory animal and produce a specific antibody. Virtually all of the true *Rubus* viruses, that is, those viruses whose natural hosts are restricted to the genus *Rubus*, either occur in low concentration or, for other reasons, have defied purification attempts. However, where some of the more damaging virus diseases affecting crops belonging to the genus *Rubus* are not restricted to that genus but infect a wide range of herbaceous and woody plants, and since these viruses can be purified, serological techniques such as ELISA can be usefully applied.

Advances in our basic knowledge of plant viruses in general has been considerable in the past decade, but advances in our knowledge and understanding of *Rubus* viruses has not changed dramatically. In fact, one could justifiably question whether there have been sufficient advances to warrant a revised edition of the handbook. I have no doubts about the value of the revised edition—although there has not been a dramatic increase in the knowledge, there have been a few major contributions and a number of minor contributions. Cumulatively, these have resulted in a significant update in the *Rubus* section of this as compared with the former handbook.

There is good reason why there have been relatively few major advances in our knowledge of *Rubus* viruses and virus diseases in recent years. The cumulative research effort devoted to *Rubus* viruses on a worldwide basis is relatively modest. Precise figures are unavailable but I would estimate that less than 10-person years are devoted to this field of research. A bibliography covering the period 1973-78 includes 76 references to *Rubus* virus diseases (Stace-Smith and Matsumoto 1979). A later compilation covering the period 1979-81, contains only 29 references to *Rubus* viruses (Stace-Smith and Matsumoto 1982), an average of about 10 citations per year.

One development that has had a positive influence on the exchange of information on *Rubus* virus diseases was the action of the Plant Protection Commission of the International Society for Horticultural Science in establishing a working group, "Virus Diseases of Small Fruits," in 1974. This working group was charged with the responsibility of organizing international symposia on small fruit virus diseases. To date, three symposia have been held, the first in Heidelberg, Federal Republic of Germany, in 1976; the second in Budapest, Hungary, in 1979; and the third in Vancouver, Canada, in 1982. In these three symposia, 8, 5, and 10 lectures, respectively, were on *Rubus* virus diseases. The proceedings were published in *Acta Horticulturae* Nos. 66, 95, and 129. Judging by the response of the participants, I anticipate that these international symposia will continue at 3-yr intervals for the foreseeable future. In addition to facilitating an exchange of information on *Rubus* virus diseases, the symposia provide a forum for keeping abreast of developments in virus diseases affecting other small fruit crops.

The ultimate objective of any research program on *Rubus* virus diseases is directed towards minimizing the adverse affects attributed to virus infections. In Western North America, we have basically reached this objective with those viruses that are transmitted by aphids. The raspberry mosaic disease, which was once widespread along the Pacific Coast of North America, is rarely a serious problem there today. The absence of mosaic is attributed to the fact that the older cultivars that supported populations of the aphid vector *Amphorophora agathonica* Hottes have been replaced by cultivars that are highly resistant to the aphid vector (Daubeney and Stary 1982). With a reduction in the importance of the aphid-borne viruses, we are experiencing what appears to be an increase in the importance of the pollen-borne viruses, particularly raspberry bushy dwarf virus (RBDV). The pollen-borne viruses present a unique problem in that there is no way to prevent their spread except by eradicating infected sources and using immune parents in breeding programs. Eradication of infected sources is not readily achieved because infected plants often exhibit either no obvious symptoms or symptoms that are so vague that conclusive diagnosis cannot be made by visual examination (Stace-Smith et al. 1982). Accurate diagnosis can be made by serological tests or sap transmission to indicator plants but, because of the time and expense involved, the extent of field surveys is curtailed. The problem can be overcome by concentrating the testing on nursery stock that is used to establish new plantings, but there is still the concern that these may become contaminated if planted near infected stock. As noted by Daubeney et al. (1982), it is preferable not to expose virus-tested stock of susceptible cultivars to pollen from infected sources. There is no information as to what

constitutes safe geographic isolation, but we assume that this distance would vary from cultivar to cultivar. In any event, we suggest that it would be unwise to establish a new planting of a susceptible cultivar adjacent to older plantings of unknown virus content.

To supply healthy planting material, certification schemes are now operating for several kinds of plants, including raspberry, and the raising of stocks for propagation is often separated from the growing of a crop for its main purpose. Special stocks that form the basis of certification schemes are built up by propagating from single virus-indexed plants. Until recently, the production of virus-tested stocks of plants that are grown as cultivars depended upon finding an uninfected plant of the cultivar to start the stock. Fortunately, this is no longer so, for methods have been devised whereby plants can be freed from some or all of the viruses that infect them. No useful clone need be abandoned because the whole clone is virus infected. The method that has found the widest application is meristem tip culture, usually taken from clonal material that has been subjected to a prolonged period of growth at a temperature that is considerably higher than would normally be used for plant production. Virus-tested raspberry clones are now available for all the newer cultivars, but some of the older cultivars that appear to be totally infected have not been revitalized by therapy procedures. It is a tedious process to produce a clone free of known viruses from a cultivar that is totally infected and, unless there is a demonstrated need for such a clone, it is doubtful whether the effort required is warranted. RBDV is of particular concern because it is common in clones that may be useful to plant breeders and a proportion of the progeny is infected. While RBDV can be eliminated from clonal stock, the process should not be necessary if the virus status of all clonal material to be used in breeding programs is established and selections that become infected are discarded.

What major advances will we see in *Rubus* virus disease research over the next decade? This is a difficult question to answer but, judging by current trends in plant virology, it is at least possible to speculate on a few possibilities. First, we will utilize advances that are being made in virus detection to extend field indexing and obtain a more accurate picture of precisely what viruses are present in the important *Rubus* growing regions of the world. A precise understanding of the problem is essential in devising control procedures and it is anticipated that, in many areas where raspberries and blackberries constitute a commercial crop, virus diseases will cease to be an appreciable concern. Second, improvements will be made in virus purification procedures to the point where some of the *Rubus* viruses whose properties are little understood will be purified, at least in minute quantities, and antiserum will be prepared using the monoclonal antibody technique. Finally, increased emphasis will be placed on developing new cultivars that are resistant or immune to the major *Rubus* virus diseases and their vectors.

## Aphid-Borne Diseases

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### Raspberry Mosaic<sup>1/</sup>

By R. H. Converse, R. Stace-Smith, and A. T. Jones

#### Additional Common Names

Red raspberry mosaic (Bennett 1927); type *b* mosaic (Harris 1933); green mottle mosaic = green mosaic (Cooley 1936*b*); raspberry Mosaic I (Harris 1939); veinbanding disease (Cadman 1952*b*) and veinbanding mosaic (Murant 1974*b*). Raspberry mosaic disease (RMD) is induced by infection with a complex of viruses. In Canada, black raspberry necrosis virus (BRNV) and rubus yellow net virus (RYNV) are together reported to induce raspberry mosaic disease in red raspberry (Stace-Smith 1956). In Europe, mosaic-affected plants usually contain, in addition, raspberry leaf mottle virus (RLMV) and raspberry leaf spot virus (RLSV). To what extent these two extra viruses are involved in the disease is not clear. Infection with each of the four viruses singly can induce symptoms in some *Rubus* species (see the chapters dealing with these specific viruses for detailed discussion on the common names associated with such symptoms) and, in some sensitive red raspberry cultivars, RLMV and RLSV induce a disease variously referred to as Mosaic 2, type *c* mosaic symptoms, or leaf spot mosaic. This disease is distinct from raspberry mosaic and is covered in detail in the chapter dealing with these two viruses.

#### History and Geographic Distribution

Wilcox and Smith (1924) first demonstrated a mosaic disease of raspberry that was induced by an agent(s) transmissible by the aphid *Amphorophora agathonica* Hottes, formerly designated *A. rubi* (Kalt.). The early literature on RMD was reviewed by Bennet (1927), Harris (1933, 1939), Stace-Smith (1956), and Cadman (1961*b*). Graft transmission was first demonstrated by Harris (1939) in Great Britain. Other papers that review RMD have been published by Converse (1966*a*, 1977), Converse et al. (1970*a*), Jones (1981*b*), Murant (1974*b*, 1976*b*), and Stace-Smith (1960*a*, 1968). Stace-Smith (1956) demonstrated that RMD in British Columbia was caused by dual infection with viruses transmitted by the aphid vector, *Amphorophora agathonica* Hottes. He named these black raspberry necrosis virus (BRNV) and rubus yellow net virus (RYNV). As already indicated, in Europe a second and distinct type of mosaic, described initially as type *c* symptoms (Harris 1933) and later as Mosaic 2 (Harris 1939) or leaf spot mosaic (Murant 1974*b*), is induced in sensitive raspberry cultivars by infection with either of the two distinct aphid-borne viruses RLMV and RLSV (Cadman 1951, 1952*d*). This chapter does not consider this second type of mosaic but does discuss the possible involvement of RLMV and RLSV in RMD.

RMD is worldwide in distribution, occurring virtually wherever red, black, or purple raspberries are grown. Crops in some regions are less severely affected than those in others, either because healthy planting material or specific cultivars are used, or because aphid vectors do not occur there. This disease was not observed in New Zealand, probably because symptomless plants were introduced and aphids of the genus *Amphorophora* do not occur (Jones and Wood 1979). In California, Oregon, and Washington, the climate and *Rubus* hosts appear to be unfavorable for the maintenance and development of large colonies of *A. agathonica*, and although RMD has been found in wild and cultivated *Rubus*, it is rare. In Great Britain, because healthy planting material is used and the acreage of cultivars that resist colonization by aphid vectors is increasing, RMD is a decreasing problem (Jones 1981c). RMD can be expected to decrease in prevalence in North America too as the acreage of aphid-resistant cultivars increases (Daubeney 1982). In parts of northern Europe and eastern North America, however, where older cultivars are still widely grown and *Amphorophora* vectors occur in large numbers, RMD is still widespread in cultivated and wild *Rubus*.

### Economic Importance

A range of symptoms is associated with RMD, from mild to severe mottling or veinbanding, dwarfing, and crumbly fruit, depending on the cultivar and the growing conditions. Yield losses due to RMD in some red raspberry cultivars were 11 to 14% in British Columbia (Freeman and Stace-Smith 1970) and 39% in Maryland (Converse 1963). Pollen abortion in affected plants of some red raspberry cultivars was twice that found in healthy plants (Freeman and Stace-Smith 1970), but drupelet set was unaffected (Daubeney et al. 1970). In Scotland, RMD was thought to decrease red raspberry yields by 28% (Wood and Anderson 1959). The effects on growth and yield of infection with the individual component viruses of the RMD complex are presented in the chapters dealing with these viruses. Strain differences within the individual viruses are possibly an additional source of variation in the severity of the disease complex, but this has not been quantified.

### Symptoms on Natural and Experimental Hosts

In cultivated black raspberry, RMD delayed foliation in the spring (Cooley 1936a). In affected plants, leaves produced during cool weather are mottled and blistered (fig. 199), but leaves formed during hot weather may be symptomless; young shoot tips are often necrotic and brittle. Canes are short and rosetted. Fruit yield is reduced and fruit is small, seedy, and of inferior flavor. Plants infected for a few years often fail to survive the winter.

In cultivated red raspberry, affected plants may show decreased vigor and may remain symptomless or exhibit mild to severe leaf mottling, blistering, and vein clearing or veinbanding, depending on the cultivars (fig. 200) and the



Figure 199. — Young shoot of black raspberry infected with raspberry mosaic disease, showing mottled, blistered leaves after leaf grafting with black raspberry necrosis virus- and rubus yellow net virus-infected red raspberry.



Figure 200. — Red raspberry cv. 'Malling Jewel' naturally infected in Scotland with raspberry mosaic disease and showing leaf puckering and veinbanding symptoms. (Copyright Scottish Crop Research Institute.)

prevailing temperature. Leaf symptoms often fade in hot weather. Pollen production, fruit yield, and quality are decreased in affected raspberry plants.

In cultivated blackberry, RMD has not been described as such, but some of the component viruses of RMD have been identified. Critical studies of their effects on growth and yield have not been made. In general, blackberry cultivars appear to be more tolerant of infection than raspberries, but plant vigor, fruit yield, and fruit quality are probably decreased by such infections.



Natural hosts of the four viruses associated with the RMD complex are all in the genus *Rubus* and are listed below.

Natural host list and RMD prevalence, where known:

*Rubus*, subgenus *Idaeobatus*

- R. idaeus* L. Widespread
- R. idaeus* L. var. *strigosus* (Michx.) Maxim. Widespread
- R. innominatus* S. Moore (Wilcox 1926)
- R. leucodermis* Dougl. (Zeller 1923; Huber and Johnson 1952)
- R. occidentalis* L. and hybrids with red raspberries. Widespread
- R. odoratus* L. (Zeller 1923)
- R. parviflorus* Nutt. (Zeller 1923)
- R. phoenicolasius* Maxim., a common wild host locally (Zeller 1923; Converse 1960)
- R. spectabilis* Pursh (Zeller 1923)

*Rubus*, subgenus *Eubatus*

- R. allegheniensis* Porter cv. 'Darrow'. Eastern U.S. (R. H. Converse, unpublished data)
- R. fruticosus* L. (aggregate species)
- R. gracilis* Roxb. (also known as *R. niveus* Wall.) (Jones 1975b)
- R. laciniatus* Willd. (Zeller 1923; Converse et al. 1970a)
- R. lasiocarpus* Thumb. var. *rosifolius* (Hook. F.) (Jones and Roberts 1977)
- R. loganobaccus* Bailey cv. 'Logan'. (Jones and Jennings 1980b)
- R. procerus* P. J. Muell. (Stace-Smith 1955a)
- Rubus ursinus* Cham. and Schlecht. (Converse and Bartlett 1979)
- R. ursinus* Cham. and Schlecht. var. *macropetalus* Dougl. (Zeller 1923)
- Rubus* species. Various named and unnamed, cultivated and wild, erect and trailing blackberries (Bennett 1927; Converse 1981; Converse et al. 1970a; Jones and Roberts 1977; Jones and Jennings 1980b)

Additional rosaceous hosts infected experimentally either by aphids or by graft inoculation:

- Fragaria vesca* L. (RYNV only) (Stace-Smith and Mellor 1957)
- Rubus albescens* Roxb. (Stace-Smith 1955a)
- R. ellipticus* Smith (Converse et al. 1970)
- R. henryi* Hemsl. and Kuntze (Cadman 1951)
- R. molaccanus* L. (Jones and Roberts 1977)
- R. saxatilis* L. (Cadman 1951)

For additional experimental herbaceous hosts of BRNV, see p. 179.

RMD has only once been reproduced experimentally. Stace-Smith (1956) found that leaves of 'Cuthbert' red raspberry remained symptomless when they were ex-

perimentally infected with BRNV, developed a fine vein chlorosis when infected with RYNV, and showed symptoms of RMD only when infected with the complex of BRNV and RYNV (fig. 201 a, b, c). Converse (1966b) found that all of the 20 'Latham' red raspberry clones collected from commercial fields in Eastern U.S. were infected with heat-labile mosaic components of the BRNV type, but that only 35% of them were additionally infected with heat-stable virus of the RYNV type.

For symptoms caused by the individual viruses of the RMD complex on various *Rubus* and non-*Rubus* hosts, see the three following chapters.

### Natural and Experimental Transmission

RMD occurs either because diseased material has been planted or because the viruses inducing RMD have been transmitted to healthy plants. None of the viruses associated with RMD are transmitted through soil or seed, but all are transmitted, probably in a semipersistent manner (Cadman 1951, 1954; Murrant 1974b), by aphids of the genus *Amphorophora*. A few aphid species other than *Amphorophora* have been found to transmit some of the component viruses experimentally, but their role in nature is uncertain. (See chapters on the individual component viruses.) The major vector in Europe is *Amphorophora idaei* Börner [also known as *A. rubi* (Kalt.) ssp. *idaei* (Börner)] and in North America, *Amphorophora agathonica* (also known as *A. rubi* (Kalt.)) (fig. 202).

The biology of *A. idaei* was investigated by Dicker (1940) and by Rautapää (1967). Four biotypes of this aphid have been recognized in Great Britain by their different abilities to colonize certain red raspberry genotypes (Briggs 1965). The biology of *A. agathonica* was studied in Michigan, Minnesota, and New York (Giles 1966; Winter 1929; Kennedy and Schaefer 1974c). Only one biotype of *A. agathonica* is recognized in North America (Converse et al. 1971).

These two *Amphorophora* species complete their life cycles on *Rubus* (that is, they are monophagous) and produce alates involved in the long-distance spread (500 m or more) of RMD-causing viruses. In Great Britain, *A. idaei* is present in crops from June to August (Jones 1976d; Converse et al. 1970a; Dicker 1940), and, in New York State, *A. agathonica* occurs from mid-June to mid-August (Kennedy and Schaefer 1974c). Apterae are active, drop to the ground when disturbed, and probably are involved in local RMD spread by their own movements and by passive movements caused by wind, rain, and passage of machinery through the crop. Virus spread is usually along rows more than across rows (Rankin 1931; Cooley 1936b). In Great Britain and The Netherlands, peak populations of *A. idaei* occur in mid-summer and early autumn (Dicker 1940; Kronenberg and de Fluiter 1951; Jones 1976d). For *A. agathonica*, peak populations occur in late June in British Columbia (Stace-Smith 1960b) and in July



Figure 201. — 'Latham' red raspberry leaves chronically infected by grafting with, left, black raspberry necrosis virus (BRNV); center, Rubus yellow net virus (RYNV) plus BRNV; right, RYNV. This illustration shows the loss of vigor characteristic of infection by BRNV plus RYNV.



Figure 202. — *Amphorophora agathonica* on *Rubus*.

and August in the United States (Bennett 1932; Huber and Schwartze 1938; Converse et al. 1970a). Natural rates of transmission of the component viruses by aphids vary with the virus, cultivar, season, and region, but annual infection rates of 60% or more have been measured (Converse et al. 1970a; Jones 1976d, 1979a). The three following chapters give specific details for the individual viruses of the RMD complex.

The component viruses of the RMD complex have been experimentally transmitted by one or more of the following methods: (1) aphids; (2) graft inoculation; and (3) inoculation of sap.

1. *Aphids*. The three following chapters give the details of transmission of individual viruses by specific aphids. The aphids listed below have been found to be vectors of one or more viruses associated with RMD:

- Amphorophora agathonica* Hottes [also known as *A. rubi*. (Kalt.)] (Bennett 1927)
- A. idaei* Börner [also known as *A. rubi*. ssp. *idaei* (Börner)] (Cadman and Hill 1947)

- A. sensoriata* Mason (Bennett 1932)
- A. rubicumberlandi* Knowlton and Allen (Huber 1939)
- Aulacorthum solani* (Kalt.) (Jones and Murrant 1972)
- Macrosiphum euphorbiae* (Thos.) (Cadman 1954; Jones 1976d)
- Macrosiphum fragariae* (Wlk.) (also known as *Sitobion fragariae* (Wlk.) (Jones 1976d)
- Illinoia rubicola* (Oestlund) (also known as *Masonaphis rubicola* and *Oestlundia rubicola*) (Bennett 1932)
- Myzus ornatus* Laing (A. T. Jones, unpublished data)

The following aphids have been found to be nonvectors of one or more of the viruses associated with RMD.

- Amphorophora rubitoxica* Knowlton (Stace-Smith 1954)
  - A. ruborum* Börner (also known as *A. rubi*. ssp. *ruborum* Börner) (Cadman 1954) *A. ruborum* is common on blackberry in Europe (Hille Ris Lambers 1950; Blackman et al. 1977)
  - Aphis rubicola* Oestlund (Bennett 1927)
  - Aphis idaei* van der Goot (Cadman 1952b)
  - Chaetosiphon fragaefolii* (Cock.) (Stace-Smith and Mellor 1957)
- Note: *C. fragaefolii* is able to acquire RYNV from graft-inoculated strawberry and infect *Rubus* but not vice-versa.

In North America, *A. agathonica* can be used in a reliable system to detect viruses of the RMD complex by infecting small, vigorous *R. occidentalis* seedlings. (See "Detection and Identification" as well as the following chapter on BRNV.)

2. *Graft inoculation*. All viruses involved in RMD can be graft transmitted to *Rubus*. Techniques used are cleft grafting, patch grafting, cane inarching, a modification of cane inarching in which cut scions are maintained in small bottles of water during the incubation period (bottle grafting), and leaf grafting (Cadman 1951; Harris 1939; van der Meer 1958). Bottle grafting is the current standard graft indexing technique in Great Britain, whereas leaf grafting is the current standard method in the United States.

Susceptible cultivars of *R. occidentalis* like 'Plum Farmer' or 'Munger' or their open-pollinated seedlings and *R. henryi* have been widely used to detect viruses of the RMD complex occurring singly or in mixtures in *Rubus* (Cadman 1951; Converse 1979; Jones 1976d; Jones and Roberts 1977). Detection of pure cultures of BRNV and RYNV was 95% reliable in *R. henryi* if three scion leaflet grafts survived for 14 days (Converse 1965).

BRNV, RLMV, and RLSV all produce similar symptoms in graft-inoculated *R. henryi* and *R. occidentalis*, and further graft inoculations are needed to specific red raspberry indicator cultivars to distinguish these viruses. (See detailed discussion of this separation in "Raspberry Leaf Mottle and

**Table 8.—Particle morphologies of viruses probably involved in the raspberry mosaic disease complex**

Virus	Morphology	Dimensions	Reference
RYNV	Unenveloped bacilliform particle with rounded ends.	80-150 x 25-31 nm	Stace-Smith and Leung 1976; Jones and Roberts 1976.
BRNV	Isometric	25-30 nm	Jones and Murrant 1972; Murrant et al. 1976.
RLMV	Possibly isometric	30 nm	Jones 1976b.
RLSV	No particles seen	—	Jones 1981a.

Raspberry Leaf Spot,” p. 183.) Additionally, *R. henryi* develops necrotic shoot tips following graft inoculation with many isolates of tobacco streak virus. This necrosis could easily be confused with that caused by BRNV, RLMV, or RLSV (Frazier 1966; Converse and Kowalczyk 1980; Jones and Jennings 1980b). (See “Tobacco Streak Virus in *Rubus*,” p. 235.)

3. *Sap inoculation.* Among the four viruses associated with the RMD complex, only BRNV has so far been successfully transmitted in sap to herbaceous plants (Cadman 1960a, 1961a; Jones and Murrant 1972; Richter 1962, 1964c), but it is transmitted only with difficulty. In *Chenopodium quinoa* Willd., BRNV typically induces small, chlorotic, local lesions within a week and systemic chlorotic mottle or necrosis a week later. (For further discussion of details, see “Black Raspberry Necrosis,” p. 178.)

### Properties of the Causal Agents

Particle morphologies of the viruses of the RMD complex are listed in table 8. (See the respective chapters on these viruses for further details.)

### Detection and Identification

In some *Rubus* cultivars, RMD produces characteristic symptoms; however, identification of the causal viruses associated with RMD in a given infected *Rubus* plant from the field is not possible by study of the symptoms produced on that plant. Although BRNV can be transmitted to *Chenopodium quinoa* test plants by inoculation of sap, this is often erratic and therefore not reliable for routine testing (Jones and Murrant 1972; Jones and Roberts 1977). No antisera have been produced against any of the causal viruses of the RMD complex so that their detection depends on assays either by (1) aphid transmission, (2) grafting, or (3) graft indexing following heat treatment. Such assays are best done in the spring when vigorous, new, succulent growth is formed on both source and indicator plants grown at about 18° to 25°C.

**1. Aphid transmission.** In North America, where the efficient vector *Amphorophora agathonica* is available and will feed on *R. occidentalis*, the use of aphids for indexing is recommended by some workers (Mellor and Stace-Smith 1979). Several *A. agathonica* are allowed to feed on the source plant for 1 day and are then transferred to young, vigorous *Rubus occidentalis* seedlings (like ‘Munger’ op, from open-pollinated seed) for 1 day in the greenhouse. The insects are then killed with an insecticide, and the test plants are observed for up to 6 wk for symptom development. (See separate chapters on the specific viruses of the RMD complex for details.) By sequentially feeding aphids on a series of black raspberry seedlings, component viruses can be separated from mixtures (Stace-Smith 1956). In Europe, the vector *A. idaei* does not readily feed on *R. occidentalis* and aphid transmission is not used for detecting infection.

**2. Grafting.** Various grafting procedures have been used. Leaf grafting is preferred in North America and bottle grafting in Europe. Young, vigorous, susceptible *R. occidentalis* seedlings (like ‘Munger’ op) are stripped of all mature leaves except those used to accept leaflet grafts. Donor leaflets from vigorous, recently matured leaves are cut, and their petioles are sharpened to a long, thin point. These are then inserted into split debled petioles of the test seedlings and are tightly wrapped with tape. Self-cohering tape is often used. (See the introductory chapter of the strawberry section of this handbook, fig. 1, for detailed illustrations of the leaf graft technique.) The grafted plants are placed in a humid environment for 1 wk to allow the grafts to become attached and are then placed on the greenhouse bench for 6 wk or more to observe symptom development. (See detailed symptom descriptions of the specific viruses.) Speed in handling the cut petioles, keeping them moist at all times during the grafting procedure, and tight binding of the graft unions are essential for successful development of the leaflet graft unions. When carefully done, wide interspecific and even intergeneric leaf grafts can be made with *Rubus* leaflet sources (Converse 1965, 1979).

In Europe, *R. henryi* and *R. occidentalis* are vegetatively propagated, and small, potted plants are bottle-grafted (inarched with a cleft graft to a donor shoot, which is maintained in a container of water fixed to a supporting stick). The graft is tightly wrapped and the donor shoot is well supported to prevent separation of the tissues (fig. 203). Successful union of such grafts may be determined by removing the donor shoot from water after about 3 to 4 wk and noting its survival. Test plants are observed for symptom development for up to several months. (See chapters on individual viruses for discussion of symptoms.)

In Scotland, bottle grafting to 'Malling Landmark' is used to detect RLMV and to 'Norfolk Giant' to detect RLSV. (See p. 185–186 for fuller details.) Leaflet grafting is also satisfactory for inoculating 'Malling Landmark' and 'Norfolk Giant' red raspberry with viruses (Converse 1981); however, separation of component viruses is not possible by grafting to *Rubus*.

In addition to separation by serial aphid transfers, RYNV can be separated from BRNV in mixed infections by leaf grafting to *Fragaria vesca* var. *semperflorens* cv. 'Alpine'. This cultivar is susceptible to RYNV but not to BRNV (Stace-Smith and Mellor 1957). Separation of RYNV and BRNV may also be achieved by leaf grafting to healthy 'Fairview' red raspberry and, after 6 mo which must include a dormancy period, selecting root propagants that show mottling (indicating the presence of only heat-labile components like BRNV) but not mosaic symptoms (BRNV plus RYNV) (Freeman and Stace-Smith 1965). The probable movement of RLMV and RLSV into roots under these conditions is unknown.

**3. Graft indexing following heat treatment.** RYNV is not eradicated from plants kept at 37°C for several weeks, but BRNV, RLMV, and RLSV usually are eradicated (Jones and Roberts 1976); however, Mellor and Stace-Smith (1979) reported an isolate of BRNV that was resistant to heat treatment.

### Control Procedures

There is no known immunity in *Rubus* to any of the viruses associated with RMD; however, control of the incidence and effects of RMD can be attempted in five ways:

1. Use of *Rubus* material free of the viruses inducing RMD.
2. Use of management methods to restrict virus spread.
3. Use of insecticides to control populations of aphid vectors.
4. Use of *Rubus* cultivars resistant to vector aphids.
5. Use of virus-tolerant *Rubus* cultivars.

**1. Use of *Rubus* material free of the viruses inducing RMD.** With the finding of suitable indicator plants, the development of indexing methods, and the application of heat treatment, it became possible in the 1950's to produce



Figure 203. — Transmission of raspberry mosaic component viruses by bottle grafting to *Rubus* sp. (Copyright Scottish Crop Research Institute.)

virus-tested clones of *Rubus* cultivars, increase them in vector-free enclosures (screenhouses and gauzeshouses or isolated sites), and distribute stocks to nurseries. Various certification schemes have been established to monitor the health of such stocks prior to their release to growers [United Kingdom (1965); Ontario Horticultural Experiment Station (1966); California Department of Agriculture (1973)]. Chambers (1954, 1961) developed *Rubus* heat treatment techniques and helped to originate a virus-tested *Rubus* stock program in Great Britain. Similar systems were developed in Canada (Bolton and Turner 1962), the United States (Converse 1964, 1966b), the Federal Republic of Germany (Baumann 1980, 1981), and France (Morand 1963). Tissue culture explant techniques were successful in eliminating the component viruses of RMD (Putz 1971) and have been combined with heat treatment to produce virus-tested *Rubus* stocks (Pyott and Converse 1981).

**2. Use of management methods to restrict virus spread.** In the past, roguing out RMD-affected plants was used in attempts to control RMD. However, single infections with viruses associated with RMD induce no symptoms in most red raspberry cultivars, so that roguing is of little value in controlling the spread of these viruses.



The desirability of planting healthy *Rubus* plants at a distance from diseased *Rubus* has long been known. Because black raspberries are more severely damaged by RMD than most red raspberries, growers in the past were advised not to plant the two crops next to each other. This can now be done safely, however, if virus-tested stocks are used. Isolation distances of 100 m or more from RMD-infected wild or cultivated *Rubus* have been recommended in the United States (Cooley 1936c). In northwestern United States, however, the general rarity of *A. agathonica* in cultivated *Rubus* (presumably because of unfavorable hosts and environmental conditions), results in only a low incidence of RMD in certified black and red raspberry plantings planted next to uncertified plants (Converse 1975; and R. H. Converse, unpublished data).

In cultivars that have some resistance to colonization by *A. idaei*, the size of the planting influences the amount of virus infection in Scotland (Jones and Murrant 1975; Jones 1979a). The larger the planting, the smaller the proportion of plants that are peripheral and that are particularly vulnerable to primary infection by viruliferous alate *A. idaei*.

As a general rule, growers should not keep *Rubus* plantings beyond the time when viruses, uncontrolled fungus diseases or insects, or adverse environmental conditions decrease fruit yield and quality below an economic level. The use of virus-tested stocks of resistant cultivars, planted as far away as possible from sources of inoculum, and treated to decrease the population of vector aphids when they are found, are useful aids in the protection of *Rubus* crops from losses caused by RMD.

**3. Use of insecticides to control populations of aphid vectors.** Although many insecticides are available that give excellent control of *Amphorophora* species on *Rubus*, none can kill viruliferous aphids before they can probe and transmit virus to plants. Thus Cadman [in Converse et al. (1970a)], Taylor and Chambers (1969), and Freeman and Stace-Smith (1970) were not able to decrease the incidence of RMD by the use of insecticides applied at an economically acceptable rate; however, insecticides that prevent the buildup of *Amphorophora* colonies within a field may be useful in decreasing secondary spread. Schaefer (1967) found that the systemic insecticides oxydemeton methyl, dimethoate, and aldicarb provided satisfactory aphid control for 30 to 100 days in New York tests, and he recommended "maintenance of a total chemical aphid control program" in *Rubus* nurseries as an important element in the production of virus-tested stock.

**4. Use of *Rubus* cultivars resistant to vector aphids.** The development of red raspberry cultivars resistant to colonization by *Amphorophora agathonica* and *A. idaei* is often used as a classical example of successful breeding for insect resistance. This work has been reviewed by Briggs (1965), Daubeney (1972, 1982), and Baumeister (1961, 1962). Schwartz and Huber (1937) were the first to

demonstrate heritable resistance in *Rubus* to feeding and colonization by *Amphorophora*. Subsequently, several North American workers evaluated *Rubus* species and cultivars for this resistance (Huber and Schwartz 1938; Schwartz and Huber 1939; Converse and Bailey 1961; Daubeney and Stace-Smith 1963; Daubeney 1966; Kennedy et al. 1973). Rapid and effective screening procedures have detected independent sources of dominant genes for immunity to colonization by *A. agathonica* (Brodel and Schaefer 1980; Daubeney 1972; Daubeney and Stary 1982; Kennedy and Schaefer 1974a, b). Several raspberry plant breeding programs in North America, particularly at Vancouver, B.C., are using these techniques to select resistant genotypes (Daubeney 1982).

In Great Britain, Briggs (1965) and Knight et al. (1959) identified four strains of *A. idaei* and developed a simple technique for the evaluation of resistance to colonization on *Rubus* seedlings. In this test, three aphids are placed on a seedling shoot tip and observed. If they walk off, the seedling is immune; if they remain and colonize, it is susceptible. Jones (1976d, 1979a) demonstrated the value of such resistance in restricting the spread of viruses transmitted by *A. idaei* and showed that even moderate resistance to *A. idaei* colonization was effective in some situations. The incorporation of resistance to *A. idaei* colonization is therefore an important aspect of British red raspberry breeding programs at the East Malling Research Station (Keep et al. 1972) and the Scottish Crop Research Institute (Jennings 1963; Jones 1976d, 1981b; Jones and Jennings 1980b). Although early selections of red raspberries in Great Britain contained the resistance gene  $A_1$ , which does not provide resistance to *A. idaei* strains 2 or 4 (Knight et al. 1959), current breeding programs are using the resistance gene  $A_{10}$  and  $A_{K49}$ , which confers resistance to the four British strains of *A. idaei* (Keep and Knight 1967).

Stace-Smith (1960b) found that when *A. agathonica* colonized certain red raspberry cultivars infected with some of the viruses associated with RMD, the aphids were unable to transmit these viruses to other susceptible raspberries. In other instances, viruliferous aphids are able to colonize but not infect cultivars that can be graft-inoculated with these viruses (Converse et al. 1970a). Although the genetics of these host responses have not been studied, they may provide additional sources of resistance to infection by RMD.

**5. Use of virus-tolerant *Rubus* cultivars.** Growers have long known that differences in the amount of damage by RMD occur among red raspberry cultivars. Furthermore, red raspberries are more tolerant to RMD than black raspberries, and purple raspberries (red x black raspberry hybrids) are intermediate in reaction. Jones and Jennings (1980b) presented quantitative data on the relative sensitivity of these three groups to infection with BRNV and concluded that the genetic control of the differences was complex, though the absence of symptoms in the purple hybrids indicated the

presence in the red raspberry of dominant genes. They also showed that the symptoms induced by infection with RLMV and RLSV in sensitive red raspberry cultivars were determined by the single dominant genes Lm and Ls, respectively.

Red raspberry cultivars like 'Glen Clova' and 'Norfolk Giant' in Great Britain and 'Willamette' in North America support low populations of vector aphids but do not readily become affected by RMD (Cadman and Fiskén 1958; Stace-Smith 1955; A. T. Jones, unpublished data). Jennings (1963) suggested that in 'Norfolk Giant' this might be due to tolerance to RMD and that such tolerance should be incorporated into *Rubus* cultivars. Jones (1976d, 1979a) felt that the field performance of 'Glen Clova' and 'Norfolk Giant' could be explained by their moderate levels of resistance to *A. idaei* colonization. The nature and inheritance of tolerance to infection by RMD require more study before tolerance to the disease can be incorporated into new cultivars.

### Remarks

Symptoms resembling RMD can also have other causes, such as (1) feeding damage caused by the aphid *Amphorophora rubitoxica* Knowlton (Stace-Smith 1954); (2) late spring frosts (Bennett 1927); (3) powdery mildew (*Sphaerotheca humuli* DC), although the powdery white growth of this fungus and the water-soaked lesions it causes on the undersides of leaves help to distinguish its symptoms from RMD (Converse 1966a); (4) leaf speckling and blotching caused by feeding of spider mites such as *Tetranychus urticae* (Koch) and of *Eriophyes gracilis* (Nal.); (5) certain chemicals (see "Viruslike Disease Symptoms in *Rubus* in Great Britain," p. 251); and (6) deficiency of soil boron (Maryland Agricultural Experiment Station 1942).

Despite the great amount of research that has been devoted to RMD in many countries for many years, a number of problems still await solution, including:

- Characterization of the component viruses and determination of their relationships to each other and to other plant viruses.
- Rapid and precise methods of identifying the component viruses in plants and vectors.
- Surveys for the occurrence of the viruses associated with RMD in the main *Rubus* growing areas of the world.
- Influence of these viruses singly and in combination on growth and yield of the main *Rubus* cultivars.

### Rubus Yellow Net

By R. Stace-Smith and A. T. Jones

### Additional Common Names

Raspberry yellow mosaic virus (Bennett 1927, 1932) is considered a probable synonym, but unequivocal evidence is lacking. Although rubus yellow net virus (RYNV) was characterized under controlled conditions and clearly distinguished from other aphid-transmitted viruses, yellow mosaic

was a field disease and in most instances a virus complex was probably involved.

### History and Geographic Distribution

RYNV was first isolated and described from naturally infected Himalaya blackberry (*Rubus procerus* P. J. Muell.) in British Columbia. Although it was rarely observed in Himalaya blackberry, it was later found in raspberry as a component virus of the raspberry mosaic disease complex (Stace-Smith 1956) that is common and has been known for many years in North America and Europe (Cadman 1961b; Jones et al. 1974; Jones and Roberts 1976).

This disease complex has been recorded from virtually every major raspberry growing area in the world, so that RYNV can be considered to have a worldwide distribution. With the widespread use of virus-free planting material, however, and the increased use of cultivars that are either aphid-immune or aphid-resistant, the incidence of the disease is decreasing in North America and Europe.

### Economic Importance

Limited observations have been made on the economic importance of RYNV. A single plant of the red raspberry cv. 'Washington' that was graft inoculated with RYNV showed no evidence of degeneration after 3 yr in a field plot (Stace-Smith 1955a). In a field trial in Canada, the yield of plants infected with RYNV and black raspberry necrosis virus (BRNV) was decreased by 43 to 78% in the first cropping year compared with 0 to 30% for plants infected with BRNV alone. Losses in dually infected plants in subsequent years, however, was 0 to 15% (Freeman and Stace-Smith 1970). In Western North America, the economic importance of RYNV is minimal because widespread use of raspberry cultivars with genes for immunity to the aphid vector has virtually precluded its spread into commercial plantations. In Great Britain and most of Europe, where a different aphid species is the vector, such cultivars have become available only recently and most plantations, therefore, still contain a large proportion of the older aphid-susceptible cultivars.

### Symptoms on Natural and Experimental Hosts

Natural hosts of RYNV are restricted to the genus *Rubus*, primarily red raspberry (*R. idaeus* L.) and black raspberry (*R. occidentalis* L.). RYNV is occasionally found in Himalaya blackberry (*R. procerus*) and other wild or cultivated species.

**Symptoms on red raspberry.** Symptoms are evident about 4 to 8 wk after inoculation by grafting or 3 to 4 wk after inoculation by aphids. Leaves of infected plants develop a netlike chlorosis of the tissue along the veins, giving the plant a pale green appearance (fig. 204). Some of the leaves are slightly cupped downward, but there is no distortion or stunting and no obvious decrease in vigor (Stace-Smith 1955a).

**Symptoms on black raspberry.** Young seedlings of black raspberry show a diagnostic netlike chlorosis 3 to 4 wk after inoculation by viruliferous aphids. The initial symptom is flecks of netlike chlorosis on the fourth or fifth leaf from the tip of the infected seedling, followed by progressive veinal chlorosis on the younger leaves. This chlorosis is typically unilateral, involving one of the basal leaflets and the lower edge of the terminal leaflet (fig. 205).

As the affected leaf expands, that portion showing netlike chlorosis develops at a slower rate, causing the affected leaflet to bend towards the chlorotic side. Beneath the first affected leaf, the older leaves remain normal, but the netlike chlorosis spreads upward towards the tip of the shoot, becoming more severe and extensive, until all affected leaves are chlorotic, stunted, and cupped downward. RYNV can also be detected in grafted black raspberry, but the chronology of disease development, so distinctive in aphid-inoculated seedlings, is less distinctive in graft-inoculated plants (Stace-Smith 1955a and unpublished data).

**Symptoms on Himalaya blackberry.** Himalaya blackberry shows considerable variations in its response to RYNV. A naturally infected clone exhibits a distinctive yellow chlorosis on some of the mature leaves but no symptoms on others or on younger leaves (Stace-Smith 1955a). Seedlings of Himalaya blackberry vary in their response; some showing no obvious symptoms and others different intensities of netlike chlorosis (R. Stace-Smith, unpublished data). The growth and vigor of infected plants is not apparently affected (Stace-Smith 1955a).

**Experimental hosts.** Tropical black raspberry (*Rubus albens* Roxb.). Symptoms in seedlings of tropical black raspberry are essentially the same as those in seedlings of *R. occidentalis*. Symptoms develop about 18 days after aphid inoculation on the third or fourth leaf from the tip of the inoculated plant. The netlike chlorosis is often more severe on one side of the petiole, resulting in a stunting of the affected *R. albens* leaf and a bending of the petiole (Stace-Smith 1955a).

*Rubus henryi*. Although Converse (1965) attributed symptoms of vein clearing, mottling, distortion, and necrosis of the leaves and shoots of *R. henryi* to RYNV, there is now doubt as to whether these were caused by RYNV alone or RYNV combined with a heat-stable strain of black raspberry necrosis virus. (See section on "Black Raspberry Necrosis Virus," p. 178.)

**Strawberry (*Fragaria vesca* L.).** Symptoms on 'Alpine' strawberry (*F. vesca* var. *semperflorens* (Duch.) Ser.) appear about 3 wk after graft inoculation, when the young leaves begin to bend downward and necrotic lesions appear at the base of the petioles. Affected leaves die within a few weeks, and lesions develop at the base of petioles of older unaffected leaves, which ultimately wilt and die. The plant is usually killed within 2 mo of graft inoculation (Stace-Smith and



Figure 204. — Symptoms of rubus yellow net virus in a systemically infected leaf of 'Washington' red raspberry.



Figure 205. — Unilateral development of rubus yellow net virus symptoms in a leaf of black raspberry.

Mellor 1957). In other clones of *F. vesca* symptoms develop more slowly than on 'Alpine', and they are not as severely affected. Although some of the graft-inoculated plants may die, others persist with only the older leaves alive (Stace-Smith and Mellor 1957).

### Natural and Experimental Transmission

RYNV is transmitted by the raspberry aphids *Amphorophora agathonica* Hottes in North America and *A. idaei* Börner in Europe. Other *Rubus*-infecting species of *Amphorophora* may serve as vectors, but, because of the widespread occurrence of *A. agathonica* and *A. idaei* on raspberry, transmission by any other species is probably relatively unimportant. Aphids can transmit the virus after an acquisition access feed of 1 hr, but frequency of transmission is greater after 4 hr. There is no latent period in the vector, but aphids require a minimum of 15 min feeding to transmit. Aphids maintain the ability to transmit the virus after feeding for 2 to 3 hr on healthy plants (Stace-Smith 1955a), but if they are starved, they may retain the virus for 1 day at 20°C and up to 4 days at 3°C (Stace-Smith 1960a).

In controlled experiments, *A. agathonica* did not transmit RYNV from raspberry to strawberry but was capable of acquiring the virus from graft-inoculated strawberry plants and transferring it to black raspberry seedlings (Stace-Smith and Mellor 1957). Efficiency of transmission is not as high as when raspberry is used as a virus source, but, using five aphids per plant, 22 of 34 test plants were infected.

Under similar test conditions, the strawberry aphid *Chaetosiphon fragaefolii* (Cock.) did not acquire and transmit RYNV from infected raspberry or strawberry plants to black raspberry (Stace-Smith and Mellor 1957).

### Properties of the Causal Agent

RYNV is readily transmitted by the aphid vectors *A. agathonica* and *A. idaei*, but it has not been transmitted mechanically (Converse et al. 1970a; Stace-Smith and Jones 1978). Unlike other viruses transmitted by *Amphorophora* species, RYNV is usually not inactivated by exposure to an air temperature of 37°C for several weeks (Stace-Smith 1960a; Converse 1966b; Jones and Roberts 1976), but it can be eradicated from small meristem tip cuttings following treatments at 37° to 39°C for 4 to 14 wk (Mellor and Stace-Smith 1979).

Particles of RYNV are bacilliform (fig. 206) and in thin sections of infected raspberry leaves are 80 to 150 nm long and 25 to 31 nm wide (Jones and Roberts 1976; Stace-Smith and Leung 1976). Both ends are rounded (figs. 206 and 207) and, in cross section, particles show an electron-translucent core about 17 nm in diameter (fig. 208). In the early stages of infection, the particles appear to be confined to the sieve tubes, but in later stages of infection the particles are found in xylem parenchyma, mesophyll, and epidermal cells. Particles occur singly or in clusters, often in degenerate



Figure 206. — Electron micrograph of sap of *R. macraei* A. Gray stained with 2% ammonium molybdate (pH 6.5), showing bacilliform particles of rubus yellow net virus. Bar represents 100 nm.

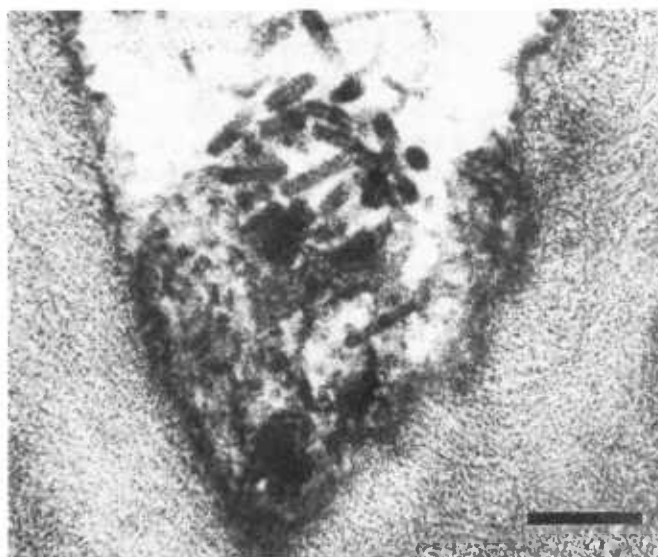


Figure 207. — Electron micrograph of thin section of *R. occidentalis* infected with rubus yellow net virus, showing longitudinal section of particles. Bar represents 200 nm.



Figure 208. — Electron micrograph of thin section of *R. occidentalis* infected with rubus yellow net virus, showing transverse section of particles and electron-translucent core. Bar represents 40 nm.



endoplasmic reticulum. Within each cluster, the particles lack orientation; some appear in cross section, others in oblique or longitudinal section.

### Detection and Identification

Other aphid-borne viruses such as black raspberry necrosis, raspberry leaf mottle, and raspberry leaf spot (see chapters on these viruses in this section, p. 178 and 183) are prevalent in raspberry and are transmitted by *Amphorophora* species, so that RYNV rarely occurs on its own in red raspberry. In combination with some of these viruses, RYNV induces veinbanding mosaic disease in North America known as raspberry mosaic disease (Cadman 1952b, 1961; Stace-Smith 1956). A plant showing veinbanding mosaic symptoms might therefore be assumed to be infected with RYNV; however, this assumption is difficult to justify because RYNV does not induce distinctive symptoms in complex infections. Moreover, graft and aphid inoculations to *R. occidentalis* will transmit the complex of viruses, and some of these will induce severe necrotic symptoms that mask those induced by RYNV. However, if aphids are fed on a source containing a virus complex and then transferred individually to test plants for short inoculation access feeds, a few of the inoculated plants will become infected with RYNV only (Stace-Smith 1955a). Additionally, heat treatment of plants or roots of plants infected with the complex will inactivate most of the viruses (Chambers 1961) but not RYNV (Stace-Smith and Mellor 1957). Such treatment has provided sources of RYNV free from other contaminating aphid-borne viruses (Stace-Smith and Mellor 1957; Converse 1965; Jones and Roberts 1976). While this technique has been useful, some mosaic sources may contain other aphid-borne viruses that are heat stable (Mellor and Stace-Smith 1979). A third method is to graft the mosaic-affected plant to strawberry (*F. vesca*), which is susceptible to RYNV but immune to most aphid-borne viruses of *Rubus* (Stace-Smith and Mellor 1957).

Although the small bacilliform particles associated with RYNV infection are unlike those of any other virus known to infect *Rubus* spp., use of electron microscopy for detection and identification is difficult because such particles are rarely seen in leaf dip preparations, and electron microscopy of thin sections of infected tissue is too laborious to be useful for routine identification.

### Control Procedures

The most effective control for RYNV is obtained by planting cultivars that are either immune or resistant to the aphid vector. (See "Black Raspberry Necrosis," p. 178, for a full discussion of control using aphid-resistant cultivars.)

RYNV is classified as a heat-stable virus (Stace-Smith 1960a); a term applied to those viruses that persist in plants held for several weeks at an air temperature that approaches the maximum at which plants can survive. The heat stability of RYNV was first reported by Stace-Smith and Mellor

(1957) and later confirmed by other workers (Chambers 1961; Converse 1965). Mellor and Stace-Smith (1979), however, showed that most small shoot tips or stem segments that were excised from plants during heat treatment and induced to root were free from RYNV.

### Black Raspberry Necrosis

By. R. Stace-Smith and A. T. Jones

#### Additional Common Names

Mild mosaic (Bennett 1927); a component of red raspberry mosaic (Stace-Smith 1956); 52V virus (Jones and Murant 1972; Jones and Roberts 1977); heat labile mosaic components (Converse 1963).

### History and Geographic Distribution

The name "black raspberry necrosis virus" (BRNV) was coined for an aphid-transmitted entity that was latent or mild in red raspberry cultivars but which induced severe tip necrosis on black raspberry seedlings (Stace-Smith 1955b). This virus, together with rubus yellow net virus (RYNV; see "Rubus Yellow Net," p. 175), was consistently associated with the raspberry mosaic disease in North America (Stace-Smith 1956). Similar entities had been observed previously but had not been clearly distinguished from other viruses associated with the mosaic disease complex. Of the aphid-borne viruses that have been isolated and described in Europe, the one initially designated "52V virus" (Jones and Murant 1972) is now equated with black raspberry necrosis (Jones and Roberts 1977). Raspberry leaf mottle and raspberry leaf spot viruses also induce a systemic necrosis in black raspberry, but they are distinguished from BRNV by inducing distinct symptoms while BRNV is usually latent in red raspberry indicators. (See "Raspberry Leaf Mottle and Raspberry Leaf Spot," p. 183.)

At one time, all clones of some of the older red raspberry cultivars were infected with BRNV and, as a consequence, the virus was introduced by farmers to many regions in the world where raspberries were grown. In Western North America, the replacement of these old cultivars with new ones immune to the main aphid vector has greatly decreased the incidence of BRNV in red raspberry, and this will probably also happen in other major raspberry growing areas where aphid-resistant or aphid-immune cultivars have recently become available.

### Economic Importance

Black raspberry cultivars vary in their response to infection, but even the most tolerant cultivars are seriously affected.

Although all red raspberry cultivars tested are susceptible to BRNV (Jones and Jennings 1980b), the economic importance of the virus depends on the cultivar and the duration of the infection. Thus, in North America, the yield of some cultivars was unaffected by infection, whereas that of others was decreased by up to 30% in the initial cropping years but lessened to stabilize at about 14% in subsequent years. The

sensitive cultivars produced smaller fruits and shorter, thinner cane than virus-free controls (Converse 1963; Freeman and Stace-Smith 1970). Freeman et al. (1969) also showed that infection with BRNV increased pollen abortion in some raspberry cultivars and that this was further increased by additional infection with RYNV.

In Great Britain, BRNV is one of the first aphid-borne viruses to infect new plantings of healthy red raspberry, and the main cv. 'Malling Jewel' may become 100% infected with BRNV by the end of its first fruiting year (Jones and Murant 1972; Jones 1976*d*, 1979*a*). In Scotland, infection of red raspberry by BRNV alone decreased the mean length of canes and mean berry weight in cvs. 'Glen Prosen' and 'Malling Leo'. This effect increased with the age of infection (Jones 1980*b* and unpublished data). Furthermore, in Great Britain, BRNV infection of cultivars such as 'Malling Jewel' is commonly accompanied by infection with several other viruses, and such multiple infections are believed to contribute to the degeneration of plantations (Cadman 1961*b*; Jones 1981*b*). For example, BRNV is found together with raspberry bushy dwarf virus in plants of cv. 'Lloyd George' affected by the degenerative disease, raspberry bushy dwarf. (See "Raspberry Bushy Dwarf," p. 229.) In controlled experiments, infection of 'Lloyd George' plants with BRNV alone induced many features of the disease, but plants inoculated with both viruses were the most obviously diseased (Jones 1967*b*). In combination with RYNV, BRNV induces raspberry mosaic disease in North America (Stace-Smith 1955*b*) and vein-banding mosaic disease in Europe (Cadman 1952*b*, 1961*b*). (See "Raspberry Mosaic," p. 168.)

### Symptoms on Natural and Experimental Hosts

**Natural hosts.** The natural hosts of BRNV are restricted to the genus *Rubus*. Natural hosts include cultivated and wild *Rubus fruticosus* Hort, *R. idaeus* L. and *R. idaeus* var. *strigosus* (Michx.) Maxim. (red raspberry), *R. occidentalis* L. (black raspberry) and *R. leucodermis* Dougl. (western black raspberry), *R. procerus* P. J. Muell. (Himalaya blackberry), 'Thornless Young' derivative of *R. ursinus* Cham. and Schlect., *R. ursinus* var. *macropetalus* Dougl., *R. lasiocarpus* Thumb. var. *rosifolius* (Hook. F.) Hara, *R. allegheniensis* Porter cv. 'Darrow'; *R. phoenicolasius* Maxim., and *R. loganobaccus* Bailey (Stace-Smith 1955*b*; Jones and Murant 1972; Jones and Wood 1979; Jones and Roberts 1977; Converse and Bartlett 1979; Jones and Jennings 1980*b*; Converse, unpublished data). Other wild and cultivated *Rubus* spp. are reported to be naturally affected by raspberry mosaic (Zeller 1923), but tests to identify the virus or viruses involved were not done.

**Experimental hosts.** In addition to red raspberry and black raspberry, BRNV has been transmitted by grafting or by aphid vectors to 'Boysen', 'Tayberry', *Rubus albens* Roxb., *R. henryi* Hemsl. and Kuntze, *R. laciniatus* Willd., *R. loganobaccus*, *R. molaccanus* L., and *R. phoenicolasius* Maxim. (Jones and Roberts 1977). The virus has also been

transmitted to the following herbaceous plants by mechanical inoculation of raspberry sap: *Chenopodium amaranticolor* Coste and Reyn., *C. quinoa* Willd., *C. murale* L., *Petunia hybrida* Vilm., *Spinacia oleracea* L., *Gomphrena globosa* L., and *Nicotiana debneyi* Domin (Jones and Murant 1972; Murant et al. 1976).

**Symptoms.** Black raspberry. Distinctive symptoms of BRNV are based on the reaction of succulent, young test plants grown in a protected greenhouse environment. Symptoms on field-grown plants are similar but are not diagnostic. Symptoms are first evident 5 to 7 days after aphid inoculation, when the shoot tip appears bent. Within a day or two of the initial bending, the tip is distinctly downcurled and brittle and the partially expanded leaves beneath the tip appear wilted. The wilting is followed by necrosis of the petiole, midribs, unfolding leaves, and the stem tip. Wilting and necrosis is a shock reaction, and, if the plant survives, later shoots produce leaves showing varying intensities of mottle. Symptoms after graft inoculations (fig. 209) are similar.

When BRNV is transmitted to black raspberry by grafting, the symptomatology is similar except that the period between grafting and the appearance of initial symptoms is 3 to 8 wk. The reaction of *R. henryi* to graft-inoculation with BRNV is similar to that of *R. occidentalis* (fig. 210) (Stace-Smith 1955*b*; Jones and Roberts 1977).

Red raspberry. Most cultivars infected with BRNV exhibit no visible symptoms but some, such as 'Malling Admiral', 'Malling Orion', 'Taylor', and 'Washington', show small chlorotic spots and mottling adjacent to leaf veins (fig. 211) (Stace-Smith 1955*b*; Jones and Roberts 1977; Jones and Jennings 1980*b*).

Other *Rubus* species. In a heated greenhouse, leaves of 'Himalaya' blackberry and tropical black raspberry (*R. albens*) may show a mild chlorotic spotting or mottling (Stace-Smith 1955*b*; Jones and Jennings 1980*b*) and *R. henryi* and *R. molaccanus* develop apical necrosis accompanied by leaf deformity and/or epinasty 4 to 8 wk after grafting; young leaves of *R. molaccanus* often show necrotic flecking (Jones and Roberts 1977). Infected *R. phoenicolasius* seedlings are less vigorous than normal but do not show obvious leaf symptoms (Jones and Roberts 1977). Under cooler conditions, infected plants of 'Boysen' and 'Tayberry' may show a chlorotic mottle (Jones and Jennings 1980*b*).

*C. quinoa* and *C. amaranticolor*. Small chlorotic/necrotic local lesions sometimes develop about 6 to 10 days after mechanical inoculation; such plants usually show systemic chlorotic flecking and/or necrosis within 2 wk (fig. 212). *C. murale* develop large necrotic local lesions but no systemic infection. *S. oleracea* plants may develop systemic chlorosis or necrosis under winter conditions (Jones and Murant 1972; Murant et al. 1976).



Figure 209. — Tip necrosis in *R. occidentalis* cv. 'Plum Farmer' graft inoculated with black raspberry necrosis virus.



Figure 210. — Tip curling prior to tip death in *R. henryi* graft inoculated with black raspberry necrosis virus.



Figure 211. — Veinal chlorotic mottle in a leaf of the red raspberry cv. 'Malling Orion' infected with black raspberry necrosis virus.

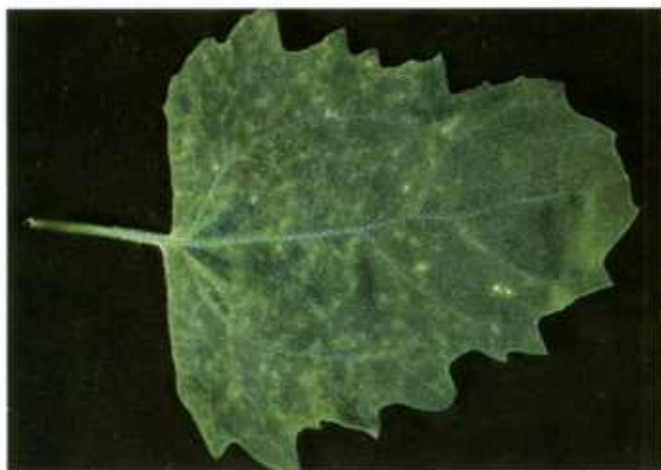


Figure 212. — Systemic chlorotic flecking and mottle in a leaf (left) and plant (right) of *C. quinoa* mechanically inoculated with the 52V isolate of black raspberry necrosis virus.

### Natural and Experimental Transmission

The natural vectors of BRNV are aphids belonging to the genus *Amphorophora*, primarily *A. agathonica* Hottes in North America and *A. idaei* Börner in Europe. Other species that may serve as minor vectors in North America include *A. sensoriata* Mason, *A. rubicumberlandii* Knowlton and Allen, and *Illinoia* (also known as *Masonaphis*) *rubicola* (Oestlund). In Europe, species other than *A. idaei* are infrequently observed in red raspberry but small populations of *Macrosiphum euphorbiae* (Thos.), *M. (Sitobion) fragariae* (Wlk.), and *Myzus ornatus* Laing have been recorded (Jones 1976d and unpublished data). While there is no evidence in Europe of field transmission by any species other than *A. idaei*, under test conditions *Aulacorthum solani* (Kalt.) and *M. euphorbiae* as well as *A. idaei* have transmitted BRNV from red raspberry to *C. quinoa* seedlings (Jones and Murant 1972; Murant et al. 1976; Jones 1976d). Attempts to transmit the virus from *C. quinoa* using these aphid species failed.

BRNV is transmitted by aphids in a semipersistent manner. In North America, all instars of *A. agathonica* can transmit BRNV, requiring minimum acquisition and transmission access feeds of 15 to 30 min and 2 min, respectively. Viruliferous aphids can continue to transmit for up to 3 to 4 h after acquisition while feeding and, depending on the temperature, up to 4 days if starved (Stace-Smith 1955b).

BRNV is readily graft transmissible from *Rubus* to *Rubus* but not from herbaceous plants such as *C. quinoa* to *Rubus* (Murant et al. 1976); BRNV is not seed-borne in red raspberry (Jones and Murant 1972).

BRNV is transmitted manually with difficulty by grinding young red or black raspberry leaves with alumina, Celite (a diatomaceous product), and 2% nicotine solution, and rubbing the inocula on leaves of *C. quinoa*. Transmission is achieved more readily from field-grown raspberry plants in spring and autumn than in summer, and from infected black raspberry than from infected red raspberry. Transmission from raspberry plants grown in the greenhouse is difficult at all times of the year (Jones and Murant 1972; Jones and Roberts 1977; Jones and Jennings 1980b).

### Properties of the Causal Agent

BRNV is difficult to maintain in greenhouse-grown *C. quinoa*, and this is especially so in summer. The virus, however, can be continuously maintained in culture by keeping inoculated *C. quinoa* plants in growth cabinets (18°C, 8000 lux, 8 h photoperiod). Systemically infected leaves from such plants are suitable for purification (Murant et al. 1976). The virus is present in very low concentration in infected raspberry and *C. quinoa* plants, with the result that only very small amounts of inadequately purified virus have been obtained. Attempts to produce an antiserum to the virus using such material were unsuccessful.

Partially purified preparations contain a few viruslike particles about 25 to 30 nm in diameter (fig. 213) (Jones and Murant 1972; Murant et al. 1976). Similar particles have been detected by electron microscopy of ultrathin sections of BRNV-infected *R. henryi*, *R. occidentalis*, and *C. quinoa*. Particles occurred in the cytoplasm in many kinds of cells and were often found arranged in single file and in plasmodesmata (fig. 214) (Murant et al. 1976; Jones and Roberts 1977).

During winter, sap of *C. quinoa* containing BRNV lost infectivity after diluting  $10^{-1}$  to  $10^{-2}$ , heating for 10 min at 50 to 52°C, and storage at 18°C for 6 to 24 h (Jones and Murant 1972).

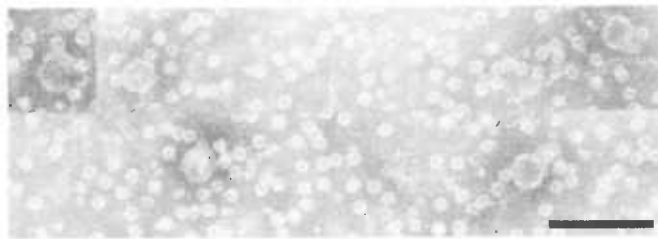


Figure 213. — Electron micrograph of a partially purified preparation of the 52V isolate of black raspberry necrosis virus showing a few viruslike particles among numerous particles of phytocystatin. Bar represents 200 nm.



Figure 214. — Electron micrograph of a thin section of *R. occidentalis* infected with black raspberry necrosis virus showing viruslike particles aligned in a row in the cytoplasm of a vascular cell. It also shows the particles within a tubule, which is continuous with a plasmodesma. Bar represents 200 nm.

### Detection and Identification

BRNV can occasionally be detected in sensitive red raspberry cultivars under field conditions in the spring and early summer by the appearance of veinal chlorotic spots. However, as most currently grown cultivars show no foliar symptoms or develop only very faint symptoms, this is of little value in detecting field infections. Consequently, insect or graft transmission to a sensitive indicator host is necessary.



The technique of choice in North America is transmission by the aphid *A. agathonica* to seedlings of black raspberry. If the plant being tested is naturally infested with this aphid, 5 to 10 aphids are transferred to black raspberry seedlings for inoculation access feeds of at least 30 min. If the plants being indexed are not already colonized by this aphid, those from a laboratory colony can be given an overnight acquisition access feed on test material before being transferred to black raspberry seedlings (Huber and Johnson 1952; Stace-Smith 1955b, Converse 1961). The technique is reliable for detecting the virus in most cultivars that can be colonized by this aphid but is unreliable for detecting virus in those not colonized by this aphid (Stace-Smith 1960b).

While *A. agathonica* readily transmits BRNV to healthy *R. occidentalis*, the European vector, *A. idaei*, does not readily feed on it and transmission of virus from diseased raspberry to *R. occidentalis* is difficult (Cadman 1961b). Detection of BRNV in Europe is therefore done by graft inoculation to *Rubus* indicator hosts and/or by inoculation of sap to *C. quinoa* plants. Inarch bottle grafting to *R. henryi* or *R. occidentalis* is usually used (Jones and Roberts 1977; Jones 1976d). Manual transmission of BRNV may be done by grinding young raspberry leaves with alumina, Celite, and 2% nicotine and rubbing the inocula on leaves of *C. quinoa*.

A disadvantage of both aphid and graft transmission is that many raspberry plants are naturally infected with several unrelated aphid-vectored viruses and the virus complex may be transmitted. Thus, BRNV frequently occurs with RYNV, the complex inducing mosaic disease. The two viruses have similar vector relations, and they are not readily separated by routine aphid transmissions. However, they may be separated by transferring individual aphids from doubly infected plants to a series of black raspberry seedlings, permitting the aphid to feed for only a short time on each seedling (Stace-Smith 1956). Another technique is to graft inoculate the complex to a healthy red raspberry plant and subdivide the grafted plant into root cuttings before the viruses have had time to invade it completely. BRNV becomes systemic sooner than RYNV, with the result that a proportion of the root cuttings are infected only with BRNV (Freeman and Stace-Smith 1965).

In Europe, BRNV is commonly found in red raspberry with two other latent viruses, raspberry leaf mottle (RLMV) and raspberry leaf spot (RLSV). All three viruses are transmitted by *A. idaei* and induce apical necrosis in *R. occidentalis* and *R. henryi*. RLMV and RLSV can be distinguished on the basis of symptom response in the red raspberry indicator cvs. 'Malling Landmark' and 'Norfolk Giant', respectively. (See "Raspberry Leaf Mottle and Raspberry Leaf Spot", p. 183.) Because BRNV induces few if any symptoms in red raspberry, however, it cannot be identified by graft indexing when in mixed infections with RLMV and/or RLSV. In these situations, successful mechanical transmission to *C. quinoa* is the only means of identifying BRNV.

## Control Procedures

The most effective control measure is to plant cultivars that are not susceptible to colonization by vector aphids. Several current raspberry breeding programs emphasize selection for resistance to these vectors and, as a result, older cultivars that lack this character are gradually being replaced (Jones and Jennings 1980b; Daubeny 1982). Immunity to *A. agathonica* is controlled by a single dominant gene derived from the cv. 'Lloyd George'. In North America, where some cultivars immune to this aphid have been grown for many years, there is no evidence of resistance-breaking *A. agathonica* biotypes (Converse et al. 1971). In Great Britain, four biotypes of *A. idaei* exist, and breeding programs have attempted to incorporate resistance to each. To be fully effective in preventing virus spread, resistance to the aphid must be extreme; however, even low levels of resistance have been shown to decrease the rate of virus spread (Jones, 1976d, 1979a, 1981b).

While resistance to *A. idaei* in Great Britain appears to prevent the spread of BRNV in red raspberry, one of the best sources of gene(s) for this character is black raspberry. Because of the severe reaction of black raspberry to BRNV infection and the possibility of transferring this sensitivity to infection to red raspberry in breeding programs, Jones and Jennings (1980b) surveyed the response to BRNV infection in more than 30 *Rubus* species and hybrids. None was immune from infection, and the study considerably expanded the number of *Rubus* species that are now known to be susceptible. There seems little prospect, therefore, of breeding raspberries immune from BRNV; however, the absence of foliar symptoms in BRNV-infected red raspberry was found to be a dominant character and the inheritance of the necrosis reaction in *R. occidentalis* was found to be complex.

Most isolates of BRNV are readily eradicated from *Rubus* spp. by heat treatment at 32° to 37°C for 1 to 4 wk (Chambers 1954; Stace-Smith and Mellor 1957; Converse 1963) or by excising and rooting tip cuttings from plants during heat treatment (Bolton and Turner 1962). Clones free of BRNV are available for virtually all commercial cultivars. Some virus isolates, however, may be more difficult to eradicate than others because Mellor and Stace-Smith (1979) reported that a virus isolate, thought to be BRNV, survived heat treatment at 39° to 42°C for 8 wk.

## Remarks

Although BRNV, raspberry leaf mottle, and raspberry leaf spot viruses seem to be separate entities, their similarity in several properties suggests that they may be related. The features that suggest that they are distinct are: (1) differences in reaction of red raspberry indicator plants; (2) frequent occurrence of the three viruses together in the same plant; (3) mechanical transmissibility of BRNV but not of RLMV or RLSV; (4) 30-nm particles, presumably those of BRNV, are readily found in thin sections of BRNV-infected black

raspberry and *C. quinoa*, but no such particles have been found in RLMV- and RLSV-infected plants; and (5) the genetic bases for reaction to the three viruses are different (Jones and Jennings 1980b). (See also "Raspberry Leaf Mottle" and "Raspberry Leaf Spot," p. 183.)

## 245 Raspberry Leaf Mottle and Raspberry Leaf Spot //

By A. T. Jones

Raspberry leaf mottle virus (RLMV, Cadman 1951) and raspberry leaf spot virus (RLSV, Cadman 1952d) are aphid-borne viruses that are latent in most red raspberry cultivars but produce symptoms in a few red raspberry indicator cultivars. Their similarity in symptoms induced in indicators, vector relations, and response to thermotherapy suggests that they may be related though distinct viruses. Because of this and the fact that in many published reports it is not clear which of the two viruses is involved in the diseases described, the two viruses will be considered together.

### Additional Common Names

The following are names used to describe the characteristic disease symptoms induced by infection with either RLMV or RLSV in sensitive cultivars: Type *c* symptom (Harris 1933), raspberry Mosaic 2 (Harris 1939), Flekkmosaikk (Bjørnstad 1953), spot mosaic (Fleckenmosaik) (Richter 1964a, b), raspberry chlorotic spot (Jordović 1963), and leaf spot mosaic (Murant 1974b).

### History and Geographic Distribution

Harris (1933) was probably the first to describe leaf spot mosaic disease in detail. He classified the many viruslike symptoms occurring in certain red raspberry cultivars in Great Britain into several kinds. One of these, termed type *c*, was characterized in cv. 'Baumforth B' by sharply defined chlorotic spots scattered randomly over the leaves. Later, Harris (1939) simplified the symptom categories into two major kinds which he termed Mosaic 1 and Mosaic 2; Mosaic 2 included the symptom previously referred to as type *c* (Harris 1933).

Cadman (1951, 1952d) showed that at least two distinct aphid-borne viruses could induce Mosaic 2 in some red raspberry cultivars but that they were symptomless in many others. Subsequently, Mosaic 2 symptoms have been reported in raspberry from Scandinavia (Bjørnstad 1953; Tapio 1961), Germany (Richter 1962a, b), Yugoslavia (Jordović 1963), U.S.S.R. (Kuznetsova and Pomazkov 1971) and France (Bouchery and Putz 1972). However, although some of these reports refer to the causal agent of the disease as "spot" or "leaf mottle," most do not distinguish between infection with RLMV and RLSV. Nevertheless, RLMV and RLSV probably occur in each of these countries. The two viruses have also been identified in New Zealand where they were probably introduced from Europe (Jones and

Wood 1979). There is also some circumstantial evidence that they were introduced into Australia and North America (Cadman 1951, 1952d; Converse 1981).

### Economic Importance

RLMV and RLSV are widespread in raspberry in Great Britain and possibly many European countries. In Great Britain, they rapidly infect newly planted stocks of the main commercial cv. 'Malling Jewel' but infect the other main cv. 'Glen Clova' less rapidly (Jones and Murant 1975; Jones 1976d, 1979a). Most red raspberry cultivars are infected symptomlessly, but a few are sensitive to infection and show severe symptoms frequently resulting in plant death (Cadman 1951, 1952d; Jones and Murant 1975; Jones and Jennings 1980b). Latent infections with these and other viruses are also believed to be involved in the degeneration of vigor of some cultivars (Cadman 1951, 1952d; Jones 1980b, 1981b). Thus, in some cultivars single infections with RLMV and RLSV impaired cane quality and decreased berry weight, and, in cv. 'Glen Prosen', RLSV infection decreased total fruit yield (Jones 1980b). It is likely that in mixed infections with other viruses, a situation which commonly prevails, latent infections with RLMV and RLSV significantly decrease plant growth and yield. The effects of either of these two viruses in mixed infections with rubus yellow net virus are not known. (See "Rubus Yellow Net," p. 175, and "Raspberry Mosaic," p. 168.)

### Symptoms on Natural and Experimental Hosts

RLMV and RLSV can occur singly but are often found together in naturally infected wild European raspberry, cultivated red raspberry, and some cultivated blackberries (Cadman 1951, 1952d; Jones and Murant 1975; Jones and Jennings 1980b; A. T. Jones, unpublished data); RLMV and RLSV have also been detected in *R. gracilis* Roxb. and RLMV in *R. occidentalis* L. (Jones 1975b). Each virus has been experimentally transmitted to several *Rubus* species, and all *Rubus* species and cultivars tested have been found susceptible (Jones and Jennings 1980b). Most are infected symptomlessly, but a few species show pronounced symptoms. The symptoms produced in sensitive plants by infections with RLMV and/or with RLSV generally are very similar.

**Symptoms on sensitive red raspberry cultivars.** Leaves of primocanes show sharply defined angular, chlorotic yellow spots about 1 to 2 mm or larger, which are randomly distributed over the leaf; leaves are often distorted (fig. 215). Leaves on fruiting canes are usually small and deformed and often show a more intense spotting than those on primocanes; some spots may merge to form large interveinal chlorotic areas (fig. 216); fruiting laterals of such affected canes are poorly developed (fig. 217). Plants become stunted and often die within 2 to 3 yr of infection (fig. 218).

Table 9 lists 13 red raspberry cultivars known to be sensitive to infection with either RLMV or RLSV. In addition, the



Figure 215. — Angular chlorotic leaf spots and distortion in a leaf of a primocane of 'Glen Clova' red raspberry infected with raspberry leaf spot virus. (Copyright Scottish Crop Research Institute.)



Figure 216. — Angular chlorotic leaf spots in leaves of 'Norfolk Giant' red raspberry infected with raspberry leaf spot virus. Left: leaf from a primocane. Right: leaf from a fruiting cane. (Copyright Scottish Crop Research Institute.)



Figure 217. — Distortion and chlorotic mottling in leaves of fruiting canes of 'Glen Clova' red raspberry field infected with raspberry leaf spot virus. Notice poorly developed fruiting laterals and dead nodes. (Copyright Scottish Crop Research Institute.)



Figure 218. — Plants of 'Glen Clova' red raspberry killed by field infection with raspberry leaf spot virus. (Copyright Scottish Crop Research Institute.)

following cultivars are reported to show symptoms characteristic of those induced by RLMV or RLSV, but no information is available on which of the viruses is involved in the disease: 'Andenken an Paul Camenzind', 'Bois Blanc', 'Deutschland', 'Frommes Erfolg', 'Frommes Vollendung', 'Hailsham', 'Harzjuwel', 'Magnum bonum', 'Norwich Market', 'Novost'kuz'mina', 'Preussen', 'Rode Radboud', 'Romy', 'Turks Fruhe Rote', and 'Zeva I' (Bjørnstad 1953; Bouchery and Putz 1972; Harris 1933, 1940; Jordović 1963; Kuznetsova and Pomazkov 1971; Richter 1964a, b).

**Symptoms on blackberries.** Plants of *R. laciniatus* Willd., *R. procerus* P. J. Muell cv. 'Himalaya Giant', 'Boysen', 'Logan', and 'Tayberry' that are graft inoculated with RLMV or RLSV are often infected symptomlessly when kept in a heated greenhouse. *R. laciniatus* and *R. procerus*, however,

**Table 9.—Red raspberry cultivars sensitive to infection with raspberry leaf mottle or raspberry leaf spot viruses**

Cultivar	Reaction to infection with —	
	Raspberry leaf mottle virus	Raspberry leaf spot virus
Baumforth B	+	S
Burnetholm	+	S
Chartham	S	•
Gertrudis	S	+
Glen Clova	+	S
Malling Delight	S	+
Malling Landmark	S	+
Norfolk Giant	+	S
Phyllis King	+	S
St. Walfried	S	+
Seedling K	S	+
Veten	S	+
Zeva Herbsternte	S	+

+ = Susceptible but shows no symptoms, S = pronounced chlorotic angular spots in leaves, and • = not tested.

Data from Cadman (1951, 1952) and Jones and Jennings (1980).

sometimes develop a transient, faint chlorotic mottle a few weeks after grafting (Jones and Jennings 1980b). Under the cooler conditions of an unheated gauze house, and particularly after pruning, new growth of 'Boysen' and 'Tayberry' infected with RLMV, but not with RLSV, showed faint transient line patterns in leaves of fruiting laterals (Jones and Jennings 1980b; A. T. Jones, unpublished data).

**Symptoms on other *Rubus* species.** *R. henryi* Hemsl. and Kuntze and *R. occidentalis* develop apical necrosis and mosaic symptoms 4 to 8 wk after graft inoculation with either RLMV or RLSV (Cadman 1951, 1952d; Jones and Jennings 1980b). *R. molaccanus* L. also shows tip necrosis, but this is generally slower to develop than in *R. henryi* or *R. occidentalis* (A. T. Jones, unpublished data). *R. saxatilis* L. inoculated with RLMV, either by grafting or by aphids, develops conspicuous interveinal chlorotic patches (Cadman 1951). RLMV and RLSV symptomlessly infect *R. phoenicolasius* Maxim. (Jones and Jennings 1980b).

#### Natural and Experimental Transmission

RLMV and RLSV are each transmitted in nature by *Amphorophora idaei* Börner (also known as *A. rubi* (Kalt.)) but not by *Aphis idaei* van der Goot (also known as *Dorsalis (Aphis) idaei* van der Goot) (Cadman 1951, 1952d; Jordović 1963). RLMV was not transmitted by *Amphorophora rubi* Börner (bramble aphid), *A. ruborum* Börner, or *Macrosiphum fragariae* (Wlk.) [also known as *Sitobion fragariae*

(Wlk.)]; a single transmission by *M. euphorbiae* (Thos.) was unconfirmed (Cadman 1954). Each virus is transmitted by grafting to *Rubus* (Cadman 1951, 1952d; Jordović 1963; Jones and Jennings 1980b). Raspberry chlorotic spot virus (probably RLSV) studied by Jordović (1963) was not transmitted through soil or seed to raspberry, nor was it mechanically transmitted to herbaceous test plants. In Scotland, neither RLMV nor RLSV have been transmitted to herbaceous plants by mechanical inoculation with sap (Cadman 1951; A. T. Jones, unpublished data). The properties of the virus transmitted mechanically by Richter (1964a) from raspberry plants with Mosaic 2 symptoms, suggest that it is raspberry bushy dwarf virus (See "Raspberry Bushy Dwarf," p. 229.)

Experimental transmissions using *Amphorophora idaei*, although possible, are beset by difficulties, the chief of which is the inherent resistance of many *Rubus* species and raspberry cultivars either to the aphid vector or to the viruses themselves (Cadman 1954, 1961b; A. T. Jones, unpublished data). In transmissions from infected red raspberry to *R. idaeus* L., *R. saxatilis*, and *R. occidentalis*, the minimum acquisition and inoculation access times required by *Amphorophora idaei* to transmit RLMV were less than 30 min and less than 60 min, respectively. The frequency of transmission was increased by extending acquisition and inoculation access times (Cadman 1954; Converse et al. 1970a). Much less precise studies have been done with RLSV (Cadman 1952d); however, both viruses were regarded as having similar vector relations (Cadman 1961b) (probably semipersistent), but further work is necessary to confirm this.

#### Properties of the Causal Agents

No information is available on the particle morphology of RLMV and RLSV. No viruslike particles were observed by electron microscopy of thin sections of infected raspberry plants (A. T. Jones and I. M. Roberts, unpublished data). A few isometric viruslike particles about 30 nm in diameter were observed in partially purified preparations obtained from raspberry plants infected with RLMV (Jones 1976b). These were not infective to *Chenopodium quinoa* Willd. plants, suggesting that they were not those of black raspberry necrosis virus, (BRNV, see p. 178), but it is not known if these particles represent those of RLMV.

Both RLMV and RLSV are heat-labile viruses and infected raspberry plants can be freed from infection with these viruses by thermotherapy (Chambers 1961; Jordović 1963; Richter 1964b).

#### Detection and Identification

In raspberry cultivars sensitive to infection with RLMV or RLSV (table 9), infection can be detected by the presence of characteristic angular chlorotic spots on the leaves (figs. 215 and 216). However, in plants with pronounced symptoms of raspberry vein chlorosis virus (see "Raspberry Vein Chlor-



osis," p. 194) or veinbanding disease (see "Raspberry Mosaic," p. 168), diagnosis may be difficult. Detection of RLMV and RLSV in cultivars of unknown response to infection or in which the viruses are latent depends on graft transmission to *Rubus* species and cultivars sensitive to either virus. The standard *Rubus* indicators, *R. henryi* and *R. occidentalis*, react similarly to RLMV and RLSV and also to BRNV (see "Black Raspberry Necrosis," p. 178) (Jones and Jennings 1980b). These viruses induce few if any symptoms in most raspberry cultivars. BRNV, however, induces few if any symptoms in most red raspberry cultivars. Thus, specific detection of RLMV and RLSV can be made by graft transmission of the raspberry cultivars sensitive to these viruses listed in table 9. Of these cultivars, 'Malling Delight', 'Malling Landmark', and 'St. Walfried' have been used to detect RLMV; and 'Burnetholm', and 'Glen Clova', and 'Norfolk Giant' to detect RLSV (Cadman 1951, 1952d; Jones and Murrant 1975; Jones and Wood 1979; Jones and Jennings 1980b).

### Control Procedures

Raspberry plants infected with these viruses can be freed from infection by heat treatment at 32° to 37°C for 10 to 20 days (Chambers 1961; Jordović 1963, Richter 1964b; Baumann 1980; A. T. Jones, unpublished data) to produce virus-tested elite mother material. A combination of heat treatment and meristem tip culture appears to be more successful than heat treatment alone (Baumann 1981). In Great Britain, most plants of the cv. 'Malling Jewel' (the main cultivar grown) derived from elite stock are reinfected with these and other aphid-borne viruses 2 to 3 yr after planting in the field (Jones and Murrant 1975; Jones 1976d). Attempts to prevent reinfection using insecticides have not been successful (Taylor and Chambers 1969).

Cadman and Fiskin (1958) observed that raspberry cultivars differed in the rate at which they became infected with RLSV in the field and found cvs. 'Malling Landmark' and 'Norfolk Giant' the most resistant. As cv. 'Malling Landmark' appeared very resistant to colonization by the aphid vector *Amphorophora idaei* and 'Norfolk Giant' did not (Cadman 1961b), they postulated two kinds of resistance mechanisms; that in cv. 'Malling Landmark' was effective against the vector, that in cv. 'Norfolk Giant' was effective against the virus. It is now known that cv. 'Norfolk Giant' also has some resistance to *Amphorophora idaei* (Knight et al. 1959; Jennings 1963; Jones 1976d, 1979a) and that much of its ability to escape infection is probably due to this. Nevertheless, the two kinds of resistance postulated by Cadman and Fiskin (1958) are currently being exploited by plant breeders to protect cultivars from the effects of infection with RLMV, RLSV, and other aphid-borne viruses (Jones 1981b).

Jones and Murrant (1975) showed that the weak resistance (minor gene resistance) to *Amphorophora idaei* in the cv. 'Glen Clova', which is sensitive to RLSV infection, was very

effective in restricting infection with RLSV, but only when grown in large plots. Under such conditions, the incidence of infection increased less than 1% per year and infected plants were largely confined to the periphery of the crop, and especially where this was adjacent to sources of the virus and its vector. In small plots and in areas of high inoculum pressure, the incidence of infection was much greater.

Later studies (Jones 1976d, 1979a), showed that major gene resistance to *Amphorophora idaei* was much more effective in restricting spread of aphid-borne viruses even under high inoculum pressure. Observations of commercial plantings of such material confirm these experimental results (Jones 1981c). Current raspberry breeding programs in Great Britain emphasize selection for resistance to this vector, and the planting of cultivars containing this resistance promises to be a major factor in decreasing the incidence and spread of RLMV and RLSV in commercial raspberry crops.

The severity of symptoms induced by infection with RLMV and RLSV in sensitive cultivars poses a serious threat should strains of *Amphorophora idaei* arise that can overcome the resistance currently being used in raspberry breeding programs. Jones and Jennings (1980b) found no source of immunity to either virus in many *Rubus* species and cultivars tested but showed that most were tolerant of infection. They found that inheritance of sensitivity to RLMV and RLSV infection was determined by single dominant genes designed *Lm* and *Ls*, respectively. Plant breeders should therefore be able to avoid introducing this sensitivity to infection with these viruses into future cultivars, although they have unwittingly allowed this to happen in the past.

### Remarks

The viruses have been detected only in Europe, New Zealand, and the U.S.S.R; however, tests to detect virus infection in raspberry in most countries are made by graft inoculation of *R. henryi* and *R. occidentalis*. Infection with these viruses in many other countries may have gone unnoticed because, as noted earlier in this chapter, these indicators will not distinguish between infection with RLMV, RLSV, and BRNV and because most red raspberry cultivars are symptomless when infected with RLMV and RLSV. RLMV and RLSV can be distinguished from BRNV in pure culture by inducing symptoms in red raspberry cultivars in which BRNV is symptomless (Jones and Jennings 1980b).

RLMV and RLSV differ from the agent of yellow spot disease of raspberry described from Poland (Basak 1974; see "Rubus Virus Diseases of Minor or Undetermined Significance," p. 248) by infecting *R. henryi* and by inducing apical necrosis in this species and in *R. occidentalis*. Furthermore, yellow spot disease affects red raspberry cultivars that are symptomless when infected by RLMV or RLSV.

Further information is needed on the relationship of RLMV and RLSV to one another and to BRNV, and on their vector relations.

## Raspberry Leaf Curl

By R. Stace-Smith and R. H. Converse

### Additional Common Names

Curl; raspberry curl; yellows. The term “yellows” was used in the early, descriptive literature (Rankin and Hockey 1922) to refer to a disease complex that included raspberry leaf curl. In Europe and the U.S.S.R., the name “leaf curl” is used to refer to a disease caused by raspberry ringspot and tomato black ring viruses (Murant 1974b). (See “Nematode-Borne Diseases,” p. 211.)

### History and Geographic Distribution

Raspberry leaf curl virus (RLCV) was one of the first virus diseases of *Rubus* recognized in North America. The early literature on the disease was reviewed by Rankin and Hockey (1922), who believed that it was transmitted by the aphid *Aphis rubiphila* Patch (now known as *A. rubicola* Oestlund). Confirmation of *A. rubicola* as a vector was provided by Smith (1925) and by Bennett (1927) who also established the existence of two types of RLCV which he called alpha and beta. Both types are restricted to North America and can infect blackberry (Bennett 1930). Otherwise, in cultivated *Rubus*, alpha curl is limited to red and purple raspberries, whereas beta curl also infects black raspberries.

Raspberry leaf curl disease occurs almost everywhere raspberries are grown in the United States and Canada. In a survey in Quebec, raspberry leaf curl disease was found in 8% of the red raspberry fields surveyed (Caron et al. 1977). Raspberry leaf curl disease is rare along the eastern seaboard south of New York and is not known on the Pacific slope. The absence of *A. rubicola* on *Rubus* on the Pacific slope explains the absence of the disease there.

### Economic Importance

Where it occurs in North America, raspberry leaf curl has the potential of being an important raspberry disease problem. Yield reductions in red raspberries of 20 to 40% and reduction in fruit quality have been reported, and infected plants may fail to survive the winter after a few seasons (Bennett 1927; Bolton 1970).

### Symptoms on Natural and Experimental Hosts

RLCV has been transmitted only to the genera *Rubus* and *Fragaria*. In the subgenus *Idaeobatus* (raspberries), natural *Rubus* hosts include *Rubus idaeus* L. and *R. idaeus* var. *strigosus* (Michx.) Maxim. (red raspberry), *R. occidentalis* L. (black raspberry), *R. neglectus* Peck (purple raspberry), *R. phoenicolasius* Maxim (wineberry); subgenus *Eubatus* hosts (blackberries, which are minor hosts) include *R.*

*allegheniensis* Porter (wild blackberry and cv. ‘Eldorado’), *R. procerus* P. J. Muell. (Himalaya blackberry), and *R. ursinus* Cham. & Schlect. (Pacific coast trailing blackberry).

**Symptoms on red raspberry.** Plants show no symptoms or, at most, a mild downcurling of the tip leaves in the current year of infection (fig. 219). The following spring, leaves on both fruiting canes and primocanes are curled and slightly yellow (fig. 220). The fruiting laterals are shortened, and there may be proliferation of the shoots, producing a rosette. New canes are stunted, numerous, and branched at the leaf axils. The plants remain stunted and are often killed in a succeeding winter. Fruit in diseased plants is small and crumbly. All red raspberry cultivars tested are susceptible to infection (Stace-Smith 1962a), although there are marked differences among cultivars in their resistance to colonization by the vector aphid, *Aphis rubicola*. (See “Natural and Experimental Transmission.”)

**Symptoms on purple raspberry.** Symptoms are milder than on other raspberries, and the cv. ‘Columbian’ was found to recover spontaneously from infection by the alpha strain of RLCV, a rare phenomenon in plant virology (Bennett 1930). Arneson and Braun (1975) felt that purple raspberry cultivars grown at that time were resistant to both alpha and beta strains of RLCV.

**Symptoms on black raspberries.** The symptoms are similar to those on red raspberry. Leaves are arched, firm, and remain small and nearly circular in outline, developing a dark greasy-green cast (fig. 221). In a chronic infection, the young canes are stiff and brittle, and frequently do not branch.

**Symptoms on blackberries.** Some blackberry cultivars show symptoms similar to those on red raspberry, whereas other cultivars remain symptomless.

Experimental hosts include: *Fragaria vesca* L. var. *semperflorens* (Duch.) Ser. cv. ‘Alpine’ (Alpine strawberry) (Stace-Smith 1962a); *Rubus albens* Roxb. (tropical black raspberry, with the beta strain (R. H. Converse, unpublished)); *R. baileyanus* Britton x *R. argutus* Link cv. ‘Lucretia’ (‘Lucretia’ dewberry) (Bennett 1930); *R. henryi* Hemsl. and Kuntze (alpha and beta strains) (R. Stace-Smith 1962a; R. H. Converse, unpublished).

**Symptoms on indicator hosts.** Symptoms on wineberry (*R. phoenicolasius*): Symptoms are usually evident 7 to 10 days after aphid inoculation. The syndrome is essentially the same as on raspberry, but is considerably more pronounced, rapid, and reliable in development. The petiole of the tip leaf is recurved downward. The leaf blade does not expand normally, and the interveinal tissue of the unexpanded portion is chlorotic. Succeeding leaves are curled and stunted, resulting in a rosette at the tip of the plant (fig. 222).

**Symptoms on *R. henryi*.** The first symptoms are evident 10 to 14 days after inoculation. The young leaves are chlorotic and develop an asymmetric twist (fig. 223). Within a month after inoculation, the shoot tips and axillary buds become necrotic.

**Symptoms on 'Alpine' strawberry.** Interveinal chlorosis is evident about 3 wk after graft inoculation. This chlorosis is visible on two or three succeeding leaves; later, infected plants tend to recover and cannot be distinguished from control plants. The presence of the latent A strain of strawberry crinkle virus does not intensify the symptoms, as it does with some of the strawberry viruses. (See "Strawberry Crinkle," p. 20.)

#### Natural and Experimental Transmission

RLCV has been transmitted by aphids as well as by patch and petiole-insert grafting (Harris 1935; Stace-Smith 1962a; Smith 1925).

*Aphis rubicola* is the only known natural vector of RLCV (Smith 1925; Bennett 1927, 1930; Converse 1962; Stace-Smith 1962a; Brodel et al. 1979) (fig. 224). *A. rubicola* transmitted alpha and beta strains of RLCV (Bennett 1930). The alpha strain was acquired after 2 hr of feeding and persisted in the aphid several days (Bennett 1927). Table 10 summarizes the known vector relationships of RLCV. The aphid is sluggish and is thought to be a rather inefficient vector (Bolton 1970). The influence of time, temperature, and light on the production of sexual forms of *A. rubicola* have been studied (Brodel and Schaefers 1979; Brodel and Schaefers 1980a).

In studies in New York, populations of *A. rubicola* reached a minor peak in late July and a major peak in early October (Schaefers 1967). RLCV transmission patterns appeared to follow the direction of prevailing winds and to be more influenced by local populations of viruliferous *A. rubicola* in a given field than by more distant occurrences (Bolton 1970). Differences exist in the resistance of red raspberry cultivars to supporting colonies of *A. rubicola*. No immunity to colonization has been found. However, significant and repeatable differences in resistance to colonization (10-30% below susceptibles) have been identified in red raspberry. Selections and cultivars like NY 632, 'Canby', 'Latham', and 'Willamette' are being used in the New York raspberry breeding program (Brodel et al. 1979; Kennedy et al. 1973).

*Aphis idaei* van der Goot is an experimental vector of RLCV (Stace-Smith 1962a). The relationships of RLCV with this aphid vector are summarized in Table 10. The aphid occurs in Eurasia but is known in the Western Hemisphere only in coastal British Columbia, where RLCV does not occur. RLCV might become a problem in red raspberry in Europe if introduced because of the widespread occurrence of *A. idaei* there.



Figure 219. — Early symptoms of alpha strain of raspberry leaf curl virus on 'Lloyd George' red raspberry.



Figure 220. — Chronic symptoms of alpha strain of raspberry leaf curl virus on 'Lloyd George' red raspberry.

*Aphis rubifolii* (Thos.), a small blackberry aphid, may be a vector of RLCV on blackberry since the disease is reported on 'Eldorado' blackberry, which is not a host of *A. rubicola* (Bennett 1930); however, in greenhouse tests, *A. rubifolii* failed to transmit RLCV (Converse 1962).





Figure 221. — Chronic symptoms of beta strain of raspberry leaf curl virus on 'New Logan' black raspberry.



Figure 222. — Symptoms of alpha strain of raspberry leaf curl virus on wineberry seedling (*Rubus phoenicolasius*).



Figure 223. — Raspberry leaf curl disease symptoms on *Rubus henryi*.



Figure 224. — *Aphis rubicola* on red raspberry shoot.

*Amphorophora agathonica* Hottes (referred to in older literature as *A. rubi*), *A. sensoriata* Mason, *Illinoia rubicola* (Oestlund) [also known as *Macrosiphum rubicola* (Oestlund)], and *Aphis spiraeicola* Patch, all aphids that feed on red raspberry, have been found to be nonvectors of RLCV (Bennett 1930; Converse 1962; Stace-Smith 1962a).

#### Properties of the Causal Agent

The alpha strain of the RLCV will not crossprotect against the beta strain (Bennett 1930); therefore, the notion that alpha and beta are strains of one virus remains unconfirmed. Movement of the alpha strain in raspberry is relatively slow and is limited to phloem tissues (Bennett 1927). Both strains of RLCV behave like circulative viruses in *A. rubicola*. Stace-Smith and Lo (1973) speculated that RLCV might be a bacilliform virus like raspberry vein chlorosis virus (see "Raspberry Vein Chlorosis," p. 194) because of the similar vector relationships of the two viruses, but subsequent examination of thin sections of RLCV-infected raspberry tissue failed to demonstrate the occurrence of such particles (R. Stace-Smith, unpublished). Matthews (1979) included RLCV as a possible member of the luteovirus group because of its vector relationships. Direct information is lacking on morphology, properties, and serological relationships of RLCV.



**Table 10.—Some vector-virus-host plant relationships of raspberry leaf-curl virus**

Vector	Vector host range	Virus strain	Transmitting stages of aphids	Minimum acquisition feed time	Inoculation threshold	Maximum retention	Transmission efficiency of aphids	References
<i>Aphis rubicola</i>	Red, black, purple raspberry; wineberry.	Alpha	All but egg	2 hr	—	Life	Single = 19%	Bennett (1927, 1930); Smith (1925).
		Beta	All but egg	—	—	—	—	Bennett (1930).
<i>Aphis rubifolii</i>	Blackberry	(?)	Field population.	—	—	—	—	Bennett (1927).
<i>Aphis idaei</i>	Red raspberry.	Alpha	—	24 hr	20 min	11 + days	Groups of 10 = 80%.	Stace-Smith (1962).

### Detection and Identification

Raspberry leaf curl disease can be readily detected in the field in raspberries by the tightly curled foliage of infected plants (fig. 225). False symptoms resembling raspberry leaf curl disease can be caused by infestations of *A. rubifolii* on blackberries (Hottes and Frison 1931), by heavy infestations of nonviruliferous *A. rubicola* on young black raspberry foliage, and by two European nepoviruses, raspberry ringspot virus and tomato black ring virus (see "Nematode-Borne Diseases" p. 211). Chronically infected raspberries are always severely dwarfed, produce few main stems, and have curled foliage. Wineberry is a rapid indicator host (by leaf grafting or by transmission with *A. rubicola*) for latent or presumptive infections, but both red and black raspberry must be inoculated to distinguish between alpha and beta curl viruses.

### Control Procedures

Control procedures include the use of certified planting stock free of this disease, avoidance of planting new fields near infected wild or cultivated raspberries, periodic inspection and roguing of infected plants, use of aphicides to limit populations of *A. rubicola*, and use of cultivars resistant to colonization by *A. rubicola*. Since the apterae of *A. rubicola* are relatively sluggish and do not readily drop off disturbed foliage, inspections and roguing programs are useful for control of raspberry leaf curl disease. A few new infected plants can be expected each year in raspberry fields in areas where the disease is prevalent, even though the above control procedures are practiced, because of the movement of viruliferous alate *A. rubicola*.

Immunity exists in 'Plum Farmer' black raspberry against both alpha and beta strains of RLCV (Converse 1962), but has not yet been used to develop immune, horticulturally desirable types of red, purple, or black raspberry. Red raspberry cultivars resistant to colonization by *A. rubicola*,



Figure 225. — Raspberry leaf curl disease symptoms on a naturally field-infected red raspberry.

however, are being developed by the New York Agricultural Experiment Station (Brodel et al. 1979; Kennedy et al. 1973).

The alpha strain of RLCV was not eliminated from infected plants held at an air temperature of 37°C for periods up to 4 wk (Stace-Smith 1962a). Heat therapy is academic, however, since cultivars are not universally infected.

### Remarks

A number of gaps exist in the basic information about both strains of RLCV. Nothing is known about the properties of the virus particles. Electron microscopy of thin sections of infected raspberry phloem tissue should provide some evidence of the morphology of the virus(es) causing this disease. Several of the basic virus-vector properties have not yet been reported, especially those for the beta strain and the inoculation threshold periods.

## Cucumber Mosaic Virus In Raspberry

By A. T. Jones

### Additional Common Names

None.

### History and Geographic Distribution

First reported from a few plants of *Rubus idaeus* L. cv. 'Lloyd George' in Scotland (Harrison 1958a). Later records of infection come from Scotland (Jones 1976a, 1980c) and the Soviet Far East (Gordejchuk et al. 1977). Fern-leaf symptoms in 'Cumberland' black raspberry plants growing adjacent to cucurbits in Pennsylvania were suggested by Zundel (1931) to be due to cucumber mosaic virus (CMV) infection because of the similarity of the symptoms to those produced by CMV in tomato. However, experimentally infected *R. occidentalis* L. showed only a mild foliar mottle (Harrison 1958a) quite unlike the symptoms observed by Zundel (1931). CMV occurs worldwide in many different crops and weed species (Francki et al. 1979), and it is likely that it occurs very occasionally in *Rubus* species worldwide.

### Economic Importance

The virus appears to be lethal in *R. phoenicolasius* Maxim. (Jones 1976a) but induces only mild foliar symptoms in red raspberry (Gordejchuk et al. 1977; Harrison 1958a; Jones 1980c) and is symptomless in cultivated brambles (Jones 1976a, 1980c). Infections are rare in *Rubus* in Scotland and are of no importance economically.

### Symptoms on Natural and Experimental Hosts

**Rubus species.** The few cultivated bramble plants found infected with CMV in Scotland were symptomless (Jones 1976a). Pale green blotching of the leaves occurs in the red raspberry cv. 'Lloyd George' with no apparent effect on plant vigor or fruiting (Harrison 1958a). A single CMV-infected plant of an unnamed red raspberry selection showed foliar chlorotic ringspot symptoms but no obvious degeneration in vigor (fig. 226A) (Jones 1980a). In the Soviet Far East, infected raspberry plants of the cultivar 'Visluha' were characterized by small leaves with bright chlorotic mottling (Gordejchuk et al. 1977). In contrast, leaves of field-infected plants of *R. phoenicolasius* were misshapen and showed areas of chlorotic blotching and line pattern, which often became bright yellow in summer (fig. 226B); plants showed a marked decline in vigor, and some plants died within 3 to 4 yr (Jones 1976a). Experimentally infected plants of *R. occidentalis* developed a green mosaic (Harrison 1958a).

**Herbaceous species.** CMV infects a wide range of herbaceous plants (Francki et al. 1979). Isolates obtained from *Rubus* species infected and induced symptoms in the following hosts: *Chenopodium amaranticolor* Coste and Reyn., *C. murale* L., *C. quinoa* Willd., and *Phaseolus vulgaris* L. — necrotic local lesions in a few days, not systemic. *Cucumis sativus* L., *Nicotiana clevelandii* Gray, *N. glutinosa* L., and *N. tabacum* L. cvs. 'White Burley' and 'Xanthi-nc' — systemic mosaic.

### Natural and Experimental Transmission

Natural transmission occurs by many aphid species and is in the nonpersistent manner (Kennedy et al. 1962). *Rubus* isolates have been transmitted between herbaceous hosts by *Amphorophora idaei* (Börn.) (formerly *A. rubi* (Kalt.)), *Macrosiphum euphorbiae* (Thos.), and *Myzus persicae* (Sulz.) (Harrison 1958a; Jones 1976a). Only *A. idaei*, however, has been shown to transmit CMV to raspberry. Although CMV is transmitted through the seed of several of its hosts (Francki et al. 1979), it was not detected in seedlings derived from CMV-infected *R. phoenicolasius* (Jones 1976a).

The virus is readily transmissible by inoculation of sap from most hosts, but transmission from *Rubus* species is sometimes difficult even when extracts are made in 2% nicotine solution. Mechanical transmission of CMV to *Rubus*, even with purified preparations, was not successful in Scotland (Harrison 1958a; Jones 1976a), but is reported from the U.S.S.R. (Gordejchuk et al. 1977).

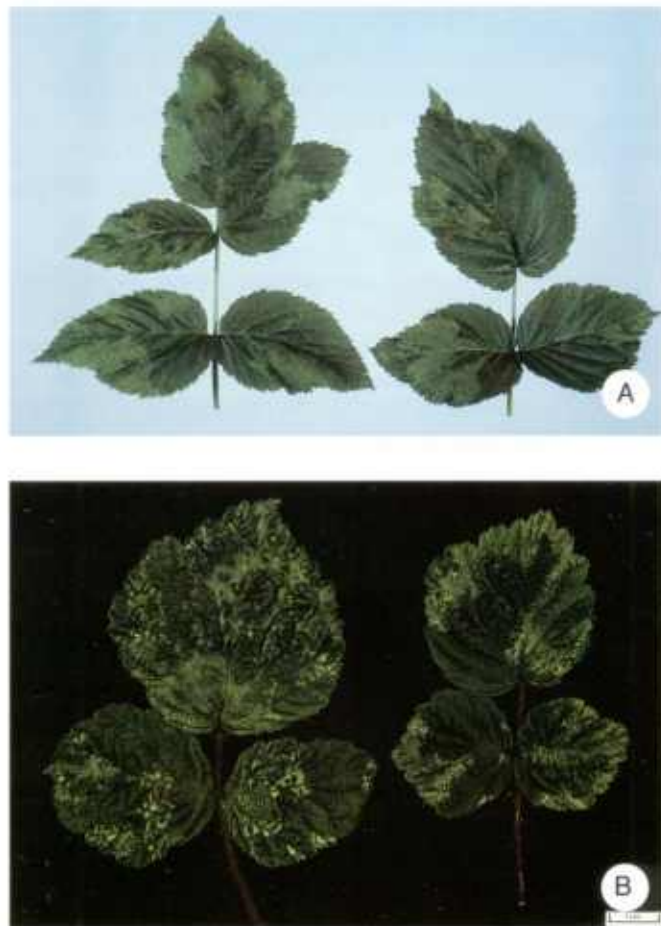


Figure 226. — Chlorotic blotches and line pattern in leaves of *Rubus* infected with cucumber mosaic virus: A, Unnamed red raspberry selection; B, *Rubus phoenicolasius*. (Copyright Scottish Crop Research Institute.)

An isolate in *R. phoenicolasius* was readily graft transmissible to *R. phoenicolasius* but did not infect *R. henryi* Hemsl. and Kuntze, *R. loganobaccus* Bailey, *R. procerus* P. J. Muell, or several red raspberry cultivars including cv. 'Lloyd George' — a cultivar found naturally infected with CMV. This suggests either that this isolate is restricted in its *Rubus* host range or that graft transmission of CMV to certain *Rubus* species is difficult.

### Properties of the Causal Agent

For a detailed description of the virus see Francki et al. (1979). All isolates of CMV are relatively unstable. In sap of *Nicotiana* species, *Rubus* isolates lost infectivity after diluting to  $10^{-3}$  to  $10^{-4}$ , heating for 10 min at 65° to 70°C, or storage at 20°C for 3 to 5 days (Harrison 1958a; Jones 1976a).

Most strains, but not all (Francki et al. 1979), can be purified by clarification with organic solvents, followed by precipitation with 10% polyethylene glycol (mol. wt. 6000) or acidification to pH 5.0 and differential centrifugation. Further purification can be achieved by sucrose density gradient centrifugation (for further details see Francki et al. 1979). The only *Rubus* isolate to be purified and examined in any detail is that from *R. phoenicolasius* (Jones 1976a). Like other CMV isolates, it has icosahedral particles about 28 nm in diameter (fig. 227), which sediment as a single component with a sedimentation coefficient of about 92 S (Jones 1976a). Strains of CMV that have been examined in more detail are known to contain a single polypeptide of about 24,500 mol. wt. and four ssRNA species; some isolates contain an additional satellite RNA species of about 100,000 mol. wt. At least one of these satellite RNA species is known to modify the symptoms induced by the satellite-free CMV strain in some hosts (Francki et al. 1979).

There are several serotypes of CMV but all *Rubus* isolates tested by Jones (1976a and unpublished) were serologically indistinguishable from the W strain, which is found commonly in British crop and weed plants (Tomlinson et al. 1973).

### Detection and Identification

Detection of infection is by inoculation of sap from *Rubus* in 2% nicotine solution to *C. quinoa*, *C. amaranticolor*, or *N. clevelandii* test plants. Identification of isolates can be made by testing the ability of sap from infected test plants to react with CMV antiserum. In agar gel double diffusion tests, particles of many CMV isolates are degraded in the absence of ethylene diamine tetra-acetate (EDTA) (Tomlinson et al. 1973; Jones 1976a); extracts from plants and the agar gel should therefore contain 0.001 M EDTA.

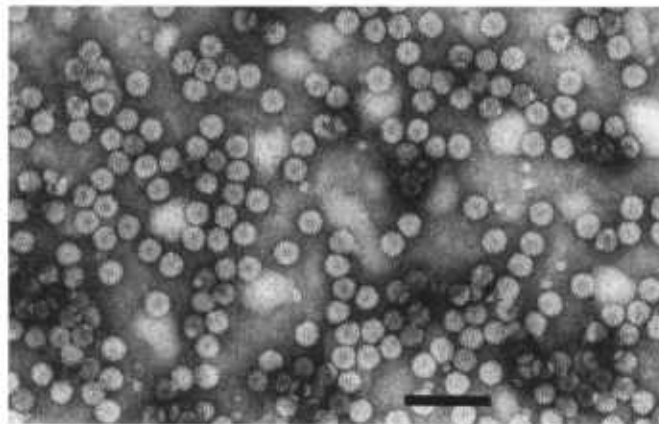


Figure 227. — Electron micrograph of a purified preparation of cucumber mosaic virus particles stained in 2% ammonium molybdate, pH 6.5. Bar represents 100 nm. (Copyright Scottish Crop Research Institute.)

### Control Procedures

Infection with CMV is rare in *Rubus*; consequently, elimination of the virus from *Rubus* has not been attempted. The possibility of propagation from CMV-infected plants should easily be avoided by indexing mother plants for virus infection.

### Remarks

CMV is not systemic in *C. quinoa* and can therefore be readily distinguished in this host from black raspberry necrosis virus (see "Black Raspberry Necrosis," p. 178), nepoviruses (see the chapters in this section dealing with nematode-borne viruses), raspberry bushy dwarf virus (see "Raspberry Bushy Dwarf," p. 229), and isolates of tobacco streak virus (see "Tobacco Streak Virus in *Rubus*," p. 235). It is also distinguishable from wineberry latent virus (WLV; see "Wineberry Latent Virus," p. 239) in *C. quinoa* as local lesions induced by WLV, although initially small, expand to 2 to 3 mm, whereas those induced by CMV remain the size of pinpoints; furthermore, the particles of WLV are filamentous and easily distinguished from CMV.

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Thimbleberry Ringspot<sup>11</sup>  
By R. Stace-Smith

### Additional Common Names

None.

### History and Geographic Distribution

The virus was described from naturally infected thimbleberry (*Rubus parviflorus* Nutt.) in British Columbia. Thimbleberry is a native understory plant widespread in the Pacific Northwest. It grows in patches under closed forest canopy or in the open, but characteristically along the edges of forest clearings and along forest creeks. The viruslike symptoms were first observed on a few plants near Vancouver, British Columbia, in 1953. The symptoms were later shown to be caused by a virus to which the name thimbleberry ringspot was applied (Stace-Smith 1958). In the intervening years,



many patches of thimbleberry have been examined for viruslike symptoms, but no additional infections have been observed (R. Stace-Smith, unpublished).

### Economic Importance

The virus is of no economic importance in thimbleberry. It was initially investigated because wild *Rubus* hosts could serve as a potential reservoir of viruses in commercial plantings. The fact that the virus has never been detected in cultivated *Rubus* spp. suggests that it is unlikely to constitute a potential problem.

### Symptoms on Natural and Experimental Hosts

Thimbleberry is the only natural host that is known. Diseased plants exhibit irregular ringspot and oakleaf markings on the leaves. The rings are faint and small as the young leaves unfold in the spring, but as the leaves expand the markings become more pronounced (fig. 228). Some leaves do not show line patterns and symptoms are restricted to patches of netlike vein chlorosis (fig. 229), varying in intensity of symptoms. Some show no or only slight mottling, whereas other leaves on the same plant show severe mottling and leaf distortion. Those plants with mild foliar symptoms show no stunting; those with severe mottling bear canes that are stunted.

**Experimental hosts.** The virus has been transmitted from thimbleberry to three *Rubus* spp., each of which was susceptible. Red raspberry (*R. idaeus* L. cv. 'Washington') developed faint chlorotic markings that took the form of ringspot or oak leaf patterns. Symptoms were not as striking as on thimbleberry. Markings could be detected readily on the leaves produced in the spring but were faint on leaves produced during the summer. The virus had no obvious effect on plant vigor or fruit yield. Black raspberry (*R. occidentalis* L. cv. 'Munger') developed a distinct mottling on some leaves (fig. 230), but more often there was a diffuse mottling rather than a definite pattern. Within a few weeks of the initial appearance of leaf symptoms, the tips of affected plants became necrotic and the axillary buds near the base of the cane sprouted, giving the plant a rosette type of growth. *R. henryi* Hemsl. and Kuntze developed symptoms about 7 wk after grafting, when chlorotic spots appeared on the young expanding leaves near the tip of the plant. Terminal buds on affected canes failed to develop and became necrotic. The chlorosis and necrosis of the first few canes to show symptoms could be considered a shock reaction since the plant recovered, and leaves on subsequent canes either showed no symptoms or at most a few chlorotic spots.

### Natural and Experimental Transmission

No direct evidence is available as to the source of the few virus-infected plants that have been found or whether the virus is spreading in nature. Although thimbleberry plants produce seed, most of the canes arise from underground rhizomes and a single infected plant may produce several canes. The outbreaks near Vancouver have been monitored



Figure 228. — Chlorotic line patterns and rings that develop on leaves of thimbleberry plants infected with thimbleberry ringspot virus.



Figure 229. — Mild symptoms of thimbleberry ringspot virus, consisting of patches of diffuse vein chlorosis, on some leaves of an infected thimbleberry plant.

for nearly three decades, and the virus has not spread to adjacent healthy plants. Further, the infected plants that have been found are confined to forested areas, well isolated from agricultural areas, suggesting that the virus had its origin in wild plants and is rarely transmitted in nature.

Despite the fact that there is no evidence of natural spread, should any natural transmission occur it could probably be attributed to one of the thimbleberry inhabiting aphids. The aphid *Illinoia* (also known as *Masonaphis*) *maxima* (Mason) is prevalent in the area, appears to be confined to the young leaves and terminal shoots of thimbleberry (Frazer and Forbes 1968), and is capable of transmitting the virus from thimbleberry to thimbleberry and from thimbleberry to black raspberry (Stace-Smith 1958). Two other aphid species, *Illinoia davidsoni* (Mason) and *Amphorophora parviflora* Hill, are occasionally found on thimbleberry and are also experimental vectors. All three species are inefficient vectors; when aphids were transferred from an infected source plant to thimbleberry seedlings, 10 aphids per test plant, a low percentage of the exposed seedlings became infected (Stace-Smith 1958).





Figure 230. — Diffuse mottle symptoms on black raspberry (*Rubus occidentalis*) cv. 'Munger' following aphid inoculation with thimbleberry ringspot virus.

### Properties of the Causal Agent

Thimbleberry ringspot virus has not been transmitted mechanically despite repeated attempts using a variety of buffers and a range of herbaceous test plants (R. Stace-Smith, unpublished). It is assumed that the virus, like most aphid-transmitted viruses affecting *Rubus* spp., is not mechanically transmissible.

The virus can be transmitted by each of the three aphid species that occur on thimbleberry in British Columbia, namely *Illionia maxima*, *I. davidsonii*, and *Amphorophora parviflora* but not by the large raspberry aphid *A. agathonica* Hottes (Stace-Smith 1958). Transmission efficiency is low, and infective aphids retain the virus for less than 1 day.

In thin section electron microscopy of infected thimbleberry leaf tissue, virus-like particles were detected. The particles were spherical, about 25 nm in diameter, and confined to single rows enclosed in tubules. Such tubules were only detected in cell walls or invaginations in cell walls between the leaf mesophyll cells (R. Stace-Smith, unpublished).

### Detection and Identification

The virus can be detected by visual examination of thimbleberry plants, particularly in the spring of the year, where the characteristic ringspot or oak leaf markings are readily seen. As the season progresses, the symptoms are less obvious, and many plants that are free of this virus develop chlorotic blotches on the upper leaf surface as a result of powdery mildew (*Sphaerotheca humuli* (DC.) Burr.) infection on the lower surface. In a superficial examination, such blotches might be confused with symptoms of viral origin. Surveys in British Columbia on the incidence of virus in the native thimbleberry have revealed no viruses other than thimbleberry ringspot, and even this virus appears to be confined to a limited area. Thimbleberry is widely distributed

in western North America, and additional infections may be detected in the future. Should plants suspected of being infected with thimbleberry ringspot be found, tentative identification could be made on the basis of the symptoms induced on the natural host. Positive identification would require graft or aphid transmission tests to *R. occidentalis* and *R. henryi*.

### Control Procedures

Thimbleberry ringspot is an unusual *Rubus* virus in that it appears to be confined in nature to the wild host. The facts that it is transmitted inefficiently by the thimbleberry-inhabiting aphids and that these aphids rarely feed on other *Rubus* spp. suggest that there is little likelihood of the virus ever being introduced into any of the commercial *Rubus* species. For the above reasons, control measures may never be required.

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### Raspberry Vein Chlorosis<sup>11</sup>

By A. T. Jones, A. F. Murrant, and R. Stace-Smith

#### Additional Common Names

Vein chlorosis mosaic (Cadman and Harris 1951); raspberry chlorotic net (Jordović 1963); Adernchlorose (Richter 1964a, b).

#### History and Geographic Distribution

The disease, which occurs naturally only in red raspberry, was first described from Scotland by Cadman (1952a) who showed that it was caused by a graft-transmissible agent, which he called raspberry vein chlorosis virus (RVCV). The disease was later found in Canada (Stace-Smith 1961), Europe (Bjørnstad 1958; Helebrant 1958; Jordović 1963; Richter 1964a; Szilágyi 1980; Tapio 1961), U.S.S.R. (Kuznetsova and Pomazkov 1971; Tiits 1962), and New Zealand (Cadman and Stace-Smith 1970; Jones and Wood 1979). The disease is widespread in Great Britain and continental Europe, common in some areas of New Zealand, but rare in Canada. It has not been reported from other raspberry growing areas such as Australia and the United States.

The virus is transmitted by the aphid *Aphis idaei* van der Goot (Cadman 1952a), which is widespread in Europe. The aphid appears not to be present in Australia and is found only infrequently in British Columbia; infection and spread of the disease in New Zealand and Canada are probably due to introduction and propagation of infected plants from Europe (Cadman and Stace-Smith 1970; Jones and Wood 1979).

#### Economic Importance

Vein chlorosis is one of the commonest diseases in raspberry in continental Europe and the U.S.S.R. This high incidence is largely caused by propagation from infected stock. Almost all stocks of some cultivars, such as 'Baumforth B', are infected with the virus. Although on its own the virus is not

lethal to raspberry, it undoubtedly affects plant vigor, especially in combination with other viruses. In some cultivars, the virus causes significant decrease of berry weight (Daubeney et al. 1970; Jones 1980d); infected plants of cv. 'Malling Leo' produced thinner canes and earlier ripening fruit than did virus-free controls (Jones 1980b). Infection of some cultivars was associated with increased pollen abortion (Freeman et al. 1969) and retarded embryo sac development (Eaton and Turner 1971).

#### Symptoms on Natural and Experimental Hosts

In nature, the virus has been found only in red raspberry (*R. idaeus* L. and *R. idaeus* var. *strigosus* (Michx.) Maxim.); only two other species (*R. loganobaccus* Bailey and *Fragaria vesca* L.) have been infected experimentally.

The following red raspberry cultivars have been reported to be infected with RVCV either naturally or by graft inoculation in Great Britain, Canada, continental Europe, and New Zealand: 'Asker', 'Badenia', 'Baumforth B', 'Burnetholm', 'Canby', 'Deutschland', 'Devon', 'Fairview', 'Gertrudis', 'Glen Clova', 'Glen Prosen', 'Golden Queen', 'Great American', 'Hailsham', 'Herbert', 'Joy', 'La France', 'Lloyd George', 'Malling Admiral', 'Malling Delight', 'Malling Exploit', 'Malling Leo', 'Malling Jewel', 'Malling Landmark', 'Malling Notable', 'Malling Orion', 'Malling Promise', 'Marcy', 'Newburgh', 'Norfolk Giant', 'Norna', 'Norwich Market', 'Park Lane', 'Preussen', 'Pyne's Royal', 'Romy', 'St. Walfried', 'Schönemann', 'Schopska alena', 'Seedling M', 'Seedling K', 'Seedling Z', 'Sumner', 'Taylor', 'Valjevka', 'Washington', 'Willamette', and 'Winkler's Seedling' (Bjørnstad 1953; Cadman 1952a; Freeman et al. 1969; Jones 1981b; Jones and Wood 1979; Jordović 1963; Richter 1964a, b; Stace-Smith 1961; Tapio 1961; A. T. Jones, unpublished data). A few North American cultivars (for example, 'Cuthbert', 'Latham', and 'Viking') seem immune to field infection and graft inoculation (Cadman 1952a; A. T. Jones, unpublished data).

'Boysen', 'Logan', 'Tayberry', *R. henryi* Hemsl. and Kuntze, *R. molaccanus* L., *R. occidentalis* L. cvs. 'Munger' and 'Plum Farmer', *R. phoenicolasius* Maxim. and *R. procerus* P. J. Muell cv. 'Bedford Giant' failed to become infected after graft inoculation (A. T. Jones, unpublished data). Stace-Smith (1961) also found *R. henryi* and *R. occidentalis* resistant to infection by graft inoculation, but found 'Logan' susceptible.

**Symptoms on red raspberry.** After graft inoculation, plants may not show symptoms until the following year, but plants grafted early in the year or inoculated by aphids may develop symptoms in 4 to 12 wk. The extent and severity of symptoms depends on host genotype, virus strain (Cadman 1952a), and growing conditions. The cvs. 'Lloyd George', 'Malling Delight', 'Norfolk Giant', and 'Washington' are good indicators (Jones et al. 1977).

Symptoms are usually less intense in the greenhouse than in the field. The most obvious symptoms appear on leaves of first-year canes as a chlorosis of the minor veins, either in patches (fig. 231) or throughout the leaf (fig. 232). When symptoms are severe, the leaf blade may become distorted by uneven growth as the leaf expands. In cv. 'Malling Delight', infected leaves may show epinasty (Jones et al. 1977).

**Symptoms on 'Logan'.** Plants graft-inoculated with a Canadian or a German isolate of RVCV developed symptoms similar to those in raspberry (Stace-Smith 1961; Richter 1964a).

**Symptoms on 'Alpine' strawberry.** Plants inoculated by *Aphis idaei* developed symptoms after 3 to 4 wk, similar to those in raspberry. In addition, veinal necrosis often developed along one of the secondary leaf veins (Jones et al. 1977).

#### Natural and Experimental Transmission

RVCV is transmitted in nature by *Aphis idaei*, but not by the aphids *Amphorophora agathonica* Hottes or *Amphorophora idaei* Börner (also known as *A. rubi* (Kalt.)) (Cadman 1952a; Stace-Smith 1961; Jordović 1963). The virus is also transmitted experimentally by grafting but not by mechanical inoculation with sap. It is not seed-transmitted in red raspberry (Jordović 1963).

*Aphis idaei* require at least 1 day to acquire RVCV, and they retain it for at least 1 day (Cadman 1952a; Stace-Smith 1961) and probably for life. Efficiency of transmission is increased after 7-day acquisition access feeds (Cadman 1952a), the greatest recorded frequency of transmission (46%) being obtained after acquisition and inoculation access periods of 7 and 30 days, respectively (Jordović 1963).

#### Properties of the Causal Agent

RVCV is not transmissible by inoculation of sap, nor has it been purified; however, electron microscopy of thin sections of raspberry leaves infected with RVCV alone showed large bacilliform particles, some about 430 to 560 × 65 to 91 nm and rounded at both ends and others shorter and rounded at only one end (fig. 233 A) (Stace-Smith and Lo 1973; Jones et al. 1974). The particles have a densely staining nucleocapsid about 50 to 70 nm in diameter surrounded by an electron-lucent zone and a unit membrane, which, in some sections, appears continuous with the endoplasmic reticulum. The nucleocapsid shows some crossbanding with a periodicity of 4 to 5 nm (fig. 233 B) (Stace-Smith and Lo 1973; Jones et al. 1974). Similar particles have been found in thin sections of viruliferous *Aphis idaei* (Murant and Roberts 1980).

In raspberry, virus particles were detected in only a small proportion of parenchyma cells of the vascular bundles and occurred singly or in groups (often within a membranous sac) in the cytoplasm and perinuclear space but not in the nucleus



Figure 231. — Patches of veinal chlorosis in a leaf of 'Lloyd George' red raspberry infected with raspberry vein chlorosis virus. (After Jones et al. 1977.)



Figure 232. — Chlorosis of the fine veins of a leaf of 'Glen Prosen' red raspberry infected with raspberry vein chlorosis virus. (After Jones et al. 1977.)

(Stace-Smith and Lo 1973; Jones et al. 1974). In *Aphis idaei*, virus particles were found in the brain and salivary glands, in the connective tissue surrounding the sucking pump and esophagus, and in the muscle cells of the sucking pump; they occurred only in the cytoplasm, not in the nuclei or perinuclear space (Murant and Roberts 1980).

The particles of RVCV closely resemble those of plant rhabdoviruses (Peters 1981), several of which are aphid-borne. By analogy with other plant rhabdoviruses, RVCV probably multiplies in its aphid vector but may have a long latent period after which the insect transmits it for life. The observations of Jordović (1963) that RVCV is transmitted most efficiently when allowed very long acquisition and inoculation feeds are consistent with this expectation.

### Detection and Identification

The characteristic clearing of the fine veins is evident in most susceptible cultivars of red raspberry. Confirmation of infection can be obtained by graft-inoculation to sensitive cultivars such as 'Lloyd George', 'Malling Delight', or 'Washington', although the presence of other viruses may make diagnosis difficult. Transmission to indicators by *Aphis idaei* is also possible, but the aphid is difficult to handle and maintain in culture. In some raspberry cultivars, the symptoms induced by rubus yellow net virus on its own (see 'Rubus Yellow Net,' p. 175) may be confused with those of RVCV, but the former are usually much less conspicuous. Pure cultures of the two viruses may be distinguished by graft-inoculation to *R. occidentalis*, which is immune to RVCV but develops a faint yellow net symptom on infection with rubus yellow net virus. Symptoms induced by either of these viruses alone are quite distinct from those of veinbanding mosaic disease. (See "Raspberry Mosaic," p. 168.)

### Control Procedures

In Scotland, the aphid vector *Aphis idaei* is usually uncommon, and roguing infected plants has helped to restrict spread (Cadman and Stace-Smith 1970). The aphid is much more common in continental Europe, but here the virus is widely disseminated in infected planting material because many stocks of the older and most popular cultivars are totally infected. They have not been freed from infection because RVCV is not inactivated in raspberry plants grown at 37°C for several weeks or months (Stace-Smith 1960; Chambers 1961; Jordović 1963). Van der Meer (1975), however, obtained plants free of RVCV by rooting excised tips from infected plants that had been kept at 35°C for 4 wk. More recently, Baumann (1982) eradicated RVCV from infected plants by a combination of thermotherapy and meristem tip culture.

Control of the aphid vector by the use of insecticides may decrease the rate of reinfection of healthy planting material, but there are no reports of the benefits of such treatment. Although red raspberry cultivars differ in susceptibility to *A. idaei*, there are no good sources of resistance to this aphid.



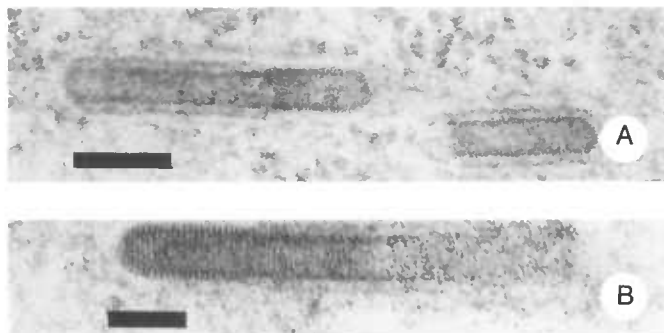


Figure 233. — Particles of raspberry vein chlorosis virus in thin sections of infected raspberry cells. Bars represent 100 nm: A, Particles rounded at one or both ends; B, cross-banding of the nucleocapsid. (Jones et al. 1977.)

The alternative approach, to breed plants with resistance to RVCV itself, is being made in Scotland. Crosses between the cvs. 'Viking' (believed to be resistant or immune to RVCV) and 'Malling Delight' (susceptible) indicate that resistance in cv. 'Viking' is due to a single recessive gene (Jones and Jennings 1980a). However, genetic control of RVCV resistance in other cultivars may be complex (A. T. Jones and D. L. Jennings, unpublished data). Nevertheless, researchers using this source of RVCV resistance, and possibly that in other *Rubus* species, may be able to incorporate resistance to RVCV into future raspberry cultivars.

#### Remarks

Viruslike particles and ultrastructural effects resembling those of RVCV were first found by Putz and Meignoz (1972) in raspberry showing symptoms of veinbanding mosaic disease. Such particles were not found in veinbanding mosaic-diseased raspberry examined by Jones et al. (1974). Furthermore, it is now known that of the two viruses believed to be responsible for raspberry mosaic disease, black raspberry necrosis virus has isometric particles about 25 nm in diameter (see "Black Raspberry Necrosis," p. 178) and rubus yellow net virus has small bacilliform particles about 80 to 150 x 25 to 31 nm (see "Rubus Yellow Net," p. 175). It therefore seems very probable that the plants examined by Putz and Meignoz (1972) were also infected with RVCV.

## Leafhopper-Borne Diseases

### Rubus Stunt

By F. A. van der Meer

#### Additional Common Names

Witches'-broom; Heksenbezen, dwergziekte; Rubus Stauche; Verzweigungskrankheit.

#### History and Geographic Distribution

Although his description of the disease is very short and undetailed, a small epidemic of Rubus stunt was very probably mentioned first by de Vries (1896). In a very old raspberry area in The Netherlands, Rietsema (1954) observed the disease as early as 1920. Wormald and Harris (1932) reported an epidemic of the disease in cultivated blackberries in Great Britain, and Prentice (1950) mentioned severe outbreaks in raspberry X blackberry hybrids (that is, 'Phenomenalberry' and 'Logan') in the same country. As cited by Ryschkow (1946), Rubus stunt was described by Vertogradova (1938) as a serious and widespread disease of raspberry in the U.S.S.R. After the First World War, the disease became disastrous in the old raspberry area in the southern parts of The Netherlands (De Fluiter and Thung 1951), and many young plantations, started with healthy planting material, were found to contain 60 to 90% infected plants in the second year after planting (van der Meer 1954). Small epidemics of the disease have been seen and reported in Bulgaria (Trifonov 1961), East Germany (Richter 1963), and Denmark (Kristensen 1962). Incidental occurrences of Rubus stunt have been reported from several other European countries such as West Germany, Hungary, Czechoslovakia, Norway (Ramsfjell 1952), and Italy (Marani et al. 1977). Recently, a disease similar to Rubus stunt has been found in black raspberries (*Rubus occidentalis* L.) in the western part of the United States (Converse et al. 1982); however, further host range studies must be done to determine if this disease is identical with Rubus stunt.

#### Economic Importance

Rubus stunt is of great potential economic importance, because crop losses can be very severe in places where the disease becomes epidemic.

#### Symptoms on Natural and Experimental Hosts

Natural infection has been found in all principal European cultivars of raspberries and in many species of wild blackberries. In all species and cultivars of *Rubus*, symptoms are basically the same, that is, numerous, small, thin, and erect canes (figs. 234 to 236) and an excessive lateral branching of the whole plant, together with phyllody and proliferations of the flowers (figs. 237 to 240). Except for cv. 'Malling Promise', which is rather tolerant and seldom shows flower malformations, all tested raspberry cultivars are equally





Figure 234. — First symptoms of Rubus stunt in red raspberry. Numerous weak and erect shoots develop from the root buds.



Figure 235. — Floricane of Rubus stunt-infected blackberry cv. 'Thornless Evergreen', showing witches'-broom growth and yellowing.

sensitive. In cultivated raspberry plantations, many diseased plants in the shock stage of infection die because they are overgrown by healthy ones. However, raspberries grown from root cuttings of infected plants and planted at a proper distance from each other seldom die and, on the contrary, may show a certain degree of regeneration. Among the great number of shoots formed, some become larger than the others and bear normal but small berries which are difficult to harvest. Fruiting laterals of such regenerated plants are always much shorter than those of healthy plants.

Plants that are already badly affected by raspberry mosaic and raspberry leaf mottle are much more sensitive to Rubus stunt, and often die within a year after infection. (See "Raspberry Mosaic," p. 168, and "Raspberry Leaf Mottle and Raspberry Leaf Spot," p. 183.)

Experimentally infected *Fragaria vesca* and strawberry cultivars show witches'-broom, phyllody of flowers, and a severe growth reduction (figs. 241 and 242). Infected plants always die within 1 or 2 yr.



Figure 236. — Rubus stunt in naturally infected wild blackberry.



Figure 237. — Various forms and stages of flower and fruit deformation in red raspberry cv. 'Radboud' affected by Rubus stunt. Normal flowers and fruits are in upper right row.



Figure 238. — Phyllody of flowers of red raspberry cv. 'Norfolk Giant'. Sepals, petals, and pistils become leaflike structures. Stamens usually remain normal.

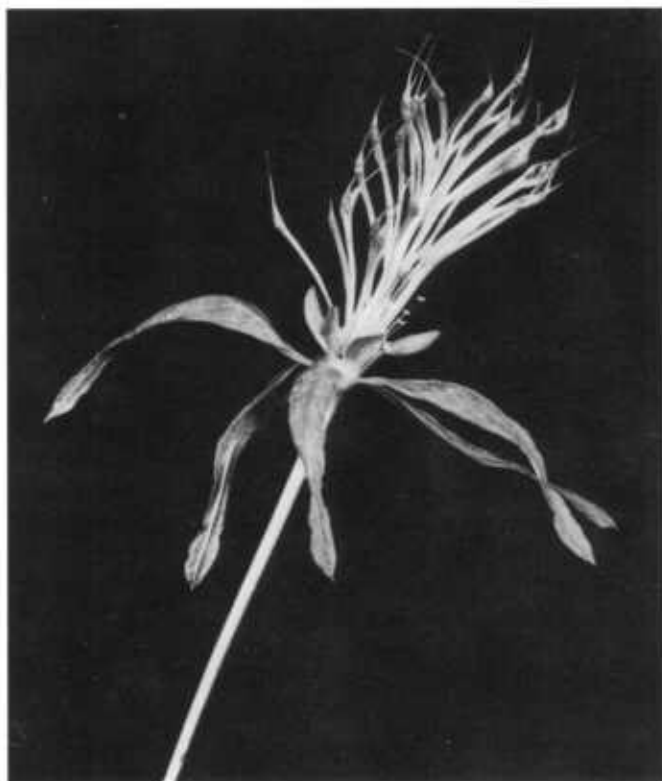


Figure 239. — Phyllody in Rubus stunt-infected wild blackberry.



Figure 240. — Proliferated flower of Rubus stunt-infected wild blackberry.

### Natural and Experimental Transmission

From results of transmission experiments and field observations, de Fluiter and van der Meer (1953) assumed that Rubus stunt is naturally transmitted, mainly by leafhoppers of the genus *Macropsis* Lewis. The species *M. fuscula* Zett. was the only common and abundantly present leafhopper in raspberry plantations during the last epidemic in The Netherlands.

In transmission experiments, *M. fuscula* was able to transmit Rubus stunt from raspberry to strawberry (van der Meer and de Fluiter 1970). The froghopper *Philaenus spumarius* L. and the leafhopper *Allygus mayri* Kbm. transmitted the Rubus stunt agent to celery (Jenser et al. 1981), whereas the leafhopper *Euscelis plebeja* Fallén transmitted the agent to white clover and *Chrysanthemum carinatum* (Lehman 1973).

Experimentally, Rubus stunt can be transmitted between *Rubus* species by graft inoculation.

Depending on the time of the growing season in which the plants are infected, the incubation time varies from 4 to 11 mo (van der Meer 1954).

**Biology of *Macropsis* species.** The eggs of *M. fuscula* overwinter in the bark of *Rubus* canes. When large numbers are present in artificial cultures, the eggs are easily detectable, but they are very difficult to find in naturally affected canes (fig. 243). Therefore, it is difficult to prevent distribution of the vector by means of planting material.



Figure 241. — Strawberry cv. 'Climax' graft-infected by Rubus stunt. Healthy plant at left.



Figure 242. — Flower of *Fragaria vesca* affected by Rubus stunt after graft inoculation.

*M. fuscula* has been reported from several European countries and from parts of western Canada (Beirne 1954; Tonks 1960) and the United States (Converse et al. 1982). On cultivated raspberries in The Netherlands, it was found in the heavily infested area but not in other raspberry-growing areas that were free of Rubus stunt. On wild blackberries, however, the insect occurred all over the country, and Rubus stunt was found in these vines in many places.

These observations have led to the opinion that infected wild blackberries do not play an important role in the severe outbreak of Rubus stunt in raspberries in The Netherlands. In laboratory experiments, however, *M. fuscula* from several wild blackberry species developed well on raspberries.

Another *Macropsis* species was found on *R. caesius* L., a trailing wild blackberry that is very common on wet soils. This species has been described by Wagner (1964) as *M.*





Figure 243. — *Macropsis fuscula*, vector of Rubus stunt: Left, adult male; right, adult female.

*brabantica*. It was not found on other blackberry species, and in laboratory experiments it was not able to develop on raspberries. *M. brabantica* probably is also able to transmit Rubus stunt because severe outbreaks of the disease in *R. caesius* have been observed in several places.

A third *Macropsis* species, *M. scotti* Edw., has been found very commonly on cultivated Himalaya blackberries (*R. procerus* P. J. Muell.) in the province of Zeeland in the southwestern part of The Netherlands. In this area, leafhoppers of the genus *Macropsis* were never found on raspberry, even where they were planted next to heavily infested 'Himalaya' blackberries, and the inability of *M. scotti* to develop on raspberries has been confirmed in laboratory experiments. According to J. T. Legg, as cited by Cadman (1961b), *M. scotti* is also able to transmit Rubus stunt. In the Netherlands, however, the disease has never been observed in 'Himalaya' blackberries whereas in England this cultivar was found infected commonly (Wormald and Harris 1932).

All three *Macropsis* species known from *Rubus* have one generation a year, and there are few morphological differences between species (Wagner 1964). All live monophagously on *Rubus* species and hibernate in the egg stage in the bark of *Rubus* canes. In The Netherlands, the first larvae appear in the middle of May, whereas the first adults appear at the end of June (de Fluiter and van der Meer 1958). Adults may be observed until the first week of October.

To establish if *Macropsis* species do occur in *Rubus* plantations, a sample of canes should be forced in a heated greenhouse at the end of April. Larvae, hatching after about 3 wk, can more readily be found on such canes than on canes in the field because of the small size and the often dark brown color of the larvae, and their tendency to hide in dark and moist places along the basal part of the canes. Larvae are immobile and can easily be handled with a small paint brush. Adults, on the contrary, are mobile and can easily be missed in plantations, even when sampling is done with nets, especially if populations are low and plantings exist of heavily thorned cultivars.

**Natural spread in raspberry.** During the last epidemic in The Netherlands, infection in raspberry plantations mainly took place in August and September of the first growing season (de Fluiter and van der Meer 1955). The first symptoms, numerous weak shoots developing from the root buds, showed up in August and September of the following year (fig. 234). In the third summer after planting, typical 'witches'-broom symptoms appeared: bunches of 5 to 10 fruiting laterals arising from single buds and showing abnormal flowers together with the development of numerous weak and thin young canes at the base of the plants (figs. 236 and 237). Symptoms never developed in the first growing season, provided that healthy planting material was used (van der Meer 1954).

During surveys in 1953 and 1954, plants in older plantations were found to be much more resistant than those in young plantations. On a number of farms, where 8- to 10-yr-old plantings were situated next to 2-yr-old plantings, the older plantings contained about 10% infected plants, whereas the young plantings contained 50 to 80% infected plants. A possible explanation for this phenomenon could be that newly planted raspberries start their growth later than do older plants and thus are physiologically younger in August and September, at which time the leafhoppers are able to transmit the Rubus stunt agent. Young shoots in such plantings keep their leaves until November, but leaves of shoots in older plantings begin to drop in July. Observations actually showed that the population on older healthy plants decreased very rapidly after the end of July, whereas on maiden plants, which were free of leafhoppers when planted, *M. fuscula* could be found until October. Diseased plants in older plantations are good host plants for the leafhoppers, probably because they keep their leaves until very late in autumn; however, they seem to be of minor importance for further spread within such old plantations.

Although *M. fuscula* is not able to develop on strawberry, it appeared able to transmit Rubus stunt from raspberry to strawberry in laboratory experiments. Infection of Rubus stunt in strawberries, however, has never been observed in the field, although in the heavily infested area strawberries and raspberries usually were grown side by side on the same farms.



**Natural spread between different *Rubus* species.** There is little exact information on the influence of infected wild blackberry species during epidemics in cultivated raspberries and blackberries; neither is there any such knowledge about the ease with which *Rubus* stunt is transmitted naturally between different cultivated species and cultivars of *Rubus*. During the last epidemic in The Netherlands, infected wild blackberries appeared of little importance in the infection of cultivated raspberries (van der Meer and de Fluiter 1962). There are reports from two areas where cv. 'Thornless Evergreen' (*R. laciniatus* Willd.) became infected, while cv. 'Himalaya' on the same farms remained healthy (Schambach 1972; Dijkstra 1973). The latter cultivar, however, was found to be susceptible during an epidemic in England (Prentice 1950).

Converse et al. (1982) found *Rubus* stuntlike symptoms common in 'Munger' black raspberries (*R. occidentalis* L.), but not at all in adjoining plantings of 'Willamette' red raspberries. Transmission of *Rubus* stunt between different cultivated *Rubus* species and between wild and cultivated *Rubus* species may be influenced by the host plant preferences of the *Macropsis* species occurring in certain areas and by the physiological condition of the hosts, which induce the leafhoppers to migrate on a large scale or may determine whether plants visited by migrating leafhoppers become infected.

It seems unlikely that the other reported vectors, *P. spumarius*, *A. mayri*, and *E. plebeja*, play an important role in transmission between *Rubus* species because they do not live specifically on *Rubus*. By chance feeding, however, they may be able to transmit the disease agent occasionally between *Rubus* and other plant species and vice versa. The very polyphagous character of *P. spumarius* marks this species as a likely vector between different plant species and families; however, froghoppers do not seem to be common vectors of witches'-broom diseases. By our best knowledge, Jenser et al. (1981) were the first to report such transmission.

### Properties of the Causal Agent

Like many witches'-broom or "yellows" diseases of plants, *Rubus* stunt was formerly attributed to a virus; however, since Doi et al. (1967) discovered mycoplasma-like organisms (MLO) in several witches'-broom diseased plant species, many other workers have found such diseases to be associated with MLO (Nienhaus and Sikora 1979; Davis and Whitcomb 1971). MLO have also been found in stunt-infected *Rubus* species, that is, in red raspberries (Murant and Roberts 1971; Muller et al. 1977), blackberries (Marani et al. 1977; van der Meer 1980; Klein et al. 1976), and black raspberries (Converse et al. 1982). Presently, it is assumed that MLO are the direct cause of *Rubus* stunt and other witches'-broom diseases of plants, although Koch's postulates are fulfilled in only a few cases (Bos 1981).

There is little information on the relationship between MLO that cause diseases in different crops. Results of Lehman (1973) and Jenser et al. (1981) show that the infective agent(s) causing *Rubus* stunt can be experimentally inoculated into non-*Rubus* test plants. In these inoculated test plants, symptoms develop that show much similarity to other MLO diseases, such as aster yellows, strawberry green petal, and clover phyllody. (See discussion, on leafhopper-borne diseases in the strawberry section of this handbook, p. 31–46.) Information is lacking, however, on the serological relationships of these MLO diseases.

Like many other MLO diseases (Whitcomb and Davis 1970), *Rubus* stunt is persistently transmitted by its vectors, which means that there is a latent period between the moment of acquisition and the moment at which the vector is able to transmit the disease agent. After this latent period, vectors usually remain infective throughout their lives.

Larvae of *M. fuscula*, born on diseased plants in the second half of May and raised on diseased plants until they became adults in the second half of July, were only able to transmit *Rubus* stunt after the fifth of August (de Fluiter and van der Meer 1958). Young larvae, from 5 to 7 days old, collected from diseased plants and subsequently raised on healthy plants, did not transmit *Rubus* stunt after they became adults, but third- and fourth-instar larvae, sampled from diseased plants in June, transmitted the disease in August, after becoming adults in the middle of July. From this, it can be estimated that the latent period of *Rubus* stunt in *M. fuscula* is about 8 wk. Once infected, *M. fuscula* remains infective until it dies. Transmission of *Rubus* stunt through the eggs has not been detected.

According to Jenser et al. (1981), the latent period of *Rubus* stunt MLO in *P. spumarius* and *A. mayri* is between 28 and 35 days, and both species remain infective during the rest of their lives.

### Detection and Identification

*Rubus* stunt is detectable by its symptoms: the development of numerous thin erect shoots together with abundant lateral branching. Phyllody of flowers is the most typical symptom. Some raspberry and blackberry cultivars may regenerate to a high degree. Such regenerated plants generally do not show flower deformation and for that reason should be indexed by grafting to sensitive raspberry cultivars, such as cvs 'Radboud' and 'Malling Landmark' or to the sensitive blackberry cv. 'Thornless Evergreen'. Attempts should be made to detect MLO in diseased plant by electron microscopy.

### Control Procedures

According to de Fluiter and van der Meer (1958), the eggs of *M. fuscula* can easily be killed by a tar oil spray in winter. According to Reitzel (1971), however, such treatment killed only half the population of the insect. Nymphs can be

controlled by spraying with parathion or other insecticides in spring. A campaign, organized by The Netherlands' Advisory Board, during which every grower was urged to spray his raspberry plantation with tar oil in winter and with parathion in spring (Slits 1954, 1955; van der Meer 1957), resulted in a good control of the disease in the heavily infested area (van der Meer and de Fluiter 1962).

Thanks to the long latent period of the disease agent in the vector, raspberries can be protected against infection by spray applications after harvest in August and September, the period in which infection takes place. Because the time of harvest is much later, such applications are usually not possible in blackberry plantations.

Blackberries must not be sprayed with tar oil because they are softer and less woody than raspberries in winter and will be damaged by this spray.

Infected plants can easily be cured by hot water treatment of dormant root cuttings or rooted shoots (Thung 1952). Treatment for 2 or 3 hr in water at 45°C appeared sufficient to inactivate the *Rubus* stunt agent.

## Remarks

To the author's knowledge, *Rubus* stunt is the only known disease of economic consequence in the genus *Rubus* that has been proved to be caused by a leafhopper-borne agent. Pierce's disease, however, probably caused by leafhopper-borne rickettsialike organisms (Mollenhauer and Hopkins 1974), appears to occur latently in *Rubus vitifolius* Cham. and Schlecht (Freitag 1951). Nichols et al. (1957) reported shoot proliferation of 'Olallie' blackberry in California. The symptoms resemble those of some leafhopper-borne diseases, but further studies have failed to associate a transmissible agent with this disorder. Witches'-broom symptoms in black raspberry have been observed in the United States by Zundel (1931) in Pennsylvania, by R. H. Converse (personal communication) in Michigan in 1963, and, again by Converse in 1980 in Oregon. With respect to the first two observations in black raspberries, no further research was done. During their last observations in Oregon, Converse et al. (1982) noticed a rapid spread of the disease in a plot of 'Munger' black raspberries, and MLO appeared to be common in sieve tubes of infected plants. Although further research is needed to confirm this, it seems likely that witches'-broom of black raspberry is a leafhopper-borne disease related to or identical with *Rubus* stunt.

### Raspberry Yellow Dwarf and Associated Diseases of *Rubus* Caused by Arabis Mosaic and Strawberry Latent Ringspot Viruses

By A. F. Murrant

#### Additional Common Names

None.

#### History and Geographic Distribution

The name "raspberry yellow dwarf" was given by Harrison (1958c) to a disease of 'Malling Exploit' red raspberry (*Rubus idaeus* L.) in Great Britain. This disease was caused by a sap-transmissible soil-borne virus. Cadman (1960b) showed that the virus was closely related to arabis mosaic virus (AMV) described by Smith and Markham (1944). AMV has since been reported throughout Great Britain in several other red raspberry cultivars (Taylor et al. 1966; Dale and Brown 1973; Cotton et al. 1978).

Another sap-transmissible soil-borne virus, strawberry latent ringspot virus (SLRV) (Lister 1964), causes a similar stunting disease in some raspberry cultivars (Taylor and Thomas 1968; Putz and Stocky 1970) and often occurs in mixed infections with AMV (Lister 1964; Dale and Brown 1973). Both viruses are transmitted by nematodes of the genus *Xiphinema*, principally *X. diversicaudatum* Micoletzky (Harrison and Cadman 1959; Jha and Posnette 1959; Lister 1964), and therefore commonly occur together in the same soils.

Both AMV and SLRV have wide natural host ranges and occur locally throughout the British Isles and continental Europe. In addition, AMV is reported from the U.S.S.R., including the Soviet Far East (Gordejchuk et al. 1977), Japan (Iwaki and Komuro 1974), New Zealand (Thomas and Procter 1972, 1977), and the United States (Waterworth 1975); SLRV is reported from New Zealand (Fry and Wood 1973), Canada (Allen et al. 1970), and the United States (Hanson and Campbell 1979). *X. diversicaudatum* occurs in Europe, U.S.S.R., Canada, the United States, Australia, and New Zealand (Pitcher et al. 1974). However, the nematode was not associated with any of the reported occurrences of these viruses in North America, all of which resulted from the importation of infected plant material.

In raspberry, both viruses have been reported from several countries outside the British Isles: AMV in France (Putz and Stocky 1971), East Germany (Richter 1964c), and the Soviet Far East (Gordejchuk et al. 1977); SLRV in France (Putz and Stocky 1970) and Italy (Vegetti et al. 1979).

#### Economic Importance

Diseases of raspberry caused by AMV and/or SLRV are locally important in England but rare in Scotland, where most of the British raspberry crop is grown; they also seem rare in continental Europe. If the outbreak is large, crop losses may be considerable because infected plants yield little or no fruit and may die. Because few or no symptoms appear in the early stages of infection, the viruses may be distributed in infected propagating materials; this can result not only in losses of crop but also in the viruses becoming established in the soil if vector nematodes are already present. It is also a potential problem in the international exchange of planting material.

#### Symptoms on Natural Hosts

Both AMV and SLRV occur naturally in red raspberry (*Rubus idaeus* L.). AMV has also been found in blackberry (*Rubus procerus* P. J. Muell.) cv. 'Himalaya Giant' (Harrison 1958c). Both viruses also infect plants in the small fruit genera *Fragaria* and *Ribes*. (See these sections of this handbook.)

Many other cultivated and wild plants have been found naturally infected with both viruses. The following is a selection of plants found infected with AMV; those in which SLRV has also been found are marked with an asterisk.

#### Cultivated Plants

*Apium graveolens* L. var. *dulce* (celery)\*, *Armoracia rusticana* Gaertn., Mey., and Scherb. (horse-radish), *Asparagus officinalis* L. (asparagus)\*, *Cucumis sativus* L. (cucumber), *Cucurbita pepo* L. (marrow), *Cyphomandra betacea* Sendt. (tamarillo, tree-tomato), *Daucus carota* L. (carrot), *Delphinium* sp.\*, *Dianthus caryophyllus* L. (carnation), *Euonymus europaea* L. (spindle tree)\*, *Forsythia* sp., *Fraxinus excelsior* L. (ash), *Humulus lupulus* L. (hop), *Jasminum officinale* L., *Lactuca sativa* L. (lettuce), *Ligustrum vulgare* L. (privet), *Melilotus officinalis* Lam. (sweet clover), *Narcissus* spp.\*, *Phaseolus multiflorus* Willd. (scarlet runner bean), *Prunus avium* L. (sweet cherry)\*, *P. domestica* L. (plum)\*, *P. persica* Batsch (peach)\*, *Rheum rhaponticum* L. (rhubarb)\*, *Rosa* spp. (rose)\*, *Syringa vulgaris* L. (lilac), *Trifolium repens* L. (white clover)\*, *Tulipa* spp. (tulip), *Vitis vinifera* L. (grapevine)\*.

#### Wild Plants

*Anagallis arvensis* L., *Arabis hirsuta* (L.) Scop., *Bellis perennis* L., *Capsella bursa-pastoris* (L.) Medic.\*, *Lamium amplexicaule* L.\*, *Mentha arvensis* L.\*, *Plantago lanceolata* L., *Polygonum aviculare* L., *P. persicaria* L., *Ranunculus repens* L., *Sambucus nigra* L.\*, *Senecio vulgaris* L.\*, *Stellaria media* (L.) Vill.\*, *Taraxacum officinale* Weber\*, *Urtica dioica* L.\*, *U. urens* L.

**Table 11.—Susceptibility of red raspberry and blackberry cultivars to British isolates of raspberry ringspot virus, tomato black ring virus, arabis mosaic virus, and strawberry latent ringspot virus<sup>1</sup>**

Cultivar	RRV (common Scottish strain)	RRV (yellow blotch strain)	TBRV (common Scottish strain)	AMV (common strain)	SLRV (common strain)
<b>Red raspberry:</b>					
Baumforth's Seedling B	+	•	•	•	•
Burnetholm	—	+	+	—	•
Cuthbert	—	+	•	•	•
Glen Clova	+	•	+	+	—
Glen Isla	+	+	+	+	+
Lloyd George	—	+	—	—(+)	—
Malling Admiral	+	—	—	+	+
Malling Delight	+	+	+	+	—
Malling Enterprise	+	+	—	—	+
Malling Exploit	+	+	+	+	+
Malling Jewel	+(E)	+	—	—(+)	+
Malling Landmark	—	+	—	•	—
Malling Leo	—	—	+	—	—
Malling Notable	+	•	—	—	•
Malling Orion	+	+	—	—	+
Malling Promise	+(E)	+	—	+	+
Norfolk Giant	+	+	+	—(+)	—
Preussen	+	•	•	•	•
St. Walfried	+	+	•	•	•
Seedling M	—	+	—	•	•
Seedling V	—	+	+	•	•
<b>Blackberry:</b>					
Himalaya Giant	•(E)	•	•	•	+
Tayberry	+	—	—	•	—

<sup>1</sup>Data are based on graft-transmission tests supplemented by data on field infection.

Note: + = susceptible; (+) = susceptible to some strains; (E) found infected with isolates of the English serotype; — = considered immune; • = not tested.

**Symptoms on red raspberry.** Table 11 lists the susceptibility of red raspberry cultivars to British isolates of AMV and SLRV. There is evidence that AMV strains differ in ability to infect some raspberry cultivars. Thus, 'Lloyd George' and 'Norfolk Giant' seem immune to common isolates of AMV, but AMV-infected plants of these cultivars were found in Scotland by Taylor et al. (1966). Subsequent graft-transmission tests by J. Chambers and A. F. Murrant (unpublished data) showed that this AMV isolate infected not only 'Lloyd George' and 'Norfolk Giant' but also 'Malling Jewel', a cultivar that has consistently failed to become infected with AMV at other sites (Taylor and Thomas 1968; Dale and Brown 1973), although becoming infected with SLRV. By contrast, the cv. 'Glen Clova' is susceptible to AMV but does not become infected with SLRV.

As with other nematode-borne virus diseases, outbreaks of AMV and SLRV occur in patches (fig. 244), reflecting the horizontal distribution of vector nematodes in the soil. However, if the disease results from the planting of infected stocks, the infected plants are of course distributed randomly throughout the crop. AMV-infected 'Malling Exploit' plants show symptoms 2 to 3 yr after planting; the young canes are stunted, and produce little or no fruit, and yellow speckling appears on the leaves (fig. 245A); conspicuous vein-yellowing or yellow net symptoms (fig. 245B) may appear on the lower leaves. AMV induces similar symptoms in the cultivars 'Malling Promise' and 'Malling Admiral', but causes leaf mottling in infected plants of 'Glen Clova' (fig. 246).





Figure 244. — An outbreak of strawberry latent ringspot virus in 'Malling Jewel' raspberry in Scotland. (After Taylor and Thomas 1968; copyright Scottish Crop Research Institute.)

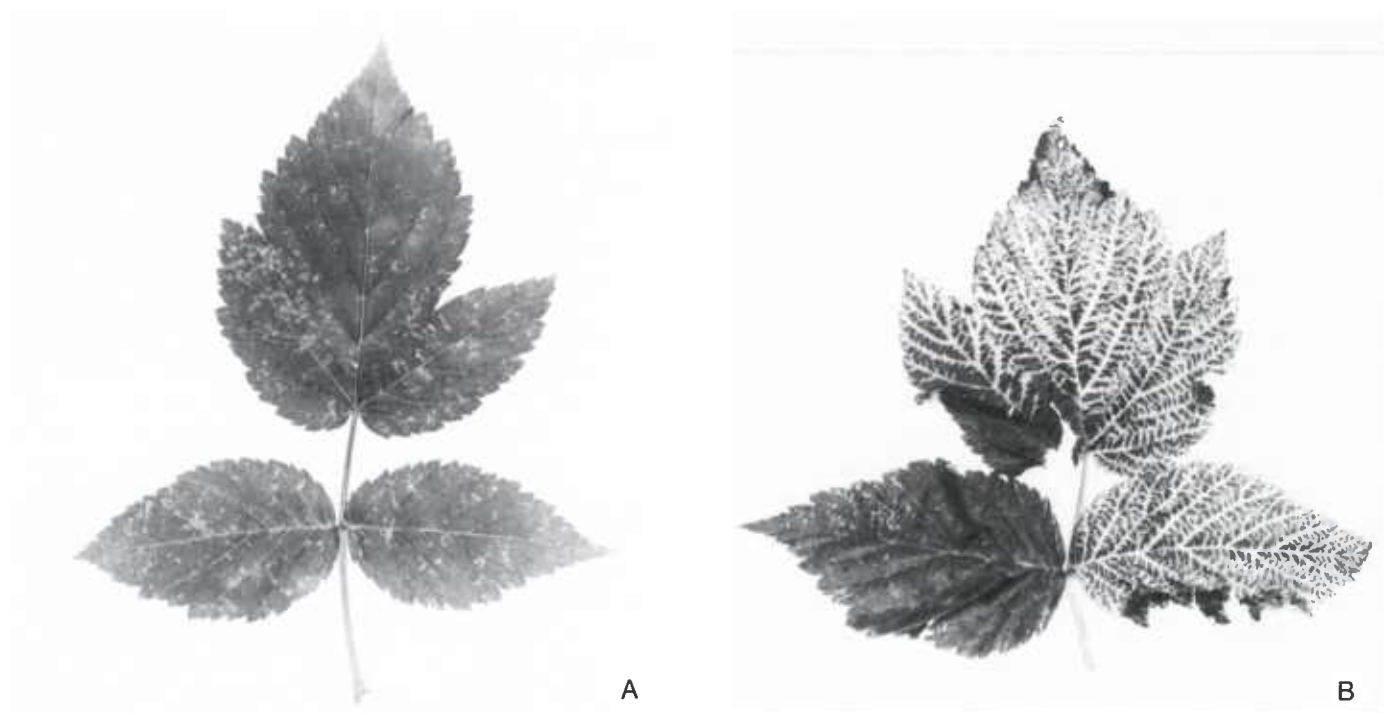


Figure 245. — Leaves of arabis mosaic virus-infected 'Malling Exploit' raspberry showing: A, yellow speck-

ling; B, vein-yellowing. (After Harrison 1958; copyright Scottish Crop Research Institute.)



Figure 246. — Leaf of arabis mosaic virus-infected 'Glen Clova' raspberry, showing mottle symptoms. (After Murrant 1981b; copyright Scottish Crop Research Institute.)

It is not known what symptoms SLRV induces in most red raspberry cultivars because it is usually found together with AMV. Lister (1964) reported that the few 'Malling Exploit' plants found infected with SLRV alone were symptomless, and plants containing both viruses looked similar to plants infected with AMV alone. However, Taylor and Thomas (1968) found that 'Malling Jewel' raspberry infected with SLRV alone were severely stunted (fig. 244) and showed foliar symptoms closely resembling those induced by AMV in 'Malling Exploit', except that vein-yellowing was not observed. Lateral shoots on the fruiting canes were poorly developed or dead, and the leaves were down-curved and yellow-blotched; leaves on primocanes showed yellow speckling (fig. 247).

**Symptoms on blackberry.** Leaves of 'Himalaya Giant' blackberry infected with AMV showed yellow mosaic (Harrison 1958c).

**Symptoms on other cultivated plants.** For the symptoms induced by AMV and SLRV in *Fragaria* and *Ribes*, see those sections of this handbook, p. 46, 131, 139 and 148.

In association with viruses of the prunus necrotic ringspot type, AMV induces rasp-leaf symptoms in cherry (Cropley 1961; East Malling Research Station 1963). In mixed infections with prune dwarf virus, SLRV induces a severe decline disease of peach (Scotto la Massese et al. 1973). Symptoms induced by AMV in some other crops are



Figure 247. — Leaf of strawberry latent ringspot virus-infected 'Malling Jewel' raspberry, showing yellow speckling. (After Taylor and Thomas 1968; copyright Scottish Crop Research Institute.)

described by the following authors: scarlet runner bean, celery, white clover, and marrow (Harrison and Winslow 1961); cherry (Cropley 1961); cucumber (Hollings 1963); hop (Bock 1966); lettuce (Walkey 1967a). Symptoms induced by SLRV were described by the following: celery (Walkey and Mitchell 1969); rose (Harrison 1967; Ikin and Frost 1976).

### Symptoms on Experimental Hosts

Both AMV and SLRV have wide experimental host ranges and infect nearly all commonly used herbaceous test plants. In detailed investigations of host range, AMV infected 93 species in 28 families (Schmelzer 1962) and SLRV infected 126 species in 27 families, most of them symptomlessly (Schmelzer 1969).

The following are some useful diagnostic hosts:

*Chenopodium amaranticolor* Coste and Reyn., *C. murale* L., and *C. quinoa* Willd.

AMV and SLRV: Chlorotic or necrotic local lesions (figs. 248 and 249); systemic chlorotic mottle (fig. 250) or necrosis.

*Cucumis sativus* L.

AMV: Chlorotic local lesions; systemic yellow spots or vein-banding, subsequently fading. The plants then stop growing.

SLRV: Chlorotic local lesions or none, systemic interveinal chlorosis or necrosis (fig. 251). In summer, subsequent leaves are symptomless but contain virus; in winter, symptoms may persist.





Figure 248. — Local lesions induced by arabis mosaic virus in *Chenopodium amaranticolor*. (After Harrison 1958; copyright Scottish Crop Research Institute.)



Figure 250. — Systemic symptoms induced by arabis mosaic virus in *Chenopodium amaranticolor*. (After Murant 1970; copyright Scottish Crop Research Institute.)

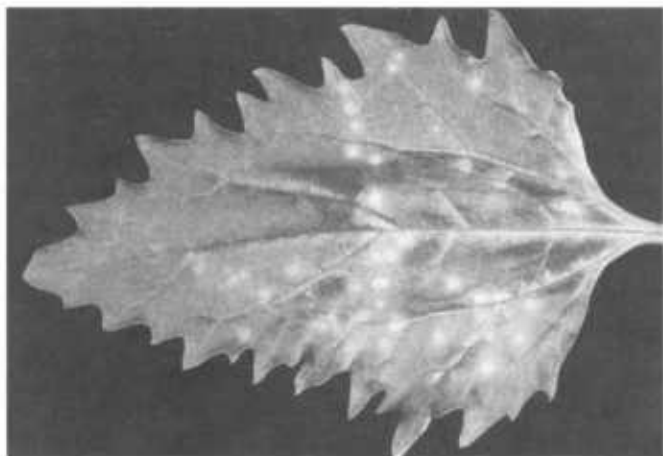


Figure 249. — Local lesions induced by strawberry latent ringspot virus in *Chenopodium murale*. (After Murant 1970; copyright Scottish Crop Research Institute.)

*Nicotiana tabacum* L. cv. 'White Burley'

AMV: Chlorotic or necrotic local lesions. Some isolates give systemic yellow spots and rings (fig. 252) and line patterns. Leaves produced later appear almost normal but contain virus.

SLRV: Symptomless systemic infection.

*Petunia hybrida* Vilm.

AMV: Local chlorotic lesions or necrotic rings. Systemic vein-clearing or chlorotic rings and line patterns. Leaves produced later are symptomless but contain virus.

SLRV: Symptomless systemic infection.



Figure 251. — Systemic interveinal chlorosis induced by strawberry latent ringspot virus in *Cucumis sativus*. (After Murant 1974; copyright Scottish Crop Research Institute.)



Figure 252. — Yellow spots and rings in a systemically infected leaf of *Nicotiana tabacum* cv. 'White Burley'. (After Harrison 1958; copyright Scottish Crop Research Institute.)

Isolates of both viruses differ in virulence, and symptoms in most hosts are milder in summer than in winter.

*Petunia hybrida* and *Nicotiana clevelandii* Gray are good hosts in which to propagate AMV. Local lesions on *Chenopodium amaranticolor* can be used to assay virulent isolates of AMV, but local lesions caused by mild isolates are too indistinct to count. For SLRV, *Cucumis sativus* is the best host for propagation and *Chenopodium murale* the most reliable local lesion host. *Cucumis sativus* and *Petunia hybrida* are useful "bait" plants for use in nematode transmission experiments with both viruses. These plants rarely become systemically infected or show symptoms following inoculation of the roots by nematodes, but the viruses may be detected in the roots or hypocotyls by inoculation of sap to *Chenopodium quinoa*.

#### Natural and Experimental Transmission

Both viruses are transmissible by grafting, by inoculation with sap, and through seed. Their natural vectors are nematodes in the genus *Xiphinema*.

Transmission of AMV and SLRV to herbaceous plants by inoculation with sap from rosaceous plants is greatly facilitated if inocula are prepared at pH 8 with 2% nicotine or 1% polyethylene glycol (mol. wt. 6000), or if powdered alumina is added to the inoculum; these additives prevent tannins from inhibiting infection.

Transmission through seed has been shown for AMV in at least 16 species in 12 plant families (Lister 1960; Lister and Murant 1967; Walkey 1967b) and for SLRV in at least 6 species (Taylor and Thomas 1968; Murant and Goold 1969; Schmelzer 1969; Allen et al. 1970; Walkey and Whittingham-Jones 1970). In many host species, more than 10%, and in some nearly 100%, of progeny seedlings are infected, commonly without showing symptoms.

The main vector of both viruses is the nematode *Xiphinema diversicaudatum* (Harrison and Cadman 1959; Jha and Posnette 1959; Lister 1964). Consequently, they often occur together in soils. In addition, there are unconfirmed reports of transmission of AMV by *X. coxi* (Fritzsche 1964) and *X. bakeri* (Iwaki and Komuro 1974) and of SLRV by *X. coxi* (Putz and Stocky 1970). Both larvae and adults of *X. diversicaudatum* transmit both viruses (Harrison and Cadman 1959; Harrison 1967; Taylor and Thomas 1968; Putz and Stocky 1970), but moulted nematodes do not retain AMV (Harrison and Winslow 1961) or, probably, SLRV. Jha and Posnette (1961) found that *X. diversicaudatum* acquired ability to transmit AMV after access to infected plants for 1 day, could inoculate bait plants in 3 days, and continued to transmit AMV after 31 days in fallow soil. Harrison and Winslow (1961) found that *X. diversicaudatum* was still able to transmit AMV after 8 mo on the virus-immune raspberry cultivar 'Malling Jewel'. SLRV and AMV were retained for up to 84 and 112 days, respectively, in *X. diversicaudatum* kept in fallow soil (Taylor and Thomas 1968). By analogy with other nematode-transmitted viruses, the viruses can probably be acquired within a few minutes, can be inoculated in a single brief feed, and do not multiply in the vector or circulate within it (Taylor 1980). Particles of both viruses are associated in a monolayer with the cuticle lining of the lumen of the odontophore, esophagus and esophageal bulb (Taylor and Robertson 1970; Taylor 1980).

Little comparative work has been done on the transmissibility of serological forms of AMV by nematodes, but serological variants of SLRV from peach and raspberry in Italy were not transmitted by a Scottish population of *X. diversicaudatum* that transmitted the type strain efficiently; moreover, nematodes of an Italian population of *X. diversicaudatum* were only inefficient vectors of the three SLRV isolates and of the type strain of AMV (Brown and Taylor 1981).

Within an outbreak area, AMV and SLRV are spread by *X. diversicaudatum*, but because the nematodes do not withstand airdrying of soil they do not carry the viruses efficiently over a distance. There is also little lateral migration of the vector, and outbreaks extend only slowly. The viruses are probably disseminated in nature in infected seeds, as are raspberry ringspot and tomato black ring viruses (Lister and Murant 1967; Murant and Lister 1967). (See "Raspberry Ringspot and Associated Diseases of *Rubus*. . .," p. 211.) The presence of infected seeds in soils, however, is less important for the survival of AMV and SLRV through periods of fallow than for raspberry ringspot and tomato black ring viruses, which persist for only 8 to 9 wk in their vector, *Longidorus elongatus*. Soils from outbreaks of AMV and SLRV seem, in fact, to contain relatively few infected weed seeds (Murant and Lister 1967; Taylor and Thomas 1968). The role of wild plants in the ecology of these and other nematode-borne viruses was discussed by Murant (1970b, 1981b).



### Properties of the Causal Agent

AMV is a definitive member of the nepovirus group; SLRV has many similar properties and has long been regarded as a nepovirus but is now considered only a tentative member of the group because of its anomalous protein composition (Harrison and Murrant 1977b; Murrant 1981a).

**AMV.** For a detailed description of the virus, see Murrant (1970a). In *Petunia hybrida* sap, the virus loses infectivity after dilution to  $10^{-3}$  to  $10^{-5}$ , after 10 min at 55° to 61°C, or after 1 to 2 wk at room temperature (Harrison 1958c); some workers report longer survival at room temperature (Hollings 1963; Schmelzer 1962). Infectivity survives for many years at -15°C. The virus may be purified by the butanol/chloroform method (Harrison and Nixon 1960) or by the procedure described on p. 218 for raspberry ringspot virus (Murrant et al. 1972; Murrant 1978). The virus particles are isometric, about 28 nm in diameter, with hexagonal outlines (fig. 253A); some of them are penetrated or partially penetrated by negative stain, others are not penetrated. The particles form three sedimenting components, T, M and B, with sedimentation coefficients ( $s_{20, w}$ ) of 53, 93, and 126 S, respectively (R. Stace-Smith, personal communication). The coat protein is a single species of mol. wt. 54,000 (Mayo et al. 1971), and the genome consists of two species of single-stranded RNA with mol. wt. ( $\times 10^6$ ) of 2.8 and 1.3 (Murrant et al. 1981).

Although serological variability exists in AMV (Bock 1966; Bercks et al. 1976), most isolates are not greatly dissimilar; however, grapevine fanleaf virus is a distantly related serotype.

**SLRV.** For a detailed description of the virus, see Murrant (1974a). In *Chenopodium quinoa* sap, the virus isolates studied by Lister (1964) lost infectivity after dilution to  $10^{-3}$  to  $10^{-5}$  or after 10 min at 52° to 58°C but were still infective after 50 days at room temperature. Some other isolates survived less well in vitro (Tomlinson and Walkey 1967; Richter and Kegler 1967). The virus may be purified by various methods: clarification with butanol/chloroform (Lister 1964), or ether/carbon tetrachloride (Richter and Proll 1970), or clarification with chloroform followed by precipitation with ammonium sulphate (Allen et al. 1970), or use of Mg-activated bentonite (Savino et al. 1979). The virus particles are isometric, about 30 nm in diameter, with hexagonal outlines. Some are penetrated by negative stain and others are not (fig. 253B). Purified preparations often contain three components, bottom (B), middle (M), and top (T). The major component (B), sediments at 126 to 130 S; sometimes components sedimenting at about 58 S (T) and 94 S (M) are also present. There are two coat protein species of mol. wt. 44 000 and 29 000 (Mayo et al. 1974), and the genome consists of two species of single-stranded RNA with mol. wt. ( $\times 10^6$ ) of 2.9 and 1.4 (Mayo et al. 1974; Murrant et al. 1981). Particles of some isolates contain a third RNA species with a molecular weight of about  $0.5 \times 10^6$ , which may be a "satellite" (Mayo et al. 1974).

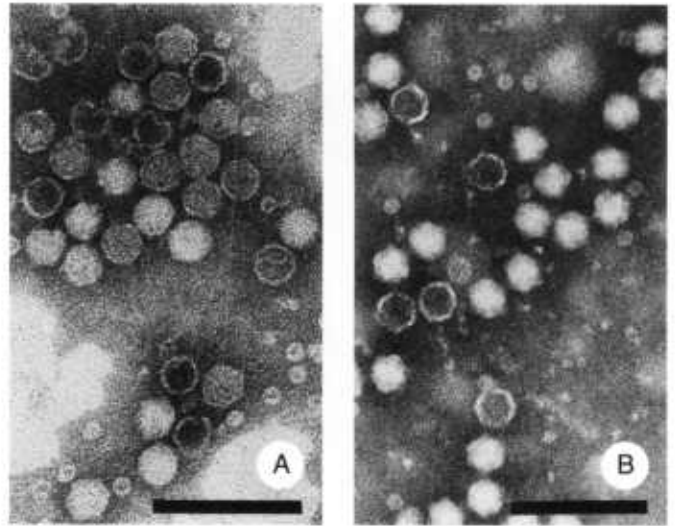


Figure 253. — Virus particles in phosphotungstate, pH 6: A, Particles of arabis mosaic virus; B, particles of strawberry latent ringspot virus. The smaller particles in both pictures are phytoferritin. Bars represent 100 nm. (Copyright Scottish Crop Research Institute.)

All isolates of SLRV studied until recently were serologically very similar, but isolates from olive and peach in Italy (Savino et al. 1979) and an isolate from raspberry, also in Italy (A. F. Murrant, unpublished data), were distinguishable from type strain by spur formation in gel diffusion tests.

### Detection and Identification

The patchy distribution of the disease, stunted plants, and characteristic leaf symptoms enable the presence of a nematode-borne virus to be readily diagnosed. To discover which of several possible viruses is present, the plants must be tested by inoculation of sap to herbaceous indicators. Any viruses transmitted must be identified by serological tests (for example, by gel diffusion tests with sap from infected *Chenopodium quinoa*) because the reactions of test plants are not sufficiently distinctive to enable the viruses to be distinguished from each other or from mild strains of other nepoviruses.

### Control Procedures

**Use of certified virus-tested planting material.** Both viruses may be distributed in infected planting material especially because few or no symptoms are visible in tolerant cultivars or in the early stages of infection in sensitive cultivars. This source of infection can be eliminated by introducing adequate certification schemes for the production of virus-tested stock. Such schemes should require that stocks for certification are not established in soil containing vector nematodes.

**Use of immune cultivars.** If the outbreak in a sensitive cultivar is small, only affected plants need to be removed and replaced with plants of an immune cultivar (see table 11). Immunity to AMV in red raspberry is inherited as a dominant

character, but at least two genes are concerned (Jennings 1964). No studies on the inheritance of immunity to SLRV have been made.

**Chemical control of vector nematodes.** Treatment of soil with the fumigants D-D (dichloropropene-dichloropropane) or methyl bromide prevented *Xiphinema diversicaudatum* from transmitting AMV in strawberry (Harrison et al. 1963; Dale and Hendy 1967). Similar fumigant nematicides, such as dazomet or dichloropropene (the active constituent of D-D), are now used commercially.

**Thermotherapy.** No information is available on the response of AMV or SLRV to thermotherapy in *Rubus*.

### Remarks

The symptoms induced by isolates of AMV and SLRV in herbaceous indicators are similar to those induced by many other *Rubus*-infecting viruses, especially by members of the nepovirus group such as cherry leaf roll, raspberry ringspot, tomato black ring, tomato ringspot, and tobacco ringspot viruses. (See papers in the *Rubus* section devoted to these viruses, p. 211–228.) Moreover, all these viruses have very wide host ranges and naturally infect many other wild and cultivated plants. Serological tests are the only satisfactory method of identification.

## 245 Raspberry Ringspot and Associated Diseases of *Rubus* Caused by Raspberry Ringspot and Tomato Black Ring Viruses//

By A. F. Murrant

### Additional Common Names

Raspberry leaf curl (Harris et al. 1943); raspberry Scottish leaf curl (Review of Applied Mycology 1957); Lloyd George yellow blotch (Cadman and Harris 1952).

### History and Geographic Distribution

The name “leaf curl” was applied by Harris et al. (1943) to a graft-transmissible disease of ‘Norfolk Giant’ red raspberry (*Rubus idaeus* L.) occurring in Scotland. It seemed the same as a disease first noticed in 1922 in ‘Baumforth’s Seedling B’. A sap-transmissible, soil-borne virus named raspberry ringspot virus (RRV) (Cadman 1956; Harrison 1956, 1958a) was found to cause this disease and also to occur in many other red raspberry cultivars showing ringspots, leaf blotches, stunting, and decline symptoms. The “yellow blotch” disease of the cv. ‘Lloyd George’ (Cadman and Harris 1952) is also caused by a strain of RRV (Murrant et al. 1968). Another soil-borne virus, tomato black ring virus (TBRV) (Smith 1946), was found to cause a similar disease in ‘Malling Exploit’ and ‘Seedling V’ (Harrison 1958b) and later in many other cultivars, including ‘Norfolk Giant’. Both viruses proved to be transmitted by nematodes in the genus *Longidorus*. Strains of the viruses occurring in Scotland

share the same vector, *L. elongatus* de Man (Harrison et al. 1961; Taylor 1962) and therefore usually occur together in the same outbreak areas.

In most red raspberry cultivars, RRV and TBRV cause ringspots on the leaves rather than leaf-curl symptoms. Because of this, and also because the disease caused by TBRV is very similar to that caused by RRV in some cultivars and has never been separately named, Cadman (1961b) suggested that “raspberry ringspot” was a better name than “leaf curl” for the disease caused by either or both viruses. Raspberry ringspot is, however, etiologically distinct from American red raspberry ringspot disease, caused by tomato ringspot virus (also nematode-borne) (see “Tomato Ringspot Virus in *Rubus*,” p. 223) and from American raspberry leaf curl disease, caused by an aphid-borne virus (see “Raspberry Leaf Curl,” p. 187).

RRV and TBRV are widespread in eastern Scotland, although of local occurrence, and are also reported in other parts of Great Britain and in continental Europe and U.S.S.R. RRV is also reported in Turkey, and TBRV in Japan. In raspberry, RRV is reported in Great Britain and The Netherlands, is widespread in the U.S.S.R. (Gordejckuk et al. 1977; M. A. Keldysh, personal communication), and has also been found in Hungary (E. Pocsai, personal communication); infection of raspberry with TBRV is reported in Great Britain and occurs rarely in the U.S.S.R. (M. A. Keldysh, personal communication). Vector nematodes (*Longidorus* spp.) occur in all these countries (Hooper 1973; Brown and Boag 1975, 1977); *L. elongatus* is also reported locally in Canada, United States and New Zealand (Hooper 1973), but the viruses are not reported there.

### Economic Importance

RRV causes a lethal disease in some raspberry cultivars, and economic losses may be considerable if the outbreak is large. TBRV is usually less damaging, but both viruses may decrease the yield of “tolerant” cultivars, as happens with TBRV in ‘Malling Exploit’ (Taylor et al. 1965). In Scotland, diseases caused by both viruses are now effectively controlled and are of much less economic importance than formerly.

The viruses cause few or no symptoms in the early stages of infection and may therefore be inadvertently distributed in infected planting material. This can result not only in losses of crop but also in the viruses becoming established in soils already containing vector nematodes. These viruses are also a potential problem in the international exchange of planting material.

### Symptoms on Natural Hosts

Both RRV and TBRV occur naturally in red raspberry (*Rubus idaeus* L.); RRV is also reported from blackberry (*R. procerus* P. J. Muell cv. ‘Himalaya Giant’) (Cadman 1960b)



Figure 254. — An outbreak of raspberry ringspot in a plantation of 'Malling Jewel' raspberry in eastern Scotland. (After Harrison 1958b; copyright Scottish Crop Research Institute.)

and *Rubus sachalinensis* Léveillé (Gordejchuk et al. 1977). Both viruses also infect plants in the small fruit genera *Fragaria* and *Ribes*. (See these sections of this handbook, p. 46, 131, 140, and 146.)

Many other cultivated and wild plants have been found naturally infected with both viruses. A selection of plants found infected with TBRV is listed below; those in which RRV has also been found are indicated by an asterisk.

#### Cultivated Plants

*Allium ascalonicum* L. (shallot), *A. cepa* L. (onion), *A. porrum* L. (leek), *A. schoenoprasum* L. (chives), *Apium graveolens* L. var. *dulce* Mill. (celery), *Asparagus officinalis* L. (asparagus), *Beta vulgaris* L. subsp. *saccharifera* (sugarbeet)\*, *Brassica oleracea* L. (cabbage), *B. napus* L. (rape), *B. napobrassica* DC. (swede), *B. rapa* L. (turnip), *Cucumis sativus* L. (cucumber), *Daucus carota* L. (carrot), *Lactuca sativa* L. (lettuce), *Lolium perenne* L. (ryegrass), *Lycopersicon esculentum* Mill. (tomato), *Medicago sativa* L. (lucerne, alfalfa), *Narcissus pseudo-narcissus* L. (daffodil)\*, *Pastinaca sativa* L. (parsnip), *Petroselinum crispum* (Mill.)

Nym. (parsley), *Prunus amygdalus* Batsch (almond), *P. avium* L. (cherry)\*, *P. persica* Batsch (peach), *Solanum tuberosum* L. (potato), *Trifolium repens* L. (white clover), *Tulipa gesneriana* L. (tulip), *Vitis vinifera* L. (grapevine)\*.

#### Wild Plants

*Capsella bursa-pastoris* (L.) Medic.\*, *Cerastium vulgatum* L.\*, *Geranium dissectum* L., *Lamium amplexicaule* L., *Myosotis arvensis* (L.) Hill\*, *Polygonum aviculare* L., *P. convolvulus* L.\*, *Spergula arvensis* L.\*, *Stellaria media* (L.) Vill.\*, *Veronica agrestis* L.\*, *V. persica* Poir\*.

**Symptoms on red raspberry.** Table 11 (p. 205) lists the susceptibility of red raspberry cultivars to British isolates of RRV and TBRV. In soils containing both RRV and TBRV, some raspberry cultivars may become infected with only one of the viruses because they are immune to the other. Thus, the widely grown cv. 'Malling Jewel' is immune to TBRV. Several cultivars, notably 'Lloyd George', 'Malling Landmark', and 'Seedling M', are immune both to TBRV and to common strains of RRV; however, a resistance-breaking strain of RRV is known and is the cause of "yellow blotch" disease (Cadman and Harris 1952; Murant et al. 1968).



Outbreaks of disease caused by RRV and TBRV occur in patches, which may vary from a few square meters to a few hectares in extent, reflecting the horizontal distribution of the vector in the soil (fig. 254). However, if the disease results from the planting of infected stocks, the infected plants are of course scattered randomly throughout the crop. In Great Britain, because spread of the viruses is now effectively controlled, the field reaction of some newer cultivars is unknown. The symptoms in long-established cultivars are as follows:

**RRV.** Conspicuous chlorotic ringspots appear on the leaves (fig. 255), usually in the spring following the year of infection, but they may become less distinct or disappear altogether in midsummer, often to return in the autumn. In addition, some cultivars, notably 'Baumforth's Seedling B' and 'Norfolk Giant', show typical leaf-curl symptoms (fig. 256): the leaves exhibit a pronounced downward curling, and are crisp and brittle to the touch. Plants of 'Baumforth's Seedling B', 'Glen Clova', 'Malling Enterprise', 'Malling Jewel', 'Malling Notable', 'Norfolk Giant', and 'Preussen' produce stunted, brittle canes, and may die within 2 to 3 yr of the first appearance of leaf symptoms. Infected plants of other cultivars are usually less vigorous than healthy ones, but are not killed, and often show few or no leaf symptoms.

**TBRV.** This virus causes a severe disease in the old cv. 'Seedling V' (Harrison 1958b), affected plants producing many short, spindly, and brittle young shoots with ill-defined chlorotic markings on the leaves. However, it causes relatively mild symptoms in most other cultivars. 'Malling Exploit' develops faint chlorotic mottling or ringspots (fig. 257) on the leaves but is otherwise little affected in the first few years after infection. Later, the canes are somewhat stunted, yield is decreased, and the fruit is deformed ("crumbly"; fig. 258) due to abortion of some of the drupelets (Taylor et al. 1965). 'Norfolk Giant' develops leaf-curling symptoms similar to those induced by RRV but is otherwise little affected by TBRV, whereas plants infected by RRV are killed.

**Symptoms on blackberry.** RRV-infected plants of 'Himalaya Giant' blackberry (*Rubus procerus* P. J. Muell.) were stunted (Cadman 1960b), but a detailed description of symptoms was not given.

**Symptoms on *R. sachalinensis*.** Symptoms were similar to those in cultivated raspberry (Gordejchuk et al. 1977).

**Symptoms on other cultivated plants.** For the symptoms induced by RRV and TBRV in *Fragaria* and *Ribes*, see these sections of this handbook, p. 46 and 131. RRV, in association with viruses of the prunus necrotic ringspot type, causes rasp-leaf symptoms in cherry (Cropley 1961; East Malling Research Station 1963). Symptoms produced by TBRV in some other crops are described by the following authors: sugarbeet



Figure 255. — Chlorotic ringspot symptoms in leaf of 'Malling Jewel' raspberry infected with raspberry ringspot virus. (After Cadman 1956; copyright Scottish Crop Research Institute.)



Figure 256. — Leaf curl symptom in 'Norfolk Giant' raspberry infected with raspberry ringspot virus. (After Murant 1981b; copyright Scottish Crop Research Institute.)





Figure 257. — Diffuse chlorotic ringspots in leaf of 'Malling Exploit' raspberry infected with tomato black ring virus. (After Taylor et al. 1965; copyright Scottish Crop Research Institute.)

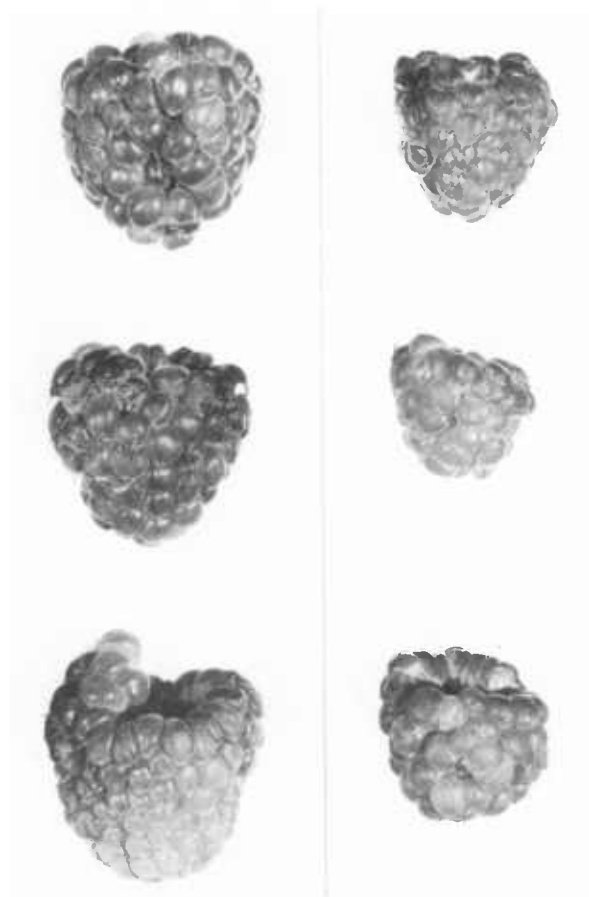


Figure 258. — Fruit of 'Malling Exploit' raspberry, (left) healthy, (right) infected with tomato black ring virus showing "crumbly fruit" symptoms. (After Taylor et al. 1965; copyright Scottish Crop Research Institute.)



Figure 259. — Lesions induced by raspberry ringspot virus in inoculated leaf of *Chenopodium amaranticolor*. (After Murrant 1981b; copyright Scottish Crop Research Institute.)

(Harrison 1957); celery (Hollings 1965); grapevine (Stellmach 1970); lettuce (Smith and Short 1959); leek and onion (Calvert and Harrison 1963); potato (Gehring and Bercks 1956; Harrison 1959); swede and turnip (Harrison 1957); tomato (Smith 1946).

#### Symptoms on Experimental Hosts

Both RRV and TBRV have very wide experimental host ranges and infect nearly all commonly used herbaceous test plants. RRV infected species in more than 14 dicotyledonous families (Murrant 1978) and TBRV infected species in more than 29 dicotyledonous families (Schmelzer 1963). The following are some useful diagnostic hosts:

##### *Chenopodium amaranticolor* Coste and Reyn.

RRV: Chlorotic or necrotic local lesions (fig. 259); no systemic infection.

TBRV: Chlorotic or necrotic local lesions; systemic necrosis or chlorotic mottle (fig. 260).

##### *Chenopodium quinoa* Willd.

RRV and TBRV: Chlorotic or necrotic local lesions; systemic chlorotic mottle or necrosis (fig. 261).

##### *Nicotiana clevelandii* Gray

RRV and TBRV: Local necrotic spots and rings; systemic veinal necrosis (fig. 262). Leaves produced later appear to "recover," that is, they look normal but contain virus.



Figure 260. — Local and systemic symptoms induced by tomato black ring virus in *C. amaranticolor*. (After Murant 1981b; copyright Scottish Crop Research Institute.)



Figure 261. — Systemic symptoms induced by raspberry ringspot virus in *C. quinoa*. (After Murant 1978; copyright Scottish Crop Research Institute.)



Figure 262. — Local necrotic rings and systemic veinal necrosis in *Nicotiana clevelandii* infected with tomato black ring virus. (After Murant 1981b; copyright Scottish Crop Research Institute.)



Figure 263. — Systemic symptoms induced by raspberry ringspot virus in *N. rustica*. (After Murant 1978; copyright Scottish Crop Research Institute.)

#### *Nicotiana rustica* L.

RRV and TBRV: Local chlorotic or necrotic spots or rings; systemic rings and line patterns with variable amounts of necrosis (fig. 263). Leaves produced later appear to “recover,” that is, they look normal but contain virus. The ‘Lloyd George’ yellow blotch strain of RRV gives symptomless local and systemic infection.

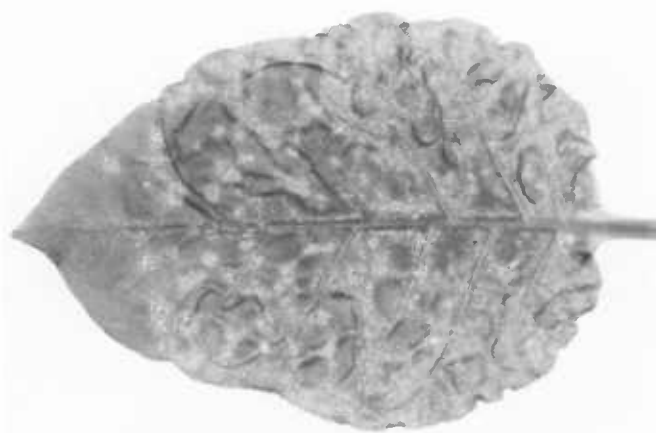


Figure 264. — Systemic symptoms induced by tomato black ring virus in *N. tabacum* cv. 'Xanthi'. (After Murant 1981b; copyright Scottish Crop Research Institute.)

*Nicotiana tabacum* L. cvs. 'White Burley' or 'Xanthi'.

RRV: Chlorotic local lesions; scattered systemic chlorotic spots and rings. Not infected by the 'Lloyd George' yellow blotch strain (table 11).

TBRV: Local necrotic spots and rings; systemic chlorotic and necrotic spots, rings, and line-patterns (fig. 264). Leaves produced later appear to "recover," that is, they look normal but contain virus (fig. 265).

*Phaseolus vulgaris* L. cv. 'The Prince'.

RRV: In winter in Great Britain, dark-brown necrotic local lesions 0.5 mm in diameter (fig. 266); in summer, chlorotic lesions or symptomless. Systemic symptoms are chlorotic mottle with variable amounts of necrosis and distortion (fig. 267). The 'Lloyd George' yellow blotch strain gives symptomless local infection and does not invade the plants systemically.

TBRV: In winter in Great Britain, dark-brown local lesions 2 mm in diameter (fig. 268); in summer, chlorotic lesions or symptomless. Systemic symptoms are chlorotic mottle with variable amounts of necrosis and distortion.

*Petunia hybrida* Vilm.

RRV and TBRV: Chlorotic local lesions, sometimes with brown necrotic margins; systemic veinal chlorosis or necrosis, or line-patterns. With TBRV, leaves produced later are symptomless but contain virus ("recovery"). RRV tends to produce persistent yellow rings and line-patterns (fig. 269) in addition to symptomless leaves; some strains of RRV induce a prominent overall yellowing or bleaching (Harrison et al. 1972b, 1974). The 'Lloyd George' yellow blotch strain of RRV gives symptomless local and systemic infection.

Isolates of both viruses differ considerably in virulence, but *Chenopodium amaranticolor* and *C. quinoa* develop symptoms with all isolates. Most hosts show milder symptoms in summer than in winter.



Figure 265. — Systemic symptoms (lower leaves) followed by apparent recovery (upper leaves) in *N. tabacum* cv. 'Xanthi' infected with tomato black ring virus. (After Murant 1981b; copyright Scottish Crop Research Institute.)

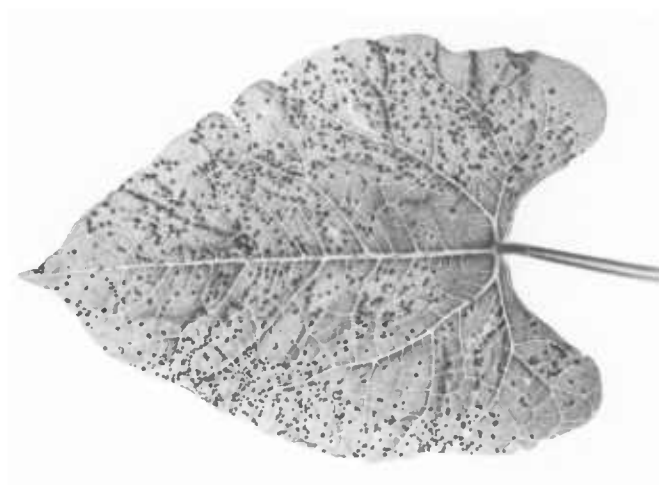


Figure 266. — Lesions induced by raspberry ringspot virus in inoculated leaf of *Phaseolus vulgaris* cv. 'The Prince'. (After Harrison 1958a; copyright Scottish Crop Research Institute.)



Figure 267. — Systemic symptoms induced by raspberry ringspot virus in *P. vulgaris* cv. 'The Prince'. (After Murant 1981b; copyright Scottish Crop Research Institute.)

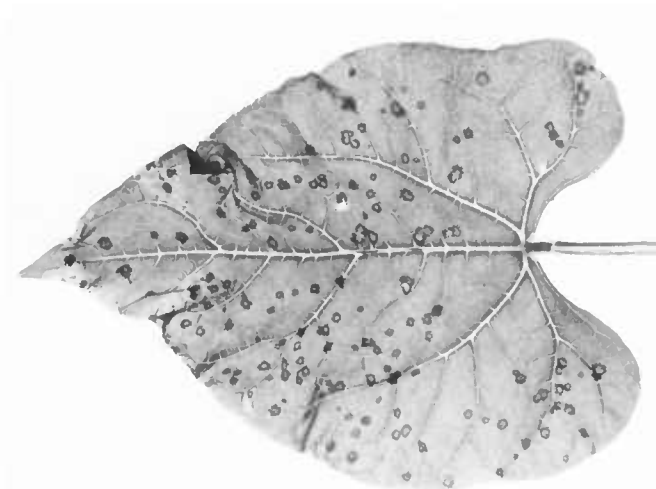


Figure 268. — Lesions induced by tomato black ring virus in inoculated leaf of *P. vulgaris* cv. 'The Prince'. (After Harrison 1957; copyright Scottish Crop Research Institute.)



Figure 269. — Systemic symptoms induced by raspberry ringspot virus in *Petunia hybrida*. (After Harrison 1958a; copyright Scottish Crop Research Institute.)

Both viruses may be propagated in *Nicotiana clevelandii* or *Petunia hybrida* and may be assayed by counting local lesions in *Chenopodium amaranticolor*. *C. quinoa* and *P. hybrida* are convenient "bait" plants for use in nematode-transmission experiments; these plants rarely become systemically infected or show symptoms following inoculation of the roots by nematodes, but the viruses may be detected in the roots or hypocotyls by sap inoculation to the leaves of further *C. quinoa* test plants.

#### Natural and Experimental Transmission

Both viruses are transmissible by grafting, by sap inoculation, and through seed. Their natural vectors are nematodes in the genus *Longidorus*.

Transmission of the viruses to herbaceous plants by inoculation with sap from rosaceous plants is greatly facilitated if inocula are prepared at pH 8 with 2% nicotine or 1% polyethylene glycol (mol. wt. 6000), or if powdered alumina is added to the inoculum. These additives prevent tannins from inhibiting infection. Transmission by inoculation with sap from herbaceous plants to rosaceous plants is extremely difficult.

Lister (1960) and Lister and Murant (1967) showed that both viruses are seed-borne in a wide range of host plants, including raspberry, strawberry, and many weed species. There are usually no symptoms in infected progeny seedlings. The viruses are transmitted through seed to up to 20% of raspberry progeny and up to 40% of strawberry progeny via either gamete; however, the presence of competing virus-free pollen greatly decreases the ability of pollen from infected plants to fertilize ovules. Transmission through pollen may therefore be of little importance in nature. Healthy plants pollinated with pollen from infected plants do not become infected.

Scottish strains of RRV and TBRV share a common vector, *Longidorus elongatus* de Man (Taylor 1962; Harrison et al. 1961) and therefore often occur together in the same outbreak



area. In contrast, English and Continental European strains of RRV and TBRV, which are serologically distinguishable from Scottish strains, are transmitted only inefficiently by *L. elongatus*; their natural vectors are, respectively, *L. macrosoma* Hooper and *L. attenuatus* Hooper (Harrison 1964; Taylor and Murant 1969). Because these nematodes have different soil-type preferences, English strains of RRV and TBRV tend to occur in separate sites.

*L. elongatus* kept in fallow soil retains infectivity with RRV and TBRV as long as 8 or 9 wk (Murant and Lister 1967; Taylor 1970). *L. macrosoma* retains the English strain of RRV for at least 34 days (Debrot 1964). Both viruses are transmitted by larvae and adults of *L. elongatus* (Harrison et al. 1961; Taylor 1962; Yassin 1968), but they do not pass through the egg, nor are they retained by the nematode after moulting (C. E. Taylor, unpublished data). By analogy with other nematode-borne viruses, it seems unlikely that RRV or TBRV multiply in their vectors or circulate within them (Taylor 1980). Particles of both viruses are associated in a specific manner with the stylet lumen or guiding sheath of *Longidorus* spp. (Taylor and Robertson 1969). The vector specificity of the viruses is determined by the composition of their coat proteins (Harrison et al. 1974; Harrison and Murant 1977a).

Within an outbreak area, the viruses are spread by the vector nematodes, but because the nematodes do not withstand airdrying of soil they do not carry the viruses efficiently over a distance. Since there is little lateral migration of the vector, outbreaks extend only slowly. The viruses are probably disseminated in nature in infected seeds, and these are also important as a continuing reservoir of the viruses in the soil, enabling them to survive periods of fallow or fasting of the vector (Murant and Taylor 1965; Murant and Lister 1967). The role of wild plants in the ecology of these and other nematode-borne viruses was discussed by Murant (1970b, 1981b).

### Properties of the Causal Agents

Both viruses are members of the nepovirus group (Harrison and Murant 1977b; Murant 1981a) and have many similar properties.

**RRV.** For a detailed description of the virus, see Murant (1978). In *Nicotiana rustica* sap, the virus loses infectivity after dilution to  $10^{-3}$  to  $10^{-4}$ , or after 10 min at 65 to 70°C or 2 to 3 wk at room temperature (Harrison 1958b). It survives for many years in sap at -15°C. The virus may be purified (Murant et al. 1972; Murant 1978) from *N. clelandii* extracts by adding *n*-butanol to 8.5% (v/v) and centrifuging at low speed. The virus is then precipitated from the supernatant fluid by adding polyethylene glycol (mol. wt. 6000) to 10% (w/v) and NaCl to 1% (w/v) and concentrated further by differential centrifugation. The virus particles are isometric, about 28 nm in diameter, with hexagonal outlines (fig. 270). Some are completely or partially penetrated by

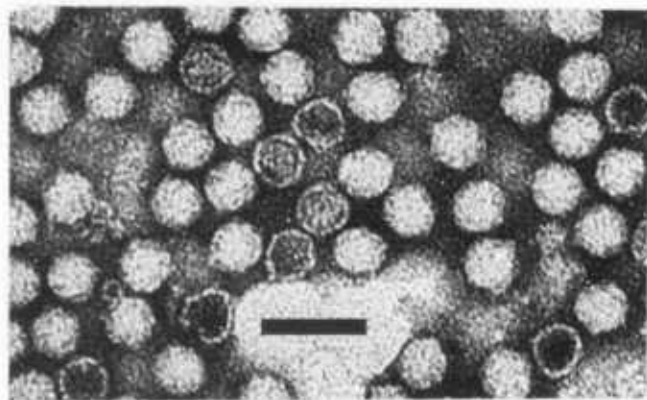


Figure 270. — Particles of raspberry ringspot virus showing some completely, some partially, and some not penetrated by phosphotungstate. Bar represents 50 nm. (After Murant 1978; copyright Scottish Crop Research Institute.)

negative stain; others are not penetrated. The particles form three sedimenting components, T, M and B, with sedimentation coefficients ( $s_{20,w}$ ) of 50, 92, and 130 S, respectively, and  $A_{260}/A_{280}$  of 0.68 (T), 1.48 (M), and 1.69 (B), respectively (Murant et al. 1972). The coat protein is a single species with a mol. wt. about 54000 (Mayo et al. 1971), and the genome consists of two species of single-stranded RNA with mol. wts. of 2.8 and  $1.4 \times 10^6$  daltons (Murant et al. 1972, 1981). Both RNA species are necessary for infectivity (Harrison et al. 1972), each carrying different genetic information. For example, the larger RNA carries the determinant for ability to infect 'Lloyd George' raspberry and the smaller RNA carries determinants for nematode transmissibility and serological specificity, that is, it contains the coat protein cistron (Harrison et al. 1972b, 1974).

Scottish isolates of RRV, both those of the common strain and those of the 'Lloyd George' yellow blotch strain, are serologically very similar to each other and to a strain causing spoon leaf disease of red currant (*Ribes rubrum* L.) in The Netherlands (Harrison 1961; Maat et al. 1962). However, these strains differ serologically from some found in England and Germany (Cadman 1960b), and this difference is correlated with a difference in transmissibility by *Longidorus* spp. (See "Natural and Experimental Transmission," p. 217). An isolate from grapevine in Germany (Vuittenez et al. 1970) was serologically very distantly related to the Scottish strain.

**TBRV.** For a detailed description of the virus, see Murant (1970a). In tobacco sap, the virus loses infectivity after dilution to  $10^{-3}$  to  $10^{-4}$ , or after 10 min at 60° to 65°C, or 2 to 3 wk at room temperature (Harrison 1957). The virus may be purified by the method described above for RRV. The particles are similar to those of RRV and, like it, sediment as three components, called T, M, and B, but with sedimentation coefficients ( $s_{20,w}$ ) of 55, 97, and 121 S, respectively (Murant 1970c). M and B components have  $A_{260}/A_{280}$  of 1.62 and 1.78, respectively (Forster 1980). The coat protein is a single species with a molecular weight of about 57000

(Murant et al. 1973), and the genome consists of two species of single-stranded RNA with mol. wts. of 2.7 and  $1.7 \times 10^6$  daltons (Murant et al. 1973, 1981). Both RNA species are necessary for infectivity (Murant et al. 1973), each carrying different genetic information (Randles et al. 1977; Harrison and Murant 1977a; Hanada and Harrison 1977). For example, the smaller RNA carries the information for nematode transmissibility and serological specificity, that is, it contains the coat protein cistron. Some isolates of TBRV also contain a "satellite" RNA, of mol. wt.  $0.5 \times 10^6$ , which appears to depend on TBRV for its replication (Murant et al. 1973).

Strains of TBRV that have been studied serologically fall into two groups, one group containing the English lettuce ringspot isolate and the German potato bouquet and grapevine strains (Harrison 1958d; Stellmach and Bercks, 1965; Vuittenez et al. 1970), the other containing the Scottish beet ringspot isolate and the German potato pseudoauctuba strain (Bercks 1962). This serological difference is correlated with a difference in specific nematode vector. (See "Natural and Experimental Transmission," p. 217.) TBRV is very distantly related to cacao necrosis and grapevine chrome mosaic viruses.

### Detection and Identification

The patchy appearance of a disease outbreak, ringspot lesions on leaves tending to fade in midsummer and, in cultivars highly sensitive to RRV, the stunted, dead, and dying plants are characteristic features; however, similar symptoms are induced by other nematode-borne viruses. Moreover, some raspberry cultivars show only mild symptoms, and even sensitive cultivars may show few or no symptoms in early stages of infection. The virus(es) present are detected by inoculation of sap to a suitable indicator, preferably *Chenopodium quinoa*, and are identified by serological tests, preferably by double diffusion in agar or agarose gel. Serological tests are essential because, although RRV and TBRV may be distinguished from each other by the reaction of *C. amaranticolor*, they cannot be reliably identified and distinguished from other nepoviruses by their effects on test plants.

### Control Procedures

**Use of certified virus-tested planting material.** Both viruses may be distributed in infected planting material, which may present a hazard because few or no symptoms are visible in tolerant cultivars or in the early stages of infection in sensitive cultivars. This source of infection can be eliminated by introducing adequate certification schemes for the production of virus-tested stock. Such schemes should include the requirement that stocks for certification are not established in soil containing vector nematodes.

**Use of immune cultivars.** If the outbreak in a sensitive cultivar is small, only affected plants need be removed to be replaced with plants of an immune cultivar (see table 11).

Immunity to RRV and TBRV in red raspberry appears to be inherited as a dominant character, but for each virus at least two genes are concerned (Jennings 1964). Although in some Scottish plantations, the 'Lloyd George' yellow blotch strain of RRV infects cultivars that are immune to the common strain, this resistance-breaking strain has not become prevalent, probably because it is poorly transmitted through seed of common weeds and therefore does not survive well in soils (Murant et al. 1968; Hanada and Harrison 1977). Cultivars immune to RRV have therefore given good control, although they are not preferred agronomically. Fortunately, the major cultivar in Scotland, 'Malling Jewel', is immune to TBRV and has never been found infected with this virus after some 30 yr in cultivation.

**Cultural methods.** The wide host ranges of the viruses and their vectors among wild and cultivated plants preclude crop rotation as the sole control measure, but not all plants are equally good hosts of vector nematodes. *L. elongatus* populations increase rapidly on strawberry, ryegrass, clovers, and many weeds such as *Stellaria media*, but decrease on barley plants, raspberry, and many vegetable crops (Taylor 1967; Thomas 1969). Therefore, in areas where RRV and TBRV are known to be troublesome, infection can be minimized by not planting raspberry after strawberry or grass/clover pasture, and by adopting good long-term weed control measures. The latter also prevent the accumulation in the soil of infected weed seeds, which act as virus sources.

**Chemical control of vector nematodes.** Treatment of the soil with D-D (dichloropropene-dichloropropane), dazomet, or pentachloronitrobenzene gave good control of *L. elongatus* and prevented spread of RRV and TBRV in strawberry and raspberry (Murant and Taylor 1965; Taylor and Murant 1965, 1968; Trudgill and Alphey 1976). Although side effects were noticed with pentachloronitrobenzene in plantings of 'Lloyd George' raspberry and sugarbeet (Taylor and Murant 1968), this chemical has been widely used and effective in Scotland. However, fumigant nematicides such as dazomet or dichloropropene (the active constituent of D-D) are now the preferred treatments in commerce. Chemical treatments are most effective and most necessary after the land has carried crops, such as strawberry, that lead to an increase in numbers of *L. elongatus*. The practice of pulverizing old raspberry canes and returning them to the soil is also effective in decreasing the numbers of *L. elongatus* (Taylor and Murant 1966).

**Thermotherapy.** Little information is available on the response of RRV and TBRV to heat treatment. In one experiment, RRV was not eliminated from 'Malling Promise' raspberry held for 3 wk at 37°C (J. Chambers, unpublished data).

### Remarks

The symptoms produced by isolates of RRV and TBRV in standard herbaceous indicators are similar to those induced

by many other viruses infecting small fruits, particularly members of the nepovirus group such as arabis mosaic, cherry leaf roll, strawberry latent ringspot, tomato ringspot, and tobacco ringspot viruses. (See papers on these viruses in the *Rubus* section.) Moreover, all these viruses have very wide host ranges and infect naturally many other wild and cultivated plants. Serological tests are the only satisfactory method of identification.

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## Cherry Leaf Roll Virus in *Rubus*

By A. T. Jones

### Additional Common Names

None.

### History and Geographic Distribution

Cherry leaf roll virus (CLRV) was first reported from blackberry plants in England (East Malling Research Station 1970, Ormerod 1972a) and later in red raspberry in New Zealand where it was found in several plantations (Jones and Wood 1978). CLRV is common in several species of trees and shrubs in Europe, North America, and the U.S.S.R. (Cropley and Tomlinson 1971; Bubaker and Pomazkov 1978), but no other reports of infection in *Rubus* are known.

### Economic Importance

The virus is common in some red raspberry plantations in New Zealand, but the incidence of infection within plantations varies from only one or two plants to over 70% (Jones and Wood 1978). Fruiting canes of infected plants usually show severe leaf symptoms and decreased vigor. The virus is thus a potentially serious disease problem; however, the precise extent of infection in New Zealand is not yet known.

Ormerod (1972a and East Malling Research Station 1970), reported three separate outbreaks of CLRV in 'Himalaya Giant' blackberry in Great Britain. The disease associated with CLRV infection was lethal in some plants (Cropley and Tomlinson 1971), but the incidence of infection is not known.

### Symptoms on Natural and Experimental Hosts

***Rubus* hosts.** In naturally infected *R. procerus* P.J. Muell. cv. 'Himalaya Giant', CLRV is reported to cause chlorotic mottling and line-pattern leaf symptoms, stunting, and sometimes death (Cropley and Tomlinson 1971). Natural infection in red raspberry in New Zealand was associated with stunted fruiting canes, which were characterized by poor and distorted leaf development. Some leaves showed line-pattern symptoms, severe chlorotic mottle, and/or ringspot symptoms late in the season (figs. 271 and 272) (Jones and Wood 1978, 1979). No symptoms were evident on primocanes. The red raspberry cultivars found naturally infected were 'Marcy', 'Lloyd George', and 'Taylor'.



Figure 271. — Chlorotic mottling in leaves of a fruiting cane of 'Marcy' red raspberry infected with cherry leaf roll virus. (Copyright Scottish Crop Research Institute.)

**Herbaceous hosts.** After mechanical inoculation with a raspberry isolate, the following herbaceous species showed symptoms:

*Chenopodium amaranticolor* Coste and Reyn., *C. foetidum* Schrad., *C. quinoa* Willd., and *Phaseolus vulgaris* L. cv. 'Market Wonder' developed chlorotic or necrotic local lesions within 6 days followed by systemic apical necrosis. *Cucumis sativus* L. (fig. 273), *Nicotiana clevelandii* Gray, and *N. tabacum* L. cvs. 'Samsun', 'White Burley', and 'Xanthi-nc' developed large chlorotic or necrotic local lesions within 4 to 5 days followed by a systemic mosaic. Local infection in *N. tabacum* was frequently characterized by the development of necrotic rings (fig. 274). *Nicotiana glutinosa* L. was often symptomlessly infected. Strains of CLRV from different natural hosts differed somewhat in host range and symptomatology (Jones 1973, 1976c).

### Natural and Experimental Transmission

Natural transmission by nematode vectors, particularly species of *Xiphinema*, was reported by Fritzsche and Kegler (1964). However, when Jones et al. (1981) reinvestigated the possibility of transmission of three strains of CLRV by 10 species of potential vector nematodes, they rarely recovered CLRV from bait plants and then only when these were





Figure 272. — Range of chlorotic mottling symptoms in leaves of 'Marcy' red raspberry infected with cherry leaf roll virus. (Copyright DSIR, New Zealand.)



Figure 273. — Cucumber seedlings mechanically inoculated with cherry leaf roll virus showing large chlorotic-necrotic local lesions and systemic mosaic. (Copyright DSIR, New Zealand.)



Figure 274. — Leaf of *N. tabacum* cv. 'Xanthi-nc' mechanically inoculated with cherry leaf roll virus and showing necrotic rings. (Copyright Scottish Crop Research Institute.)



growing concurrently in pots containing CLRV-infecter plants. They attributed these few infections to contamination and concluded that nematode transmission was unlikely to account for the widespread occurrence of CLRV in woody hosts. For more detailed discussion on this subject, see Jones et al. (1981).

Recent information on the spread of CLRV in walnut orchards in California confirms that nematodes are not involved but provides good circumstantial evidence that CLRV is transmitted via pollen to the plant pollinated (Mircetich et al. 1980). Further experimental work is needed to prove that this is so and to determine if this method of transmission also occurs in *Rubus* and other natural hosts.

CLRV is seed-borne, often to a large proportion of seedlings, in many natural (Callahan 1957a; Schimanski and Schmelzer 1972; Cooper 1976) and experimental (Lister and Murant 1967; Tomlinson and Walkey 1967) hosts and can infect seed of some species via both ovule and pollen (Callahan 1957a, b). However, no information is available on seed transmission in *Rubus*.

CLRV is transmitted experimentally by mechanical inoculation, readily to herbaceous hosts, and less readily to natural host species of *Betula*, *Prunus*, *Rheum*, and *Sambucus* (Tomlinson and Walkey 1967; Hansen and Stace-Smith 1971; Jones 1973; Cooper and Atkinson 1975).

CLRV is graft transmissible in many woody hosts, but there are no reports of this in *Rubus*.

### Properties of the Causal Agent

For a detailed description of the virus, see Cropley and Tomlinson (1971), Jones and Mayo (1972), and Walkey et al. (1973). Many isolates of CLRV have been described, and most are relatively stable in sap of herbaceous hosts. Infectivity in sap of *C. quinoa* or *N. clelandii* usually survives diluting  $10^{-3}$  to  $10^{-4}$ , heating for 10 min at 55 to 60°C, and storing for 4 to 8 days at room temperature (Cropley and Tomlinson 1971). Virus preparations free from most contaminating host components can be prepared in the following way: Extract sap of CLRV-infected *C. quinoa* or *N. clelandii* in 0.1 M phosphate buffer (pH 7) and clarify either by freeze/thawing, or treatment with organic solvents, or ammonium sulphate, or combinations of these.

Further clarification and concentration is by differential centrifugation and sucrose density gradient centrifugation. Purified preparations of CLRV contain isometric particles about 28 nm in diameter, some of which are penetrated by negative stain (fig. 275). Particles sediment as two nucleoprotein components with sedimentation coefficients of about 115 S and 128 S, and in some preparations a nucleic acid-free component of about 54 S is detectable. The  $A_{260/280}$  value of mixtures of the 2 nucleoprotein components is about 1.6. Particles of several different strains of CLRV have been

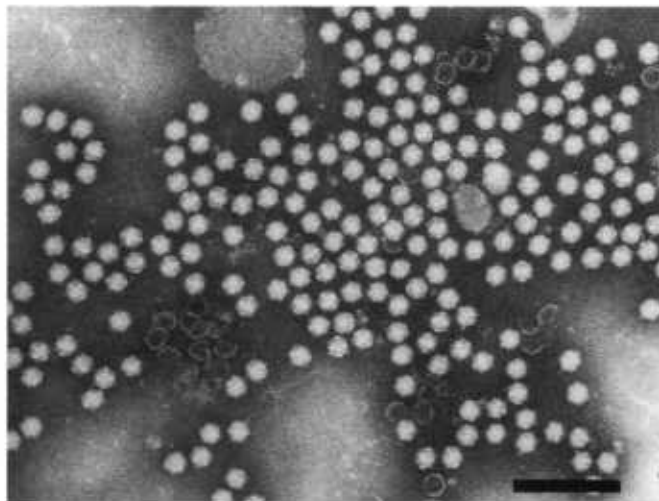


Figure 275. — Electron micrograph of a purified preparation of cherry leaf roll virus stained in 2% phosphotungstate, pH 6.5. Some particles are penetrated by the stain. Bar represents 150 nm. (Copyright Scottish Crop Research Institute.)

shown to contain a single polypeptide species of 54000 mol. wt. and 2 RNA species of estimated mol. wt.  $2.1 \times 10^6$  and  $2.4 \times 10^6$ , which are contained in the 115 S and 128 S particles, respectively (Jones and Mayo 1972; Walkey et al. 1973).

Many serotypes of CLRV are known (Jones and Murant 1971; Jones 1976c; Cooper and Edwards 1980); indeed, isolates from different plant genera tend to be serologically distinct (Jones 1976c; Cooper and Edwards 1980). Ormerod (1975), however, reported that one isolate from blackberry was serologically identical to the type strain of CLRV from cherry but that this blackberry isolate differed in symptomatology in herbaceous hosts from two other isolates from blackberry. Raspberry isolates of CLRV tested from various parts of New Zealand were serologically indistinguishable from one another when tested against antiserum to CLRV from American dogwood (*Cornus nuttallii* Aud.) (Jones and Wood 1978).

### Detection and Identification

Although disease symptoms were associated with CLRV infection of blackberry and raspberry (East Malling Research Station 1970; Jones and Wood 1978), the virus is best detected by mechanical inoculation of herbaceous test plants, such as *Chenopodium* spp. or cucumber, and is then best identified by serological tests. Because of the antigenic differences among CLRV isolates, some may fail to react with diluted antiserum to a given isolate.

### Control Procedures

In the absence of firm evidence on the mode of transmission of CLRV in *Rubus*, control measures are difficult to prescribe in detail. Much of the incidence in raspberry in New Zealand was attributed by Jones and Wood (1978) to propagation from infected material. The planting of material derived from

virus-tested elite stock should restrict further unwitting spread of this virus; however, if CLRV is pollen transmitted in *Rubus*, as it is suspected to be in walnut (see "Natural and Experimental Transmission"), much of the incidence observed in raspberry plantations in New Zealand could be the result of natural spread. If this is so, eradication of affected plants in the only known means of controlling spread. Resistant or immune cultivars have not been reported.

No attempts have been made to cure infected *Rubus* plants from CLRV, but the virus was eliminated from rhubarb by meristem-tip culture (Walkey 1968).

### Remarks

Although CLRV appears not to be spread readily if at all by nematodes (Jones et al. 1981), it is regarded as a nepovirus because of the properties of its particles (Jones and Mayo 1972; Harrison and Murrant 1977b). It induces symptoms in *Chenopodium* and *Nicotiana* species similar to those caused by many nepoviruses (see the nematode-borne diseases papers of the *Rubus* section of this handbook, p. 204–228) and by some isolates of tobacco streak virus (see "Tobacco Streak Virus in *Rubus*," p. 235) and can only be identified with certainty by serological tests.

### 245 Tomato Ringspot Virus in *Rubus* //

By R. Stace-Smith and R. H. Converse

### Additional Common Names

Raspberry yellow blotch curl (Chamberlain 1938); raspberry decline (Zeller and Braun 1943); Himalaya blackberry mosaic (Alcorn et al. 1955); raspberry ringspot (Vaughan et al. 1951).

### History and Geographic Distribution

The symptoms now attributed to infection with tomato ringspot virus (TomRSV) were not recorded in the early raspberry virus literature in North America. In retrospect, the virus was probably in those plantings where the mosaic complex (see "Raspberry Mosaic," p. 168) and raspberry leaf curl (see "Raspberry Leaf Curl," p. 187) were first observed, but the symptoms were probably attributed to one of those viruses. The first description of TomRSV in raspberry was from Ontario, Canada (Chamberlain 1938). The new disease was named "yellow blotch-curl" and, although conclusive evidence on the identity of the causal virus was not obtained, there is little doubt that the virus involved was TomRSV.

The next record of the disease was from Oregon, where Zeller and Braun (1943) proposed the name "raspberry decline" for a disease that lacked leaf or cane symptoms but was characterized by crumbly fruit and a decline in plant growth and productivity. Raspberry decline was shown to be graft transmissible and the pattern of spread in the field led the authors to speculate that spread of infection involved

some underground factor. Although raspberry decline was not shown to be caused by TomRSV, the evidence today leaves little doubt that the virus involved was TomRSV.

Vaughan et al. (1951) used the term "ringspot" to describe a disease that was generally found in red raspberry throughout Oregon and Washington. This disease was later found in British Columbia (Stace-Smith 1962b) and was shown for the first time to be caused by TomRSV. This virus is now known to occur throughout the raspberry growing areas of the United States and Canada. Field spread is restricted to those areas where certain vectors of the genus nematode *Xiphinema* occur.

The geographic distribution of TomRSV in *Rubus* is confined to the temperate regions of North and South America. To a very limited extent, the virus has been distributed in infected clones to other parts of the world. Reports to date indicate that the virus has been isolated from cultivars originating in North America, Yugoslavia (Jordović et al. 1972a), and the U.S.S.R. (Gordejchuk et al. 1977).

### Economic Importance

TomRSV is considered to be one of the most widespread and economically important virus diseases of *Rubus* in North America. Loss is difficult to assess because variability in symptoms depends on the cultivar and duration of infection. The ringspot disease, caused by TomRSV, is the most common virus disease seen in field-grown red raspberries in Oregon and Washington (Converse et al. 1970). The virus is also prevalent in Eastern United States, where it is associated with a crumbly berry condition in red raspberry (Keplinger et al. 1968).

### Symptoms on Natural and Experimental Hosts

TomRSV has a wide experimental and natural host range; species in more than 35 dicotyledonous and monocotyledonous families are susceptible (Stace-Smith 1970b). In nature, the virus occurs mostly in ornamentals and woody or semiwoody plants. Transmission by sap inoculation is readily achieved to herbaceous hosts but with difficulty to woody hosts.

**Symptoms on red raspberry.** Symptoms of TomRSV in red raspberry are variable, depending to a large extent on the cultivar and the duration of the infection. Plants develop symptoms associated with a shock reaction in the year following infection. As new foliage is produced in the spring, leaves may show yellow rings, line-patterns, or a fine yellow vein chlorosis (figs. 276 and 277). Symptoms that develop remain visible throughout the growing season, but leaves that develop during hot weather usually show no symptoms. Those plants that have been infected for more than one year either develop no symptoms on the new foliage or show ring and line-patterns on only one or two leaves.



Figure 276. — Leaf of red raspberry, cv. 'Williamette', naturally infected with tomato ringspot virus, showing ringspot markings.



Figure 277. — Leaf of red raspberry, cv. 'Williamette', naturally infected with tomato ringspot virus, showing netlike chlorosis along leaf veins.

The pattern of spread of TomRSV is characteristic of nematode-borne viruses. Patches of infected plants form a circular pattern with the chronically infected plants in the center of the patch and the recently infected plants, showing shock symptoms, at the margin. In such infections, the chronically infected plants of some cultivars are dwarfed in the spring, foliage is slower to develop (fig. 278), and the primocanes have a distinctly bronze cast in comparison with healthy plants (Converse and Stace-Smith 1971). Cultivars that are severely affected by TomRSV (for example, 'Fairview') show cane death in the spring, and those canes that do survive produce small downcurled leaves that mature early and abscise early in the fall (Freeman and Stace-Smith 1968).

The effect of TomRSV infection on plant yield depends to a large extent on the cultivar. In a study involving 10 cvs., 'Lloyd George', 'Avon', 'Latham', 'Glen Clova', and 'Meeker' showed a reduction in yield in the third cropping year, whereas 'Canby', 'Carnival', 'Malling Jewel', 'Matsqui', and 'Puyallup' showed no significant yield reduction (Freeman et al. 1975). Drupelet set also varied with the cultivar. 'Avon', 'Fairview' and 'Lloyd George' showed a particularly adverse affect (fig. 279); 'Matsqui', 'Puyallup,' and 'Newburgh' were affected to a lesser extent, and TomRSV infection did not affect drupelet set in 'Canby', 'Carnival', 'Glen Clova', 'Latham', 'Malling Jewel', and 'Meeker' (Daubeney et al. 1975).



Figure 278. — Red raspberry plant, cv. 'Puyallup', infected with tomato ringspot virus showing delayed foliation associated with chronic infection (left) as compared with healthy plant (right).

**Symptoms on blackberry.** The effects of TomRSV on blackberry cultivars have not been investigated, but transmission experiments have been done with a 'Himalaya' blackberry plant with what is thought to be TomRSV plus a virus causing feather vein symptoms (Alcorn et al. 1955). These experiments indicate that TomRSV may cause a variety of leaf symptoms, including small chlorotic spots scattered over the leaf blade, large yellow blotches at the base of the leaflets, conspicuous veinal chlorosis, and oak leaf patterns in 'Himalaya' blackberry. The absence of these





Figure 279. — Fruit from red raspberry, cv. 'Fairview', infected with tomato ringspot virus, showing reduced drupelet set, abnormal shapes, and small size (left and right) as compared with fruit from a healthy plant (center).

symptoms in grafted plants of 'Boysen', 'Nectar', 'Youngberry', and seedlings of 'Chehalem', 'Logan', and the native trailing blackberry (*R. ursinus* Cham. and Schlecht.) suggests that these cultivars are either mildly affected or immune.

**Symptoms on indicator hosts.** Since TomRSV affects a wide range of herbaceous hosts, many species have been utilized for detection and diagnosis. The host reactions are valuable for detecting virus infections, but for identification of the virus, confirmatory tests (see "Detection and Identification," p. 226) must be employed (Stace-Smith 1970b). The most useful indicator hosts are as follows:

*Chenopodium quinoa* Willd. (fig. 280) and *C. amaranticolor* Coste and Reyn. (fig. 281).

Small chlorotic local lesions; systemic apical necrosis.

*Cucumis sativus* L. (cucumber)

Local chlorotic lesions; systemic chlorosis and mottle.

*Nicotiana tabacum* L. (tobacco)

Necrotic local spots or rings; systemic etched ring or line-patterns on a few leaves; later leaves symptomless (fig. 282).

*Phaseolus vulgaris* L. (bean)

Chlorotic local lesions; systemic rugosity and necrosis of tip leaves.

*Prunus persica* (L.) Batsch (peach)

Seedlings show no symptoms on inoculated leaves; systemically infected leaves show netlike chlorosis, and tip leaves are distorted and stunted (fig. 283).

### Natural and Experimental Transmission

**Natural transmission:** Natural transmission in *Rubus* spp. is thought to be exclusively by means of nematode vectors belonging to the genus *Xiphinema*, particularly *X. americanum* Cobb and *X. rivesi* Dalmasso. These nematodes can be found in raspberry plantings where TomRSV spread is occurring (McElroy 1977; Forer and Stouffer 1982). The



Figure 280. — Local lesions on *Chenopodium quinoa* caused by inoculation with tomato ringspot virus-infected *Rubus* spp.



Figure 281. — Systemic symptoms on *C. amaranticolor*. Photograph taken 15 days after sap inoculation from a tomato ringspot virus infected red raspberry plant.

virus moves more rapidly from plant to plant along the rows than it moves between rows, producing oblong patches. Where active spread is occurring, the virus moves from plant to plant along rows at an annual rate of about 2 m (Converse and Stace-Smith 1971). Most new infections occur in plants adjoining infected plants. New infections in raspberry may also arise from the feeding of viruliferous nematodes that have acquired the virus by feeding on an infected weed host. Infected chickweed (*Stellaria media* (L.) Cyrillo) as well as





Figure 282. — Tobacco, cv. 'Haranova', showing lesions on inoculated leaf 7 days after sap inoculation from a tomato ringspot virus infected raspberry plant.



Figure 283. — Peach seedling showing systemic chlorosis and tip distortion. Photograph taken 18 days after sap inoculation from a tomato ringspot virus-infected red raspberry plant.

many other weed species can harbor the virus in a symptomless condition (Converse and Stace-Smith 1971; Forer and Stouffer 1982).

The virus is transmissible through a small percentage of seed from an infected raspberry plant (Braun and Keplinger 1973). The possibility exists that a healthy plant could be infected by pollen from an infected source, but there is no experimental evidence of pollen transmission.

**Experimental transmission:** Transmission from raspberry to raspberry can be achieved by grafting. Experimental transmission to raspberry by means of viruliferous nematodes has not been demonstrated, but it is assumed that techniques similar to those used with other host plants (Téliz et al. 1966) would succeed with *Rubus* spp.

Sap transmissions from infected raspberry to herbaceous host plants are readily achieved, provided succulent leaf tissue is used as the source of inoculum. A phosphate buffer (0.02M, pH. 7.5) containing 2% nicotine is satisfactory, as is 0.05 M phosphate, pH 7.0, containing 2% polyvinylpyrrolidone (mol. wt. 10,000) (Martin and Converse 1982). The most useful indicator hosts are *Chenopodium quinoa* or *Cucumis sativus*.

### Properties of the Causal Agent

TomRSV is a member of the nepovirus group (Harrison and Murrant 1977b). It has three types of isometric particles about 28 nm in diameter, sedimenting at 53S, 119S, and 127S, and containing respectively 0, 40, and 43% single-stranded RNA. The two RNA species, mol. wt.  $2.4 \times 10^6$  and  $2.8 \times 10^6$ , are both required for infection. Each particle contains 60 molecules of a single coat polypeptide, mol. wt. 55000. The virus has a wide natural host range, primarily woody or semiwoody cultivated species and herbaceous and perennial weed species, and may cause ringspot or mottle shock symptoms followed by recovery. Natural transmission is by means of the nematode vectors *X. americanum* and *X. rivesi*.

### Detection and Identification

Infection in raspberry may be detected by field examinations, particularly if surveys are conducted in the spring when foliar symptoms are most pronounced (Vaughan et al. 1951; Converse et al. 1970). Field surveys detect a high proportion of new infections (that is, plants infected during the previous growing season) but a low proportion of chronically infected plants. Some sensitive cultivars (for example, 'Washington' and 'Fairview') show foliar markings on at least a few canes in most years; other cultivars show no symptoms.

Since the absence of symptoms does not necessarily mean absence of infection, visual examinations must be supplemented with tests capable of detecting latent infections. Two tests are useful, namely sap transmissions from *Rubus* to herbaceous hosts or direct serological tests on *Rubus* sap. Bioassay and agar gel serology can be effectively used with foliage produced in the spring, but as summer progresses, it becomes difficult to detect infections (Converse 1976). The enzyme-linked immunosorbent assay (ELISA) technique is more reliable and can be used to detect TomRSV in leaf, stem, bud, and root samples from infected plants even late in the autumn (Converse 1978). Serological tests have the added advantage that the virus is identified. Bioassays may detect the virus, but it is only tentatively identified on the basis of the symptoms induced on a range of herbaceous indicator hosts.

### Control Procedures

TomRSV infections are usually not detected at an early stage of development in field plantings. Infections are not detected until the virus has spread to produce circular patches of unthrifty plants. At this stage, control of the virus is difficult. Plants showing symptoms could be removed and replaced

with healthy plants; but, unless measures were taken to destroy the viruliferous nematodes by soil fumigation, the virus would soon spread into the healthy plants in the replanted area.

A more effective course would be to delay action until yield loss resulting from infection was sufficient to warrant complete removal of both healthy and diseased plants from problem fields. The soil would have to be fumigated with a nematicide before being replanted to a susceptible host. Treatment of established red raspberry plants with the nematicide phenamiphos (58 kg/ha) or dibromochloropropene (64 kg/ha) reduced existing populations of *X. americanum* by half but did not reduce the spread of TomRSV in the field. If roguing is practiced in an attempt to reduce spread of this disease, removing a band at least five red raspberry plants wide together with weeds and suckers beyond those exhibiting symptoms may be helpful in limiting spread of TomRSV in an established field (R. H. Converse, unpublished data).

Precautions should be taken to avoid TomRSV problems when new raspberry plantings are established. These are particularly important if nematode surveys have detected populations of *X. americanum* in the area to be planted. The vector nematodes themselves cause little damage to red raspberry unless the virus is also present (McElroy 1977). The source of inoculum may be excluded from the new planting if planting stock that is certified as free from TomRSV (and other recognized *Rubus* viruses) is used to establish the new planting. If previous history suggests there is a possibility of TomRSV becoming a problem, growers should avoid those cultivars (for example, 'Avon', 'Canby', 'Fairview', 'Lloyd George', and 'Puyallup') that are known to be particularly damaged by the virus.

#### **Tobacco Ringspot Virus in *Rubus*** By R. Stace-Smith

##### **Additional Common Names**

None.

##### **History and Geographic Distribution**

Tobacco ringspot virus (TRSV), a member of the nepovirus group, was first isolated from a wild erect blackberry plant in North Carolina in 1965 (Rush et al. 1968). It was later (Rush and Gooding 1970) found in four native *Rubus* species — *R. allegheniensis* Porter, *R. argutus* Link, and *R. flagellaris* Willd. plus an unidentified *Rubus* sp. in North Carolina. The only record of the virus being isolated from a cultivated blackberry is from British Columbia (Stace-Smith and Hansen 1974).

##### **Economic Importance**

None, primarily because there are essentially no commercial raspberry or blackberry plantings in those areas of North America where TRSV and its nematode vector are endemic.

##### **Symptoms on Natural and Experimental Hosts**

TRSV has a wide natural and experimental host range. The virus causes a ringspot disease of tobacco, cucumber, Easter lily, hydrangea, iris, and *Pelargonium*. It also causes blueberry necrotic ringspot (see "Necrotic Ringspot of Blueberry," p. 114), soybean bud blight, and chlorotic or necrotic spotting in many other annual and perennial crops (Stace-Smith 1970a).

Infected native *Rubus* spp. in North Carolina showed faint to severe ringspots, mottling and mosaic, yellow line patterns, leaf distortion, and stunting of infected foliage (fig. 284).

An unidentified blackberry cultivar growing in a backyard at Kamloops, British Columbia, exhibited rasp leaf symptoms similar to those induced on cherry by cherry rasp leaf virus.



Figure 284. — Symptoms in wild blackberry (*Rubus* sp.) associated with tobacco ringspot virus infection: A, Ringspots; B, mottling and leaf distortion. (Courtesy G. V. Gooding, Jr., North Carolina State University.)

(See "Cherry Rasp Leaf Virus in *Rubus*," p. 241.) Two infected plants were found, and most of the leaves in these plants showed some symptoms. The only virus that was isolated from these plants was TRSV which was assumed responsible for the rasp leaf symptoms (R. Stace-Smith, unpublished data).

TRSV has a wide experimental host range, but the following species have been used for assay, propagation, or diagnostic purposes:

*Cucumis sativus* L. Chlorotic local lesions, systemic mottling, or dwarfing, severe apical distortion.

*Nicotiana tabacum* L., *N. glutinosa* L., and *N. clevelandii* Gray. Necrotic local lesions that frequently develop into rings or ringspots; systemic ring or line-patterns; leaves produced later show no symptoms.

*Chenopodium amaranticolor* Coste and Reyn. and *C. quinoa* Willd. Local necrotic dots; usually no systemic reaction.

*Vigna unguiculata* L. Walp. Necrotic local lesions, systemic necrosis, apical necrosis, and wilt.

*Phaseolus vulgaris* L. Necrotic spots on inoculated leaves; systemically infected leaves show spots and rings, and the growing tip becomes necrotic.

#### Natural and Experimental Transmission

The virus was isolated from 17 plant species indigenous to North Carolina, including 4 *Rubus* spp. All naturally infected plants were collected from areas near fields containing TRSV infected tobacco plants. It is assumed that weed hosts such as *Rubus* spp. serve as reservoirs of the virus and as acquisition sources for vectors. Mites (*Tetranychus* sp.) have been reported to be inefficient vectors (Thomas 1969), but the American dagger nematode *Xiphinema americanum* Cobb was reported as an efficient vector (McGuire 1964), and it is assumed that most field spread is attributable to this nematode. The taxonomy of the complex species, *X. americanum*, is in question and the true species is thought to be limited in its distribution to the eastern part of the U.S.A. and Canada (Lamberti 1980).

Experimental transmission can be achieved by screening American dagger nematodes from soil collected from the root area of infected plants, adding the nematodes to cucumber seedlings, and, after a few weeks, assaying the cucumber roots by mechanical inoculation or serology. However, this technique is cumbersome and would not be used in routine assay. Sap transmission can be achieved by triturating a small piece of leaf tissue from infected *Rubus* spp. with buffer (0.01 M phosphate buffer, pH 7.5 containing 2% nicotine) and inoculating to cucumber or any of several other herbaceous test plants.

#### Properties of the Causal Agent

TRSV is a member of the nepovirus group (Harrison and Murrant 1977b). It has three types of isometric particles sedimenting at 51, 91, and 126S and containing 0, 35, and 43% RNA, respectively. The two RNA species, mol. wt. 1.4

$\times 10^6$  and  $2.4 \times 10^6$ , are both required for infection. The 91 S particle contains one strand of the smaller RNA; the 126 S particle contains either one strand of the larger or two strands of the smaller RNA. Natural transmission is primarily by means of the nematode vector *X. americanum* or, in some species, by seed (Stace-Smith 1970a).

#### Detection and Identification

The virus can be detected by mechanical inoculation from infected *Rubus* spp. to one of many herbaceous test plants (see "Symptoms on Natural and Experimental Hosts," p. 227); however, since the geographical distribution, natural host range, and vector relationships of the virus are similar to those of tomato ringspot virus (see "Tomato Ringspot Virus in *Rubus*," p. 223) serological tests are essential for positive identification. Evidence to date suggests that most isolates that may be found in *Rubus* are serologically indistinguishable from the "common strains" of TRSV (Rush and Gooding 1970; Stace-Smith and Hansen 1974) based upon the absence of spur formation in agar gel diffusion tests. Although the ELISA technique has not been used for detection of TRSV in *Rubus*, the technique would undoubtedly be effective if extensive indexing or mass screening were being considered.

#### Control Procedures

No problem requiring control procedures has been detected to date. A problem would only arise if plantings were made in soils having a history of TRSV in cultivated crops such as tobacco. Since *Rubus* spp. are not grown to any extent in those regions where TRSV is prevalent, it is unlikely that this virus will become a problem in cultivated raspberry or blackberry. No information is available on the therapy of TRSV-infected *Rubus* plants.



### Raspberry Bushy Dwarf<sub>11</sub>

By A. F. M<sub>1</sub>urant

#### Additional Common Names

Loganberry degeneration virus (Legg 1960); raspberry yellows virus (Cadman 1952c); probably raspberry line-pattern virus (Basak 1971).

#### History and Geographic Distribution

A widespread decline disease of the red raspberry cv. 'Lloyd George' in Great Britain was called "bushy dwarf" by Cadman and Harris (1951) and "symptomless decline" by Cadman (1952c). A sap-transmissible virus consistently associated with this disease was called raspberry bushy dwarf virus (RBDV) by Cadman (1961b) and reported (Cadman 1963) to be serologically related to apple chlorotic leaf spot virus, which has flexuous filamentous particles. Barnett and M<sub>1</sub>urant (1970), however, disproved this relationship and showed that RBDV has quasi-isometric particles about 33 nm in diameter. Cadman (1961b) found no evidence for transmission of RBDV by aphids or through soils, although it appeared to spread rapidly in the field. Later, he reported that RBDV is transmitted through seed and pollen and infects the pollinated plant (Cadman 1965); this was confirmed by M<sub>1</sub>urant et al. (1974) who concluded that transmission in pollen is probably the only means by which RBDV spreads in the field.

Confusingly, red raspberry plants infected with RBDV alone, either by manual inoculation (Barnett and M<sub>1</sub>urant 1970) or by pollination (M<sub>1</sub>urant et al. 1974), do not show bushy dwarf disease. Jones (1979b) showed that this disease is probably caused by mixed infection with RBDV and black raspberry necrosis virus (BRNV) or even by infection with BRNV alone. (See "Black Raspberry Necrosis Virus," p. 178.) As a further complication, recent evidence (Jones et al. 1982) shows that RBDV is, under some conditions, associated with the raspberry yellows disease of Cadman (1952c) and is therefore synonymous with raspberry yellows virus; however, other yellowing diseases of raspberry may have other causes. (See "Blackberry Calico," p. 245.)

RBDV also has other effects. It induces drupelet abortion ("crumbly fruit") (M<sub>1</sub>urant et al. 1974; Dauben<sub>1</sub>y et al. 1978), a condition that can also be induced by other viruses and other factors. Jones and M<sub>1</sub>urant (1972) noted a similarity between RBDV and raspberry line-pattern virus reported from Poland (Basak 1971), but no serological studies on the relationship between these viruses have been reported. Dauben<sub>1</sub>y et al. (1978), however, found an association between RBDV infection and presence of chlorotic line-pattern or interveinal chlorosis in the leaves of some red raspberry cultivars in North America. In addition, Barnett and M<sub>1</sub>urant (1970)

showed that RBDV is serologically identical to loganberry degeneration virus, which is associated with loganberry degeneration disease (Legg 1960; Ormerod 1970; 1972b).

RBDV probably occurs throughout the world wherever susceptible cultivars are grown. In red raspberry and 'Logan' ('Loganberry'), it is reported from western Europe (Cadman 1961b; Barnett and M<sub>1</sub>urant 1970; Desvignes and Savio 1975; Converse and Casper 1977), North America (Converse 1973; Dauben<sub>1</sub>y et al. 1978); New Zealand (Fry and Wood 1978), Australia (Guy et al. 1982), U.S.S.R. (M. A. Keldysh, personal communication; Knight and Barbara 1981; Jones et al. 1982), South Africa (Kooyman et al. 1982), and Chile (Auger and Converse 1982). A strain of the virus also occurs in black raspberry in the United States (Converse 1973; M<sub>1</sub>urant and Jones 1976).

#### Economic Importance

In Great Britain, RBDV rarely occurs alone in raspberry plants in the field. It is therefore important mainly as a component of mixed infections with viruses, most of them aphid-borne. It is undoubtedly a major factor in the virus-induced decline of the red raspberry cv. 'Lloyd George' after its heyday in the 1930s.

In pot experiments Jones (1979b) found that cane height, cane diameter, and fruit size of 'Lloyd George' raspberry were decreased significantly by infection with RBDV but especially by joint infection with RBDV and BRNV, which induced symptoms resembling the classical bushy dwarf disease. Most red raspberry cultivars now grown commercially in Great Britain (notably 'Malling Jewel', 'Glen Clova', and 'Malling Admiral') do not become infected with RBDV, so that the virus is at present of little economic importance there; however, it could become important with the planting of new, susceptible cultivars (for example, 'Malling Leo' and 'Glen Prosen') or if a newly detected resistance-breaking strain (Knight and Barbara 1981, D. J. Barbara and A. T. Jones, unpublished data) becomes prevalent. Its effects on fruit quality (crumbly fruit) could be of particular importance; however, not all susceptible cultivars are as sensitive as 'Lloyd George' to mixed infection with RBDV and BRNV.

In North America and New Zealand, bushy dwarf disease is not reported, although RBDV occurs in many cultivars of red raspberry, including 'Lloyd George' (Converse 1973; Dauben<sub>1</sub>y et al. 1978; Jones and Wood 1979). Perhaps this is because the RBDV-infected plants do not also contain BRNV. The two main vectors of BRNV, *Amphorophora agathonica* Hottes and *A. idaei* Börner (also known as *A. rubi* (Kalt.)), are not found in New Zealand, and the cv. 'Lloyd George' is not colonized by *A. agathonica*, the only one of these aphids that occurs in North America. In New Zealand, yellows disease caused by RBDV is prevalent in most years and, in the main cv. 'Marcy', is accompanied by crumbly fruit (Wood and Todd 1976; Fry and Wood 1978;



Jones and Wood 1979); it is regarded as a serious disease, although there are no estimates of yield loss (G. A. Wood, personal communication).

In Western North America, the prevailing cv. 'Willamette' is immune, but newer cultivars (for example, 'Meeker' and 'Skeena') are susceptible (Daubeney et al. 1978, Daubeney 1982). Daubeney et al. (1978) found that RBDV caused significant decreases in yield and in percentage drupelet set in a breeder's selection although it did not significantly affect cane height or cane diameter.

In a more recent experiment (Daubeney et al. 1982), significant reductions in cane height and diameter, as well as in fruit yield, were observed in the cvs. 'Canby', 'Lloyd George', and 'Meeker'; 'Creston' appeared relatively tolerant. In black raspberry, in contrast to red raspberry, RBDV caused a nonsignificant increase in fruit yield although it significantly impaired vegetative growth (Converse 1973).

### Symptoms on Natural Hosts

In nature, RBDV has been found only in species of *Rubus*: Subgenus *Idaeobatus* (raspberries)

Natural hosts reported are *Rubus idaeus* L. (red raspberry), *R. occidentalis* L. (black raspberry), *R. phoenicolasius* Maxim. (wineberry), *R. sachalinensis* Léveillé, and *R. vulgatus* ssp. *buschii* Roz.

Subgenus *Eubatus* (blackberries)

Not found occurring naturally in any species in this subgenus except in *Rubus macropetalus* Dougl. and also in five blackberry-raspberry hybrids: 'Boysen', 'Cascade', 'Logan', 'Marion', 'Merton', and 'Olallie'.

**Symptoms on red raspberry.** Table 12 lists a selection of modern cultivars that are susceptible to RBDV, together with some that have never been found infected in the field. A complete list of published records is given by Jones et al. (1982). When infected with RBDV alone, either naturally or experimentally (by manual inoculation, grafting or pollination), plants of many susceptible cultivars are symptomless. Under some circumstances, however, symptoms may be expressed, as follows:

1. In the cv. 'Lloyd George,' RBDV usually causes no symptoms on its own in Great Britain (Barnett and Murant 1970; Murant et al. 1974), but Jones (1979b) showed that in mixed infection with BRNV it causes "bushy dwarf" disease (Cadman and Harris 1951). Canes of affected plants are shorter than normal, are prone to autumn fruiting, and produce leaves that are downcurled and greasy-looking (fig. 285). Young canes are slow to appear in spring and the shoots are chlorotic and red-tinged.

The "bushy dwarf" syndrome is difficult to recognize unless uninfected "control" plants are available for comparison. A mild form of the disease may be caused by BRNV on its own



Figure 285. — Plant of 'Lloyd George' red raspberry showing bushy dwarf disease. (Copyright Scottish Crop Research Institute.)

(Jones 1979b). No other red raspberry cultivar is reported to show this symptom, although many are susceptible to infection with both RBDV and BRNV. In North America, RBDV was found to cause stunting and reduction in yield of 'Canby', 'Lloyd George', and, especially, 'Meeker' (Daubeney et al. 1982).

2. Infected plants of at least some cultivars may show "crumbly fruit" symptoms due to drupelet abortion (Murant et al. 1974; Wood and Todd 1976; Daubeney et al. 1978). Expression of this symptom seems to depend on environmental factors because not all infected plants produce crumbly fruit, and those that do so in one season may not do so in another.

3. Infected plants of cultivars given in italics in table 12 may develop "yellows" (Cadman 1952c). The etiology of this disease has long remained obscure, but recent evidence summarized by Jones et al. (1982) shows that it is associated with infection by RBDV. In the field, symptoms occur in late spring in the lower leaves as a vivid chlorosis of the veins; the chlorosis later extends into the leaf lamina (vein-banding) and broadens until the whole interveinal area is chlorotic (fig. 286). The whole or part of a leaf may be affected.

In some cultivars, particularly in Canada and New Zealand, the chlorosis may take the form of line-patterns or interveinal chlorosis (Daubeney et al. 1978; Jones and Wood 1979). Symptoms may gradually progress to affect leaves throughout the plant but tend to become less prominent in midsummer. Most leaves produced late in the season show no symptoms. Expression of symptoms seems to depend on environmental factors because plants of even the most sensitive cultivars may show symptoms in some years but not in others. The disease is now uncommon in Great Britain but seems prevalent in New Zealand (Jones and Wood 1979). RBDV may not be associated with all types of yellowing in *Rubus*. For example, neither the "calico" disease of the cv. 'Puyallup' (Johnson 1972), the symptoms of which closely

**Table 12.—Susceptibility of some red raspberry and black raspberry cultivars to RBDV<sup>1</sup>**

Red raspberry:					
‘Canby’	+	‘Malling Admiral’	–†	‘Nootka’	(–)†
‘Chilcotin’	(–)†	‘Malling Delight’	–	‘Norfolk Giant’	+
‘Creston’	+	‘Malling Exploit’	–	‘Red Antwerp’	+
‘Glen Clova’	–†	‘Malling Jewel’	–†	‘Rode Radboud’	+
‘Glen Isla’	–†	‘Malling Joy’	–†	‘Schönemann’	(–)†
‘Glen Moy’	–	‘Malling Landmark’	+	‘September’	(–)
‘Glen Prosen’	+	‘Malling Orion’	–†	‘Skeena’	+
‘Great American’	+	‘Malling Promise’	–†	‘Taylor’	+
‘Haida’	–	‘Marcy’	+	‘Veten’	+
‘Leo’	+	‘Matsqui’	(–)	‘Willamette’	(–)
‘Lloyd George’	+	‘Meeker’	+	‘Zeva Herbsternte’	(–)†
Black raspberry:					
‘Cumberland’	(–)	‘Bristol’	(–)	‘New Logan’	+
‘Black Hawk’	(–)	‘Munger’	+	‘Plum Farmer’	+

<sup>1</sup>A more complete list of published records is given by Jones et al. (1982).

+ = susceptible; cultivars given in italics are those in which yellows symptoms have been recorded.

(-) = cultivars not found infected in the field.

- = cultivars not infected in the field or by graft inoculation with a Scottish isolate.

† = cultivars recently found to be infected by graft inoculation with a Russian isolate (Knight and Barbara 1981 and unpublished data). These workers also found 'Malling Delight' and 'Zeva Herbsternte' infected in the field in southern England. These results are contrary to previous experience in Canada, New Zealand, and Great Britain. Recent tests (D. J. Barbara and A. T. Jones, unpublished data) indicate that the Russian isolate is a resistance-breaking strain.

resemble yellows disease, nor the calico diseases of 'Chehalem', 'Logan', and 'Marion' blackberry (Converse and Kowalczyk 1980) seem to be associated with RBDV infection.

**Symptoms on black raspberry.** Table 12 gives details of cultivar susceptibility; field-infected plants of cvs. 'Munger', 'New Logan', and 'Plum Farmer', and graft-inoculated plants of cv. 'Munger' were symptomless (Converse 1973). Seedlings infected through seed show no symptoms.

**Symptoms on wineberry (*R. phoenicolasius*).** No symptoms were observed in a field-infected plant, but faint transient line-patterns were observed in grafted plants soon after inoculation (Jones 1977).

**Symptoms on *R. sachalinensis* and *R. vulgatus* ssp. *buschii*.** Most plants were symptomless, but some showed "yellows" (Jones et al. 1982).

**Symptoms on 'Marion' berry.** Plants showing "yellows" in New Zealand contained RBDV (Jones et al. 1982).

**Symptoms on 'Boysen' berry.** Field-infected plants were symptomless (Converse 1973).

**Symptoms on 'Logan' berry.** Affected plants were symptomless but "weak" and gave decreased cane weights and fruit yields (Legg 1960).

**Symptoms on 'Merton' berry.** Affected plants showed chlorotic mottle (Jones et al. 1982).

**Symptoms on experimental hosts.** Barnett and Murant (1970, 1971) obtained experimental infection in 55 species in 12 dicotyledonous families. Experimental hosts include: *Fragaria vesca* L. (Barnett and Murant 1970); *Rubus bartoni* Newton cv. 'Ashton Cross' (Jones et al. 1982); *R. henryi* Hemsl. and Kuntze (Barnett and Murant 1970); *R. laciniatus* Willd. (Jones 1977); *R. molaccanus* L., *R. parviflorus* Nutt., and *R. parvifolius* L. (Jones et al. 1982); *R. procerus* P. J. Muell. cv. 'Himalaya Giant' (Jones 1977); *Cydonia oblonga* Mill. cv. 'C7/1' (Desvignes and Savio 1975; Jones et al. 1982); *Chenopodium amaranticolor* Coste and Reyn. (Cadman 1961b); *C. murale* L. (Barnett and Murant 1970);

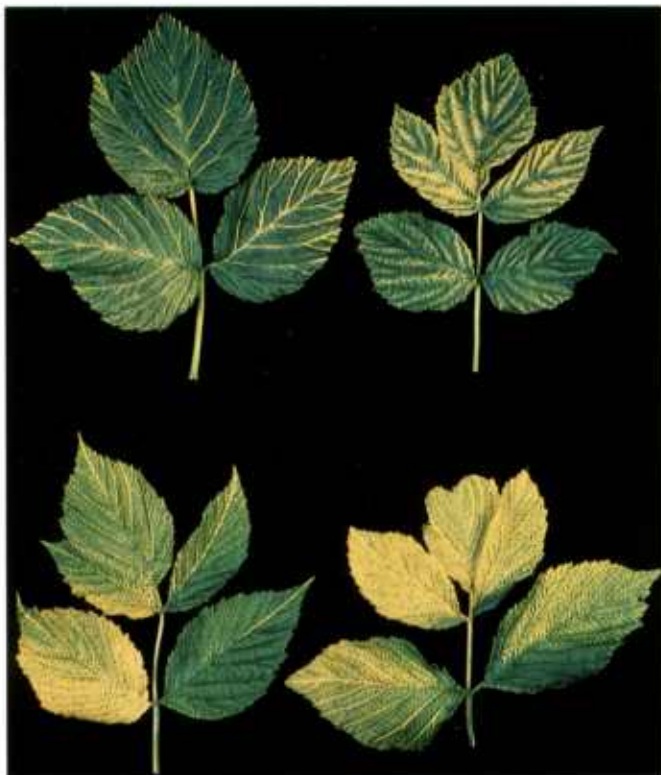


Figure 286. — Leaves of raspberry bushy dwarf virus-infected 'Norfolk Giant' red raspberry showing (upper left to lower right) progressive stages in development of yellows symptoms. (After Jones et al. 1982; copyright Scottish Crop Research Institute.)

*C. quinoa* Willd. (Cadman 1961b); *Cucumis sativus* L. (Cadman 1961b); *Nicotiana clevelandii* Gray (Barnett and Murant 1970); and *Phaseolus vulgaris* L. cv. 'The Prince' (Barnett and Murant 1970).

**Symptoms on indicator hosts.** (a) Graft-inoculated plants *Rubus henryi*: transient chlorotic mottle, easily overlooked; this distinguishes RBDV from BRNV, raspberry leaf spot, and raspberry leaf mottle viruses, which induce severe tip necrosis in this indicator. (See "Black Raspberry Necrosis," p. 178, and "Raspberry Leaf Mottle and Raspberry Leaf Spot," p. 183).

*R. molaccanus*: prominent chlorotic line-patterns, interveinal yellowing (fig. 287).

*Cydonia oblonga* cv. 'C7-1': yellow line-patterns; leaves produced later display prominent yellow vein-banding (fig. 288) or are entirely yellow.

(b) Manually inoculated plants

*Chenopodium amaranticolor*: transient chlorotic local lesions may appear in 4 days, especially in spring and autumn; systemic chlorotic rings and line-patterns develop after 7 days (fig. 289).

*C. murale*: inoculated leaves show sunken necrotic rings; no systemic infection.

*C. quinoa*: transient chlorotic local lesions may appear in 4 to 7 days, especially in spring and autumn; systemic chlorotic



Figure 287. — Prominent interveinal yellowing induced by raspberry bushy dwarf virus in *Rubus molaccanus*. (After Jones et al. 1982; copyright Scottish Crop Research Institute.)



Figure 288. — Raspberry bushy dwarf virus-infected leaves of *Cydonia oblonga* cv. 'C7-1', showing prominent yellow vein-banding. (After Jones et al. 1982; copyright Scottish Crop Research Institute.)



Figure 289. — Chlorotic rings and line-patterns induced by raspberry bushy dwarf virus in systemically infected leaf of *Chenopodium amaranticolor*. (After Barnett and Murant 1970; copyright Scottish Crop Research Institute.)

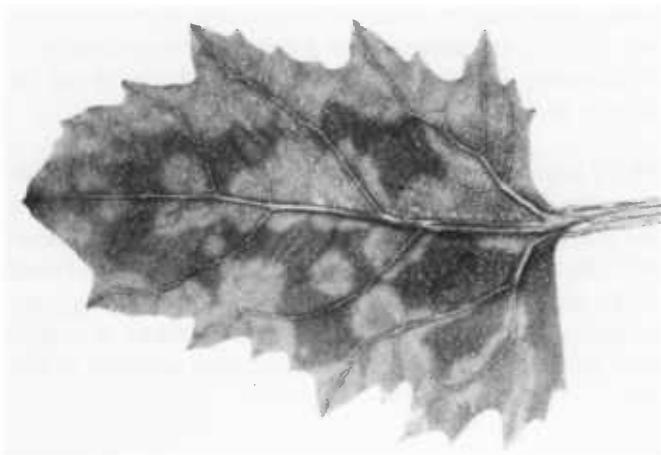


Figure 290. — Chlorotic rings and line-patterns induced by raspberry bushy dwarf virus in systemically infected leaf of *C. quinoa*. (After Barnett and Murant 1970; copyright Scottish Crop Research Institute.)

spots develop after 7 days followed by light- and dark-green mosaic and ring patterns (fig. 290). This plant is useful for propagating the virus.

*Cucumis sativus*: transient chlorotic lesions in inoculated leaves; transient systemic mottling.

*Nicotiana clevelandii*: symptomless systemic infection.

*Phaseolus vulgaris* cv. 'The Prince': In winter, minute brown local lesions (fig. 291) develop after 3 days; no systemic infection. This plant is useful for quantitative assays in winter or when grown in controlled environment chambers (20°C, 5000 lux, 16-h photoperiod). Lesion numbers are increased by keeping the plants in darkness for 1 day before inoculation and by using phosphate buffer in the inoculum. Lesion development is inhibited by a component of *C. quinoa* sap, which occurs in increased concentration in plants grown in long days and high light intensities (Barnett and Murant 1970).

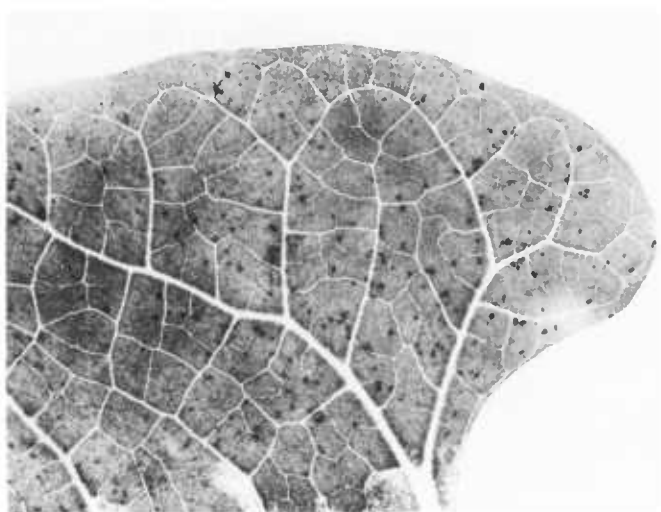


Figure 291. — Minute local lesions induced by raspberry bushy dwarf virus in leaf of *Phaseolus vulgaris* cv. 'The Prince'. (After Barnett and Murant 1970; copyright Scottish Crop Research Institute.)

### Natural and Experimental Transmission

Experimentally, RBDV is transmissible by grafting and by mechanical inoculation. Sap from raspberry is more infective in spring and autumn than in summer, and inocula are best prepared at pH 8 with 2% nicotine or 1% polyethylene glycol (mol. wt. 6000). *Chenopodium quinoa* is the most sensitive test plant. RBDV is detectable in leaves, flower parts, pollen, and seed of infected raspberry.

RBDV is transmitted through raspberry seed (Cadman 1965; Converse 1973; Murant et al. 1974). The virus may enter seed via either gamete, although less readily via pollen, and transmission was greatest (77%) when both parents were infected (Murant et al. 1974); only 1 to 2% seed transmission was found in *Fragaria vesca* and none in *Chenopodium quinoa*. Jones (1977) found 15% seed transmission in *Rubus phoenicolasius*. Pollination of healthy or infected raspberry flowers with infected pollen may result in the production of crumbly fruit (Murant et al. 1974).

In raspberry and 'Logan', the virus carried in pollen infects not only the progeny seedlings but also the pollinated plant (Cadman 1965; Ormerod 1970). Murant et al. (1974) found that the virus did not spread to plants that were prevented from flowering for 3 yr and concluded that transmission in pollen is probably the only means of spread in the field; most plants that were near to RBDV sources became infected in the first two or three flowering seasons.

### Properties of the Causal Agent

For a detailed description of the virus, see Murant (1976b). In *Chenopodium quinoa* sap, typical isolates from *Rubus idaeus* lose infectivity when diluted  $10^{-4}$ , heated for 10 min at 65°C, or stored for 4 days at 22°C (Barnett and Murant 1970). An isolate from *R. occidentalis* lost infectivity when diluted  $10^{-2}$  or after storage for 2 to 3 hr at room temperature (Murant and Jones 1976). Isolates of both types were still infective in lyophilized leaf tissue after 6 yr (A. F. Murant, unpublished data). The virus is purified (Barnett and Murant 1970; Murant 1976b) by acidifying *C. quinoa* extracts to pH 4.8 and concentrating the virus from pellet or supernatant fractions by precipitation from 8% polyethylene glycol, (mol. wt. 6000) + 0.8% NaCl, and differential centrifugation.

In the electron microscope, the virus particles are stable in uranyl acetate or uranyl formate negative stain but disrupt in phosphotungstate; they are isometric, about 33 nm in diameter (fig. 292), but appear somewhat pleiomorphic because they partially collapse on the grid. They form a single sedimenting component with a sedimentation coefficient ( $s_{20,w}$ ) of 115 S in 0.05 M citrate buffer, pH 6 or 7. Fractions from this zone have  $A_{260}/A_{280}$  of 1.62. The particles contain a single major protein of mol. wt. about 29,000 daltons and three species of single-stranded RNA with mol. wt. of 2.2, 0.9, and  $0.4 \times 10^6$ .



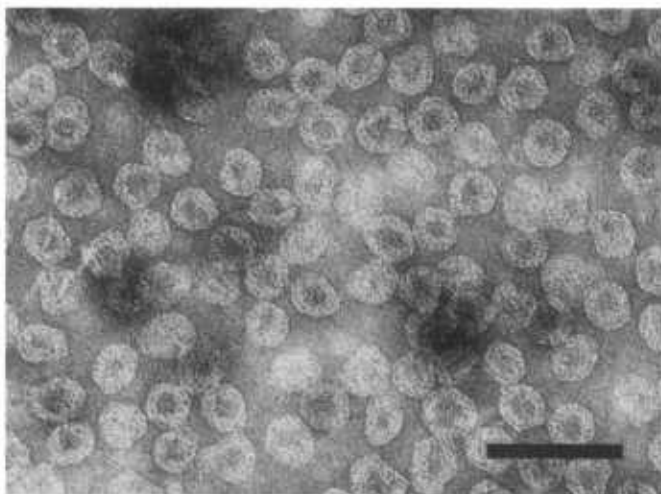


Figure 292. — Particles of raspberry bushy dwarf virus in uranyl formate. Bar represents 100 nm. (After Barnett and Murant 1970; copyright Scottish Crop Research Institute.)

All tested isolates from red raspberry (from Great Britain, France, Canada, New Zealand, and U.S.S.R.) were serologically indistinguishable (A. F. Murant, unpublished data), but two isolates from *R. occidentalis* differed slightly from *R. idaeus* isolates in gel double-diffusion serological tests as well as in properties in vitro (Murant and Jones 1976). It was suggested that species-specific strains could have arisen as a result of transmission exclusively through pollination.

The particle morphology of RBDV and its transmission through pollination suggest that it may have affinities with ilarviruses, but it differs from them in the number and size of its RNA molecules and in the sedimentation behavior of its particles.

#### Detection and Identification

Although under some conditions RBDV may be associated with symptoms of decline, yellowing, line-patterns, or crumbly fruit, it more often infects plants without inducing obvious symptoms. Presence of the virus is detected by one or more of the following methods:

1. Manual inoculation to *Chenopodium quinoa*. Identity of the virus is confirmed serologically by double diffusion tests in agarose gels.
2. Grafting to *Rubus molaccanus* and/or *Cydonia oblonga* cv. 'C7-1'.
3. Enzyme-linked immunosorbent assay (ELISA).

#### Control Procedures

The only method of controlling RBDV is by planting resistant cultivars (table 12). In red raspberry, a single dominant gene confers resistance or immunity to the Scottish type strain of RBDV (Jones et al. 1982), but Knight and Barbara (1981) found a more complex situation in tests with a Russian isolate. They suggested that resistance is controlled by dominant complementary genes. In susceptible cultivars,

some control may be achieved by planting virus-free stocks and siting fruiting plantations at a distance from infected wild or cultivated *Rubus*; plants in cane nurseries must not be allowed to flower.

RBDV is somewhat resistant to thermotherapy but it has been eliminated from red raspberry (Murant et al. 1974; Mellor and Stace-Smith 1976) and from black raspberry (Converse 1973) by keeping infected plants at or above 36°C for several weeks and propagating from the shoot tips that appears subsequently. 'Canby' red raspberry, all stocks of which were infected with RBDV, was freed from infection in this way.

#### Remarks

The characteristic symptoms in *Chenopodium quinoa* and symptomless infection of *Nicotiana clevelandii* distinguish RBDV from most nepoviruses found in *Rubus* (see "Nematode-Borne Viruses" in this section, p. 204–228), except perhaps strawberry latent ringspot virus; the nepoviruses also differ from RBDV in particle morphology (regular isometric particles with hexagonal outlines, some penetrated by negative stain). The type of symptom in *C. quinoa* also distinguishes RBDV from black raspberry latent virus (see "Tobacco Streak Virus in *Rubus*," p. 235), which is more difficult to transmit by manual inoculation and has regular isometric particles. Black raspberry latent virus (see "Tobacco Streak Virus in *Rubus*," p. 235) resembles RBDV in particle morphology and transmission through pollination but causes severe systemic necrosis in *C. quinoa* and *Phaseolus vulgaris*.

The economic importance of RBDV is difficult to assess, but some loss in yield and fruit quality is certainly caused. RBDV assumes greater importance in mixed infections with other viruses. Because the planting of resistant cultivars is the only method of control, breeders should, if possible, not release new cultivars that are susceptible to RBDV.

## Vectors Unknown

### Tobacco Streak Virus in *Rubus*

By R. Stace-Smith

#### Additional Common Names

Strawberry necrotic shock virus (Frazier et al. 1962); black raspberry latent virus (Converse and Lister 1969).

#### History and Geographic Distribution

The history of our understanding of the occurrence of tobacco streak virus (TSV) in *Rubus* spp. is difficult to trace with any degree of certainty. The reason for this is that at the time the work was done and reported there was no indication that the virus involved was either distantly or closely related to TSV. The first virus involved, strawberry necrotic shock virus (SNSV), had not even been transmitted mechanically from strawberry to herbaceous hosts at the time it was shown to occur in *Rubus* spp. (Frazier 1966). Later, a virus isolated from strawberry plants infected with strawberry necrotic shock was identified as TSV (Stace-Smith and Frazier 1971). In this review, therefore, I am equating SNSV with TSV.

The second virus that features in the history of TSV in *Rubus* spp. is black raspberry latent virus (BRLV), a virus isolated from clones of black raspberry and red raspberry in Eastern United States (Converse and Lister 1969). At the point this work was reported, the authors had not succeeded in producing an antiserum and, since there were no characteristic properties that suggested that it might be a strain of TSV, they concluded that it was a new virus. Later, when an antiserum was available against BRLV, a serological relationship to some strains of TSV was demonstrated (Jones and Mayo 1975; Brunt and Stace-Smith 1976). Therefore I am equating BRLV with TSV.

The first indication of the natural occurrence of TSV in *Rubus* spp. was obtained when Frazier (1966) grafted leaves from 'Boysen' and 'Logan' plants into strawberry, producing necrotic shock symptoms. Several plants of 'Olallie', a trailing blackberry cultivar, 'Himalaya' blackberry (*R. procerus* P. J. Meull), and the Pacific coast trailing blackberry (*R. ursinus* Cham. and Schlecht.) indexed negative. Converse and Lister (1969) found the virus to be widely distributed in most cultivars of black raspberry in Eastern United States but rare in red raspberry. Converse (1972) found a strain of tobacco streak virus to be prevalent in black raspberry cultivars in Oregon and Washington. In all instances, affected plants were symptomless. Stace-Smith and Brunt (1974) and Converse and Bartlett (1979) found the virus to be widespread in wild Pacific coast trailing blackberry (*R. ursinus*). The virus was equally prevalent in agricultural and nonagricultural areas, indicating a long association between the virus and its host.

TSV is prevalent in black raspberry in both Eastern and Western United States (Converse and Lister 1969; Converse 1972). The virus has been detected in some blackberry cultivars in California (Frazier 1966) and has been isolated from a number of blackberry cultivars originating in the United States and imported into Scotland (Jones and Mayo 1975), Canada (Brunt and Stace-Smith 1976), Australia (Guy et al. 1982), and possibly Yugoslavia (Perišić and Babović 1978). It has also been isolated from a blackberry selection originating in Australia and imported into New Zealand (Jones and Wood 1979). The virus rarely infects red raspberry, and the reports of its occurrence are confined to the United States and Canada (Converse and Lister 1969; Peterson and Corbett 1980; Stace-Smith et al. 1982).

The occurrence of the virus in wild *Rubus* appears to be confined to a single species, *R. ursinus* (Stace-Smith and Brunt 1974; Converse and Bartlett 1979). This species occurs in a narrow zone along the Pacific coast, extending from California to British Columbia. The high level of TSV infection encountered in virus surveys done in British Columbia and Oregon suggests that the virus is generally prevalent in *R. ursinus* throughout its geographical range.

Although the natural geographical distribution of TSV in *Rubus* appears to be restricted to United States, Canada, and Australia, the virus has been recovered from other plants in many areas of the world (Fulton 1971).

#### Economic Importance

No information is available on the economic importance of TSV in *Rubus* spp. The fact that the virus appears to be symptomless in several black raspberry, red raspberry, and blackberry cultivars suggests that the damage is negligible. Some infected red raspberry plants were slower to break dormancy in the spring than virus-free plants (Jones and Mayo 1975), and infected 'Santiam' blackberry plants produced significantly fewer primocanes than virus-free plants (Converse 1978). These two observations provide evidence that the virus is not completely symptomless in *Rubus* hosts, and it must be recognized that, even though TSV is mild or symptomless in commercial cultivars, it may contribute to a decline in productivity of field plantings, alone or acting synergistically with other viruses (Fulton 1981).

#### Symptoms on Natural and Experimental Hosts

The natural host range of TSV is very wide, including monocotyledonous and dicotyledonous hosts and both woody and herbaceous hosts. The symptoms induced in these hosts are so variable as to be of little diagnostic value. The virus is known to cause necrotic symptoms in tobacco, pea, bean, and potato; mottling symptoms in cotton and dahlia; and ringspot symptoms in tomato. Most of the herbaceous hosts recover from the virus and produce foliage that is systemically infected but exhibits no symptoms of infection. In contrast, woody hosts generally exhibit no symptoms

when initially infected and remain symptomless following systemic invasion of the virus. Infections can therefore only be detected by graft or sap transmissions to a sensitive host or by serological indexing.

As would be expected, a virus with a wide natural host range also has a wide experimental host range. In one extensive test, Fulton (1948) inoculated 169 species with TSV and succeeded in recovering the virus from 87 of the inoculated species. Even this test underestimates the experimental host range of the virus since virus preparations are unstable. With improvements in techniques in inoculation and in stabilizing the virus, many of the species that did not become infected in earlier tests are known to be hosts of the virus (Fulton 1981).

Symptoms are as variable on the experimental hosts as they are on the natural hosts. For this reason, it is virtually impossible to identify the virus with any degree of certainty on the basis of symptoms induced on a variety of experimental hosts. Symptoms on *Nicotiana tabacum* L. (most cultivars) (fig. 293) include necrotic rings and, sometimes, solid necrotic spots on the inoculated leaves, which usually appear within 4 days of inoculation. Systemic symptoms appear on the younger leaves a few days later and consist of necrotic lines that follow the small leaf veins. Succeeding leaves are free of necrotic symptoms and appear healthy, although virus can be recovered from such leaves. Other commonly used virus indicator hosts, such as cucumber, bean, and *Chenopodium quinoa* Willd., develop necrotic or chlorotic local lesions followed by systemic necrosis or chlorosis. The variability in host reaction is extreme because there is a high degree of strain variation in TSV. None of the indicator hosts is entirely reliable in identifying all strains, although the symptom sequence in *N. tabacum* is helpful in at least providing a tentative diagnosis.

**Symptoms on *Rubus* hosts.** As a generalization, symptoms of TSV infections cannot be detected in field plantings of red raspberries, black raspberries, or blackberries, although there is some question in the literature with respect to the symptom response in infected blackberry clones. Frazier (1966) first detected TSV in degenerate-appearing 'Boysen' plants but, since he later detected the virus in vigorous plants with a strong chlorotic leaf pattern, he concluded that none of the symptoms on the 'Boysen' plants could be ascribed to the virus. Jones and Wood (1979) isolated TSV from 'Scoresby Selection' bramble, which showed chlorotic ringspot and line-pattern symptoms. These symptoms may have been induced by TSV, and the fact that several symptomless clones of 'Scoresby Selection' have indexed negative for TSV (Guy et al. 1982 and R. Stace-Smith, unpublished results) leaves this possibility open.

The virus is prevalent in blackberry cultivars in British Columbia, and some of the infected plants show strong mosaic symptoms, whereas others are symptomless. Similarly, all commercial 'Logan' plantings that have been indexed



Figure 293. — Local lesions in *Nicotiana tabacum* cv. 'Haranova' 5 days after inoculation with a *Rubus* isolate of tobacco streak virus.

in British Columbia have proven to be infected with TSV, yet none of these plants show viruslike symptoms (R. Stace-Smith, unpublished results). The conclusions from these observations are that TSV does not induce foliar symptoms in blackberry cultivars but that cultivars infected with TSV often contain other viruses that do induce foliar symptoms.

In contrast to the absence of symptoms in naturally infected commercial cultivars, some *Rubus* hosts develop a severe necrotic reaction following graft inoculation. Frazier (1966) reported that several seedling clones of 'Himalaya' blackberry, seedling clones of 'Logan', 'Ollalie', wineberry (*R. phoenicolasius* Maxim), and *R. henryi* Hemsl. and Kuntze react by becoming necrotic at the side of the graft union. The necrosis spreads basally or distally through one or more internodes, usually causing wilting and death of the grafted shoot. The virus did not become systemic in these plants. Jones and Mayo (1975), using a different isolate of TSV, observed the same necrotic reaction in *R. henryi*, *R. phoenicolasius* and *R. procerus* (fig. 294) as did Converse and Kowalczyk (1980) in *R. ursinus* cv. 'Marion'. Other *Rubus* hosts, such as black raspberry, red raspberry, 'Logan', and 'Boysen', are invaded systemically following graft inoculation but remain symptomless (Frazier 1966; Jones and Mayo 1975).

### Natural and Experimental Transmission

TSV, like many other members of the ilarvirus group (Matthews 1981), is known to be transmitted through the seed of many of its natural and experimental hosts (Mandahar 1981; Kaiser et al. 1982). The evidence is too sparse to speculate as to whether seed and pollen transmission are the major means of natural spread of the virus, but these mechanisms are undoubtedly important in the dissemination and survival of the virus. Occurrence of TSV is usually low and erratic, and this has led to speculation that insect vectors may be involved. In tobacco, infections are more prevalent near the periphery of the field, suggesting that the source of





Figure 294. — Shock reaction (necrosis below the graft) in *Rubus procerus* ('Himalaya' blackberry) graft-inoculated with a *Rubus* isolate of tobacco streak virus. (Copyright Scottish Crop Research Institute.)

the virus is infected perennial weed species and that transmission is by some insect vector. Although a number of attempts have been made to demonstrate insect transmission, the only vectors implicated to date are species of thrips. Evidence that thrips (*Frankliniella* sp.) may serve as a vector was first obtained in Brazil (Costa and da Costa Lima Neto 1976). More recently, two species of thrips [*Thrips tabaci* Lindeman and *Frankliniella occidentalis* (Pergande)] have been implicated as vectors in the United States (Kaiser et al. 1982).

There is considerable evidence for seed and pollen transmission in *Rubus* spp. Converse and Lister (1969) reported about 6% infection in seedlings arising from seed of infected black raspberry plants. They also transmitted the virus to healthy black raspberry plants by hand pollinating with infected pollen. Converse (1980) found that TSV spread more rapidly into black raspberry and 'Boysen' plantings that were permitted to flower as compared with those that were deflowered. He concluded that virus transmission could have taken place by flower-visiting vectors or by viruliferous pollen moved by wind or insects. An alternate explanation was proposed by Kaiser et al. (1982), namely that thrips

could have been responsible for TSV spread to both flowering and deflowered *Rubus* hosts.

Experimental transmission to or from *Rubus* hosts can be achieved either by grafting or by sap inoculation. Leaf or approach grafts were first used by Frazier (1966) to transmit the virus from 'Boysen' to *Fragaria* and *Rubus* indicator plants. Sap transmission from *Rubus* to *Rubus* has not been achieved, but Jones and Mayo (1975) succeeded in infecting black raspberry seedlings by inoculating them with purified virus preparations. The virus is readily transmitted from infected *Rubus* hosts to herbaceous hosts, provided appropriate buffers are used. Buffers that have been used include 2% nicotine (Jones and Mayo 1975), 2% nicotine plus aluminum oxide powder (Converse and Lister 1969), or 1% nicotine plus 1% polyvinylpyrrolidone (Brunt and Stace-Smith 1976).

### Properties of the Causal Agent

TSV has been designated as the type member of the ilarvirus group (Matthews 1981). The particles are quasi-isometric, averaging about 28 nm in diameter (fig. 295). Three particle types are normally separated upon sucrose density gradient centrifugation, with  $s_{20, w}$  values of approximately 90, 98, and 113 S. Differences in sedimentation rates are due to different size groups of the virus particles. Those sedimenting most rapidly averaging 35 nm in diameter, the middle group averages 30 nm, and the slowest group averages 27 nm. Particles are fragile and deform readily; hence fixation in glutaraldehyde or formaldehyde is recommended for good electron micrographs (Fulton 1981; Matthews 1981). All three components contain nucleoproteins with maximum absorption at 260 nm and minimum absorption at 242 nm. Unfractionated preparations have an  $A_{260}/A_{280}$  ratio of about 1.60. The particles contain a single protein species with an estimated mol. wt. of 28700. Particles contain four linear ssRNA with approximate mol. wt. of 1.1 (RNA-1), 0.9 (RNA-2),  $0.7 \times 10^6$  daltons (RNA-3), and  $0.3 \times 10^6$  daltons (RNA-4). Besides RNAs 1 to 3, coat protein or RNA-4 is required for infectivity. The physiochemical properties of the various strains that have been isolated from *Rubus* (Converse and Lister 1969; Converse 1972; Jones and Mayo 1975; Brunt and Stace-Smith 1976) are essentially the same as those of other strains of TSV (Fulton 1981).

### Detection and Identification

Since TSV induces no obvious symptoms in *Rubus*, infections cannot be detected by visual observation. Techniques such as graft transmission, sap transmission, or serology must be used. Frazier (1966) detected the virus in 'Boysen' by graft transmission to sensitive *Fragaria* and *Rubus* hosts. At the time that work was done, however, the virus was not known to be sap transmissible from *Rubus* hosts. Since sap transmission is a much simpler procedure, it has superseded graft transmission as a detection technique. The most widely used herbaceous indicator plant for sap transmission is *C. quinoa* (fig. 296), although other hosts



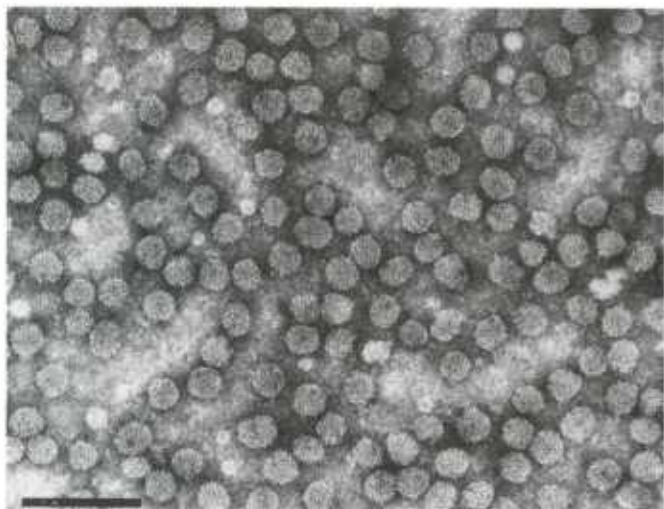


Figure 295. — Electron micrograph of a purified preparation of a *Rubus* isolate of tobacco streak virus, fixed in glutaraldehyde and negatively stained. Bar represents 100 nm.



Figure 296. — Local lesions in *Chenopodium quinoa* 6 days after inoculation with a *Rubus* isolate of tobacco streak virus.



Figure 297. — Local lesions in *Phaseolus vulgaris* (bean, cv. 'Black Turtle') 5 days after inoculation with a *Rubus* isolate of tobacco streak virus.



Figure 298. — Systemic symptoms in *Nicotiana clelandii* 12 days after inoculation with a *Rubus* isolate of tobacco streak virus.

such as cucumber, bean (fig. 297), and *Nicotiana clelandii* Gray (fig. 298) are equally effective (Brunt and Stace-Smith 1976). The most reliable plant source is expanding leaf tissue in the first growth from dormant plants; slow-growing plants from the greenhouse or the summer growth on field-grown plants are poor sources of inoculum (Converse and Lister 1969; Brunt and Stace-Smith 1976). The sap should be extracted in nicotine or polyvinylpyrrolidone to minimize inactivation by host polyphenols.

Although sap transmission is the most widely used technique for TSV detection, serological techniques are applicable, particularly for extensive field surveys; however, because of the diversity of serological relationships among TSV strains, failure of virus isolates to react with antisera to some other isolates need not indicate the absence of TSV. Tests should include antisera to several TSV isolates.

Converse (1976) successfully detected TSV in *Rubus* by the agar gel double diffusion technique. The most consistent results were obtained by grinding leaf tissue in a buffer of 0.1 M Tricine, pH 8, plus 4% polyethylene glycol. As noted by Converse (1976), TSV cannot be identified on the basis of symptoms produced on herbaceous hosts and, since isolates must be identified serologically, direct seroassay in the *Rubus* sap eliminates the need for the bioassay step. As with detection of some other sap-transmissible viruses in *Rubus*, the most useful serological technique may be the enzyme-linked immunosorbent assay (ELISA) technique (Converse 1979).

#### Control Procedures

Of the many viruses that are known to infect *Rubus* hosts, TSV appears to be one of the least significant in terms of economic importance. For this reason, it is questionable whether any special control measures are required other than the standard precaution of establishing all new plantings with

virus-tested stock. Fulton (1981) noted that the virus is of minor importance on most hosts in North America and no controls have been necessary. This general statement is probably true with respect to *Rubus* hosts, although it must be recognized that controlled experiments on the possible adverse effects associated with TSV infection, singly or in complex with other viruses, have not been done.

The virus is known to be more prevalent in some *Rubus* hosts than in others. The virus, for example, is rarely found in red raspberry. Provided care is taken to establish new red raspberry plantings with virus-tested clones, the virus will probably remain rare in red raspberry. In contrast, the virus is prevalent in black raspberry and some blackberry cultivars. The reason for the high incidence is not known but, until recently, plantings were probably established with infected stock. Possibly, virus incidence will remain low in new plantings that are established with healthy clones, although preliminary evidence (Converse 1980) is not encouraging.

Virus-tested planting stock is available for most *Rubus* cultivars so virus eradication procedures are rarely required; however, should it be necessary to eradicate TSV from any clone, the virus is amenable to eradication by heat therapy (Converse 1978).

## Wineberry Latent Virus

By A. T. Jones

### Additional Common Names

Initially code named Rp7 by Jones (1975).

### History and Geographic Distribution

Wineberry latent virus (WLV) was first reported by Jones (1974). He isolated it from a symptomless plant of *R. phoenicolasius* Maxim. originally imported from the United States but grown in the field at Invergowrie, Scotland, for at least 10 yr (Jones 1977). The geographical origin of the virus is therefore not known.

### Economic Importance

Probably none.

### Symptoms on Experimental Hosts

***Rubus* hosts.** The single *R. phoenicolasius* plant found infected with WLV was symptomless. This source plant also contained raspberry bushy dwarf virus (RBDV, see "Raspberry Bushy Dwarf," p. 229) but was free from other known *Rubus* viruses (Jones 1977). A culture of WLV free from RBDV was obtained in herbaceous test plants by passing through *Lycopersicon esculentum* Mill., which is immune to RBDV. However, as WLV was not transmissible to *Rubus* spp. by mechanical inoculation, these species were inoculated by grafting with tissue from the dually infected *R. phoenicolasius* field plant. In these graft inoculation tests, WLV infected the *R. idaeus* L. cvs. 'Lloyd George',

'Malling Landmark', 'Norfolk Giant', and 'St Walfried', and *R. loganobaccus* Bailey, *R. mollacanus* L., *R. occidentalis* L., *R. phoenicolasius*, and *R. procerus* P. J. Muell. None of the plants showed symptoms apart from line-pattern in the leaves of some grafted plants of *R. mollacanus*, *R. phoenicolasius*, and *R. procerus*; however, identical symptoms are produced in these species by RBDV alone, suggesting that these symptoms are not caused by WLV. *R. henryi* Hemsl. and Kuntze; *R. idaeus* cvs. 'Glen Clova', 'Malling Enterprise', and 'Malling Jewel'; and *R. laciniatus* Willd. appear to be resistant to infection with WLV by graft inoculation.

**Herbaceous hosts.** After mechanical inoculation, the following herbaceous species show symptoms: *Chenopodium album* L., *C. amaranticolor* Coste and Reyn., *C. foetidum* Schrad., *C. murale* L., *C. quinoa* Willd., and *Tetragonia expansa* Murr. develop small necrotic local lesions in 5 to 8 days, which enlarge to form large necrotic spots or rings (fig. 299). Inoculated leaves of *C. ambrosioides* L. and *Gomphrena globosa* L. develop red rings in about 7 days (fig 300). *Beta macrocarpa* L., *Lycopersicon esculentum*, *Spinacia oleracea* L., and *Catharanthus roseus* (L.) G. Don, also known as *Vinca rosea* L., are symptomlessly infected. The virus is systemic (but weakly so) only in a few *Chenopodium* species. *Datura stramonium* L., several *Nicotiana* spp., *Petunia hybrida* Vilm., and *Phaseolus vulgaris* L. were not infected with WLV (Jones 1977).



Figure 299. — Necrotic local lesions in a leaf of *Chenopodium amaranticolor* 20 days after inoculation with wineberry latent virus. (Copyright Scottish Crop Research Institute.)



Figure 300. — Local red rings caused by inoculation with wineberry latent virus in a leaf of *Gomphrena globosa*. (Copyright Scottish Crop Research Institute.)

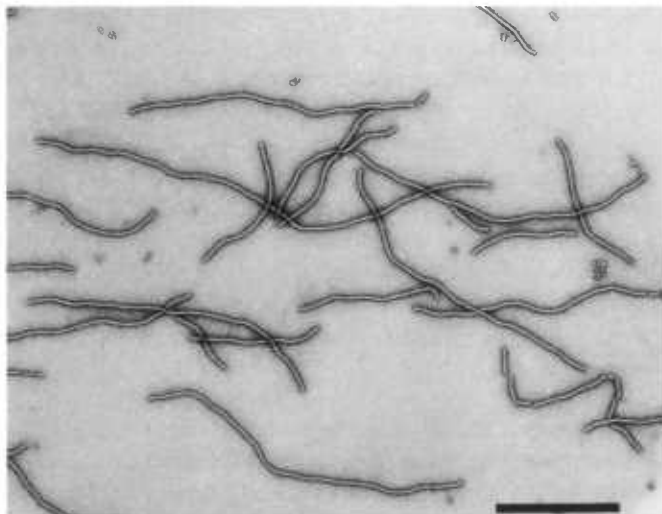


Figure 301. — Electron micrograph of a purified preparation of wineberry latent virus stained in 2% phosphotungstate, pH 6.5; it shows aggregated and fragmented particles. Bar represents 500 nm. (Copyright Scottish Crop Research Institute.)

### Natural and Experimental Transmission

The natural mode of transmission of WLV is not known. It is transmissible experimentally to *Rubus* species by grafting. It is also transmissible to several herbaceous species, but not to *Rubus*, by inoculation of sap. It is not seed transmitted in *R. phoenicolasius* (Jones 1977). *Macrosiphum euphorbiae* (Thos.) failed to transmit WLV to *C. quinoa* when given short or long acquisition feeds on WLV-infected *C. quinoa* (Jones 1975a and unpublished data). No spread from the single naturally infected plant of *R. phoenicolasius* to *R. phoenicolasius* or to other *Rubus* spp. appears to have occurred in Scotland.

### Properties of the Causal Agent

WLV has flexuous filamentous particles with a modal length in sap of *C. quinoa* of about  $510 \times 12$  nm (fig. 301). In *C. quinoa* sap, WLV lost infectivity after diluting to  $10^{-3}$  to  $10^{-4}$ , heating for 10 min at 65° to 70°C, or storing for 8 to 16 days at 18°C.

WLV has been only partially purified, and, of several purification methods studied, the following was found most useful (Jones 1977). Sap from WLV-infected *C. quinoa* was extracted in 0.05 M tris-HCl buffer (pH 7) containing 0.2% thioglycerol and 10% (v/v) chloroform, and the virus was precipitated from the aqueous phase with 7% polyethylene glycol (mol. wt. 6000) + 0.1 M NaCl. Virus recovered from the resuspended precipitate was then further concentrated and clarified by differential centrifugation and/or sucrose density gradient centrifugation. The virus yield and clarification achieved varied with the time of year and other undetermined factors, but particles purified in this way were always both aggregated end-to-end and fragmented (fig. 301 and Jones 1977). Preparations of particles recovered from sucrose density gradients had  $A_{260}/A_{280}$  values of about 1.26.

Although morphologically similar to potexviruses (Koenig and Lesemann 1978), WLV reaches lower concentrations in plants and is less stable in plant sap than most potexviruses. Furthermore, it is serologically unrelated to any of four potexviruses: potato virus X, hydrangea ringspot, narcissus mosaic, and white clover mosaic viruses (Jones 1977).

The virus also differs in properties from two other filamentous viruses reported to occur in *Rubus*, namely bean yellow mosaic (Provvidenti and Granett 1974) and bramble yellow mosaic. (See "Bramble Yellow Mosaic," p. 243.)

Although RBDV was once thought to be serologically related to apple chlorotic leaf spot virus (Cadman 1963), which has filamentous particles, it is now known that this is not true and that RBDV has isometric particles. (See "Raspberry Bushy Dwarf Virus," p. 229.) WLV differs from apple chlorotic leaf spot virus in several properties, and the two viruses are serologically unrelated (Jones 1977).

### Detection and Identification

WLV can be detected and identified in *Rubus* only by mechanical transmission to herbaceous test plants followed by serological tests. Transmissions from infected plants kept in a heated greenhouse are often less reliable than those from field-grown plants.

### Control Procedures

As the virus has been isolated from only a single plant, no attempts have been made to free plants from infection. However, in the absence of information on its distribution, mode of transmission, and effects in raspberry, it would seem prudent to destroy any exotic plants found infected in the field. The possibility of propagation from infected plants should be avoided by indexing raspberry mother plants for virus infection by inoculating leaf extracts to *C. quinoa*.

### Remarks

Further studies on the virus particle may show affinities with existing viruses or virus groups and thus provide indications as to its possible mode of transmission. Difficulties in producing preparations of virus particles in an unaggregated state have hindered these studies.

Interestingly WLV was found in association with RBDV, and in the limited experimental *Rubus* host range tested, red raspberry cultivars known to be resistant to RBDV were also resistant to WLV. However, not all *Rubus* species that are susceptible to RBDV were also susceptible to WLV (Jones 1977).

In *C. quinoa*, WLV induces large spreading local lesions unlike the pinpoint lesions induced by cucumber mosaic virus. (See "Cucumber Mosaic Virus in Raspberry," p. 191). Also, in this host, WLV does not induce systemic symptoms, and is thus unlike black raspberry necrosis virus (see "Black Raspberry Necrosis," p. 178), bramble



yellow mosaic virus (see "Bramble Yellow Mosaic," p. 243), nepoviruses (see "Nematode-Borne Viruses" of this *Rubus* section, p. 204–228), raspberry bushy dwarf virus (see "Raspberry Bushy Dwarf," p. 229), and isolates of tobacco streak virus (see "Tobacco Streak Virus in *Rubus*," p. 235). Furthermore, apart from bramble yellow mosaic virus, each of these viruses has isometric or quasi-isometric particles.

## 245 Cherry Rasp Leaf Virus in *Rubus*

By A. T. Jones

### Additional Common Names

None.

### History and Geographic Distribution

Cherry rasp leaf disease was first described by Bodine and Newton (1942) in cherry trees in Western United States and British Columbia, Canada. Later workers showed that the disease was associated with a virus that was transmitted mechanically and also by the nematode *Xiphinema americanum* Cobb. to herbaceous plants (Nyland 1961; Nyland et al. 1969). The only report of this virus in *Rubus* is from red raspberry sent from Quebec, Canada, to Scotland (Jones and Badenoch 1981); this is also the only report of the virus occurring outside the western seaboard of North America.

### Economic Importance

The extent of infection in commercial raspberry crops is not known. The few infected plants detected in Scotland showed no obvious symptoms, and the effects on growth and yield are not known.

### Symptoms on Natural and Experimental Hosts

***Rubus* hosts.** Naturally infected plants of a red raspberry seedling showed no obvious foliar symptoms when kept in an unheated gauze house. No symptoms developed in plants of *R. bartonianus* Peck cv. 'Ashton Cross'; *R. henryi* Hemsl. and Kunze, *R. idaeus* L. cvs. 'Glen Clova', 'Malling Jewel', and 'Norfolk Giant'; *R. laciniatus* Willd.; and *R. occidentalis* L. infected by graft inoculation (Jones et al. 1985).

**Herbaceous hosts.** Cherry isolates of cherry rasp leaf virus (CRLV) have been found symptomlessly infecting many weed species in CRLV-affected cherry orchards. No information is available for the *Rubus* isolate; however, this isolate was mechanically transmitted to several herbaceous plants and was symptomless in most. The following developed symptoms: *Chenopodium amaranticolor* Coste and Reyn. and *C. quinoa* Willd. showed occasional faint chlorotic local lesions in 5 days followed by a weak systemic vein-clearing or mottle in 7 to 8 days (figs. 302 and 303). *C. murale* developed necrotic local lesions in 4 to 5 days (fig. 304) followed by a pronounced systemic mottle or severe systemic necrosis in 8 to 10 days (fig. 305). *Cucumis sativus* L. cv. 'National Pickling' and *Phaseolus vulgaris* L. cv. 'The Prince' developed faint chlorotic local lesions in 7 days



Figure 302. — Systemic chlorotic vein-clearing in leaves of *C. quinoa* infected with cherry rasp leaf virus. (Copyright Scottish Crop Research Institute.)



Figure 303. — Systemic mottle in leaves of *Chenopodium quinoa* infected with cherry rasp leaf virus. (Copyright Scottish Crop Research Institute.)

and symptomless systemic infection. Several other species became infected symptomlessly (Jones et al. 1985). Cherry isolates of CRLV differ in the severity of symptoms they cause but, unlike all the cherry isolates reported by Hansen et al. (1974), the *Rubus* isolate infected *Spinacia oleracea* L.

### Natural and Experimental Transmission

Apart from experimental transmission by mechanical inoculation with sap and by grafting, no information is available for the *Rubus* isolate. Natural transmission of CRLV in cherry orchards, however, is by the nematode *Xiphinema americanum*, which appears to be an efficient vector (Nyland et al. 1969; Hansen et al. 1974). In one experiment, *X. diversicaudatum* (Micol.) Thorne failed to transmit CRLV (Nyland et al. 1969).

Cherry isolates are seed-borne in *C. quinoa* and *Taraxacum officinale* Weber and have been detected in pollen of infected cherry (Williams et al. 1963). The role of pollen infection in seed transmission was not determined. The pattern of spread in cherry orchards suggests that transmission by nematodes is the only natural means of spread (Hansen et al. 1974).



### Properties of the Causal Agent

For a detailed description of the virus, see Stace-Smith and Hansen (1976a) and Jones et al. (1985). The virus shows many of the properties of nepoviruses. In sap of *C. quinoa*, the *Rubus* isolate survived dilution to  $10^{-4}$  but not  $10^{-5}$ ,



Figure 304. — Necrotic local lesions in a *C. murale* leaf caused by cherry rasp leaf virus infection. (Copyright Scottish Crop Research Institute.)



Figure 305. — Severe systemic necrosis in *C. murale* caused by cherry rasp leaf virus. (Copyright Scottish Crop Research Institute.)

heating for 10 min at 55° but not 60°C, and storage for at least 16 days at 18° or 4°C (Jones et al. 1985). The virus is relatively unstable and difficult to purify in quantity; however the following two methods seem better than most others: (1) Extract infected tissue in cold 0.5 M borate buffer containing 0.05 M EDTA + 0.02 M mercaptoethanol (pH 6.5), clarify with ammonium sulphate (15 g/100 ml extract), and concentrate the virus by differential centrifugation (Stace-Smith and Hansen 1976b). (2) Extract infected tissue in 0.1 M tris-HCl + 0.2% thioglycerol (pH 7) and chloroform (1 g leaf: 2 ml buffer: 2 ml chloroform). Precipitate the virus from the aqueous phase by adding 4% polyethylene glycol (mol. wt. 6000). Further clarification and concentration is by differential centrifugation and resuspending pellets in 0.01 M tris-HCl + 0.2 M NaCl (pH 7) (Jones et al. 1985).

Purified virus preparations contain isometric particles about 28 nm in diameter. Few particles observed in the electron microscope are penetrated by negative stain (fig. 306). Preparations of particles of the *Rubus* isolate sediment as two nucleoprotein components with sedimentation coefficients of about 89 S and 115 S. Particles of CLRV isolates studied contain three polypeptides of estimated mol. wts. of 26,000, 23,000 and 21,000 and two RNA species of mol. wts.  $2.56 \times 10^6$  and  $1.26 \times 10^6$  daltons, and appear to contain a genome-linked protein necessary for infectivity (Jones et al. 1985).

All cherry isolates appear to be serologically indistinguishable (Hansen et al. 1974; Stace-Smith and Hansen 1976b), and the *Rubus* isolate was serologically indistinguishable from a cherry isolate (Jones et al. 1985).

### Detection and Identification

No symptoms occurred in the few red raspberry plants infected naturally or in graft-inoculated plants of *R. henryi*, *R. idaeus* cvs. 'Glen Clova' and 'Norfolk Giant', or *R. occidentalis*, species used as indicators for other *Rubus* viruses (Jones et al. 1985). Detection, therefore, has relied on mechanical transmission to herbaceous test plants; however at certain times of the year, only very faint symptoms develop in *C. quinoa* and *N. clevelandii*, which could make detection difficult. The use of species such as *Chenopodium murale* and *Cucumis sativus* that develop more diagnostic symptoms might help overcome this. The virus can be identified only by serological tests.

### Control Procedures

The cherry isolate is efficiently transmitted by *Xiphinema americanum*, and it is likely that the *Rubus* isolate is also spread by this means. Control measures, therefore, are the same as for other nematode-transmitted viruses (see the chapters on "Nematode-Borne Diseases" in the *Rubus* section, p. 204-228). Virus-detection tests can be used to eliminate infected plants from material to be propagated, thus preventing the widespread distribution of infected material. No attempts have been made to eliminate the virus from infected plants.

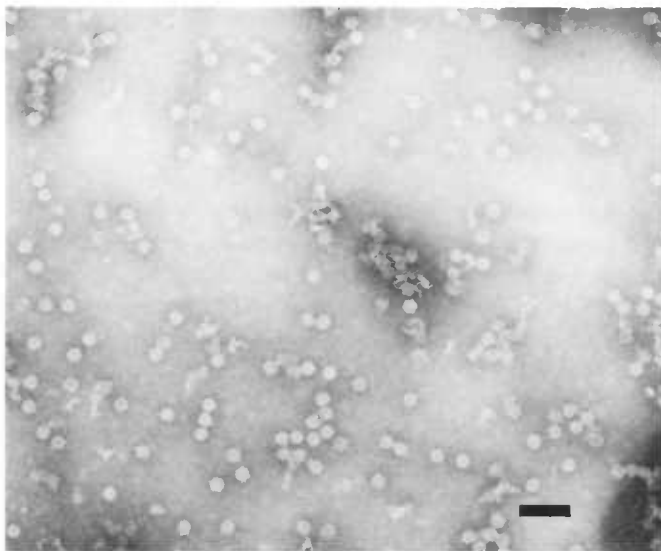


Figure 306. — Electron micrograph of a purified preparation of cherry rasp leaf virus particles stained in 2% ammonium molybdate, pH 6.5. Bar represents 100 nm. (Copyright Scottish Crop Research Institute.)

#### Remarks

Symptoms of the virus in some herbaceous hosts could be confused with those of some nepoviruses (see above) or isolates of tobacco streak virus (see "Tobacco Streak Virus in *Rubus*," p. 235), and the virus can be identified unequivocally only by serological tests. The virus is serologically unrelated either to nepoviruses, or to the virus causing cherry Eola rasp leaf or other viruses inducing enations in cherry (Stace-Smith and Hansen 1976a).

#### Bramble Yellow Mosaic

By D. J. Engelbrecht

#### Additional Common Names

None.

#### History and Geographic Distribution

Bramble yellow mosaic disease, caused by bramble yellow mosaic virus (BrYMV), was found in an isolated patch of wild trailing blackberry (*Rubus rigidus* Smith) in the Western Cape Province of South Africa and described by Engelbrecht and van der Walt (1974).

#### Economic Importance

Unknown.

#### Symptoms

**Natural host.** Pronounced yellow mosaic and line-pattern symptoms develop on the leaves of the only known natural host, *R. rigidus*, during early spring (fig. 307). Usually, no distinctive symptoms are detectable on the foliage of young canes after early spring. Furthermore, as the season advances, the yellow areas on affected leaves tend to bleach to a bright calico.



Figure 307. — Leaves of *Rubus rigidus* naturally infected with bramble yellow mosaic virus, showing yellow mosaic and line-patterns.

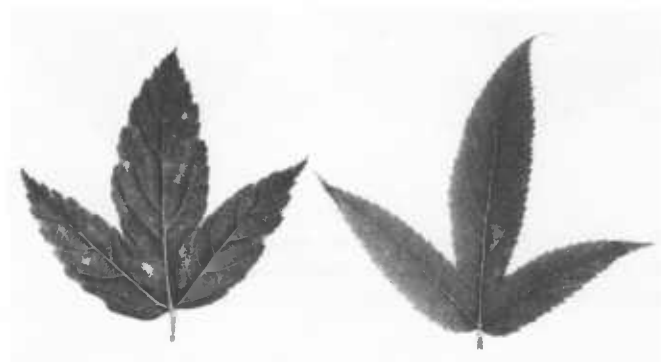


Figure 308. — Mild transient mottle on a leaf of *Rubus henryi* experimentally infected with bramble yellow mosaic virus.

**Experimental hosts by grafting.** Leaves of grafted *R. henryi* Hemsl. and Kuntze developed a mild transient chlorotic mottling (fig. 308). On 'Royal Sovereign' (*Fragaria* x *ananassa* Duch.), a mild mottling accompanied by a pronounced veinal necrosis was evident. Symptoms persisted in affected plants; however, in *Fragaria vesca* L. cv. 'EM-1' symptoms were more severe and progressive. An initial interveinal chlorosis (fig. 309), followed by a leaf-necrosis and dwarfing, led to the eventual death of affected plants.

**Experimental hosts by sap transmission.** *Chenopodium murale* L. is an excellent host for the detection of BrYMV by mechanical inoculation with infected blackberry or strawberry plant sap, but the virus is difficult to transfer from *C. murale* to other herbaceous hosts. Large irregular yellow chlorotic lesions developed on inoculated *C. murale* leaves



Figure 309. — Interveinal chlorosis on a leaf of *Fragaria vesca* newly infected with bramble yellow mosaic virus.



Figure 310. — Bramble yellow mosaic virus symptoms on *Chenopodium murale*. Left, irregular necrotic lesions on inoculated leaf; right, systemic necrotic rings.

after 7 to 10 days. The lesions, which gradually coalesced, became necrotic. This was followed by the development of systemic yellow necrotic rings (fig. 310).

Symptoms were also observed on the following plants tested:

*C. quinoa* Willd. Pinpoint necrotic lesions appeared 10 to 15 days after inoculation, followed by a mild systemic chlorotic mottling.

*Gomphrena globosa* L. cv. 'Rosé'. Local white necrotic spots surrounded by purple halos.

*Nicotiana tabacum* cv. 'White Burley'. Chlorotic spots on inoculated leaves about 3 wk after inoculation. Virus was not recovered from symptomless secondary tobacco leaves.

Species that did not show symptoms and from which virus could not be retrieved included *Cucumis sativus* L.

(cucumber), *Petunia hybrida* Vilm. (petunia), and *Phaseolus vulgaris* L. (bean) (Engelbrecht 1963, 1976).

### Transmission

**Natural spread.** Unknown in *Rubus*.

**Graft transmission.** BrYMV transmission by cane inarching and runner-to-cane inarching has been demonstrated for *R. henryi* and strawberry, respectively. Back transmission of BrYMV to *F. vesca* cv. 'EM-1' plants, following inarching of the stems of infected *C. murale* plants, was also confirmed (Engelbrecht 1976).

**Mechanical transmission.** BrYMV is easily transmitted mechanically from blackberry and strawberry to *C. murale*, provided nicotine is added to the extracting solution. Infected *R. henryi* failed to yield virus.

**Seed transmission.** Progeny seedlings raised from systemically infected *C. murale* showed 86 to 100% infected seedlings. Symptoms usually appeared 2 to 3 wk after transplanting and were similar to the systemic reaction, though milder, on the mother plant.

### Detection

The mosaic and line-pattern symptoms on leaves of affected blackberry plants can be readily recognized throughout the growing season.

### Identification

BrYMV possesses filamentous particles with a modal length of 730 nm (Engelbrecht 1976). The characteristic particles have only been detected in sap from *C. murale* infected with BrYMV from blackberry and experimentally infected strawberry. The virus remains infective in *C. murale* sap after dilution to  $10^{-3}$  or 10 min at 50°C or 8 days at 20°C. Infectivity was lost at pH 6.0 and after prolonged freezing but was only slightly decreased after 24 h at -20°C. Attempts to purify sufficient virus for antiserum production have not yet succeeded.

### Control Measures

No information is available.

### Remarks

This virus differs in properties from two other filamentous viruses reported to occur in *Rubus*, namely, bean yellow mosaic (Provvidenti and Granett 1974) and wineberry latent virus (Jones 1974). (See "Wineberry Latent Virus," p. 239.) A previous suggestion (Engelbrecht 1976) that BrYMV virus be classified as a potyvirus on the basis of particle length must be considered premature.



## Blackberry Calico

By R. H. Converse

### Additional Common Names

Loganberry calico, Boysenberry calico, Chehalem calico, Marion calico, yellow blotch mosaic; blackberry calico disease (BCD).

### History and Geographic Distribution

The disease was described by Wilhelm (1951) and Wilhelm et al. (1951). It occurs in California and Oregon and probably is present wherever U.S. Pacific coast trailing blackberry cultivars like 'Boysen', 'Logan', 'Thornless Logan' ('Loganberry'), and 'Marion' are grown throughout the world.

### Economic Importance

The disease almost universally infects some cultivars like 'Thornless Logan' and 'Chehalem', which nevertheless bear productive, horticulturally acceptable crops. In California, 'Thornless Logan' with BCD grown without irrigation may develop yellow areas in leaves of floricanes, which may then become damaged by sun and wind, especially in dry years.

### Symptoms on Natural and Experimental Hosts

On naturally infected clones of 'Boysen', 'Chehalem', 'Marion', 'Ollalie', and 'Thornless Logan', chlorotic areas appear on leaves of floricanes at fruiting time. The chlorosis may be blotchy and without pattern or may occur as distinct rings and line patterns. Various shades of yellow, verging on white, occur and occasionally some red coloration (fig. 311). Leaves exhibiting severe calico symptoms may wither and drop off in periods of high light intensity and drought stress.

On graft-inoculated 'Boysen', 'Marion', 'Thornless Logan', and 'Young' blackberries in the greenhouse, BCD caused symptoms similar to those expressed naturally in the field, but often only a few leaves showed symptoms, which were sometimes limited to a few, very small, chlorotic spots on leaf blades (Converse and Kowalczyk 1980). Symptom expression of BCD in floricanes appears to be favored by high light intensity, and some BCD-infected cultivars like 'Thornless Logan' rarely develop symptoms in the greenhouse or outdoors in predominantly cool, cloudy growing conditions.

### Natural and Experimental Transmission

Much Pacific coast blackberry nursery stock, particularly 'Thornless Logan', is infected with BCD. BCD has been observed to spread naturally in the field in 'Chehalem' and 'OR-US 1600' blackberries. The wild Pacific coast trailing blackberry (*Rubus ursinus* Cham. & Schlecht.) has been found to be naturally infected (R. H. Converse, unpublished data).

Experimentally, BCD has been transmitted by approach graft to 'Boysen', 'Marion', 'Thornless Logan', 'Young', and *R. ulmifolius* Schott var. *inermis* (Willd.) Focke (Converse and Kowalczyk 1980; Wilhelm 1951; Wilhelm et al. 1951).

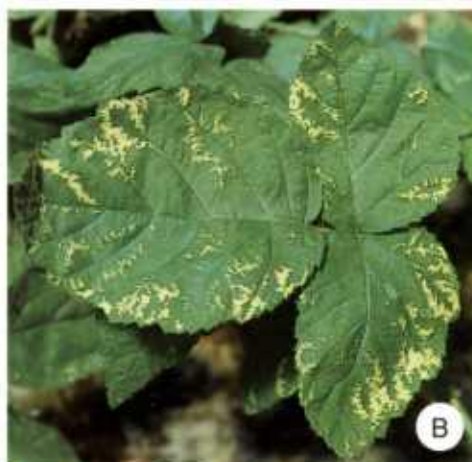


Figure 311.—Calico disease symptoms in leaves of (A) 'Logan' and (B) 'Marion' blackberries.

Symptoms did not develop after BCD was graft inoculated to *R. henryi* Hemsl. and Kuntze, *Fragaria vesca* L. var. *semperflorens* (Duch.) Ser. cv. 'Alpine', or after sap transmission to *Nicotiana tabacum* L. cv. 'Turkish' (Williams and Wagnon 1970). Contradictory results were obtained when BCD was approach grafted to *R. procerus* P. J. Muell. and *R. occidentalis* L. (Wilhelm et al. 1951; Williams and Wagnon 1970; Converse and Kowalczyk 1980).

Vector transmission of BCD has not been studied. A leaf variegation that was graft transmissible from wild *R. allegheniensis* Porter to blackberry and black raspberry was reported in Maryland (Horn 1948). Its relationship to BCD is unknown. Johnson (1972) has reported the graft transmissibility of a calico disorder in 'Puyallup' red raspberry. BCD sources grafted to 'Puyallup' failed to induce any symptoms (Converse and Kowalczyk 1980).

### Properties of the Causal Agent

Nothing is known of the nature of the causal agent of BCD except that it is graft transmissible and that raspberry bushy dwarf virus is not necessarily associated with it. Comparable graft transmission studies indicate that BCD from



'Chehalem' blackberry had a different host range and symptomatology than BCD from 'Marion' and 'Thornless Logan' blackberry (Converse and Kowalczyk 1980). The names "Chehalem calico," "Loganberry calico," "Boysenberry calico," and "Ollalie calico" will probably continue to be useful in designating individual variants of BCD until their etiology is better understood.

### Detection and Identification

'Logan' seedlings and 'Marion' blackberry have been used as indicator hosts for BCD in graft transmission studies (Wilhelm et al. 1951; Converse and Kowalczyk 1980). On 'Marion', the minimum incubation period in the greenhouse after grafting varied from 6 mo to 2 yr. Exposing grafted 'Marion' plants to winter dormancy conditions may shorten incubation time and heighten symptoms. Many trailing blackberries are infected with tobacco streak virus. (See "Tobacco Streak Virus in *Rubus*," p. 235.) 'Marion-65', a clone that is free from known viruses and viruslike diseases, is killed back along the shoot below the graft for a few nodes when approach-grafted with a plant infected with tobacco streak virus, so that it is not possible to use 'Marion-65' as an indicator for BCD in plants infected with both diseases. In such cases, 'Logan' seedlings can be used as indicators.

### Control Procedures

The principal control method is the use of Pacific coast trailing blackberry cultivars that are known to be free of BCD. Recently, a heat-treated, shoot-tip-propagated clone of 'Thornless Logan' from Canada (R. Stace-Smith, unpublished results) has been found to be free of BCD and has been released as 'Thornless Logan AC-2', completing a collection of clones of major Pacific coast blackberry cultivars that are known to be free from BCD (Converse and Kowalczyk 1980).

Heat treatment of 'Thornless Logan' plants at 37°C for 17 days did not eliminate BCD (Williams and Wagnon 1970), but the AC-2 clone was derived from a shoot tip that was propagated from a 'Thornless Logan' plant held for 35 days at 37°C (R. Stace-Smith, unpublished results).

### Remarks

BCD is very poorly understood. The demonstration of its graft transmissibility allows distinctions to be made between this infectious disease and purely genetic disorders, with which it has often been confused in the past. Because BCD is so widely distributed in many Pacific coast blackberry clones, often without visible symptoms, its possible deleterious effects in *Rubus*, alone and in combination with other viruses or viruslike diseases, have been largely ignored. Further research on the identification and relationships of the causal agent(s) and its rapid identification will permit the true economic impact of BCD to be more correctly evaluated and its control to be improved.

## Miscellaneous Virus and Viruslike Conditions and Disorders

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### Apple Mosaic Virus in *Rubus*

By G. [Baumann, R.] [Casper, and R. H.] [Converse]

### Additional Common Names

None.

### History And Geographic Distribution

Apple mosaic virus (ApMV), a member of the ilarvirus group, was found in samples of symptomless plants of *Rubus ursinus* Cham. and Schlecht., *R. idaeus* L., and *R. occidentalis* L. on both coasts of the United States (Converse and Casper 1975). Latent infection of red raspberry (*R. idaeus* L.) plants by ApMV was reported in Germany (Baumann et al. 1982). These authors also reported the association of ApMV with yellow mottling and line-pattern symptoms in one plant of red raspberry cv. 'Schoenemann', which was found in northwest Germany.

### Economic Importance

The economic importance of ApMV in *Rubus* is not yet known.

### Symptoms on Natural and Experimental Hosts

Natural hosts of ApMV include the genera *Malus* (Bradford and Joley 1933), *Prunus* (Barbara 1980), *Rosa* (Fulton 1952, 1968), *Aesculus* (Sweet and Barbara 1979), *Betula* (Gotlieb and Berbee 1973), *Humulus* (Albrechtova et al. 1979), and *Rubus* (Converse and Casper 1975). Although ApMV is generally symptomless in infected *Rubus*, infected plants of the other genera mentioned frequently show yellow or white leaf mosaic and line-pattern symptoms. Virus isolates from naturally infected hosts have been transmitted by grafting to a wide range of species in the genera *Aesculus*, *Betula*, *Chaenomeles*, *Crataegus*, *Cydonia*, *Fragaria*, *Malus*, *Prunus*, *Pyrus*, *Rosa*, and *Sorbus*. Mosaic and leaf-pattern symptoms were produced in each of these genera (Posnette 1963; Gotlieb and Berbee 1973; Sweet and Barbara 1979).

Latent occurrence of ApMV was found in *Rubus ursinus* Cham. and Schlecht. cv. 'Boysen' (California and Oregon), in *R. occidentalis* L. cvs. 'Alleghany' and 'Munger' (Massachusetts and Oregon, respectively), and in *R. idaeus* cvs. 'Willamette' (Oregon), 'Korbfüller', and 'Schoenemann' (Germany).

In a red raspberry field in Germany (cv. 'Schoenemann'), in which latent infection by ApMV had been identified (Baumann et al. 1982), one plant was found showing conspicuous leaf symptoms. These consisted of a bright yellow-white mosaic (fig. 312), sometimes accompanied by



Figure 312.—A, Bright yellow mottling and line-pattern on leaves of red raspberry plants from which apple mosaic virus has been isolated; B, leaf symptoms of red raspberry cv. 'Malling Landmark' graft inoculated with the apple mosaic virus isolate from 'Schoenemann'.

small, light-green rings and spots. No fruit symptoms were observed, and there appeared to be no significant reduction in the growth of canes showing leaf symptoms.

The disease was transmitted by shoot or bark shield grafts to virus-indexed red raspberry plants (cvs. 'Schoenemann', 'Malling Landmark', and 'Baumforth's Seedling B') which developed leaf symptoms similar to those shown by the original source plant. *Prunus serrulata* L. cv. 'Shirofugen' grafted with bark shields from visibly infected 'Schoenemann' developed necrosis and gum production around the inserted bark shields. However, the reaction was less severe than that usually demonstrated by *Prunus* necrotic ringspot virus (Baumann et al. 1982).

The original source plant as well as the experimentally infected woody hosts were found to react in ELISA tests with ApMV antiserum. No other known *Rubus* viruses were found in these plants. The ApMV isolate from the 'Schoenemann' field plant showing symptoms was transmitted by sap inoculation from infected raspberry to several herbaceous hosts, producing the symptoms noted, which in general resemble those of previously described ApMV isolates on the following hosts:

*Cucumis sativus* L. cv. 'Straight Eight' developed green-yellow local lesions on the rubbed cotyledons. Sometimes, chlorotic spots developed on the first true leaves, often leading to their death.

*Chenopodium quinoa* Willd. Chlorotic-necrotic lesions developed on the inoculated leaves, and systemic symptoms consisted of sharply defined water-soaked rings, lines, and spots.

*C. amaranticolor* Coste and Reyn. Inoculated plants showed chlorotic-necrotic lesions on the rubbed leaves but no systemic symptoms.

*C. capitatum* (L.) Asch. Numerous small chlorotic lesions developed on the inoculated leaves and single chlorotic rings around dark-green or necrotic centers on systemically infected leaves.

*Phaseolus vulgaris* L. cvs. 'Pinto' and 'Black Turtle' showed severe vein necrosis on the inoculated leaves followed by tip necrosis.

*Vigna unguiculata* (L.) Walp. Inoculated leaves showed chlorotic lesions and red or brown necrotic spots and lines. The tips curled downward, and the plants stopped growing.

*Nicotiana clevelandii* Gray, *N. rustica* L., *N. tabacum* L. 'White Burley', and *Petunia hybrida* Vilm. did not become infected.

#### Natural and Experimental Transmission

Converse and Casper (1975) stated that field transmission of ApMV occurred in Oregon where root graft transmissions were unlikely to have occurred when plants of red raspberry, black raspberry, and blackberry that had indexed free from ApMV were planted in the field. About 92% of red raspberry plants became infected by ApMV within 24 mo and 16% of the blackberry plants became infected after 12 mo. ApMV is not known to spread naturally other than by root grafting in any of its other reported hosts (Fulton 1972). Thus, the mode of field transmission of ApMV in its *Rubus* hosts requires further investigation.

Experimental transmission of the yellow mosaic and line-pattern found in the 'Schoenemann' plant in West Germany was conducted by bottle grafting (shoot grafting) or bark shield grafting (Baumann et al. 1982). Symptoms developed on leaves of inoculated plants 7 mo after inoculation when the experiment was started in August, but 4 wk after it was started in March or April. The 'Schoenemann' isolate of ApMV was transmitted by sap inoculation to herbaceous hosts when buds from infected red raspberry plants were ground in 0.05 M phosphate buffer (pH 7.0) containing 2% polyvinylpyrrolidone (mol. wt. 10000) (Martin and Converse 1982) and rubbed on leaves of young herbaceous indicator plants.

### Properties of the Causal Agent

ApMV is a member of the ilarvirus group and is an isometric virus for which particles 25 to 26 and 29 to 30 nm in diameter have been reported, corresponding to sedimentation coefficients of 88 and 117S, respectively (de Sequiera 1967). Only the heavier particles are infective, but all are serologically and electrophoretically homogenous. The  $A_{260}/A_{280}$  for ApMV is about 1.5 (Fulton 1972). The RNA content of ApMV has not been determined directly, but it is 16% for the distantly serologically related *Prunus* necrotic ringspot virus. The mechanism of natural transmission in *Rubus* is unknown; however, ApMV is commonly transmitted by vegetative propagation and natural root grafts of other woody hosts.

### Detection and Identification

ApMV was easily detected and identified by ELISA using antiserum against the RV-1 (rose virus-1) isolate of apple mosaic virus (Casper 1973). Tests by agar gel diffusion are also possible (Converse and Casper 1975). Indexing should be done between the end of March and the end of April using leaf buds of very young primocanes. Results of tests conducted later in the season may not be reliable.

### Control Procedure

The use of certified planting stock and the selection of nuclear stock materials shown to be free from ApMV by serological tests are recommended. Both apple mosaic and rose mosaic viruses have been easily eliminated from cultivars of several rosaceous hosts other than *Rubus* by brief hot air therapy (Nyland and Goheen 1969).

### Remarks

In Oregon, plants of red raspberry cv. 'Willamette' which had indexed positive for ApMV were also found to react positively with tomato ringspot virus antiserum. Tobacco streak virus was found to occur together with ApMV in *Rubus ursinus* 'Boysen' and also in *R. occidentalis* cvs. 'Alleghany' and 'Munger' (Converse and Casper 1975). The significance of double infections in the occurrence of ApMV in U.S. *Rubus* is not known.

Since the 'Schoenemann' isolate of ApMV has not yet been transmitted from herbaceous hosts to red raspberry plants, it is not known whether more than one ApMV strain exists in *Rubus* or whether the yellow mosaic and line pattern found in 'Schoenemann' is due to a mixed infection by ApMV and an unknown *Rubus* virus. Symptoms of the 'Schoenemann' isolate of ApMV and raspberry bushy dwarf virus (see 'Raspberry Bushy Dwarf,' p. 229) in sap inoculated *Chenopodium quinoa* are very similar to each other and can be differentiated by ELISA (Baumann et al. 1982).

## 245 *Rubus* Virus Diseases of Minor or Undetermined Significance

By R. Stace-Smith

Those involved in compiling literature on virus diseases affecting a particular crop are always faced with the awkward task of attempting to relate some of the early published work, usually of a preliminary and descriptive nature, with more comprehensive reports that followed. We would prefer to have the tidy situation where early observations and reports could be correlated with what is known today to form a more comprehensive picture of how the significance of individual diseases has changed with the introduction of new cultivars and modified cultural practices. Alas, such a utopian state is not possible with respect to the *Rubus* virus diseases. It is almost impossible to relate some of the earlier work, compiled on the basis of acceptable standards at the time, with the various diseases that are recognized today.

This is not meant to downgrade the value of the earlier work. With the many developments in the science of plant virology, the accepted criteria as to what body of information is necessary to designate a causal agent as "new" or "previously undescribed" has been upgraded considerably. The literature contains many examples of known viruses redescribed under new names or viruses so inadequately described that it is doubtful that they are really new. The purpose of this chapter is to include a brief description of a few viruses that exist in the literature but whose identity is still uncertain and to record reports of isolation of viruses from *Rubus* crops that are of local or minor significance. A similar chapter was included in "Virus Diseases of Small Fruits and Grapevines" (Converse 1970b).

**Tobacco necrosis virus.** Tobacco necrosis virus (TNV) is detected by transmission to herbaceous indicator plants from root extracts of a wide range of herbaceous and woody plants. The virus occasionally invades woody crop plants systemically and, in such instances, TNV can be transmitted to indicator plants from aboveground plant parts. Although sporadic disease losses have been reported, TNV is not usually considered to cause any significant damage to a crop. Cadman (1961b) reported that TNV was commonly isolated from roots of field-grown raspberry plants in Scotland. The



virus has not been isolated from other raspberry-growing areas, probably because most inoculations are made from aboveground plant parts.

**Tobacco rattle virus.** Tobacco rattle virus (TRV) infects many species of cultivated and wild plants in many parts of the world. Many of the infected plants develop symptoms, but others can be infected symptomlessly. Symptomless infections also occur when TRV is confined to the roots of infected plants. Cadman (1961*b*) reported that the virus was occasionally isolated from the roots of raspberry plants in Scotland.

**Black raspberry streak.** This virus disease was allotted a full chapter in "Virus Diseases of Small Fruits and Grapevines" (Converse 1970*a*). However, since no new information has been published since 1970 and since little is known about the disease, it was considered appropriate to relegate it to the minor disease category. The disease was named for the characteristic faint blue or gray streaks that develop on or under the surface bloom of young canes. Positive identification of the disease is difficult because streak development is influenced by environmental conditions. Other than black raspberry, there is no satisfactory indicator host. Graft and dodder transmission have been demonstrated, but the mechanism of natural spread is unknown.

**Bean yellow mosaic virus.** Provvidenti and Granett (1974) reported the isolation of a severe strain of bean yellow mosaic virus from several plants in New York State, including two red raspberry plants. No information is given as to whether the infected raspberry plants showed any symptoms.

**Blackberry sterility.** Blackberry sterility is occasionally a major problem in nurseries and fruiting fields in some areas of eastern and central United States, where blackberries are grown. In affected plants the flowers appear normal but none or only a few of the drupelets develop (fig. 313). Some of the affected plants may exhibit a mosaic pattern on the leaves, but, as noted by Hemphill (1970), this symptom may be caused by the presence of other viruses. Further, sterility in *Rubus* may be induced by environmental and genetic factors (Hemphill 1970). At present, it is not possible to say whether there is a virus, distinct from the viruses that are known to affect *Rubus*, which induces sterility symptoms.

**Necrotic fernleaf mosaic.** The disease was described on the basis of observations and grafting experiments on a single 'Cuthbert' red raspberry plant in Ontario, Canada (Chamberlain 1941). Leaves of the affected plant were small, narrow, and extensively serrated, giving a "fernleaf" appearance. In addition, leaves of the affected plant exhibited ringspot markings, necrotic spots, stunting, and retarded foliation, symptoms that are similar to the yellow blotch curl disease of red raspberry in Ontario (Chamberlain 1938). Clones of these two field diseases have not been retained, so at this time one



Figure 313.—Blackberry sterility disease: A, Completely sterile flowers of infected 'Lawton' blackberry; B, partially to completely sterile flowers of an unnamed blackberry cultivar. (Courtesy D. D. Hemphill, University of Missouri.)

can only speculate on the identity of the viruses involved. As noted by Freeman and Stace-Smith (1968), circumstantial evidence strongly suggests that the yellow blotch curl disease was caused by infection with tomato ringspot virus. One could speculate further that necrotic fernleaf mosaic disease was induced by a complex of viruses, one of which was tomato ringspot virus (see "Tomato Ringspot Virus in *Rubus*," p. 223).

**Raspberry yellow spot.** Yellow spot was the name applied to a virus disease of wild and cultivated raspberries in Poland (Basak 1974). The most characteristic symptom of the disease, as the name implies, is the yellow spotting of the leaves. The spots are of variable size and shape, scattered irregularly over the leaflets, occasionally so numerous that they cover most of the leaf blade, giving the entire plant a yellow cast. As the season progresses, the yellow color gradually fades. The spotting causes uneven growth of the leaflets and results in leaf curling and deformity. Plants that are severely affected are stunted. Intensity of the symptoms



varies from year to year depending upon weather conditions—particularly severe symptoms occur in years with cold springs.

The virus was graft transmitted and induced symptoms on *Rubus procerus* P. J. Muell., *R. phoenicolasius* Maxim., and *R. occidentalis* L. *R. henryi* Hemsl. and Kuntze and *R. xanthocarpus* Rur. and Franch., which were infected by grafting, did not show symptoms. The virus was also transmitted from red raspberry to black raspberry seedlings by means of the aphid *Amphorophora idaei* Börner, and it was concluded that this aphid is the vector under natural field conditions. Wild raspberries appear to be the main source of the disease; the virus is transmitted from them by aphids to commercial plantings.

While direct comparisons have not been made with other raspberry viruses that are transmitted by *A. idaei*, Basak (1974) is of the opinion that raspberry yellow spot differs from virus diseases reported from other countries.

#### **Rubus Diseases in Great Britain With Presumed but Unproved Viral Etiology**

By A. T. Jones

**A mottling disease of 'Bedford Thornless' blackberry.** This disease has been observed in Scotland. It is characterized by a chlorotic mottling of leaves, often adjacent to the main veins. Symptoms are most pronounced on leaves on fruiting canes (fig. 314) and are most evident in late spring. Similar symptoms have been observed in 'Bedford Giant' blackberry plants in England and Scotland. No virus was detected by mechanical inoculation of sap from such affected plants to herbaceous test plants. However, graft inoculation of diseased material to *Rubus* indicators detected raspberry leaf mottle and raspberry leaf spot viruses in some affected plants but not others, whereas scions from all diseased plants induced tip necrosis in graft-inoculated *R. occidentalis* suggesting that black raspberry necrosis virus, raspberry leaf mottle virus, or raspberry leaf spot virus were present. Whether these viruses are causal agents of the disease is not known. (See "Black Raspberry Necrosis," p. 178.)

**Crinkle and sterility of 'Bedford Thornless' blackberry.** This disease is characterized by a mottling and severe crinkling of the leaves, especially in fruiting canes (fig. 314). Most laterals are sterile (fig. 315). The cause of this disease is not known, but no virus was detected by mechanical inoculation of sap from such plants to herbaceous test plants.

**Leaf curling in 'Norfolk Giant' red raspberry.** A single plant of this cultivar showing severe curling of the young leaves of primocanes (fig. 316) was observed in southern England. The plant contained raspberry bushy dwarf virus (see "Raspberry Bushy Dwarf," p. 229) but was apparently free of other mechanically transmissible viruses. The agent of the



Figure 314.—Leaves from fruiting cane of 'Bedford Thornless' blackberry from plants affected with: mottling (left), crinkle (right), and unaffected (center). (Copyright Scottish Crop Research Institute.)



Figure 315.—Fruiting canes of 'Bedford Thornless' blackberry from plants affected with: mottling (left), crinkle and sterility (right), and unaffected (center). (Copyright Scottish Crop Research Institute.)



Figure 316.—A cane of 'Norfolk Giant' red raspberry showing small, tightly curled leaves at the tip. (Copyright Scottish Crop Research Institute.)

disease was graft transmitted to other 'Norfolk Giant' plants in which it induced leaf curling. Although the symptoms closely resemble those of raspberry leaf curl that occurs in North America (see "Raspberry Leaf Curl," p. 187), no symptoms occurred in graft-inoculated plants of *R. henryi*, *R. occidentalis*, and *R. phoenicolasius* 3 mo after inoculation.

## Viruslike Disease Symptoms in *Rubus* in Great Britain

By A. T. Jones

A number of viruslike symptoms in *Rubus* spp. are known to be caused by factors other than virus infection. These are most frequently symptoms induced by chemical or pest damage or by genetic causes. A discussion of viruslike symptoms in North America *Rubus* was prepared by Converse (1970d).

### Chemical Damage

Several chemicals frequently used in the management of raspberry and blackberry plantations are known to induce some damage in these crops. At least three chemicals have been observed to induce viruslike symptoms in some *Rubus* cultivars in Great Britain.

**Fenitrothion (an organophosphorus insecticide).** In Scotland, Woodford and Gordon (1978) observed that plants of the red raspberry cvs. 'Malling Admiral' and 'Malling Delight' showed numerous chlorotic spots and flecks in leaves sprayed a few days previously with fenitrothion (fig. 317). Greenhouse experiments confirmed that these symptoms in these two cultivars and somewhat milder symptoms in cv. 'Glen Clova' were due to their response to the chemical; cv. 'Malling Jewel' showed no symptoms after spraying. They also observed that the leaf symptoms were more pronounced after plants were sprayed in bright sunlight. Such symptoms could easily be confused with virus infection or could mask symptoms due to virus infection; however, in commerce, symptoms would tend to develop uniformly on almost all sprayed plants.

**Glyphosate (a translocated herbicide).** Plants of the red raspberry cv. 'Glen Clova' have been observed with extensive proliferation of fruiting laterals; leaves of such laterals tended to be thin and distorted (fig. 318). Such plants occurred along the periphery of a plantation adjacent to a crop sprayed several months earlier with glyphosate, and such symptoms are believed to be due to spraydrift. Individual plants of the cvs. 'Glen Clova' and 'Malling Jewel' showing similar symptoms have also been observed in plantations where spot treatment with glyphosate has been used for weed control. Similar symptoms frequently occur in many *Rubus* cultivars in North America after plants have been accidentally sprayed with glyphosate during in-row weed control operation. Symptoms frequently take several months to develop after application of the chemical, and great care should be taken to avoid drift to crops.

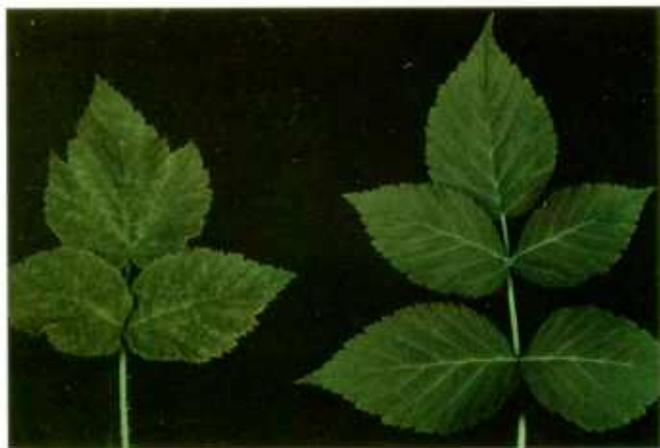


Figure 317.—Chlorotic mottling in a leaf of 'Malling Admiral' red raspberry sprayed with fenitrothion (left) and sprayed with water (right). (Copyright Scottish Crop Research Institute.)



Figure 318.—Proliferation of fruiting laterals and thin, deformed leaves of 'Glen Clova' red raspberry caused by spray drift of glyphosate. (Copyright Scottish Crop Research Institute.)

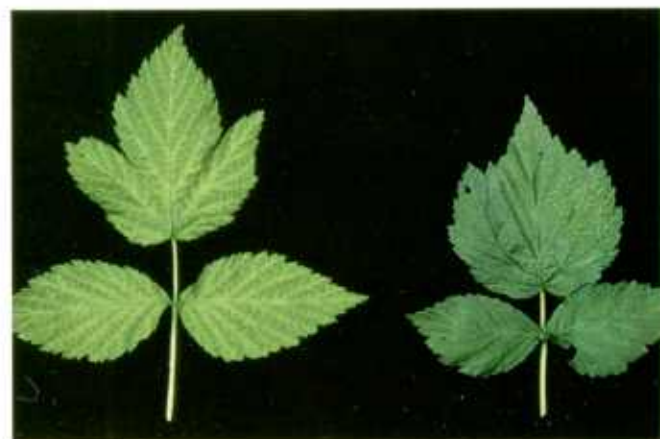


Figure 319.—Leaves of young primocanes of 'Glen Clova' red raspberry showing chlorotic veinbanding (left) and veinclearing (right) symptoms following treatment of the plantation with bromocil. (Copyright Scottish Crop Research Institute.)



**Bromocil (a soil-acting herbicide).** A few weeks after application of this chemical in the spring, leaves on new canes of 'Glen Clova' red raspberry and 'Tayberry' often show a mild chlorosis. This frequently takes the form of a veinbanding or vein clearing symptom (fig. 319). Plants tend to grow out of this symptom, and thus it is distinguishable from veinbanding mosaic disease (see "Raspberry Mosaic," p. 168), the symptoms of which usually persist in plants and are more extensive.

### Pest Damage

In Great Britain, the only pest damage likely to be confused with symptoms due to virus infection is that caused by the raspberry leaf and bud mite *Phyllocoptes gracilis* Nal. (also known as *Eriophyes gracilis* Nal.). Feeding damage on leaves of primocanes and fruiting canes of many red raspberry, blackberry, and 'Tayberry' cultivars causes chlorotic spots and blotches of the upper leaf surface. On the lower leaf surface, areas of feeding damage appear pale green when compared with the grayish bloom of the unaffected leaf surface (fig. 320 and 321). Although symptoms can easily be confused with those of virus infection, the characteristic appearance of the chlorotic areas on the lower leaf surface and examination of such leaf areas under the microscope for evidence of these small, translucent, cigar-shaped eriophyid mites should prevent misidentification. In exposed raspberry plantations, mites do not normally reach sufficient numbers to induce feeding damage, but in sheltered areas, and especially in hot dry summers, numbers of mites may increase enormously. Under such favorable conditions, mites may not only induce symptoms in many leaves but can also cause malformation and uneven ripening of the fruit (Gordon and Taylor 1976).

### Genetic Causes

*Rubus* species are prone to genetic abnormalities of one kind or another and, although many can be identified as such, a few are similar to those induced by virus infection. Perhaps one of the most common abnormalities is leaf chlorosis in raspberry seedlings. In some of these instances, such chlorosis is associated with infection with raspberry bushy dwarf virus (see "Raspberry Bushy Dwarf", p. 229), but in others it is not (Jones et al. 1982), presumably being caused by genetic abnormality.

The following are some of the kinds of genetically induced changes that may be confused with virus infection.

**Crumbly fruit.** This is a condition in which some drupelets fail to set, often producing malformed fruits. The reduced number of drupelets that do set are often enlarged and cohere imperfectly so that the fruit crumbles when picked. Although infection with some viruses, for example, raspberry mosaic disease, raspberry bushy dwarf, tomato black ring, and tomato ringspot viruses, is associated with this condition in some raspberry cultivars in Great Britain and North America, it is also known that in many instances it is induced by



Figure 320.—Raspberry leaf and bud mite (*Phyllocoptes gracilis*) damage in red raspberry. Upper leaf surface shows chlorotic spots and blotches. (Copyright Scottish Crop Research Institute.)

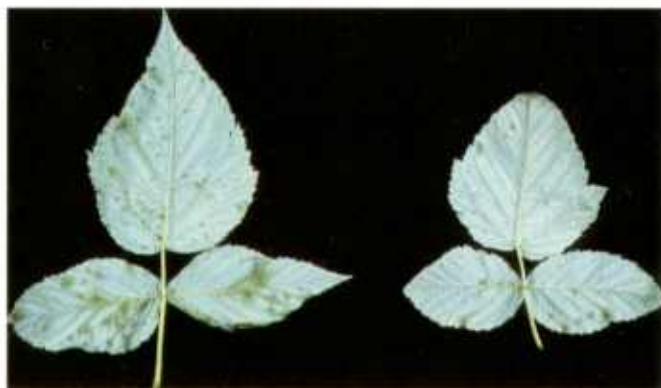


Figure 321.—Raspberry leaf and bud mite (*Phyllocoptes gracilis*) damage in red raspberry. Lower surface of the same leaf shown in figure 320, showing pale green areas of feeding damage. (Copyright Scottish Crop Research Institute.)



Figure 322.—Leaves of a red raspberry seedling showing frilly leaf symptom. (Copyright Scottish Crop Research Institute.)

genetic alterations in the plant. Some of these are associated with gene mutation, meiotic irregularities, and degeneration of the embryo sac (Murant et al. 1973; Jennings 1967; Daubeney et al. 1967, Viridi et al. 1972a,b). Suitable tests on affected plants to determine the presence of virus should be used to determine the cause of the condition.

**Frilly leaf.** This condition, first noted by Knight et al. (1959), occurred in self-bred progeny of the red raspberry cvs. 'Baumforth A' and 'Canby'. Affected plants have distorted young leaves with chlorotic flecks (fig. 322), and the stems fail to elongate. The condition in cv. 'Baumforth A' is determined by gene *fr*, which is linked with gene A, which confers resistance to the aphid *Amphorophora idaei* Börner.

A somewhat similar condition has been observed by A. T. Jones and D. L. Jennings (unpublished observations) in raspberry seedlings (figs. 323 and 324). However, although the symptoms were pronounced when plants were grown in a heated greenhouse, they were inconspicuous or absent when grown outdoors. No symptoms developed in apparently normal seedlings from the same family or in the *Rubus* virus indicators *R. henryi* Hemsl. and Kuntze, *R. occidentalis* L., and the *R. idaeus* L. cvs. 'Malling Landmark' and 'Norfolk Giant' when these were graft inoculated with tissue from affected plants.

**Catkin in cv. 'Malling Jewel'.** This condition, reported by Jennings (1977) and believed to be due to a mutation, was characterized by some canes of a plant bearing catkinlike flowers. The flower parts on such canes were replaced by bractlike structures of decreasing size (figs. 325 and 326) produced a catkinlike effect.

**Lateral-leaf crinkle in cv. 'Glen Clova'.** An aberrant cane of this red raspberry cultivar, showing severely crinkled leaves which were free from chlorosis (fig. 327), was reported in Scotland by Jennings (1977). As raspberry leaf curl virus is not known to occur in Scotland and the condition did not recur, it is suspected that a mutation from a recessive to a dominant condition was involved.



Figure 324.—Enlargement of figure 323 to show detail of frilly leaf symptom. (Copyright Scottish Crop Research Institute.)



Figure 325.—A fruiting cane of 'Malling Jewel' red raspberry showing 'catkin' symptom of the flowers. (Copyright Scottish Crop Research Institute.)



Figure 323.—Red raspberry seedlings. Left: normal seedling; right: seedling showing frilly leaf symptom. (Copyright Scottish Crop Research Institute.)





Figure 326.—Detail of 'catkin' symptom in fig. 325.  
(Copyright Scottish Crop Research Institute.)



Figure 327.—Lateral-leaf crinkle symptom in a fruiting cane of 'Glen Clova' red raspberry. (After Jennings 1977; copyright Scottish Crop Research Institute.)

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