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INTERNATIONAL PLANT
GENETIC RESOURCES INSTITUTE

FAO/IPGRI TECHNICAL GUIDELINES

FOR THE

SAFE MOVEMENT OF SMALL FRUIT GERmplasm



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In collaboration with the
**Small Fruit Virus Working Group
of the
International Society for
Horticultural Science**

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INTRODUCTION

Collecting, conservation and utilization of plant genetic resources and their global distribution are essential components of international crop improvement programmes.

Inevitably, the movement of germplasm involves a risk of accidentally introducing plant quarantine pests* along with the host plant material; in particular, pathogens that are often symptomless, such as viruses, pose a special risk. In order to minimize, this risk, effective testing (indexing) procedures are required to ensure that distributed material is free of pests that are of quarantine concern.

The ever-increasing volume of germplasm exchanged internationally, coupled with recent rapid advances in biotechnology, has created a pressing need for crop-specific overviews of the existing knowledge in all disciplines relating to the phytosanitary safety of germplasm transfer. This has prompted FAO and IPGRI to launch a collaborative programme for the safe and expeditious movement of germplasm, reflecting the complementarity of their mandates with regard to the safe movement of germplasm. FAO, as the depository of the International Plant Protection Convention of 1951, has a long-standing mandate to assist its member governments to strengthen their Plant Quarantine Services, while IPGRI's mandate - *inter alia* - is to further the collecting, conservation and use of the genetic diversity of useful plants for the benefit of people throughout the world.

The aim of the joint FAO/IPGRI programme is to generate a series of crop-specific technical guidelines that provide relevant information on disease indexing and other procedures that will help to ensure phytosanitary safety when germplasm is moved internationally.

The technical guidelines are produced by meetings of panels of experts on the crop concerned, who have been selected in consultation with the relevant specialized institutions and research centres. The experts contribute to the elaboration of the guidelines in their private capacities and do not represent the organizations to whom they belong. FAO, IPGRI and the contributing experts cannot be held responsible for any failures resulting from the application of the present guidelines. By their nature, they reflect the consensus of the crop specialists who attended the

* The word 'pest' is used in this document as it is defined in the revised edition of the International Plant Protection Convention. It encompasses all harmful biotic agents ranging from viroids to weeds.

meeting, based on the best scientific knowledge available at the time of the meeting. The experts that have contributed to this document are listed after this introduction.

The technical guidelines are written in a short, direct, sometimes 'telegraphic' style, in order to keep the volume of the document to a minimum and to facilitate updating. The guidelines are divided into two parts: The first part makes general recommendations on how best to move germplasm of the crop concerned and mentions available intermediate quarantine facilities when relevant. The second part covers the important pests and diseases of quarantine concern. The information given on a particular pest or disease does not pretend to be exhaustive but concentrates on those aspects that are most relevant to quarantine. Where possible, acronyms for viruses are according to Hull *et al.* (1991)**

The present guidelines were developed at a meeting held in Corvallis, Oregon from 13 to 15 August, 1992 in collaboration with the Small Fruit Virus Working Group of the International Society of Horticultural Science (ISHS). The meeting was hosted by the USDA-ARS National Clonal Germplasm Repository.

** Hull, R., Milne, R.G. & Van Regenmortel, M.H.V. 1991. A list of proposed standard acronyms for plant viruses and viroids. *Arch. Virol.* **120**:151-164.

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GENERAL RECOMMENDATIONS

- Plant material should be obtained directly from the lowest risk source available: This could be one of the institutions listed below where virus-tested germplasm is maintained, or one of the other sources listed in the same country or continent than to move it over or longer distances.
- All material should be kept in containment and undergo indexing as well as therapy procedures, if necessary, and retesting before being released.
- Germplasm that is infected with a pathogen of quarantine concern which cannot be eradicated readily should be maintained in containment until suitable therapy methods become available and have been applied successfully. In case healthy germplasm is available from other sources, the infected material should be destroyed.

TECHNICAL RECOMMENDATIONS

Small fruit germplasm can be moved as pollen, seed, *in vitro* material or as vegetative propagules.

A. Pollen

- Pollen should be collected from pathogen-tested plants in containment.
- Pollinated mother plants and progeny seedlings derived from other pollen sources should be tested for pollen-transmitted viruses and virus-like agents known to occur in that crop.
- Imported pollen, found to carry arthropod pests and fungal pathogens of bees, should be destroyed or treated and retested.

B. Seed

- Seed should be free of pulp, air-dried, inspected for the absence of insect pests and fumigated when necessary.
- Seed may be surface-disinfected with 0.5% sodium hypochlorite for 10 minutes at room temperature to avoid externally seed-borne pathogens.
- Seeds should be germinated in sterilized potting media and grown in an insect-proof facility.
- Seedlings should be tested for seed-transmitted viruses.

C. *In vitro* material

- *In vitro* material should be derived from pathogen-tested sources.
- *In vitro* material obtained from other sources should be tested for viruses and other systemic pathogens known to occur in that crop.
- For the movement of *in vitro* material, neither charcoal, fungicides or antibiotics should be added to the medium.
- *In vitro* material should be shipped in transparent containers and visually inspected for bacteria, fungi and arthropods. contaminated material should be treated or destroyed.

D. Vegetative propagules

- Vegetative propagules should be derived directly from sources that have been pathogen-tested following the recommendations in this guide and found free from infection.
- Vegetative propagules from sources not tested for pathogens should only be used when *in vitro* material or seed is not available.
- Runner tips or stem cuttings should be obtained in preference to roots, rooted cuttings or rooted plants.

- Vegetative propagules should be washed free of soil, visually inspected, and treated for external arthropod and nematode pests by either fumigation with an ovicidal material or dipping in an appropriate insecticide.
- Vegetative propagules should be established in a sterilized potting mix and maintained in a containment facility. A systemic insecticide (e.g. aldicarb or oxamyl) should be placed in the root zone at the time of planting to kill any possible arthropods and foliar nematodes within the plant tissue.
- Once established, vegetative propagules should be indexed according to the tidelines outlined below.

E. Disease indexing

Many of the pathogens found in small fruit species are latent and cannot be detected by visual assessment. It is essential that all material, including plants derived from tissue culture, be extensively tested.

To optimize the sensitivity of the biological tests, donor source plants and indicator plants should be actively growing and be free from pests and diseases. These conditions are best provided in insect-proof glasshouse facilities with good temperature control to maintain 20-25°C and supplementary high light intensity during 14-18 h per day.

Mechanical inoculation. Tests are best made in early spring when plants are actively growing. Soft, young donor plant leaves or, where possible for *Vaccinium*, flower petals, should be homogenized in a 2% nicotine alkaloid solution in water. Concentrated nicotine alkaloid is a very hazardous and poisonous substance and should be handled with great care. If finger inoculation is used, plastic gloves should be worn, and inhalation of the fumes be avoided as much as possible. In many cases 0.05 M phosphate buffer with 2% polyvinyl pyrrolidone (PVP; MW 40,000) is a good alternative. A suitable abrasive should be used either in the extraction buffer (e.g. Celite) or dusted on the test plant leaves prior to inoculation (e.g. 300-mesh carborundum). Test plants should be inoculated as soon as the extract is made and should be rinsed with water immediately after inoculation. Cucumber cotyledons should be inoculated before the tip of the first true leaf is

exposed more than 3-4 mm; *Chenopodium quinoa* should be inoculated when less than 15 cm tall; *Nicotiana bethamiana* should be used when 3-6 leaves are suitable for inoculation and before flower buds appear. Plants should be observed for symptoms for up to three weeks after inoculation.

Graft-inoculation. Actively growing donor and recipient plants should be used. Bottle grafts are often used for *Ribes*, *Rubus* and *Vaccinium* and leaflet grafts for *Fragaria*. Grafts are considered successful if leaflet grafts appear viable after two weeks and bottle grafts after three to four weeks. Depending on the virus and host species, symptoms may appear after incubation times ranging from weeks (*Fragaria*) to two growing seasons (reversion disease in *Ribes*). Details are given in the respective descriptions.

F. Therapy

Thermotherapy applied to well established plants for several weeks, followed by excision of explants from treated plants, is the method of choice for the four genera. Explants are regenerated on a nutrient medium *in vitro* to fully developed plants. It should be noted that not all plants resulting from a thermotherapy are pathogen-free. Therefore, successfully regenerated plants should be indexed according to the procedures recommended in these guidelines. In the case of commercial varieties, evaluation for trueness to horticultural type is necessary.

Fragaria

Well rooted plants in large porous pots should be placed in a growth chamber at ambient temperature. A gradual 2°C increase per day should be employed to a constant 38°C. Plants should be held at 38°C for at least 21 days (longer if the virus is heat stable, e.g. strawberry veinbanding virus). Explants of 0.5 mm or smaller should be excised and regenerated on a nutrient medium *in vitro* to whole plants. Testing for pathogens should be done after plants have gone through natural winter dormancy.

Vaccinium

Vaccinium corymbosum is very sensitive to high temperature. Plant growth at 38°C is improved by placing plants in a CO₂-enriched atmosphere (1200 ppm CO₂). New shoot cuttings 10-20 mm long are taken after 6 weeks of growth under these conditions. After treating cut ends with rooting hormone (1000 ppm IBA), cuttings

are successfully regenerated in mist propagation beds. The pH of the rooting medium in the mist bed should be between 3.5 and 5.5.

Rubus

After 3 weeks of thermotherapy at 36-38°C meristem tips of 0.5 mm should be excised and regenerated. Alternatively, *Rubus* primocane tips, 5 mm long, can be mist-bed propagated after a longer treatment period (6-8 weeks), following rooting hormone dip treatment of the base of cuttings. Also root pieces may be heat-treated.

Ribes

Use adequate nutrients to assure vigorous growth. *Ribes* may not tolerate constant temperatures above 35°C during thermotherapy. Three weeks at 36°C has been adequate to eliminate several viruses of *Ribes*. In cases where meristem culture is difficult for tissue culture, prolonged exposure (up to 6 weeks) at temperatures below 36°C may permit production of some clean plants from 5-10 mm shoot tips.

General References

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DESCRIPTIONS OF PESTS

Fragaria spp. (strawberry)

Viruses

1. Iarviruses

Two ilarviruses are reported in *Fragaria* - tobacco streak virus (TSV) and *Fragaria chiloensis* ilarvirus (FCIV). They have quasi-isometric particles of 22-35 nm in diameter; for FCIV bacilliform particles up to 57 nm long have also been reported.

Symptoms

TSV is symptomless or causes mild transient symptoms on commercial strawberry cultivars. FCIV is symptomless in *F. chiloensis*.

Host range

TSV occurs in commercial strawberry cultivars and is known to have a wide natural host range including both herbaceous and woody plants. FCIV occurs in wild *F. chiloensis*.

Geographical distribution

TSV was reported from Australia, Israel and North America; occurs probably worldwide. FCIV was detected in Chile in the Andes and coastal mountain areas.

Transmission

Thrips (*Frankliniella occidentalis* and *Thrips tabaci*) have been reported as vectors of TSV in several annual crops. No vectors are known for FCIV. Both viruses are transmitted through pollen and seed.

Therapy

Heat therapy (38°C) for six weeks followed by tissue culture of meristem tips 0.5 mm in length should eliminate TSV in a high percentage of resulting plants.

Indexing

ELISA is reliable for leaves, seeds and pollen. Leaflet grafted *F. vesca* indicator clones show symptoms 10-30 days after grafting. New growth may appear normal but plants remain infected.

References

- Kaiser, W.J., Wyatt, S.D. & Pesho, G.R. 1982. Natural hosts and vectors of tobacco streak virus in eastern Washington. *Phytopathology* **72**:1508-1512.
- Spiegel, S., Martin, R.R., Leggett, F., ter Borg, M. & Postman, J. 1993. Characterization and geographical distribution of a new ilarvirus from *Fragaria chiloensis*. *Phytopathology* **83**:991-995.
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2. Nepoviruses

Five nepoviruses are important in *Fragaria* spp.: arabis mosaic (ArMV), raspberry ringspot (RRSV), strawberry latent ringspot (SLRSV), tomato black ring (TBRV) and tomato ringspot (ToRSV). Virus particles are isometric, about 30 nm in diameter, profiles angular.

Symptoms

Newly infected plants may show blotchy leaf mottling and occasional necrosis on leaf tissue; infected plants become progressively stunted and eventually die.



Fig. 1. Necrosis of young leaves in *Fragaria vesca* var. *sempreflorens* 'Alpine' two weeks after grafting from a plant infected with tobacco streak virus. (Dr. R.H. Converse, Horticultural Crops Research Laboratory, Corvallis)

Host range

Nepoviruses occur in *Fragaria* spp. and many other wild and cultivated hosts. The experimental host range includes many commonly used test plants.

Geographical distribution

AMV, RRSV, SLRSV and TBRV occur in strawberry in Europe. ToRSV occurs in native *F. chiloensis* along the coast of northern California and in commercial strawberry in North America.

Transmission

In nature transmitted by nematode vectors belonging to the Dorylaimidae. A high level of pollen and seed transmission occurs in *Fragaria* spp.

Therapy

No information is available for *Fragaria*.

Indexing

Nepoviruses are readily detected by ELISA. However, many serological variants are described that may not react with all antisera. Nepoviruses can be mechanically transmitted to herbaceous hosts, which are satisfactory for virus detection but not for identification.

Reference

Sections on nepoviruses in: *Virus Diseases of Small Fruits*. Ed. R.H. Converse. United States Department of Agriculture, Agriculture Handbook No. 631, Washington, D.C.

3. Pallidosis**Cause**

The pallidosis agent (PA) is a probable virus of undetermined morphology and identity. Pallidosis disease (PD) is associated with several high molecular weight double-stranded RNA (dsRNA) bands of $4.3\text{-}5.2 \times 10^6$ daltons in diseased plants.

Symptoms

In strawberry cultivars PA is either latent or causes very mild, non-diagnostic symptoms.

Fig. 2. Pallidosis disease - left: distorted, chlorotic leaves in graft-inoculated *F. virginiana* clone UC-10; right: healthy plant. Dr. R.H. Converse, Horticultural Crops Research Laboratory, Corvallis)



Host range

Fragaria spp.

Geographical distribution

Australia (probably introduced from the US), Canada and USA.

Transmission

PA is seed-borne in *F. vesca* and spreads in the field by unknown means.

Therapy

Meristem tip culture.

Indexing

PA causes epinasty, distortion, chlorosis and dwarfing in graft-inoculated *F. virginiana* clones UC-10 or UC-11, and no symptoms in graft-inoculated *F. vesca* clones. 'The presence of dsRNA bands in the 4.3-5.2 x 10⁶ dalton range allows presumptive identification of PA in complex infections where other viruses can mimic the symptoms of PA on clones UC-10 or UC-11.

References

Fulton, J.P. 1987. Strawberry pallidosis. pp. 55-56. In: *Virus Diseases of Small Fruits*. Ed. R.H. Converse. United States Department of Agriculture, Agriculture Handbook No. 631, Washington, D.C.

Yoshikawa, N. & Converse, R.H. 1990. Strawberry pallidosis disease: distinctive dsRNA species associated with latent infections in indicators and in diseased strawberry cultivars. *Phytopathology* **80**:543-548.

4. Strawberry crinkle virus (SCrV)

A rhabdovirus with membrane-bound bacilliform particles, 69 x 190-380 nm.

Symptoms

Infected cultivars may be symptomless. Sensitive strawberry cultivars may have dwarfed, distorted, crinkled, or otherwise deformed leaves with chlorotic to necrotic vein-associated spots. Symptoms are more severe when other viruses are present.

Host range

Fragaria spp.

Geographical distribution

Worldwide.

Transmission

By strawberry aphids (mainly *Chaetosiphon fragaefolii*) in a persistent circulative fashion. There is a 14 days minimum latent period from acquisition to transmission.



Fig. 3. Strawberry crinkle in *Fragaria vesca* clone UC-4. (Dr. R.H. Converse, Horticultural Crops Research Laboratory, Corvallis)

Therapy

Thermotherapy and meristem tip culture.

Indexing

Leaflet grafting onto clones of *F. vesca*. Symptoms vary from slight angular epinasty to severe leaflet crinkling. Petiole and stolon lesions, often causing bending at the lesion, are diagnostic symptoms for SCrV infection.

Reference

Frazier, N.W., Sylvester, E.S. & Richardson, J. 1987. Strawberry crinkle. pp. 20-25. In: *Virus Diseases of Small Fruits*. Ed. R.H. Converse. United States Department of Agriculture, Agriculture Handbook No. 631, Washington, D.C.

5. Strawberry latent C virus (SLCV)

A rhabdovirus with membrane-bound bacilliform particles, 68 x 190-380 nm.

Symptoms

Infected cultivars may be symptomless. In indicator plants *F. vesca* clone UC-5 SLCV produces diagnostic symptoms of severe epinasty in new leaves, followed by dwarfing, mottling and distortion of subsequent leaves without epinasty; clones UC-4 and UC-6 are symptomless.



Fig. 4. Symptoms of strawberry latent C virus several months after grafting on *Fragaria vesca* 'EMK'.
(Dr. R.H. Converse, Horticultural Crops Research Laboratory, Corvallis)

Host range

Fragaria spp.

Geographical distribution

Eastern North America and Japan.

Transmission

In nature SLCV is persistently transmitted by *Chaetosiphon fragaefolii* aphid. Experimentally, it also has been transmitted by leaflet grafting and by dodder.

Therapy

Thermotherapy and meristem tip culture.

References

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- Yoshikawa, N., Inouye, T. & Converse, R.H. 1986. Two types of rhabdovirus in strawberry. *Ann. Phytopathol. Soc. Japan* 52:437-444.

6. Strawberry mild yellow-edge**Cause**

A luteovirus with isometric particles of 25 nm diameter and a potyvirus with rod-shaped particles of about 14 x 500 nm are associated with the disease.



Fig. 5. Symptoms of strawberry mild yellow-edge disease in *Fragaria vesca* clone UC-4 45 days after grafting: mottling of young leaves and premature senescence. (Dr. R.H. Converse, Horticultural Crops Research Laboratory, Corvallis)

Symptoms

Strawberry cultivars are symptomless carriers of strawberry mild yellow-edge disease (SMYED). In combination with other viruses, symptoms vary and include chlorosis, stunting and marginal or overall chlorosis.

Host range

In addition to cultivated strawberries, SMYED has been found in nature in *F. chiloensis* in Chile and in several *Duchesnea* spp. in Japan.

Geographical distribution

Worldwide.

Transmission

Naturally by aphids, mostly in the genus *Chaetosiphon*, in a persistent or circulative manner. *C. fragaefolii* seems to be the major vector. Experimentally, SMYED can be graft- or aphid-transmitted to *F. vesca* indicators.

Therapy

Thermotherapy and meristem tip culture.

Indexing

Leaflet grafting onto sensitive clones of *F. vesca*. Clone UC-4 and 'Alpine' seedlings usually show typical symptoms within 3 weeks after inoculation while clone UC-6 remains symptomless. Potex virus particles can be detected in crude sap of SMYED-infected plants by ELISA and by immunosorbent electron microscopy.

References

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- Spiegel, S. & Martin, R.R. 1992. Detection of strawberry mild yellow-edge disease in micropropagated strawberry plantlets. *Acta Hort.* **308**:61-68.

7. Strawberry mottle virus (SMoV)

A virus with isometric particles about 32-37 nm in diameter.

Symptoms

SMoV exhibits great strain variation. Severe strains of SMoV may reduce vigour and fruit yield. SMoV frequently occurs along with other aphid-borne viruses which results in more severe symptoms.

Host range

Fragaria spp., *Duchesnea chrysantha*, *D. indica* and *Potentilla sunaica*.

Geographical distribution

Worldwide.

Transmission

SMoV is transmitted naturally in a semipersistent manner by *Chaetosiphon* spp. and *Aphis gossypii*. SMoV is experimentally transmitted by these and five other genera of aphids. Mechanical transmission to *C. quinoa* is possible but inconsistent.

Therapy

SMoV is one of the easiest of the strawberry viruses to eliminate by thermotherapy. Entire plants grown at constant 37°C were freed of SMoV in 10-14 days. Meristem tip culture was effective in eliminating SMoV from cultivars.



Fig. 6. Symptoms of a moderate strain of strawberry mottle virus in *fragaria vesca* 'EMC'. (Dr. R.H. Converse, Horticultural Crops Research Laboratory, Corvallis)

Indexing

Leaflet grafting or transmission by *C. fragaefolii* to *F. vesca* clones UC-4, UC-5 and UC-6, as well as *F. vesca* 'Alpine' seedlings. Initial symptoms on young leaves vary from asymmetric net vein chlorosis and epinasty to leaf tip necrosis. Mild strains cause persistent leaf mottling symptoms. Intermediate strains cause fine vein clearing and fusing but no mottle. Leaves are small and distorted.

References

- Mellor, F.C. & Krczal, H. 1987. Strawberry mottle. pp. 10-16. In: *Virus Diseases of Small Fruits*. Ed. R.H. Converse. United States Department of Agriculture, Agriculture Handbook No. 631, Washington, D.C.
- Yoshikawa, N. & Converse, R.H. 1991. Purification and some properties of strawberry mottle virus. *Ann. Appl. Biol.* **118**:565-576.

8. Strawberry pseudo mild yellow-edge virus (SPMYEV)

A carlavirus with rod-shaped particles, 12 x 625 nm.

Symptoms

Infected strawberry cultivars are symptomless.

Host range

Fragaria spp., *Duchesnea chrysantha*, *D. indica*, and *Rubus parvifolius*.

Geographical distribution

Japan, USA.



Fig. 7. Stippling symptom on an old 'leaf of *Fragaria vesca* 'Alpine', characteristic of strawberry pseudo mild yellow-edge virus infection. (Dr. R.H. Converse, Horticultural Crops Research Laboratory, Corvallis)

Transmission

Naturally transmitted by the aphids *Chaetosiphon fragaefolii* and *Aphis gossypii* in a semipersistent manner.

Therapy

Unknown.

Indexing

ELISA or dot immunoblot assay. Leaflet grafting onto *F. virginiana* clone UC-10 and *F. vesca* clones UC-4 and UC-5. On *F. vesca*, older leaves become mottled, may exhibit vein yellowing or stippling, later turn necrotic, and die prematurely. These symptoms closely resemble those caused on this indicator species by strawberry mild yellow-edge disease.

References

- Frazier, N.W. 1987. Strawberry pseudo-mild yellow edge. pp. 63-64. In: *Virus Diseases of Small Fruits*. Ed. R.H. Converse. United States Department of Agriculture, Agriculture Handbook No. 631, Washington, D.C.
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9. Strawberry vein banding virus (SVBV)

A caulimovirus with isometric particles of 40-50 nm diameter.

Symptoms

Non-diagnostic chlorosis may develop in some susceptible strawberry cultivars. Distinctive symptoms only occur in indicator plants.

Host range

Fragaria spp.

Geographical distribution

Australia, Brazil, Europe, Japan and North America.

Fig. 8. Symptoms of strawberry vein banding virus infection in *Fragaria vesca* 'Alpine'. (Dr. R.H. Converse, Horticultural Crops Research Laboratory, Corvallis)



Transmission

In nature, SVBV is aphid-transmitted in a semipersistent manner, primarily by *Chaetosiphon fragaefolii*. Five other aphid genera have transmitted this virus experimentally.

Therapy

Thermotherapy and meristem tip culture.

Indexing

Leaflet grafting onto *F. vesca* clone UC-6 or *F. virginiana* clone UC-12. The virus is very unevenly distributed among leaves of infected cultivars and indicator plants and is best detected in symptom-bearing leaves. SVBV is detectable by dot hybridization using homologous cDNA probes.

References

- Frazier, N.W. & Morris, T.J. 1987. Strawberry vein banding. pp. 16-20. In: *Virus Diseases of Small Fruits*. Ed. R.H. Converse. United States Department of Agriculture, Agriculture Handbook No. 631, Washington, DC.
- Stenger, D.C., Mullin, R.H. & Morris, T.J. 1988. Isolation, molecular cloning, and detection of strawberry vein banding virus DNA. *Phytopathology* **78**:154-159.

Diseases of unknown etiology

1. Chlorotic fleck

Cause

Probably a virus.

Symptoms

Latent in most strawberry cultivars.

Host range

Fragaria spp.

Geographical distribution

Reported only from the state of Louisiana in the USA.

Transmission

Experimentally to *F. vesca* and *F. virginiana* by leaflet grafting and by the aphid *Aphis gossypii*. Not mechanically transmissible.

Therapy

Thermotherapy, 35°C for 40 days or longer, and meristem tip culture.



Fig. 9. Chlorotic fleck symptoms in *Fragaria vesca* indicator plant.
(Dr. R.H. Converse, Horticultural Crops Research Laboratory, Corvallis)

Indexing

By leaflet grafting to *F. vesca* and *F. virginiana* indicator clones. In *F. vesca* indicators young leaves are distorted; vein clearing followed by small chlorotic flecks is sometimes evident. Chlorotic fleck agent is not clearly distinguished from other viruses by its symptomatology.

Reference

Fulton, J.P. 1987. Strawberry chlorotic fleck. p. 60. In: *Virus Diseases of Small Fruits*. Ed. R.H. Converse. United States Department of Agriculture, Agriculture Handbook No. 631, Washington, D.C.

2. June yellows of strawberry

Cause

Unknown. No viruslike particles nor other recognizable pathogens were observed in tissues exhibiting symptoms of this non-graft-transmissible strawberry disorder. However, a group of double-stranded RNA (dsRNA) species have been associated with June yellows (JY) symptoms in strawberry.

Symptoms

Leaf blades of affected cultivated strawberry plants develop clearly delimited, usually sectorial, chlorotic areas. Symptoms diminish in warm weather. White streaks may also appear and persist in leaf blades. Plants affected with JY usually become progressively stunted over a few seasons and die prematurely.

Host range

Fragaria spp.

Geographical distribution

Worldwide.



Fig. 10. Symptoms of June yellows disorder in 'Cambridge Favourite'. Left: leaf from a healthy plant; other leaves show delimited chlorotic areas. (Scottish Crop Research Institute, Invergowrie)

Transmission

In seed and by vegetative propagation. JY behaves as if it were a genetic trait and can be transmitted by either parent of a seedling. JY may not appear in the clonal progeny of a seedling for a number of asexual generations and then reappear. JY is not transmissible by grafting, by sap inoculation, or by vectors.

Therapy

JY has not been eliminated by thermotherapy or meristem tip culture.

Indexing

There is no satisfactory procedure for detecting JY in symptomless plants, such as potential parental lines for breeding. The dsRNAs that have been associated with JY are present at too low a level for routine testing and have only been found in plants exhibiting JY symptoms. Progeny testing by selfing is the best currently available technique for screening potential parents to avoid JY in seedlings. Much otherwise excellent strawberry genetic material is currently inaccessible to plant breeders because of the threat of JY transmission to progeny.

References

- Hughes, J. d'A. 1989. Strawberry June yellows - a review. *Plant Path.* **38**:146-160.
- Watkins, C.A., Roberts, I.M. & Jones, A.T. 1992. Ultrastructural changes associated with June Yellows in strawberry and with leaf yellowing symptoms of viral and genetic origin in *Fragaria*, *Rubus* and *Ribes*. *Ann. Appl. Biol.* **121**:151-160
- Watkins, C.A., Jones, A.T., Mayo, M.A., & Mitchell, M.J. 1990. Double-stranded RNA analysis of strawberry plants affected by June yellows. *Ann. Appl. Biol.* **117**:73-83.
- Wills, A.B. 1987. Strawberry June yellows. pp. 69-72. In: *Virus Diseases of Small Fruits*. Ed. R.H. Converse. United States Department of Agriculture, Agriculture Handbook No. 631, Washington, D.C.

3. Leafroll

Cause

Unknown. No viruslike particles or other identified pathogens were observed in tissues exhibiting symptoms.

Symptoms

In cultivated strawberries and *Fragaria* indicator plants, the diagnostic symptom is downward roiling of leaflet margins, varying from slight rolling to complete inward rolling of both leaflets to form a tubular shape. Leaves are chlorotic, rugose and exhibit vein clearing. All cultivars are susceptible, and there is no latency. The disease, while limited in occurrence, is very damaging to infected plants.

Host range

Fragaria spp.

Geographical distribution

Northeastern part of North America and Kazakhstan.

Transmission

Natural spread occurs by unknown means. The agent is transmitted by grafting, but not mechanically.

Therapy

This disease agent is not inactivated by thermotherapy.



Fig. 11. Symptoms of strawberry leafroll in *Fragaria vesca* clone UC-5. (Dr. R.H. Converse, Horticultural Crops Research Laboratory, Corvallis)

Indexing

Leafroll is self-evident in cultivated strawberries and in many *Fragaria* species. *F. vesca* clone UC-3 inoculated with leafroll by leaflet grafting is very susceptible, while *F. vesca* clones UC-4 and UC-6 are symptomless. After grafting, the incubation period may be as long as six months.

Reference

Frazier, N.W. 1987. Strawberry leafroll. pp. 61-62. In: *Virus Diseases of Small Fruits*. Ed. R.H. Converse. United States Department of Agriculture, Agriculture Handbook No. 631, Washington, D.C.

4. Vein yellowing

Cause

Probably a virus.

Symptoms

In cultivated strawberries and *Fragaria vesca* indicator plants, the diagnostic symptom is very striking vein yellowing. This symptom is obvious in spring and autumn but is masked in summer. The disease is not damaging to infected plants.

Host range

Fragaria spp.

Geographical distribution

Japan.



Fig. 12. Strawberry vein yellowing disease. (Dr. N. Yoshikawa, Iwate University, Morioka)

Transmission

Natural spread occurs by unknown means. Experimentally, it is transmitted by leaflet grafting. Aphid transmission tests using *Chaetosiphon fragaefolii* and *Aphis gossypii* were negative. Not mechanically transmissible.

Therapy

Not reported.

Indexing

F. vesca clones UC-4, UC-5 or UC-6 show symptoms one to two weeks after grafting of symptomless, infected leaflets.

Reference

Yoshikawa, N. & Inouye, T. 1988. Strawberry viruses occurring in Japan. *Acta Hort.* **236**:59-67.

Prokaryotic diseases - 'MLOs'

1. Aster yellows

Cause

Aster yellows (AY), a non-cultivable mollicute, often referred to as a mycoplasma-like organism (MLO). Strawberry green petal MLO which is identical to clover phyllody MLO, has been assigned to Type III, one of three major AY Types.

Symptoms

Cultivated strawberries become dwarfed, with chlorotic, cupped leaves, turning red, later brown and flat on the ground. Flowers show virescent petals and various degrees of phyllody and flower proliferation, depending on the AY strain, cultivar and time of infection. No immunity or latency are known for AY in *Fragaria*.

Host range

In addition to *Fragaria* spp., AY has a host range of more than 300 species in 50 families.

Geographical distribution

North America. Similar diseases occur in Europe, Russia and Japan.

Transmission

By leafhoppers. Western North American strains of AY are transmitted by 27 leafhopper species, of which *Macrostelus fascifrons*, *Colladonus montanus*, and *C. geminatus* have experimentally transmitted AY to or from *Fragaria* but do not favour *Fragaria* as a host. Therefore other leafhoppers may be 'important in the spread of AY to strawberry in western North America. Eastern strains of AY are only transmitted by *M. fascifrons*.

Therapy

No specific information for strawberry. In periwinkle, AY was eliminated by hot water therapy.

Indexing

AY produces symptoms on all *Fragaria* species and cultivars tested, and generally is eventually lethal in cultivars. AY is difficult to distinguish from green petal when the two mollicutes are co-existent. Antisera against AY do not recognize green petal MLO (non-cultivable mollicute) and vice versa.

References

- Chiykowski, L.N. 1987. Aster yellows in strawberry. pp. 31-34. In: *Virus Diseases of Small Fruits*. Ed. R.H. Converse. United States Department of Agriculture, Agriculture Handbook No. 631, Washington, D.C.
- Clark, M.F. & Davies, D.L. 1984. Mycoplasma detection and characterization. pp 108-109. In: *East Malling Res. Stn. Annu. Rpt.* 1983.
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2. Strawberry green petal

Cause

A phloem-limited non-cultivable mollicute, often referred to as a mycoplasma-like organism (MLO). The pathogen is also known to cause clover phyllody and was recently classified as Type III of aster yellows MLO (non-cultivable mollicute).

Fig. 13. Strawberry green petal disease - green flower petals and affected (above), dwarfed leaves with chlorotic margins (below). (Dr. R.H. Converse, Horticultural Crops research Laboratory, Corvallis)



Symptoms

In infected strawberry cultivars, petals adhere, turn green and eventually may turn pink. In later stages of the disease petals may become leaflike (phylloid). Fruits remain small, hard and green. Foliage is dwarfed, cupped and chlorotic. Plants form few, shorter runners and die within a few months.

Host range

Fragaria spp. and several species of clover (*Trifolium* spp.).

Geographical distribution

North America (where it is particularly serious in northeastern Canada), Australia, Europe, New Zealand, and Russia.

Transmission

The disease agent is transmitted in nature in a persistent manner by several leafhopper species, but only *Aphrodes bicincta* has been demonstrated to transmit to and from strawberry. Other species such as *Macrostelus fascifrons* and species of *Euscelis* are involved in field spread in clover and other plant species. Transmitted experimentally by dodder and graft-inoculation, but not by mechanical inoculation of sap extracts.

Therapy

Propagation of rapidly growing shoots from heat-treated plants was effective in eradicating the agent from infected periwinkle. Application of tetracycline antibiotics can cause the remission of symptoms.

Indexing

Green petal disease is evident in infected cultivars. It can be distinguished from other aster yellows types by bioassay using dodder to infect celery, a differential host in which the pathogen is symptomless. Can be detected and identified serologically by ELISA from infected strawberry sap.

References

- Chiykowski, L.N. 1962. Clover phyllody and strawberry green petal diseases, caused by the same virus in eastern Canada. *Can. J. Bot.* **40**:397-404.
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- Posnette, A.F. & Ellenberger, C.E. 1963. Further studies of green petal and other leafhopper-transmitted viruses infecting strawberry and clover. *Ann. Appl. Biol.* **51**:69-83.

3. Witches'-broom and multiplier disease

Cause

A non-cultivable mollicute, often referred to as a mycoplasma-like organism (MLO).

Symptoms

Witches'-broom (WB) infected strawberries are dwarfed, bushy, sterile, have numerous crowns, small leaves with spindly petioles, and severely broomed daughter plants on short stolons. Strawberry plants infected with multiplier disease (MD) are dwarfed with leaflets cupping upwards, and petioles short and erect. Crown proliferation and short rulers are also characteristic. The few small flowers that are formed bear normal-sized fruit.

Fig. 14. Witches'-broom and multiplier disease in cv. 'Sparkle'. (Dr. R.H. Converse, Horticultural Crops Research Laboratory, Corvallis)



Host range

WB and MD are known only in *Fragaria* spp.

Geographical distribution

North America.

Transmission

Leafhoppers are suspected but unproved vectors.

Therapy

No specific information for strawberry.

Indexing

Fragaria virginiana clone UC-12 is the best graft-inoculated indicator host for WB, and *F. chiloensis* for MD. Both diseases cause severe crown proliferation and dwarfing in graft-inoculated indicator plants.

References

Converse, R.H. 1987. Strawberry witches'-broom and multiplier diseases. pp. 66-68. In: *Virus Diseases of Small Fruits*. Ed. R.H. Converse. United States Department of Agriculture, Agriculture Handbook No. 631, Washington, D.C.

- Heimann, M.F., Spear, R.N., & Jeffers, S.N. 1992. Mycoplasma-like organisms associated with strawberry multiplier disease. *Phytopathology* **82**: 1133 (abstract).
- Sehgal, O.P. & Boone, D.M. 1963. Multiplier disease of strawberry in Wisconsin. *Plant Dis. Repr* **47**: 46-48.

Prokaryotic diseases-bacteria

1. Strawberry angular leaf spot

Cause

Xanthomonas fragariae Kennedy & King.

Symptoms

Minute water-soaked lesions on the lower leaf surface enlarge to become angular spots delimited by small veins. Lesions are translucent when viewed by transmitted light, but dark green when viewed with reflected light. A viscous bacterial exudate may form on lesions under high moisture conditions. When it dries, the exudate forms a whitish, scaly film. Plants may be defoliated when disease is severe.

Host range

Fragaria spp.

Geographical distribution

Brazil, Ethiopia, France, Italy (Sicily), USA, Venezuela.

Biology and transmission

Primary inoculum comes from overwintering leaf debris. The pathogen is very resistant to desiccation and other adverse conditions. Very little is known about the epidemiology.

Therapy

None is recommended.

Indexing

It is recommended, when clones are put into tissue culture, to routinely place the initial explants for 2 or 3 weeks on a medium that promotes the growth of bacteria

(e.g. 40 g Trypticase Soy Agar in 1000 ml of water, or on media containing peptone plus sucrose or glucose). Those meristems that remain free of bacterial growth are transferred to a medium allowing meristem growth.

References

- Bradbury, J.F. 1977. *Xanthomonas fragariae*. CMI Descriptions of Pathogenic Fungi and Bacteria No. 558. Commonwealth Agricultural Bureaux, Slough.
- Maas, J.L. 1984. Angular leaf spot. pp. 41-42. In: *Compendium of Strawberry Diseases*. Ed. J.L. Maas. APS Press, St. Paul.

2. Strawberry bacterial wilt

Cause

Pseudomonas solanacearum E.F. Smith.

Symptoms

The earliest field symptoms are dropping of the older leaves of youngest plants. Infected plants may show wilting during the hottest period of the day and recover during cooler periods.

Host range

Not known if isolates of *P. solanacearum* from strawberry are specific for strawberry, or if isolates of *P. solanacearum* from other hosts are also pathogenic to strawberry.

Geographical distribution

Japan, Taiwan.

Biology

Infected plants die within a few days after the appearance of the first symptoms. Older plants appear resistant to the bacterium; *P. solanacearum* invades only a limited number of xylem trachea cells even in young plants. Parenchymatous tissues are attacked preferentially and large lysigenous cavities are formed and become filled with bacteria.

References

- Hsu, Y.H. 1987. Control of bacterial wilt of strawberry by planting with healthy seedlings. *Plant Prot. Bull. Taiwan* **29**:199-201.

Kawaguchi, K., Ohta, K. & Goto, M. 1981. Studies on bacterial wilt of strawberry plants caused by *Pseudomonas solanacearum*. 2. beta-D-glucogallin, the antibacterial substance detected in the tissues of strawberry plants. *Ann. Phytopathol. Soc. Japan* **47**:520-527.

3. Marginal chlorosis of strawberry

Cause

An elongated bacterium has been uniquely associated with this disease.

Symptoms

Dwarfing of plants, cupping and yellowing of leaves, especially at leaflet margins.

Host range

Cultivated strawberry.

Geographical distribution

Widespread and serious in France and Spain in nurseries and in fruiting fields.

Transmission

Marginal chlorosis spreads in the field by unknown means.

Therapy

No information.

Indexing

Self-indicating.

Reference

Nourrisseau, J.G., Lansac, M. & Garnier, M. 1993. Marginal chlorosis, a new disease of strawberries associated with a bacteriumlike organism. *Plant Dis.* **77**:1055-1059.

Fungal diseases

1. *Alternaria* leaf spot

Cause

Alternaria alternata f.sp. *fragaria* Dingley.

Symptoms

Circular to irregular brown to black lesions, 2-5 mm in diameter, develop on upper leaf surfaces and may have dark reddish purple margins. The lesions on the lower leaf surface appear greyish brown. Elongate, dark, depressed lesions may occur on and kill petioles and fruit.

Host range

Cultivated strawberries.

Geographical distribution

Belgium, France, Japan, Korea, Netherlands, New Zealand.

Biology and transmission

This fungus species is not morphologically distinguishable from other *A. alternata* forms, however, the form attacking strawberry leaves is a distinct pathotype from other *Alternaria* forms that commonly infect ripe strawberry fruit. Flowers and fruits may also be infected, but this occurs less frequently. The disease spreads by air-borne spores (conidia) from infected plant material. Disease development is favoured by humid, warm conditions and overcrowding of plants. Cultivars differ in susceptibility to *Alternaria* leaf spot; highly sensitive cultivars may be killed by severe attacks.

References

- Bal, E., Gilles, G., Creemers, P. & Verheyden, C. 1983. Problems and control of *Alternaria* leaf spot on strawberries. *Med. Fac. Landbouww. Rijksuniv. Gent* **48**:637-646.
- Cho, J.T. & Moon, B.J. 1980. Occurrence of strawberry black leaf spot caused by *Altenaria alternata* (Fr.) Keissler in Korea. *Korean J. Plant Prot.* **19**:221-227.
- Dingley, J.M. 1970. Records of fungi parasitic on plants in New Zealand, 1966-68 *N.Z.J. Agric. Res.* **13**:325-337.

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2. Anthracnose

Cause

Colletotrichum spp.; *C. fragariae* A.N. Brooks, *C. acutatum* Simmonds, *C. gloeosporioides* Penz. [*Glomerella cingulata* (Ston.) Spauld. & Schrenk].

Symptoms

Symptoms range from fruit rot to stolon, petiole, and leaf lesions and crown rot. Fruit rot begins as light to dark brown, slightly depressed lesions on unripe or ripe fruits with pink to buff masses of spores (conidia) on the lesion surface. Fruit lesions are firm and dry. Lesions on petioles and fruit trusses are dark, elongate, slightly depressed, and often develop into a canker that kills the petiole or fruit truss. Blossoms of highly susceptible cultivars may be attacked and killed. Anthracnose black leaf spots developing on young expanding leaves are round and 3 mm or less in diameter. Strawberry plants affected by crown rot develop a reddish brown, firm rot or streaking in interiors of crowns of wilting plants. Infected plants generally wilt suddenly (collapse) and die.

Host range

In addition to strawberry, *Cassia obtusifolia* for *C. fragariae*; *C. acutatum* and *C. gloeosporioides* each have a large host range of other fruit, vegetable, and non-food plants.

Geographical distribution

Almost worldwide.

Biology and transmission

Spores from infected plants are blown or carried by splashing water or insects to healthy plants. Ground cover (plastic, straw, etc.) greatly affects spore dispersal from fruit infections. High moisture and temperature favour disease development. The fungi are able to over-season on some alternative hosts and in infected plant debris in soil. The disease may be carried to healthy fields with nursery plants if their stolons or petioles are infected.

References

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3. Fusarium wilt

Cause

Fusarium oxysporum f.sp. *fragariae* Winks & Williams.

Symptoms

Symptom development of this systemic disease in strawberry is favoured by high temperature, which cause leaves of infected plants to wilt and die rapidly. Sudden plant collapse is similar to that caused by some other wilt diseases. Leaf chlorosis may or may not develop. Crowns show distinct reddish brown discoloration, and as the disease advances, the lower crown tissues may decay completely.

Host range

Cultivated strawberries.

Geographical distribution

Australia, Japan, Korea.

Biology and transmission

Fusarium wilt is a soil-borne disease that begins as infection of roots and crowns and becomes systemic. The fungus may be isolated from petioles of infected plants. The fungus can survive in soil in infected strawberry plant debris, but it does not infect other crop or cover plants in rotation with strawberry. The disease is favoured by high temperature and plant stress during fruiting. Infection by *F. oxysporum* f.sp. *fragariae* should not be confused with other *Fusarium* spp. that also infect strawberry roots or fruit. Cultivars differ in susceptibility to Fusarium wilt, thus it is possible that the pathogen may be carried systemically in symptomless and apparently healthy plant material of resistant cultivars.

Reference

Wilhelm, S. 1984. Fusarium wilt (yellows). pp. 97-98. In: *Compendium of Strawberry Diseases*. Ed. J.L. Maas. APS Press, St. Paul.

4. Phytophthora crown rot

Cause

Phytophthora cactorum (Lebert & Cohn) Schröt. Strawberry crown rot is induced by specific strains of *P. cactorum* pathogenic to strawberry and not to apple or other crops. Not to be confused with strawberry fruit rot, also caused by *P. cactorum* strains that occur in temperate regions.

Symptoms

The youngest leaves wilt suddenly and often turn blush-green. Wilting quickly spreads to include all leaves, typically within a few days. Crowns show necrosis and intensive browning of the vascular tissue. Generally, roots are unaffected until the entire crown is destroyed.

Host range

Cultivated strawberries.

Geographical distribution

Europe, Australia and USA.

Biology and transmission

The fungus is a common soil-inhabitant in temperate regions, however, apparently only certain strains infect strawberry crowns while most strains infect strawberry fruit. Strawberry isolates apparently are not pathogenic to other crop hosts or

weed species. Warm, wet weather favours infection and high temperature enhances disease development. Stress conditions and short day-length promote symptom development. Specificity of conditions necessary for symptom development is important, otherwise infections may remain latent in plants.

References

- Seemüller, E. 1984. Crown rot (vascular collapse). pp. 83-85. In: *Compendium of Strawberry Diseases*. Ed. J.L. Maas. APS Press, St. Paul,
- Seemüller, E. & Schmidle, A. 1979. Einfluss der Herkunft von *Phytophthora cactorum*-Isolaten auf ihre Virulenz an Apfelrinde, Erdbeerrhizomen und Erdbeerfrüchten. *Phytopathol. Z.* **94**:218-225.

5. Strawberry black root rot

Cause

Black root rot is a collective descriptive term for a complex of diseases having more than one possible causal agent. Symptoms may be brought on by injurious environmental conditions such as freezing or water logging of the soil, by pathogenic fungi or nematodes, or most likely, by a combination of these factors. The fungi *Rhizoctonia* spp., *Pythium* spp. and many others have been associated with black root rot, as have *Pratylenchus* spp. nematodes.

Symptoms

Symptoms on roots generally are apparent when infections of lateral and main roots occur. Dark lesions enlarge and girdle roots, eventually infecting the entire root system. Cortex tissues are rotted, while steles of roots remain white. Quite often, cortical tissues of roots can be pulled away from steles.



Fig. 15. Strawberry black root rot. (Dr. J.L. Maas, USDA-ARS Fruit Laboratory, Beltsville)

Geographical distribution

Black root rot occurs world-wide, however specific biotic causes may be localized.

Biology

The disease is generally associated with certain soil types, especially those with high clay contents. The disease is not of a systemic nature.

Reference

Wilhelm, S. 1984. Black root rot. p. 90 In: *Compendium of Strawberry Diseases*. Ed. J.L. Maas. APS Press, St. Paul.

6. Strawberry red stele (red core)

Cause

Phytophthora fragariae var. *fragariae* Hickman.

Symptoms

Young roots rot first at the tip, and when the root is cut open lengthwise, the stele above the rot is red. As the rot progresses, lateral roots are quickly destroyed, giving the main roots a 'rat-tail' appearance. The red discoloration of the stele often progresses to the crowns of susceptible plants. Only roots are infected. The red stele disease ranges in extent from sporadic infection of root tips in strawberry cultivars with the highest resistance to total root destruction in those with the lowest resistance. Infected plants in the field may occur in irregular patches rather than in rows. Diseased susceptible plants become stunted, produce small or no fruit, and eventually die.

Host Range

Species of *Dryas*, *Geum*, *Potentilla*, and *Rubus* have been infected experimentally and *Rubus* spp. may become infected naturally in infested areas, posing potential risks to strawberry plantings if they are transferred as planting material from infested soil to clean soil that may be later used for strawberry production. This pathogen is not to be confused with *P. fragariae* var. *rubi*, the cause of *Phytophthora* root rot of *Rubus* (see *Rubus* diseases).

Geographical distribution

Temperate strawberry growing regions.

Biology and transmission

At least 15 physiologic races of *P. fragariae* are known worldwide, and strawberry cultivars differ in susceptibility to the races. Spores of the fungus (zoospores) are released in soil during wet, cool periods. These spores germinate on tips of main or lateral roots and infection proceeds toward the stele of the main root. Roots begin to rot from the tip a few days after infection and eventually, rotted roots containing zoospores become incorporated into the soil. The zoospores may germinate to produce more zoospores, or they may remain inactive in soil for many years before germinating. The fungus may be moved to new areas in infected roots of susceptible or resistant plants, plant debris, or in infested soil adhering to plant roots. High rainfall, poor soil drainage, and low temperature favour the disease.

References

- Montgomerie, I.G. 1984. Red stele root rot. pp. 79-83. In: *Compendium of Strawberry Diseases*. Ed. J.L. Maas. APS Press, St. Paul.
- Pepin, H.S. 1967. Susceptibility of members of the Rosaceae to races of *Phytophthora fragariae*. *Phytopathology* **57**:782-784.

7. Verticillium wilt

Cause

Verticillium albo-atrum Reinke & Berth. and/or *V. dahliae* Kleb.

Symptoms

Outer leaves of the strawberry plant show marginal and interveinal browning and eventually collapse. Inner leaves are stunted, but tend to remain green and turgid until the plants die. The wilting symptom generally distinguishes *Verticillium* wilt from other root and crown diseases that cause wilting of all leaves at the same time. First-year strawberry plantings are affected most severely.

Host range

A large number of crop and weed species.

Geographical distribution

Throughout temperate zones of the world.

Biology and transmission

Pathogens can over-season in infected plant debris as small, dormant structures (microsclerotia). Infection occurs in the soil through spores (conidia) or hyphae in

contact with plant roots. Infections destroy roots, causing plants to wilt and die. The disease is systemic; *Verticillium* can be readily isolated from petioles of infected plants. Verticillium wilt is often most prevalent and severe in arid or semiarid, irrigated areas and is favoured by periods of environmental stress, such as high temperature and drought. Disease Spread from plant to plant is negligible. *Verticillium* may be disseminated to new locations by water, wind, on infected planting stock, in crop or weed debris, or in soil. Strawberry cultivars differ in susceptibility to this wilt and isolates of *Verticillium* differ in pathogenicity.

References

- Maas, J.L. & Galletta, G.J. 1989. Germplasm evaluation for resistance to fungus-incited diseases. *Acta Hort.* **265**:461-472.
- Maas, J.L., Galletta, G.J. & Draper, A.D. 1989. Resistance in strawberry to races of *Phytophthora fragariae* and to isolates of *Verticillium* from North America. *Acta Hort.* **265**:521-526.
- Wilhelm, S. 1984. Verticillium wilt. pp. 87-90. In: *Compendium of Strawberry Diseases*. Ed. J.L. Maas. APS Press, St. Paul.

Ribes spp. (currant, gooseberry)

Viruses

1. Alfalfa mosaic virus (AMV)

Virus particles are bacilliform and of different lengths; the longest usually about 60 nm. All particles are approximately 16 nm wide.

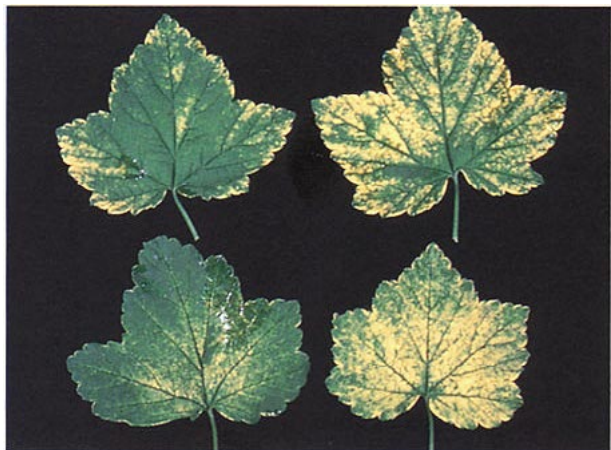


Fig. 16. Spring symptoms of interveinal white mosaic in red currant, caused by alfalfa mosaic virus. (Dr. R.H. Converse, Horticultural Crops Research Laboratory, Corvallis)

Symptoms

Interveinal white mosaic. Leaves of affected plants show yellow or white patches which often occur between the main veins. Leaves produced later in the season may show only yellow or white flecking on the outer margins.

Host range

Although AMV has a wide natural host range extending over several genera, *Ribes* seems to be infected only rarely.

Geographical distribution

AMV occurs worldwide, but is reported in *Ribes* only from Europe.

Transmission

AMV is transmitted in nature by many species of aphids in a non-persistent manner and also through the seed of some hosts. Experimentally, AMV is transmitted by mechanical inoculation of sap to herbaceous test plants and by graft-inoculation.

Therapy

No specific information for *Ribes*.

Indexing

Mechanical inoculation of sap extracted in 2% nicotine alkaloid solution to *Chenopodium quinoa* (see underlined note of caution on p. 10), which develops both local lesions and systemic mosaic. The development of systemic symptoms in *C. quinoa* distinguishes these isolates from cucumber mosaic and tobacco rattle viruses which do not infect this host systemically. The presence of AMV should be confirmed serologically to distinguish it from other possible viruses.

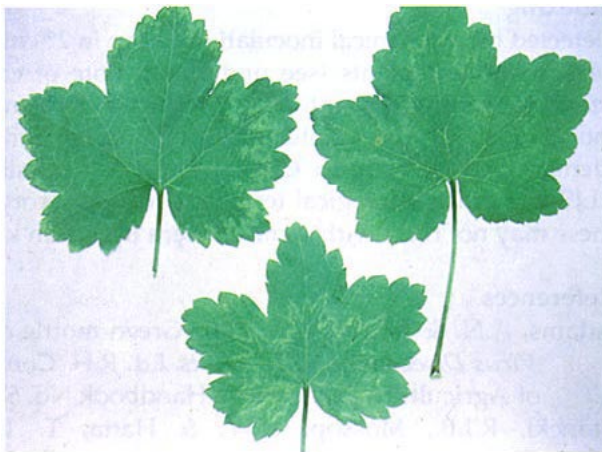
References

- Jaspers, E.M.J. & Bos, L. 1980. Alfalfa mosaic virus. CMI/AAB Descriptions of Plant Viruses No. 229. Commonwealth Agricultural Bureaux, Slough.
- van der Meer, F.A. Interveinal white mosaic. pp. 153-155. In: *Virus Diseases of Small Fruits*. Ed. R.H. Converse. United States Department of Agriculture, Agriculture Handbook No. 631, Washington, D.C.

2. Cucumber mosaic virus (CMV)

Virus particles are isometric, about 30 nm in diameter.

Fig. 17. Green mottle disease in red currant, caused by cucumber mosaic virus. (Dr. R.H. Converse, Horticultural Crops Research Laboratory, Corvallis)



Symptoms

Green mottle disease in *Ribes*. Symptoms are variable, depending on the *Ribes* species and cultivar infected as well as time of infection and environmental conditions. Symptoms in sensitive material are chlorotic line-patterns, often associated with leaf veins, or mottling; they are most obvious in rapidly growing plants in spring and in fully expanded leaves.

Host range

CMV has a very wide host range covering several genera. *Ribes* seems to be infected only occasionally.

Geographical distribution

World-wide in many crop species. Has been found in Europe in *Ribes*.

Transmission

Transmitted in a non-persistent manner by many species of aphids, including those that colonize *Ribes* spp., and through the seed of some host plants. Transmitted experimentally by grafting and by mechanical inoculation of sap extracts to herbaceous plants.

Therapy

Eradicated by thermotherapy at 36°C for 3 weeks, followed by apical tip propagation.

Indexing

Detected by mechanical inoculation of sap in 2% nicotine alkaloid solution to many herbaceous test plants (see underlined note of caution on p. 10). *Chenopodium quinoa* is sensitive to infection producing local necrotic lesions a few days after inoculation; it is not infected systemically. Serological tests are necessary to identify and distinguish CMV from other possible viruses. Also detectable by ELISA or other serological tests, but several serotypes of the virus are known and these may not react with some antisera in certain kinds of tests.

References

- Adams, A.N. & Thresh, J.M. 1987. Green mottle of black currant. pp. 136-137. In: *Virus Diseases of Small Fruits*. Ed. R.H. Converse. United States Department of Agriculture, Agriculture Handbook No. 631, Washington, D.C.
- Francki, R.I.B., Mossop, D.W. & Hatta, T. 1979. Cucumber mosaic virus. CMI/AAB Descriptions of Plant Viruses No. 213. Commonwealth Agricultural Bureaux, Slough.
- van der Meer, F.A. 1987. Green mottle of red currant. pp. 145-146. In: *Virus Diseases of Small Fruits*. Ed. R.H. Converse. United States Department of Agriculture, Agriculture Handbook No. 631, Washington, D.C.

3. Gooseberry vein banding virus (GVBV)

Possibly a member of the badnavirus group; in infected plants small unenveloped bacilliform particles of about 30 x 130 nm were found.

Symptoms

Chlorotic banding along the main veins of leaves which may be distorted in severely affected plants. Symptoms are most evident in early spring growth and become less obvious later in the season. Symptom expression is also greatly influenced by cultivar and species.

Host range

Reported only from *Ribes* spp. More research is required to determine the properties of isolates in the three cultivated *Ribes* spp.

Geographical distribution

Common in Europe and in countries of the former Soviet Union. Found in North America and New Zealand probably as a result of importing infected material.

Fig. 18. Symptoms of gooseberry vein banding virus infection in red currant.
(Dr. J. Postman, USDA-ARS-NCGR, Corvallis)



Transmission

Transmitted in a semi-persistent manner by several species of aphids, including *Aphis grossulariae*, *A. schneideri*, *Cryptomyzus ribis*, *Hyperomyzus lactucae*, *Myzus persicae* and *Nasonova ribisnigri*. Transmitted experimentally by grafting and, with great difficulty, by mechanical inoculation of sap to some herbaceous plants.

Therapy

Eradicated from red currant by thermotherapy at 38°C for 3 weeks followed by shoot-tip propagation. Gooseberry is heat sensitive, so that thermotherapy of infected plants is more difficult. The virus has been eradicated from plants by meristem tip culture alone, and a combination of thermotherapy at a lower temperature, followed by meristem tip culture.

Indexing

Graft-inoculation to sensitive gooseberry indicator cultivars or clones such as 'Leveller' or B1385/81.

References

Adams, A.N. & Posnette, A.F. 1987. Gooseberry vein banding. pp. 129-130. In: *Virus Diseases of Small Fruits*. Ed. R.H. Converse. United States Department of Agriculture, Agriculture Handbook No. 631, Washington, D.C.

- Adams, A.N. & Thresh, J.M. 1987. Vein clearing and vein net disease of black currant. pp. 137-138. In: *Virus Diseases of Small Fruits*. Ed. R.H. Converse. United States Department of Agriculture, Agriculture Handbook No. 631, Washington, DC.
- Jones, A.T., McGavin, W.J. & Watkins, C.A. 1992. Recent studies in Scotland on the aetiology of virus and virus-like diseases of small fruit crops. *Acta Hort.* **308**:31-35.
- van der Meer, F.A. 1987. Red currant vein banding. pp. 143-145. In: *Virus Diseases of Small Fruits*. Ed. R.H. Converse. United States Department of Agriculture, Agriculture Handbook No. 631, Washington, D.C.
- Wood, G.A. 1991. Three graft-transmissible diseases and a variegation disorder of small fruit in New Zealand. *N.Z.J. Crop Hortic. Sci.* **19**:313-323.

4. Nepoviruses

Virus particles are isometric, about 30 nm in diameter with a angular outlines; a proportion of the particles are penetrated by negative stains.

Symptoms

Symptoms vary depending on the cultivar, virus and environmental conditions. Symptoms may range from chlorotic mottling or line-pattern to mosaic; symptomless infection also occurs. Symptoms are most obvious in early spring growth and are often less noticeable as the season progresses.

Host range

Nepoviruses infect a wide range of hosts covering many genera, including several weed species.



Fig. 19. Chlorotic mottling in red currant, caused by tomato ringspot virus. (Dr. J. Postman, USDA-ARS-NCGR, Corvallis)

Geographical distribution

Nepoviruses occur worldwide, but infection in *Ribes* is not common. Arabis mosaic virus (ArMV), strawberry latent ringspot virus (SLRSV), raspberry ringspot virus (RRSV) and tomato black ring virus (TBRV) are reported in *Ribes* in Europe. Tomato ringspot virus (ToRSV) occurs in North America.

Transmission

Transmitted in nature through soil by nematodes of the genus *Longidorus* (RRSV, TBRV) and *Xiphinema* (ArMV, SLRSV, ToRSV) and through seed of some hosts. Transmitted experimentally by grafting and by mechanical inoculation of sap extracts to herbaceous test plants.

Therapy

ToRSV was eradicated from red currant by thermotherapy for three weeks at 38°C followed by apical tip propagation. No information available for the other nepoviruses in *Ribes*.

Indexing

Detected by mechanical inoculation of sap extracted in 2% nicotine alkaloid solution to herbaceous test plants (see underlined note of caution on p. 10). *Chenopodium quinoa* produces chlorotic or necrotic local lesions followed by systemic necrosis. However, serological tests are necessary to identify and distinguish nepoviruses from one another and from other viruses. ELISA alone is not recommended for the detection of RRSV, TBRV and ToRSV as a range of serological variants exist that may not react with certain antisera.

References

Sections on nepoviruses in: *Virus Diseases of Small Fruits*. Ed. R.H. Converse. United States Department of Agriculture, Agriculture Handbook No. 631, Washington, D.C.

5. Tobacco rattle virus (TRV)

TRV has straight tubular particles of two predominant lengths; the longer are about 190 nm and the shorter 50-115 nm, depending on the virus isolate. Particle width is approximately 23 nm.

Symptoms

Reported only from red currant and *R. sanguineum* in which the virus induces chlorotic or yellow mosaic and line-pattern symptoms. Symptoms are restricted to only a few branches and usually only in the first-formed leaves of these branches.

Fig. 20. Yellow mosaic and line-pattern symptoms in *Ribes sanguineum*. (Dr. R.H. Converse, Horticultural Crops Research Laboratory, Corvallis)



Host range

TRV infects a wide range of host plants that extends over several genera, but infects *Ribes* only rarely.

Geographical distribution

Reported in *Ribes* only from the Netherlands and Germany, but the virus probably occurs world-wide.

Transmission

The virus is transmitted through soil by nematodes in the genera *Paratrichodorus* and *Trichodorus* and also through the seed of some hosts. Transmissible experimentally by grafting and by inoculation of sap extracts to herbaceous test plants.

Therapy

None reported for *Ribes* as plants are infected only rarely.

Indexing

Detected by mechanical inoculation of sap, extracted from symptom-bearing leaves of *Ribes* in 2% nicotine alkaloid solution, to *Chenopodium amaranticolor* or *C. quinoa* in which TRV induces spreading necrotic local lesions within a few days; most virus isolates do not become systemic. See underlined note of caution on p. 10. Serological tests and/or electron microscopy of infected test plants are necessary to identify and distinguish TRV from other possible viruses. The virus cannot be detected reliably by ELISA with the available antisera because of the wide range of serological variants that exist in nature.

References

- Robinson, D.J. & Harrison, B.D. 1989. Tobacco rattle virus. AAB Descriptions of Plant Viruses No. 346. Association of Applied Biologists, Wellesbourne.
- van der Meer, F.A. 1987. Leaf pattern of red currant. pp. 150-152. In: *Virus Diseases of Small Fruits*. Ed. R.H. Converse. United States Department of Agriculture, Agriculture Handbook No. 631, Washington, D.C.

Diseases of unknown etiology

1. Black currant yellows

Cause

Unknown.

Symptoms

Symptoms are noticeable in early spring as chlorotic or yellow flecks in newly expanding leaves. These flecks and spots increase in number and may merge to produce an overall yellowing of the leaf. Leaves emerging during the more rapid phase of summer growth are often symptomless.

Host range

Reported only in Ribes.



Fig. 21. Black currant yellows in 'Baldwin'. (Scottish Crop Research institute, Invergowrie)

Geographical distribution

Reported only in Britain, but similar symptoms have been observed in black currant plants in Europe and New Zealand.

Transmission

No vector has been identified. Natural spread in the field is slow. Experimentally, the agent is transmissible by grafting, but not by mechanical inoculation of sap extracts to herbaceous plants.

Therapy

Attempts to eradicate the agent from infected plants by thermotherapy were not successful.

Indexing

Graft-inoculation to the sensitive black currant cultivars 'Baldwin' or 'Amos Black'.

Reference

Thresh, J.M. 1987. Black currant yellows. pp. 140-141. In: *Virus Diseases of Small Fruits*. Ed. R.H. Converse. United States Department of Agriculture, Agriculture Handbook No. 631, Washington, D.C.

2. Reversion of red and black currant

Cause

Unknown. Reports that the causal agent is potato virus Y or a mycoplasma-like organism have not been confirmed by other workers.



Fig. 22. Reversion disease of black currant, symptoms of infection with strain E. Infected plants are characterized by the absence of downy hairs and the-brighter colour of flower buds (left) compared to the healthy plant (right). (Scottish Crop Research Institute, invergowrie)

Symptoms

Symptoms do not usually appear until at least one year (sometimes longer) after infection and then often in only a few branches. Full systemic infection may take up to three to five years. At least two strains are recognized: the common European strain (E), and a more virulent strain (R) present in Scandinavia, eastern and central Europe, and countries of the former Soviet Union. Flower bud symptoms induced by these strains are quite distinct. In black currant, infection with strain E results in decreased hairiness of the sepals of the unopened buds so that they appear more brightly coloured compared with the grey, downy appearance of normal buds. In addition to these symptoms, infection with strain R also results in much darker pigmentation of the sepals and, most characteristically, sepal division to form 10 instead of the usual 5 sepals ('double' flowers). Symptoms in leaves are much less diagnostic, being influenced greatly by cultivar and season. Some sensitive black currant cultivars show a transient chlorotic line-pattern usually associated with the main veins. Other leaf symptoms include decreased marginal serrations and number of main veins, and a smaller basal sinus. Flower bud and leaf symptoms are much less obvious in red currant.

Host range

Only reported from *Ribes* spp.

Geographical distribution

Common in Europe, countries of the former Soviet Union, New Zealand; not reported from the Americas.



Fig. 23. Reversion disease of black currant, symptoms of infection with strain R. Infected plants are characterized by darker pigmentation of the sepals and 'double' flowers, as well as reverted leaves (left) compared to the healthy plant (right). (Scottish Crop Research Institute, Invergowrie)

Transmission

Transmitted in nature by the black currant gall mite, *Cecidophyopsis ribis*. These mites colonize young buds in late spring and early summer and their feeding induces the formation of distinctive galling of the buds. Tens of thousands of mites may be found in a single galled bud. Plants affected with reversion are more susceptible to infestation by mites than healthy plants. Experimentally, the reversion agent is transmitted by grafting, but not by mechanical inoculation of sap to herbaceous plants.

Therapy

Reversion-free plants have been obtained from infected ones by thermotherapy of plants and tip grafting the heat-treated shoots to healthy black currant seedlings.

Indexing

In black currant, flower bud symptoms are the most reliable for diagnosis; some cultivars also show obvious leaf symptoms. In red currant, symptoms are much less 'noticeable than in black currant. Because of the slow development of symptoms and the erratic distribution of the agent in infected plants, they should be observed for two flowering seasons. For uncertain material and for red currant, graft-inoculation to reversion-sensitive black currant cultivars such as 'Baldwin', 'Ojebyn' and 'Silver Gieters' is necessary. Because of the erratic distribution of the reversion agent in plants, scions for grafting should be selected from three separate branches of each plant to be tested. Grafted plants should be observed for symptoms for two flowering seasons.

References

- Adams, A.N. & Thresh, J.M. 1987. Reversion of black currant. pp. 133-136. In: *Virus Diseases of Small Fruits*. Ed. R.H. Converse. United States Department of Agriculture, Agriculture Handbook No. 631, Washington, D.C.
- Campbell, A.I. 1965. The inactivation of black currant reversion by heat therapy. pp. 89-92. *Long Ashton Res. Stn. Report for 1964*.
- Wood, G.A. 1991. Three graft-transmissible diseases and a variegation disorder of small fruit in New Zealand. *N.Z.J Crop Hortic. Sci.* **19**:313-323.

3. Wildfire of black currant**Cause**

Unknown.

Symptoms

Symptoms occur in spring as chlorotic spotting of the leaves; spots may coalesce to form large chlorotic areas along and between the main veins. Sometimes chlorosis takes the form of rings or streaks. Tissues in the centre of chlorotic areas become thin and translucent. In severely affected plants leaves are deformed and small. Symptoms become less intense in leaves produced during hot summer conditions.

Host range

Reported only in *Ribes*.

Geographical distribution

Reported only from Siberia and the Far East of the former Soviet Union.

Transmission

Transmitted in nature by the aphid *Aphis grossulariae*, but the mode of transmission is not known. Experimentally, the agent of the disease is readily transmitted between *Ribes* plants by grafting, but not by mechanical inoculation of sap to herbaceous plants.

Therapy

Prolonged thermotherapy of entire plants or, for shorter periods, shoot tips, is reported to free plants from infection.

Indexing

Observations of characteristic symptoms in infected plants or in graft-inoculated plants of the sensitive Russian black currant cultivar 'Lisavenko's Black'. Symptoms appear 3-5 weeks after graft-inoculation.

Reference

Kalinitchenko, A.N. & Gladkych, V.I. 1976. Wildfire of black currants. *Acta Hort.* **66**:85-89.

4. Yellow leaf spot of currant**Cause**

Unknown.

Symptoms

Red currant plants may show chlorotic or yellow spotting, often more noticeable at the outer leaf margins, but also found scattered over the whole leaf. Symptom expression is very dependent on the cultivar and growing season. Black currant seems to be symptomlessly infected.

Host range

Reported only from red currant.

Geographical distribution

Reported only from Europe.

Transmission

No information on natural spread, but the agent is graft-transmissible.

Therapy

No information.

Indexing

Detected only by symptoms in plants or by graft-inoculation to the sensitive red currant cultivars 'Laxton No.1' or 'Fay's Prolific'. However, mechanical inoculation of sap to *Chenopodium quinoa* should be attempted to determine the presence of alfalfa mosaic virus which can induce symptoms in red currant similar to yellow leaf spot.

Reference

van der Meer, F.A. 1987. Yellow leaf spot of red currant. pp. 152-153. In: *Virus Diseases of Small Fruits*. Ed. R.H. Converse. United States Department of Agriculture, Agriculture Handbook No. 631, Washington, D.C.

Prokaryotic disease

Full blossom of currant

Cause

The causal agent appears to be a non-cultivable mollicute, often referred to as a mycoplasma-like organism (MLO).

Symptoms

Flower malformations are most typical and include some of the following: absence of stamens, presence of more than one style, petals with sepal-like appearance, leaf-like development of petals or sepals, and misshapen berries.

Host range

Reported only in *Ribes*.

Geographical distribution

Reported only from Czechoslovakia.

Transmission

No information on natural spread. Experimentally, the causal agent can be transmitted by graft-inoculation.

Therapy

No information.

Indexing

Detected by symptoms on flowering plants or in graft-inoculated plants of the red currant cultivar 'Houghton Castle'.

References

- Rakus, D., Kralik, O. & Brack, J. 1974. Mycoplasma V. *Ribes houghtonianum* Jancz. nakazene 'plnokvetosti'. *Sbornik Ustav Vedeckotechmckych Informaci Ochrana Rostlin* **10**:307-309.
- van der Meer, F.A. 1987. Full blossom of red currant. pp. 155-157. In: *Virus Diseases of Small Fruits*. Ed. R.H. Converse. United States Department of Agriculture, Agriculture Handbook No. 631, Washington, D.C.

Fungal diseases**1. American powdery mildew****Cause**

Sphaerotheca mars-uvae (Schwein.) Berk. & Curt.



Fig. 24. American powdery mildew (*Sphaerotheca mars-uvae*) on black currant. (Dr. P.R. Bristow, Washington State University, Puyallup)

Symptoms

The lower portion of plants usually show the first symptoms. Tips of young shoots are attacked and may become distorted. Leaves are covered on both sides with a white mycelium which turns rusty-brown with age. Leaves and berries in all stages are attacked. Infected berries may be dwarfed, roughened and cracked. Severe infection reduces shoot growth, thus having an adverse impact on the crop in the following year.

Host range

Chiefly on gooseberry (*Ribes grossularia* and *R. hordeolum*), but also on currants (*R. sativum*, *R. rubrum* and *R. nigrum*).

Geographical distribution

Asia, Europe, North America, Australia and New Zealand.

Biology and transmission

The disease is favoured by cool, humid and rainy periods during spring and early summer. The main source of infection in the spring is ascospores released from overwintering cleistothecia. Cleistothecia form on living and on fallen leaves. On gooseberries the pathogen normally overwinters as mycelium in dormant buds. Wind-borne conidia formed on infected plant parts cause secondary infections.

Reference

Purnell, T.J. & Sivanesan, A. 1970. *Sphaerotheca mors-uvae*. CMI Descriptions of Pathogenic Fungi and Bacteria No. 254. Commonwealth Agricultural Bureaux, Slough.

2. Anthracnose (leaf spot)**Cause**

Drepanopezlia ribis (Kleb.) Höhn.
(anamorph: *Gloeosporidiella ribis* (Lib.) Petr.).

Symptoms

The disease is a leaf spot or anthracnose of currants and gooseberries. The primary symptom is small, circular or irregular spots on both leaf surfaces. Spots are dark brown and coalesce when numerous. Minute, grey acervuli develop in lesions.

Fig. 25. Anthracnose (*Drepanopeziza ribis*) on black currant. (Dr. P.R. Bristow, Washington State University, Puyallup)



Leaves become chlorotic and drop prematurely. Yellowing of leaves is more pronounced on gooseberries. All succulent plant parts including petioles, peduncles and fruit are susceptible, but leaf spotting is the most pronounced symptom. On fruit the spots resemble flyspecks. Severely infected berries crack open and drop.

Host range

Ribes spp., including currants (*R. rubrum*, *R. nigrum*) and gooseberries (*R. grossularia*, *R. hirtellum*).

Geographical distribution

Australia, Europe, Japan, New Zealand and North America.

Biology and transmission

Primary inoculum are ascospores produced in apothecia on overwintering leaves beneath plants. Ascospores are air-borne. Conidia produced in acervuli on leaf lesions are waterborne. Conidia are inoculum for the repeating secondary cycle.

References

- Blodgett, E.C. 1936. The anthracnose of currant and gooseberry caused by *Pseudopeziza ribis*. *Phytopathology* **26**:115-152.
- Booth, C. & Waller, J.M. 1979. *Drepanopeziza ribis*. CMI Descriptions of Pathogenic Fungi and Bacteria No. 638. Commonwealth Agricultural Bureaux, Slough.

Rubus spp. (blackberry, raspberry)

Viruses

1. Blackberry calico virus (BCV) (see also wineberry latent virus)

A carlavirus with flexuous, rod-shaped particles about 15 x 627 nm in length. Very similar to, and possibly the same as wineberry latent virus.

Symptoms

Yellow spots, blotchy chlorosis and ring-and-line patterns appear first on floricanes leaves. Similar symptoms develop on primocane leaves in hot weather. In some cases, red pigmentation of the chlorotic areas also occurs. Infected plants may be symptomless in cool weather.

Host range

Wild and cultivated *Rubus ursinus*, blackberry x raspberry hybrids such as boysenberry and loganberry.

Geographical distribution

North America and possibly through imported material in Europe.



Fig. 26. Blackberry calico disease in 'Marion' blackberry. (Dr. R.H. Converse, Horticultural Crops Research Laboratory, Corvallis)

Transmission

The virus spreads very slowly in the field by unknown means. Experimentally, BCV is graft-transmissible to other *Rubus* plants and is mechanically transmitted with great difficulty to *Nicotiana occidentalis* accession PI-c.

Therapy

Thermotherapy for 35 days at 37°C.

Indexing

Serological detection of BCV by ELISA is probably the fastest and most sensitive technique currently available. BCV is very irregularly distributed within naturally infected *Rubus* plants. Therefore, multiple sampling is needed for virus detection in a given plant. BCV can be detected by grafting onto loganberry seedlings which should then be grown out of doors through two complete growing seasons to permit symptom expression.

References

- Converse, R.H. 1987. Blackberry calico. pp. 245-246. In: *Virus Diseases of Small Fruits*. Ed. R.H. Converse. United States Department of Agriculture, Agriculture Handbook No. 631, Washington, D.C.
- Jones, A.T., Mitchell, M.J., McGavin, W.J. & Roberts, I.M. 1990. Further properties of wineberry latent virus and evidence for its possible involvement in calico disease. *Ann. Appl. Biol.* **117**:571-581.
- van der Meer, F.A. 1989. *Nicotiana occidentalis*, a suitable test plant in research on viruses of small fruit crops. *Acta Hortic.* **236**:27-35.

2. Black raspberry necrosis virus (BRNV)

Virus particles are isometric, about 25-30 nm in diameter and many are penetrated by negative stains.

Symptoms

Induces severe tip necrosis and mosaic in black raspberry. Symptomless in most red raspberry and blackberry cultivars, but some may show a veinal chlorotic mottle or line-pattern during early growth.

Host range

Rubus spp. and a few herbaceous test species.

Fig. 27. Veinal chlorotic mottle caused by black raspberry necrosis virus. (Dr. R.H. Converse, Horticultural Crops Research Laboratory, Corvallis)



Geographical distribution

Probably occurs wherever *Rubus* is grown. Usually occurs as a complex infection with one or more of the following viruses: raspberry leaf mottle, raspberry leaf spot, and *Rubus* yellow net to cause diseases known as veinbanding mosaic disease or raspberry mosaic. In Australasia, where the main vector aphid species are absent, little or no spread occurs apart from propagation of infected material.

Transmission

Transmitted in nature in a semi-persistent manner by the aphids *Amphorophora idaei* in Europe and *A. agathonica* in North America. Possibly other *Rubus*-infesting aphids are also able to act as vectors. Transmitted experimentally by grafting and, with great difficulty, by mechanical inoculation of sap extracts in 2% nicotine alkaloid solution to herbaceous indicator plants (see underlined note of caution on p. 10).

Therapy

Eradicated from infected plants by thermotherapy although a heat-stable isolate was reported in Canada.

Indexing

Detected by transmission to *R. occidentalis* (black raspberry) or *R. henryi* which develop apical tip necrosis and mosaic. However, identification is dependent on distinguishing it from raspberry leaf mottle and raspberry leaf spot viruses which react similarly on these indicators. BRNV is symptomless in the red raspberry indicators of the other two viruses. Mechanical inoculation is difficult to achieve because of low virus concentration in raspberry plants, but sap extracts in 2% nicotine alkaloid solution when inoculated to *Chenopodium quinoa* sometimes

induce necrotic local lesions followed by systemic chlorotic flecks or mottle. See underlined note of caution on p. 10.

Reference

Stace-Smith, R. & Jones, A.T. 1987. Black raspberry necrosis. pp. 178-183. In: *Virus Diseases of Small Fruits*. Ed. R.H. Converse. United States Department of Agriculture, Agriculture Handbook No. 631, Washington, DC.

3. Bramble yellow mosaic virus (BrYMV)

A probable potyvirus with flexuous particles of about 15 x 730 nm.

Symptoms

Pronounced yellow mosaic and line-pattern in *R. rigidus* that persists through the growing season.

Host range

Rubus spp.

Geographical distribution

Reported only from South Africa.

Transmission

Natural mode of spread is not known. Transmitted experimentally by grafting and by mechanical inoculation of sap extracts to herbaceous plants.

Therapy

No information.

Indexing

Readily transmitted from *Rubus* by mechanical inoculation of sap extracts in 2% nicotine alkaloid solution to several herbaceous species (see underlined note of caution on p. 10). *Chenopodium morale* is sensitive to infection and develops large local chlorotic or yellow lesions that are irregular in size and later become necrotic, and systemic chlorotic or yellow rings. Graft-inoculated *R. henryi* develop a transient chlorotic mottle. No antiserum is available to the virus.

Reference

Engelbrecht, D.J. 1987. Bramble yellow mosaic. pp. 243-244. In: *Virus Diseases of Small Fruits*. Ed. R.H. Converse. United States Department of Agriculture, Agriculture Handbook No. 631, Washington, D.C.

4. Cucumber mosaic virus (CMV)

Virus particles are isometric and about 30 nm in diameter.

Symptoms

Severe chlorotic blotching and line-pattern, leaf deformation, decreased vigour and plant death in wineberry (*R. phoenicolasius*), chlorotic mottling and blotching in some raspberry cultivars and symptomless infection in blackberry.

Host range

Rubus spp.

Geographical distribution

The virus occurs worldwide in many plant species. Reported in *Rubus* only from Britain and Eastern Russia.

Transmission

Transmitted in nature in a non-persistent manner by many species of aphids, including those that colonize *Rubus*; seed-transmitted in many hosts but not detected in *Rubus*. Also transmitted experimentally by grafting and by mechanical inoculation of plant sap to herbaceous plants.

Therapy

No information for *Rubus*.

Indexing

Readily detected by mechanical inoculation of sap in 2% nicotine alkaloid solution to many herbaceous test plants (see underlined note of caution on p. 10). *Chenopodium quinoa* is sensitive to infection producing local necrotic lesions; it is not infected systemically. Serological tests are necessary to identify and distinguish CMV from other possible viruses. Several serotypes are known and these may not react with some antisera in certain kinds of tests.

References

- Francki, R.I.B., Mossop, D.W. & Hatta, T. 1979. Cucumber mosaic virus. CMI/AAB Descriptions Plant Viruses No. 213. Commonwealth Agricultural Bureaux, Slough.
- Jones, A.T. 1987. Cucumber mosaic virus in raspberry. pp. 191-192. In: *Virus Diseases of Small Fruits*. Ed. R.H. Converse. United States Department of Agriculture, Agriculture Handbook No. 631, Washington, D.C.

5. Iarviruses

At least two iarviruses have been reported in *Rubus*, namely apple mosaic virus (ApMV) and tobacco streak virus (TSV) and its serological relatives. Iarvirus particles are quasi-isometric and range in diameter from 23-35 nm.

Symptoms

Iarvirus infection appears to be symptomless in *Rubus*. However, in Germany ApMV infection of some red raspberry cultivars was associated with conspicuous yellow spots and line-pattern.

Host range

Iarviruses infect a wide range of plant species. ApMV is especially common in woody perennials, including *Malus*, *Prunus*, *Rosa* and *Humulus* as well as *Rubus* species. TSV infects in addition to *Rubus* several herbaceous species including *Fragaria*.

Geographical distribution

The viruses are reported worldwide in various crops. In *Rubus*, TSV is reported only from Australia and North America and ApMV only from Germany and North America.

Transmission

Transmitted through seed and possibly through pollen. Thrips have been implicated in pollen transmission of some isolates in experiments using herbaceous plants.

Therapy

No information for *Rubus*. ApMV has been eliminated from Rosaceae by thermotherapy.

Indexing

Readily detected in young leaf tissue in early spring by mechanical inoculation of sap in 2% nicotine solution to many herbaceous plants (see underlined note of caution on p. 10). While the response of test plants varies with the virus isolate, *Chenopodium quinoa* is sensitive to infection with most isolates. The symptoms are local chlorotic or necrotic lesions, followed by systemic necrosis or 'chlorotic rings and line-patterns. Serological tests are necessary to identify and distinguish ApMV and TSV isolates from other possible viruses. Several serotypes of these two viruses are known, which may not react with some antisera in certain kinds of tests.

References

- Baumann, G., Casper, R. & Converse, R.H. 1987. Apple mosaic virus in *Rubus*. pp. 246-248. In: *Virus Diseases of Small Fruits*. Ed. R.H. Converse. United States Department of Agriculture, Agriculture Handbook No. 631, Washington, D.C.
- Jones, A.T. & Mayo, M.A. 1975. Further properties of black raspberry latent virus, and evidence for its relationship to tobacco streak virus. *Ann. Appl. Biol.* **79**:297-306.
- Stace-Smith, R. 1987. Tobacco streak virus in *Rubus*. pp. 235-239. In: *Virus Diseases of Small Fruits*. Ed. R.H. Converse. United States Department of Agriculture, Agriculture Handbook No. 631, Washington, D.C.

6. Nepoviruses

Virus particles are isometric and about 30 nm in diameter, with angular outlines; a proportion of the particles are penetrated by negative stains. The following nepoviruses have been reported in *Rubus*: arabis mosaic virus (ArMV), cherry leaf roll virus (CLRv), strawberry latent ringspot virus (SLRSV), raspberry ringspot virus (RRSV), tomato black ring virus (TBRV), cherry rasp leaf virus (CRLV), tobacco ringspot virus (TRSV), tomato ringspot virus (ToRSV) and *Rubus* Chinese seed-borne virus (RCSbV).

Symptoms

Symptoms vary depending on the cultivar, virus and environmental conditions. Symptoms may range from chlorotic mottling, line-pattern, mosaic, vein yellowing to leaf curling; symptomless infection also occurs. There is usually a progressive decline in vigour and plants become stunted. Some cultivars are prone to develop 'crumbly' fruit following infection with some nepoviruses. Symptoms are most obvious in early spring growth and are often less noticeable as the season progresses.

Host range

Nepoviruses infect a wide range of plants in many genera, including many weed species.

Geographical distribution

Nepoviruses occur worldwide, but are most common in Europe and North America. ArMV, CLRv, SLRSV, EtRSV and TBRV are reported in *Rubus* from Europe; CRLV and TRSV from North America; ToRSV from North America and Chile. CLRv has also been detected in red raspberry in New Zealand. RCSbV was detected in *Rubus* seed imported into England from China.

Fig. 28. Chlorotic mottling in red raspberry infected with cherry leaf roll virus. (Dr. AT. Jones, Scottish Crop Research Institute, Invergowrie)



Transmission

With the exception of RCSbV, for which little information exists, and of CLRV, nepoviruses are transmitted in nature through soil by nematodes of the genus *Longidorus* (RRSV, TBRV) and *Xiphinema* (ArMV, SLRSV, TRSV, ToRSV). CLRV was not transmitted experimentally by nematodes; evidence exists for its pollen transmission in some host species. All are transmitted, often to a high frequency, through the seed of some hosts. Nepoviruses can be transmitted experimentally by grafting and by mechanical inoculation of sap extracts to herbaceous test plants.

Therapy

No information available for nepoviruses in *Rubus*, but see the Nepovirus item in the strawberry section.

Indexing

Detected by mechanical inoculation of sap extracted in 2% nicotine alkaloid solution to herbaceous test plants (see underlined note of caution on p. 10). *Chenopodium quinoa* is sensitive to infection, producing chlorotic or necrotic local lesions followed by systemic mosaic or necrosis. However, serological tests are necessary to identify and distinguish nepoviruses from one another and from other possible viruses. ELISA alone is not recommended for detecting CLRV, RRSV, TBRV and ToRSV as a range of serological variants exist that may not react with certain antisera.

References

- Barbara, D.J., Ashby, S.C. & McNamara, D.G. 1985. Host range, purification and some properties of *Rubus* Chinese seed-borne virus. *Ann. Appl. Biol.* **107**:45-55.
- Jones, A.T. 1985. Cherry leaf roll virus. AAB Descriptions of Plant Viruses No. 306. Association of Applied Biologists, Wellesbourne.
- Jones, A.T., McElroy, F.D. & Brown, D.J.F. 2981. Tests for transmission of cherry leaf roll virus using *Longidorus*, *Paralongidorus* and *Xiphinema* nematodes. *Ann. Appl. Biol.* **99**:143-150.
- Jones, A.T., Mitchell, M.J. & Brown, D.J.F. 1989. Infectibility of some new raspberry cultivars with arabis mosaic and raspberry ringspot viruses and further evidence for variation in British isolates' of these two nepoviruses. *Ann. Appl. Biol.* **115**:5769.
- Murant, A.F. 1970. Arabis mosaic virus. CMI/AAB Descriptions of Plant Viruses No. 16. Commonwealth Agricultural Bureaux, Slough.
- Murant, A.F. 1970. Tomato black ring virus. CMI/AAB Descriptions of Plant Viruses No. 16. Commonwealth Agricultural Bureaux, Slough.
- Murant, A.F. 1974. Strawberry latent ringspot virus. CMI/AAB Descriptions of Plant Viruses No. 126. Commonwealth Agricultural Bureaux, Slough.
- Murant, A.F. 1978. Raspberry ringspot virus. CMI/AAB Descriptions of Plant Viruses No. 198. Commonwealth Agricultural Bureaux, Slough.
- State-Smith, R. & Hansen, A.J. 1976. Cherry rasp leaf virus. CMI/AAB Descriptions of Plant Viruses No. 159. Commonwealth Agricultural Bureaux, Slough.

7. Raspberry bushy dwarf virus (RBDV)

Virus particles are quasi-isometric and about 33 nm in diameter. They are disrupted in phosphotungstate, but stable in uranyl acetate and uranyl formate negative stains.

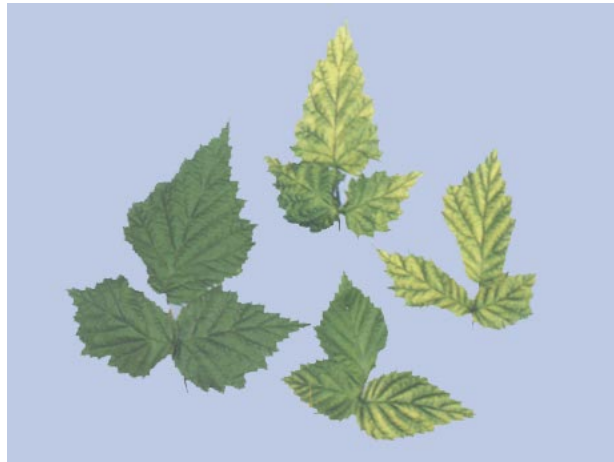
Symptoms

Often symptomless in *Rubus* plants. However, in sensitive raspberry cultivars and under ill-defined environmental conditions, it is the cause of yellows disease, characterized by yellowing of interveinal areas of leaves. These areas can merge to form rings or line-patterns or complete yellowing of the leaf. In mixed infections with aphid-borne viruses in raspberry it causes degeneration in vigour. In some cultivars it is the cause of 'crumbly' fruit.

Host range

Rubus spp.

Fig. 29. Leaves on the right show yellowing caused by raspberry bushy dwarf virus. (Dr. G. Wood, Horticulture and Food Research Institute of New Zealand, Auckland)



Geographical distribution

Worldwide. One strain, termed RB, seems restricted to central Europe, Russia and Siberia and to isolated areas in England and some parts of western Europe.

Transmission

In nature, RBDV is transmitted through seed of *Rubus* hosts and also through infected pollen to the plant pollinated. Transmitted experimentally by grafting and by mechanical inoculation of sap extracts to herbaceous plants.

Therapy

Not readily eradicated by thermotherapy alone. However, thermotherapy for several weeks at 36°C followed by propagating from shoot tips or apical meristems has been successful in eradicating the virus from plants.



Fig. 30. Crumbly fruit caused by raspberry bushy dwarf virus, compared to healthy (left). (Dr. G. Wood, Horticulture and Food Research Institute of New Zealand, Auckland)

Indexing

Readily transmitted from *Rubus* by mechanical inoculation of sap extracts in 2% nicotine alkaloid solution to several herbaceous species (see underlined note of caution on p. 10). *Chenopodium quinoa* is sensitive to infection and develops occasional local chlorotic or necrotic lesions and systemic chlorotic spots and line-patterns. Serological tests are necessary to identify and distinguish RBDV from other possible viruses. ELISA readily detects RBDV and is more reliable than bioassay for detecting black raspberry isolates which appear to have a low specific infectivity.

References

- Barbara, D.J., Jones, A.T., Henderson, S.J., Wilson, S.C. & Knight, V.H. 1984. Isolates of raspberry bushy dwarf virus differing in *Rubus* host range. *Ann. Appl. Biol.* **105**:49-54.
- Murant, A.F. 1987. Raspberry bushy dwarf. pp. 229-234. In: *Virus Diseases of Small Fruits*. Ed. R.H. Converse. United States Department of Agriculture, Agriculture Handbook No. 631, Washington, D.C.

8. Raspberry leaf mottle virus (RLMV)

No information on morphology and taxonomy.

Symptoms

Induces severe tip necrosis and mosaic in black raspberry. Most red raspberry and blackberry cultivars are infected symptomlessly or show only a transient chlorotic mottle or line-pattern in early spring growth. However, a few red raspberry cultivars show sharply defined angular chlorotic or yellow spots randomly distributed over the leaf to give a mosaic appearance. Leaves of affected plants are often smaller in size and deformed. Symptoms are usually more severe on leaves of fruiting canes than of primocanes and affected plants die within 3-5 years of infection. In mixed infections with rubus yellow net virus in sensitive red raspberry cultivars, it causes veinbanding mosaic disease, characterized by chlorosis of the lamina adjacent to the main veins; in severely affected plants leaves become puckered and distorted.

Host range

Rubus spp.

Geographical distribution

Common in Europe and detected in Australia and New Zealand probably as a result of importing infected material. Circumstantial evidence suggests it also

occurs in North America. Usually occurs as a complex infection with one or more of the following viruses: black raspberry necrosis, raspberry leaf spot, and Rubus yellow net to cause diseases known as veinbanding mosaic disease or raspberry mosaic.

Transmission

Transmitted in nature in a non-persistent manner by the aphid *Amphorophora idaei* and possibly by other aphids feeding on *Rubus* spp. Transmitted experimentally by grafting, but not by mechanical inoculation of sap extracts to herbaceous plants.

Therapy

Eradicated from plants by thermotherapy.

Indexing

Graft-inoculation to cultivars 'Mailing Delight', 'Mailing Landmark', 'St. Walfried' and 'Veten'. The commonly used virus indicators, *R. henryi* and *R. occidentalis* develop severe tip necrosis followed by mosaic when infected, but they react similarly to infection with some other aphid-borne viruses of *Rubus*.

References

- Guy, G.L., McGechan, J., Sampson, P.Y. & Stace-Smith, R. 1982. Occurrence of viruses in *Rubus* cultivars and species in Australia. *Acta Hort.* **129**:31-39.
- Jones, A.T. 1987. Raspberry leaf mottle and raspberry leaf spot. pp. 183-187. In: *Virus Diseases of Small Fruits*. Ed. R.H. Converse. United States Department of Agriculture, Agriculture Handbook No. 631, Washington, D.C.
- Jones, A.T. 1991. *Rubus* host range of rubus yellow net virus and its involvement with other aphid-borne latent viruses in inducing raspberry veinbanding mosaic disease. *Ann. Appl. Biol.* **118**:331-338.

9. Raspberry leaf spot virus (RLSV)

No information on morphology and taxonomy.

Symptoms

Induces severe tip necrosis and mosaic in black raspberry. Most red raspberry and blackberry cultivars are infected symptomlessly or show only a transient chlorotic mottle or line-pattern in early spring growth. However, a few red raspberry cultivars show sharply defined angular chlorotic or yellow spots randomly distributed over the leaf to give a mosaic appearance. Affected leaves are often smaller in size and deformed, and plants die within 3-5 years of infection. Symptoms are usually more severe on leaves of fruiting canes than of primocanes.

Fig. 31. Angular chlorotic spots caused by raspberry leaf spot virus. Left leaf from infected primocane, right from infected fruiting cane. (Dr. R.H. Converse, Horticultural Crops Research Laboratory, Corvallis)



Host range

Rubus spp.

Geographical distribution

Common in Europe and detected in Australia and New Zealand probably as a result of importing infected material. Probably of limited distribution in Australia. Circumstantial evidence suggests it also occurs in North America. Usually occurs as a complex infection with one or more of the following viruses: black raspberry necrosis, raspberry leaf mottle and *Rubus* yellow net to cause diseases known as veinbanding mosaic disease or raspberry mosaic.

Transmission

Transmitted in nature in a non-persistent manner by the aphid *Amphorophora idaei* and possibly by other aphids feeding on *Rubus* spp. Transmitted experimentally by grafting, but not by mechanical inoculation of sap extracts to herbaceous plants.

Therapy

Eradicated from plants by thermotherapy.

Indexing

Identified only by symptoms in sensitive red raspberry cultivars, or by graft-inoculation to such cultivars. Indicator cultivars include 'Burnholm', 'Glen Clova', 'Norfolk Giant' and 'Phyllis King'. The commonly used virus indicators *R. henryi* and *R. occidentalis* develop severe tip necrosis followed by mosaic when infected, but they react similarly to infection with some other aphid-borne viruses of *Rubus*.

Reference

Jones, A.T. 1987. Raspberry leaf mottle and raspberry leaf spot. pp. 183-187. In: *Virus Diseases of Small Fruits*. Ed. R.H. Converse. United States Department of Agriculture, Agriculture Handbook No. 631, Washington, D.C.

10. Raspberry vein chlorosis virus (RVCV)

Electron microscopy of ultrathin sections of infected leaves show large bacilliform particles about 65-91 x 430-560 nm which are rounded at each end. Particles have a densely staining nucleocapsid, about 50 x 70 nm in diameter, showing cross-banding with a periodicity of 4-5 nm, and surrounded by an electron-lucent zone and a unit membrane. No particles were observed in the nucleus of infected cells.

Symptoms

Symptoms are most noticeable on leaves of primocanes, appearing as a chlorosis of the fine veins, either in patches or throughout the leaf. The extent and severity of the symptoms depends on the cultivar and growing conditions.

Host range

Found only in red raspberry.

Geographical distribution

Canada, Europe, New Zealand.

Transmission

Transmitted in nature in a persistent manner by the aphid *Aphis idaei*. Transmitted experimentally by grafting, but not by mechanical inoculation of sap extracts or through seed of infected plants.

Therapy

Not eradicated from infected plants by thermotherapy alone, but plants free from the virus were obtained when this treatment was followed by rooting excised shoot tips or by meristem tip culture.



Fig. 32. Chlorosis of the fine veins caused by raspberry vein chlorosis virus. (Scottish Crop Research Institute, Invergowrie)

Indexing

Graft-inoculation to indicator red raspberry cultivars such as ‘Malling Delight’, ‘Lloyd George’ or ‘Washington’.

References

- Jones, A.T., Murant, A.F. & Stace-Smith, R. 1987. Raspberry vein chlorosis. pp. 194-197 In: *Virus Diseases of Small Fruits*. Ed. R.H. Converse. United States Department of Agriculture, Agriculture Handbook No. 631, Washington, D.C.
- Jones, A.T. & Wood, G.A. 1979. The virus status of raspberries (*Rubus idaeus* L.) in New Zealand. *N.Z. J. Agric. Res.* **22**:173-182.

11. Raspberry yellow spot virus (RYSV)

No information on morphology and taxonomy.

Symptoms

Diffuse chlorotic or yellow spots of regular size randomly distributed over the leaf. In the most severe instances the whole leaf may appear yellow. Affected leaves are often smaller in size and deformed. Symptoms are usually more severe on leaves of fruiting canes than of primocanes.

Host range

Only reported from ‘Malling Promise’ red raspberry.

Geographical distribution

Reported only from Poland, but similar symptoms have been observed in plants of ‘Malling Promise’ raspberry in Scotland.

Transmission

Reported to be transmitted in nature in a semi-persistent manner by the aphid *Amphorophora idaei*. Transmitted experimentally by grafting.

Therapy

Eradicated from infected plants by thermotherapy.

Indexing

Graft-inoculation to ‘Malling Promise’ red raspberry.

Reference

- Basak, W. 1974. Yellow spot - a virus disease of raspberry. *Bull. Acad. Pol. Sci. Cl. V. Ser. Sci. Biol.* **22**:47-51.

12. Rubus yellow net virus (RYNV)

Virus particles are bacilliform, rounded at both ends and measure 25-31 x 80-150 nm, with an electron-dense core about 17 nm in diameter; a possible member of the 'badnavirus' group.

Symptoms

Often symptomless when alone in raspberry; in sensitive cultivars it produces a chlorosis of the fine veins to form a net appearance. Infected plants show no obvious decrease in vigour. However, the virus is usually found in mixed infections with other aphid-borne viruses. In combination with some of these in sensitive raspberry cultivars it induces raspberry veinbanding mosaic disease characterized by a chlorosis along the lamina adjacent to the main veins. Such multiple infections greatly decrease plant vigour.

Host range

A few species in the genus *Rubus*.

Geographical distribution

Probably worldwide, wherever *Rubus* is grown. Usually occurs as a complex infection with one or more of the following viruses: black raspberry necrosis, raspberry leaf spot, and raspberry leaf mottle to cause diseases known as veinbanding mosaic disease or raspberry mosaic.



Fig. 33. Chlorosis of the fine veins forming a net appearance, caused by Rubus yellow net virus. (Dr. R.H. Converse, Horticultural Crops Research Laboratory, Corvallis)

Transmission

Transmitted by the raspberry aphids *Amphorophora agathonica* in North America and *A. rubi* in Europe. Other *Rubus*-infesting aphids may also serve as vectors. The vector transmits after an acquisition feed of 1 hour. There is no latent period in the vector, and aphids lose the capacity to transmit the virus after feeding for 2-3 hours.

Therapy

RYNV is classified as a heat-stable virus, in that it survives in plants held for several weeks at an air temperature of 37°C, near the maximum at which plants can survive. Small shoot tips or meristems excised from heat-treated plants and induced to root may be free of the virus.

Indexing

When on its own in plants, RYNV can be detected by graft-inoculation to *R. macraei* in which the virus induces distinct interveinal clearing and yellowing. Symptoms are less clear in graft-inoculated *R. occidentalis*. However, as RYNV is usually found in complex with other aphid-borne viruses, graft or aphid transmission to *R. occidentalis* will transmit the complex of viruses and some of them will induce necrotic symptoms that mask those induced by RYNV. Thermotherapy of plants infected with the virus complex will inactivate most other viruses, leaving RYNV free from contaminating viruses.

References

- Jones, A.T. 1991. *Rubus* host range of Rubus yellow net virus and its involvement with other aphid-borne latent viruses in inducing raspberry veinbanding mosaic disease. *Ann. Appl. Biol.* **118**:331-338.
- Stace-Smith, R. & Jones, A.T. 1978. Rubus yellow net virus. CMI/AAB Descriptions of Plant Viruses No. 188. Commonwealth Agricultural Bureaux, Slough.
- Stace-Smith, R. & Jones, A.T. 1987. Rubus yellow net. pp. 175-178. In: *Virus Diseases of Small Fruits*. Ed. R.H. Converse. United States Department of Agriculture, Agriculture Handbook No. 631, Washington, D.C.

13. Wineberry latent virus (WLV) (see also blackberry calico virus (BCV))

Virus particles are flexuous filaments about 12 x 620 nm which showed no serological relationship to viruses with particles of similar size. However, it shows several similarities to blackberry calico carlavirus.

Fig. 34. Necrotic local lesions in *Chenopodium amaranticolor* 20 days after inoculation with wineberry latent virus. (Scottish Crop Research Institute, Invergowrie)



Symptoms

In mixed infections with raspberry bushy dwarf virus (RBDV) WLW induced pronounced venal chlorotic line-pattern and/or chlorotic blotches, symptoms typical of calico disease.

Host range

Reported *only* in *Rubus* spp.

Geographical distribution

Detected in Scotland in wineberry (*R. phoenicolasius*) originally imported from USA.

Transmission

No information on natural transmission. Not seed transmitted in wineberry. Transmitted experimentally by grafting and by mechanical inoculation of sap extracts to herbaceous plants.

Therapy

No information.

Indexing

WLW from wineberry, co-infected with RBDV, was readily transmitted from *Rubus* in 2% nicotine alkaloid solution to herbaceous plants (see underlined note of caution on p. 10). However, a Dutch culture of a virus that may be similar to WLW, but free from RBDV, was transmitted in this way only with great difficulty. In *Chenopodium quinoa* and *C. amaranticolor*, WLW induces necrotic local lesions that gradually expand in size; it is only weakly systemic in these species. Electron microscopy and/or serological tests are necessary to identify and distinguish WLW from other possible viruses. WLW was not detected by ELISA in *Rubus*.

References

- Converse, R.H. 1987. Blackberry calico. pp. 245-246. In: *Virus Diseases of Small Fruits*. Ed. R.H. Converse. United States Department of Agriculture, Agriculture Handbook No. 631, Washington, D.C.
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- Jones, A.T., Mitchell, M.J., McGavin, W.J. & Roberts, I.M. 1990. Further properties of wineberry latent virus and evidence for its possible involvement in calico disease. *Ann. Appl. Biol.* **117**:571-581.

Prokaryotic diseases - 'MLOs'

1. Boysenberry decline

Cause

Unknown, possibly a non-cultivable mollicute, often referred to as mycoplasma-like organism (MLO).



Fig. 35. Symptoms of boysenberry decline in flowers (top) and fruits (bottom) of boysenberry. Flower symptoms are shortened stamens, with styles and stigmas enlarged; fruitlets from infected plants have the appearance of a hard green cone. (Dr. G. Wood, Horticulture and Food Research Institute of New Zealand, Auckland)

Fig. 36. Fruiting canes of plants affected by boysenberry decline, showing proliferating shoot growth and red down-curling leaves (right), compared to shoot growth of a healthy plant on the left. (Dr. G. Wood, Horticulture and Food Research Institute of New Zealand, Auckland)



Symptoms

Abnormally large flowers with crinkled petals, and large distorted sepals. Stamens are shortened, styles and stigmas enlarged, giving the young fruitlet formed in the flowers the appearance of a hard green cone. Large, sharp spines develop on flower stems. Fruits fail to develop and remain as small, hard, hairy cones. Shoot growth proliferates on the fruiting canes, growing to a metre or more in length, and develops small down-curling leaves which become distinctly red in colour. Symptoms do not occur on the primocanes.

Host range

Confined to boysenberry, but suspected symptoms have been found on loganberry and several blackberry cultivars following graft-inoculation.

Geographical distribution

Found only in New Zealand.

Transmission

Transmitted by grafting. Suspected to be transmitted by the bramble leaf hopper *Ribautiana tenerrima*, but this has not been confirmed.

Therapy

No information available.

Indexing

Grafting to boysenberry,

Reference

Wood, G.A. 1991. Three graft-transmissible diseases and a variegation disorder of small fruit in New Zealand. *N.Z.J. Crop Hortic. Sci.* **19**:313-323.

2. *Rubus* stunt

Cause

The causal agent is believed to be a non-cultivable mollicute, often referred to as mycoplasma-like organism (MLO).

Symptoms

The disease is characterized by thin, spindly canes showing excessive lateral branching, to form a witches-broom appearance, proliferation of flowers and phyllody.

Host range

All raspberry and blackberry cultivars as well as raspberry x blackberry hybrids seem infectible.

Geographical distribution

Reported only from Southern Britain, Europe and Russia.

Transmission

Transmitted in nature in a persistent manner by leafhoppers, mainly in the genus *Macropsis*. The main vector in Europe is *M. fuscula*. Also transmitted experimentally by froghoppers (spittle bugs) and by graft-inoculation to *Rubus* and plants of other genera.



Fig. 37. Floricane of blackberry showing witches' broom symptoms typically of *Rubus* stunt disease. (Dr. R.H. Converse, Horticultural Crops Research Laboratory, Corvallis)

Therapy

Eradicated from plants by thermotherapy in hot water (45°C) for 2-3 hours.

Indexing

Graft-inoculation to sensitive cultivars such as ‘Malling Landmark’ raspberry or ‘Thornless Evergreen’ blackberry.

References

- Murant, A.F. & Roberts, I.M. 1971. Mycoplasma-like bodies associated with *Rubus* stunt disease. *Ann. Appl. Biol.* **67**:389-393.
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Prokaryotic diseases - bacteria**1. Crown and cane gall****Cause**

Two species of *Agrobacterium* are associated with the crown gall syndrome on *Rubus*: *A. tumefaciens* (E.F. Smith & Towns.) Conn and *A. rubi* (Hildebr.) Starr & Weiss. The latter is associated with galls on canes.

Symptoms

Galls typically form in the crown area or on roots. They may also form at pruning wounds or natural splits in canes. Initially galls are soft and become hard with age. Severely infected canes exhibit a number of symptoms including stunting, foliar chlorosis, small and seedy fruit, wilting and at times death. Galls blacken and die during winter. New galls erupt the following spring in the vicinity of old galls. With time plants become progressively less vigorous.

Host range

Broad, including all species of *Rubus*.

Geographical distribution

Worldwide.

Biology and transmission

Infection occurs through wounds. Wounds can result from natural causes (lateral root formation, leaf scars) or mechanical damage (pruning, training practices, cultivation, harvesting, insect feeding, frost, etc.). Infection results in the uncontrolled synthesis of plant hormones stimulating host cells to abnormally multiply. Temperatures below 15°C delay symptom development leading to latent infections. Symptoms develop at 20°C. Infection is inhibited above 32°C. Once the pathogen's DNA becomes incorporated into the genome of the host, new galls develop even though the pathogen dies during winter

Reference

Moore, L.N. 1991. Crown and cane gall. pp. 39-40. In: *Compendium of Raspberry and Blackberry Diseases and Insects*. Eds. M.A. Ellis, R.H. Converse, R.N. Williams & B. Williamson. APS Press, St. Paul.

2. Fireblight**Cause**

Erwinia amylovora (Burr.) Winslow *et al.*

Symptoms

Characteristic water-soaked lesions that produce abundant bacterial ooze. Diseased parts of canes become necrotic and purplish black. Infected berries do not mature; they become dry, brown, and very hard.

Host range

Fireblight is a common and very serious disease of many rosaceous plants, but on *Rubus* cane fruits it is relatively uncommon and rarely of economic importance.

Geographical distribution

Fireblight on *Rubus* has occasionally been reported from eastern USA.

Biology and transmission

The initial inoculum source is probably from overwintering cankers on *Rubus*. Isolates from *Rubus* infect only *Rubus*, those from apples and pears are not pathogenic on *Rubus*. Warm temperature and light rain favour infections.

Reference

Ries, S.M. 1991. Fireblight. pp. 40-41. In: *Compendium of Raspberry and Blackberry Diseases and Insects*. Eds. M.A. Ellis, R.H. Converse, R.N. Williams & B. Williamson. APS Press, St. Paul.

3. Hairy root

Cause

Agrobacterium rhizogenes (Riker *et al.*) Conn.

Symptoms

Infected parts develop abnormally fibrous roots.

Host range

Rubus spp., *Malus* spp. and *Rosa* spp.

Geographical distribution

Unknown.

Biology and transmission

The bacterium is exclusively a wound pathogen and soil-borne and may be readily transmitted with nursery stock.

Reference

Ellis, MA. 1991. Hairy root. p. 42 In: *Compendium of Raspberry and Blackberry Diseases and Insects*. Eds. M.A. Ellis, R.H. Converse, R.N. Williams & B. Williamson. APS Press, St. Paul.

Fungal diseases

1. Blackberry rust

Cause

Phragmidium violaceum (C.F. Schultz) Winter, a macrocyclic autoecious rust fungus.

Symptoms

The disease affects most plant parts including leaves, leaf veins, petioles, flowers and fruit. Symptoms on leaves are the most common. On the upper surface of leaves, symptoms first appear as yellowish-red blotches and develop in about ten days into purple or red circular spots (up to 4 mm in diameter). These spots often have yellow to brown centers. On the lower leaf surface beneath the spots, golden yellow powdery pustules (uredinia) appear. When the disease is severe, leaf edges may curl, the entire leaf may turn chlorotic and drop prematurely. Uredinia eventually turn black as telia develop. Pustules are larger on susceptible genotypes than on resistant ones.

Host range

European blackberry (*Rubus fruticosus*) and some cultivated blackberries.

Geographical distribution

Common in Europe and the Middle East. The disease also occurs in Australia, Chile and New Zealand.

Biology and transmission

The pathogen most likely overwinters as teliospores on old leaves. One report indicates that the pathogen overwinters as a perennial mycelium on stems and produces urediniospores directly in the spring,

Reference

Washington, W.S. 1991. Blackberry rust. pp. 32-33. In: *Compendium of Raspberry and Blackberry Diseases and Insects*. Eds. M.A. Ellis, R.H. Converse, R.N. Williams & B. Williamson. APS Press, St. Paul.

2. Cane and leaf rust

Cause

Kuehneola uredinis (Link) Arthur, a macrocyclic autoecious rust fungus.

Symptoms

Large, lemon yellow pustules of powdery urediniospores split the bark in late spring on infected floricanes. In early summer, small yellow uredinia develop on the lower surfaces of the leaves of these floricanes. Severe infection causes premature defoliation. Only rarely are rust symptoms observed on fruit.

Host range

This rust attacks blackberry and blackberry hybrids. It occurs only rarely on red and black raspberries.

Geographical distribution

North America and Australia.

Biology and transmission

The fungus probably overwinters on canes as mycelium or latent uredinia. Urediniospores infect leaves on the same or other floricanes during the growing season. Telia develop on leaves in late fall and basidiospores from germinating teliospores infect adjacent leaves of primocanes. Disease development is favoured.

by wet conditions. Cane and leaf rust is not systemic and care should be taken not to confuse its identification with the systemic orange rusts caused by *Arthuriomyces peckianus* and *Gymnoconia nitens*.

Reference

Ellis, M.A. 1991. Cane and leaf rust p. 28. In: *Compendium of Raspberry and Blackberry Diseases and Insects*. Eds. M.A. Ellis, R.H. Converse, R.N. Williams & B. Williamson. APS Press, St. Paul.

3. Downy mildew

Cause

Peronospora sparsa Berk.

Symptoms

Angular lesions occur on upper surfaces of leaves, first yellow, later turning red to purple. On lower leaf surfaces opposite these lesions, pink to tan spots appear on which spore masses may be borne. Systemically infected plants may produce distorted leaves with a mosaic pattern of small, reddish lesions, later becoming necrotic with chlorotic margins. Suckers and infected canes from such plants may be stunted with reddish blotches on leaves and canes. Infected fruit is dull, without sheen. Berries infected early turn red prematurely, shrivel and harden, while those infected late may split in two and become shriveled. Pedicels may become infected, turning red.

Host range

Rubus and *Rosa* species.

Geographical distribution

Worldwide on *Rubus*, except in South America.

Biology and transmission

Air-borne spores of this obligate parasite are produced on infected plants during cool, wet nights and are wind-disseminated. These spores can infect foliage, blossoms and berries. Cool, moist conditions favour infection. In some areas, oospores develop in early summer in infected leaves and sepals. Systemic infections may occur, especially in blackberry-raspberry hybrids, like boysenberry, in which intercellular mycelium may spread throughout the plant. Inoculum from *Rubus* and *Rosa* species is cross-infective.

Reference

Gubler, W.D. 1991. Downy Mildew. pp. 15-16. In: *Compendium of Raspberry and Blackberry Diseases and Insects*. Eds. M.A. Ellis, R.H. Converse, R.N. Williams & B. Williamson. APS Press, St. Paul.

4. Late leaf rust**Cause**

Pucciniastrum americanum (Farl.) Arthur.

Symptoms

This rust disease is not systemic. On mature leaves many small spots develop and turn yellow and eventually brown before the leaves die in autumn. Small uredinia filled with powdery yellow spores are formed on the underside of infected leaves. Badly infected leaves drop prematurely, and the canes of highly susceptible cultivars may be bare by early autumn. Flower calyces, petioles, and fruit at all stages of development are also attacked. On fruit, uredinia develop on individual drupelets, producing yellow masses of urediniospores.

Host range

Many cultivated red and purple raspberries and some wild red raspberries.

Geographical distribution

Canada, USA.

Biology and transmission

The causal organism is heteroecious and macrocyclic. It produces spermogonia and aecia on white spruce (*Picea glauca*) and uredinia and telia on *Rubus* spp. White spruce is the most common host for the aecial stage. The main uredinial hosts are red and purple raspberries.

Reference

Ellis, M.A. 1991. Late leaf rust. pp. 30-31. In: *Compendium of Raspberry and Blackberry Diseases and Insects*. Eds. M.A. Ellis, R.H. Converse, R.N. Williams & B. Williamson. APS Press, St. Paul.

5. Orange rust**Cause**

Arthuriomyces peckianus (Howe) Cummins & Y. Hirats. (demicyclic) and *Gymnoconia nitens* (Schwein.) F. Kern & Thurst. (endocyclic); both rust fungi are autoecious.

Fig. 38. Orange rust caused by *Gymnoconia nitens* native North American blackberry *Rubus alumnus*. (Dr. J. Postman, USDA-ARS-NCGR, Corvallis)



Symptoms

First symptoms in spring are the spindly and clustered young shoots. Characteristic are the bright orange aecia on the lower leaf surface. Plants infected with the demicyclic form *Arthuriomyces peckianus* develop dark telia. Plants become systemically infected, although primocanes and floricanes may appear healthy.

Host range

The demicyclic form attacks mostly black raspberries, whereas the endocyclic form predominates on blackberries. Red raspberries are immune.

Geographical distribution

Asia, Australia, Europe and North America.

Biology and transmission

The fungi overwinter as systemic, perennial mycelium. *Gymnoconia nitens* may also overwinter as teliospores. Disease development is favoured by cool and wet conditions. Orange rust is easily confused with the non-systemic cane and leaf rust caused by *Kuehneola uredinis*.

Reference

Kleiner, W.C. & Travis, J.W. 1991. Orange rust pp. 26-28. In: *Compendium of Raspberry and Blackberry Diseases and Insects*. Eds. M.A. Ellis, R.H. Converse, R.N. Williams & B. Williamson. APS Press, St. Paul.

6. *Phytophthora root rot*

Cause

Six different *Phytophthora* species have been reported on roots of *Rubus*.

<u>Species</u>	<u>Location</u>
<i>P. fragariae</i> Hickman var. <i>rubi</i> Wilcox & Duncan	Eastern N. America* Western N. America** UK and Europe***
<i>P. cactorum</i> (Lebert & Cohn) Schröt.	USA and UK
<i>P. citricola</i> Sawada	USA, UK and Poland
<i>P. cryptogea</i> Pethybr. & Laff.	USA and Australia
<i>P. drechsleri</i> Tucker	UK
<i>P. cambivora</i> (Petri) Buisman	UK

* formerly *P. fragariae* Hickman

** formerly *P. erythroseptica* Pethybr.

*** formerly *P. megasperma* (type 2) Drechsler

Symptoms

Both primocanes and floricanes may exhibit symptoms. Infected primocanes may rapidly wilt and collapse. Collapse can occur at any time during the spring. On succulent young primocanes dark, water-soaked lesions form at the base. Wilting usually begins at the tip of primocanes progressing downward. Primocanes which survive may die during the winter. Affected floricanes typically produce weak lateral shoots. Leaves turn yellow, wilt or scorch along the margins or between the veins. Severely affected floricanes may wilt and die during flowering or before harvest. Scraping the epidermis from roots of infected plants reveals a characteristic reddish brown discoloration. A distinct margin is usually evident at the interface of diseased and healthy root tissues. Extension of necrosis into the crown is common.

Host range

Red raspberry (*Rubus idaeus*) and some of its hybrids. Black raspberry (*R. occidentalis*) may also be infected.

Geographical distribution

Worldwide. Serious outbreaks have occurred in Australia, Europe, North America and the UK. For distribution of species, see above.

Biology and transmission

Once present, *Phytophthora* spp. persist primarily as mycelium in recently infected tissues or as dormant oospores that are released into soil as infected tissues die and decompose. Oospores may remain viable for a number of years in soil in the absence of a host. Oospores are insensitive to environmental extremes and chemicals (fungicides and fumigants) applied to soil. The soil moisture requirement for germination of oospores is high.

References

- Bristow, P.R., Daubeny, H.A., Sjulín, T.M., Pepin, H.S., Nestby, R. & Windom, G. 1988. Evaluation of *Rubus* germplasm for reaction to root rot caused by *Phytophthora erythroseptica*. *J. Am. Soc. Hortic. Sci.* **113**:588-591.
- Duncan, J.M. & Kennedy, D.M. 1989. The effect of waterlogging on *Phytophthora* root rot on red raspberry. *Plant Pathol.* **38**:161-168.
- Wilcox, W.F. 1991. *Phytophthora* root rot. pp. 34-36. In: *Compendium of Raspberry and Blackberry Diseases and Insects*. Eds. M.A. Ellis, R.H. Converse, R.N. Williams & B. Williamson. APS Press, St. Paul.
- Wilcox, W.F., Scott, P.H., Hamm, P.B., Kennedy, D.M., Duncan, J.M., Brasier, C.M. & Hansen, E.M. 1993. Identity of a *Phytophthora* species attacking red raspberry in Europe and North America. *Mycol. Res.* **97**:817-831.

7. *Verticillium* wilt (bluestem or bluestripe wilt)

Cause

Verticillium albo-atrum Reinke & Berth. and/or *V. dahliae* Kleb.

Symptoms

On black and purple raspberry, leaves of infected fruiting canes may turn yellow and then wilt and die. Infected canes are often stunted and may turn entirely blue or blue on one side before they die. Infected vessels generally have a red coloration. Symptoms on red raspberry are usually less severe than on black raspberry. Leaflets on infected plants often fall before the petioles drop. Cane discoloration is not as evident as on black raspberry. Plants may survive for years, but are stunted and suckering is reduced. On blackberries the infected canes wilt,

leaves turn yellow and become brown and necrotic, Cool weather in the autumn may lead to a disappearance of symptoms. Canes do not turn blue, as they do in infected black raspberry. Infected fruiting canes may survive the winter, leaf out and set fruit, but as the fruits ripen during the summer, the canes usually collapse.

Host range

Black, purple and red raspberry and blackberry.

Geographical distribution

Northern part of the USA and along the Pacific Coast, particularly in California.

Biology and transmission

The fungus survives as microsclerotia or as melanized hyphal fragments bound within plant debris or free in the soil. Hyphae from these structures penetrate root hairs or the root cortex directly and enter xylem vessels. The fungi can also penetrate through breaks or wounds in the roots. Movement within the host occurs by growth of the hyphae through the vessels and by movement of conidia in the transpiration stream. Upon death of infected plant parts, new fungal survival structures are formed and returned to the soil where they survive for up to 14 years.

Reference

Ellis, M.A. 1991. Verticillium wilt. pp. 36-37. In: *Compendium of Raspberry and Blackberry Diseases and Insects*. Eds. M.A. Ellis, R.H. Converse, R.N. Williams & B. Williamson. APS Press, St. Paul.

8. White root rot

Cause

Vararia spp. (Lachnocladiaceae), various basidiomyceteous fungi.

Symptoms

These pathogens cause a root rot, dieback and death of plants. *Vararia* spp. induce yellowing, wilting and dieback of canes which is especially rapid in young plants. Roots and crowns are also rotted and characteristically covered by a white mycelial mat. White rhizomorphs may develop on the surface of these parts. Similar symptoms have been observed in New Zealand, but unrelated basidiomyceteous fungi have been reported.

Host range

This disease has been reported on field-infected raspberry, on loganberry and once on plum. Other rosaceous plants have been artificially inoculated.

Geographical distribution

Australia, New Zealand.

Biology and transmission

Vararia can probably survive in soil for several years on infected roots or canes. It spreads within a planting via root contact and less often by the spread of infected roots and canes through cultivation. Disease severity is greatest when the soil moisture is low and soil temperatures are high.

References

- Pascoe, I.G., Washington, W.S. & Guy, G. 1984. White root rot of raspberry in Victoria is caused by a *Vararia* species. *Trans. Br. Mycol. Soc.* **82**:723-726.
- Washington, W.S. 1991. White root rot. pp. 38-39. In: *Compendium of Raspberry and Blackberry Diseases and Insects*. Eds. M.A. Ellis, R.H. Converse, R.N. Williams & B. Williamson. APS Press, St. Paul.

Vaccinium spp. (blueberry, cranberry)**Viruses****1. Blueberry red ringspot virus (BRRV)**

A caulimovirus with isometric particles of about 42-46 nm in diameter.



Fig. 39. Symptoms of blueberry red ringspot virus on highbush blueberry: red rings enclosing green leaf tissue. (Dr. J. Postman, USDA-ARS-NCCR, Corvallis)

Symptoms

Infected plants are usually symptomless until fruit is formed. Then red rings or reddish spots 2-6 mm in diameter may develop on the upper sides of leaves. The rings often enclose green leaf tissue. Similar spots and rings form on the epidermis of canes. Occasionally rings form on the fruit of some cultivars. Leaves often develop reddish autumn coloration several weeks before those of healthy plants.

Host range

Vaccinium corymbosum and *V. australe*.

Geographical distribution

USA.

Transmission

BRRV spreads in New Jersey but not in Michigan or in Oregon. The vector is suspected, but not proven, to be a mealybug (*Dysmicoccus* spp.). The virus is not mechanically transmitted.

Therapy

BRRV is difficult to eliminate by propagating from new growth following extended thermotherapy.

Indexing

Chronic infections of BRRV are not known to be latent when leaf symptoms reach their height after harvest and before normal autumn foliar coloration develops. At other times of the year BRRV is difficult to detect by symptomatology. Identity can be confirmed by ELISA in symptomatic leaves.

References

- Hepp, R.F. & Converse, R.H. 1987. Blueberry red ringspot virus detection in crude sap of highbush blueberry plants. *Plant Dis.* **71**:536-539.
- Ramsdell, D.C., Kim, K.S. & Fulton, J.P. 1987. Red ringspot of blueberry. pp. 121-123. In: *Virus Diseases of Small Fruits*. Ed. R.H. Converse. United States Department of Agriculture, Agriculture Handbook No. 631, Washington, D.C.
- Ramsdell, D.C. 1994. Red ringspot. In: *Compendium of Blueberry and Cranberry Diseases*. Eds. F.L. Caruso & D.C. Ramsdell. APS Press (in press).

2. Blueberry scorch virus (BBScV)

A flexuous, rod-shaped carlavirus, about 610-700 nm in length. Virus particles contain a positive sense, single-stranded RNA genome. Sheep Pen Hill disease (SPHD) in New Jersey, USA is caused by a closely related virus.

Symptoms

Some cultivars react with complete necrosis of flowers and leaves that eventually leads to bush death, others do not seem to be adversely affected by infection. Some cultivars show a flower necrosis combined with a marginal leaf chlorosis. Symptoms usually appear on one or a few branches the first year and progressively spread to affect the entire bush in subsequent years.

Host range

Highbush blueberry (*Vaccinium corymbosum*).

Geographical distribution

USA.

Transmission

BBScV has been experimentally transmitted by the blueberry aphid *Fimbriaphis fimbriata*. Field studies involving placement of healthy 'trap' bushes in commercial plantings with scorch disease have shown that transmission is directly correlated with the colonization of trap plants by blueberry aphids. BBScV is graft-transmissible.



Fig. 40. Complete necrosis of flowers and leaves of highbush blueberry caused by blueberry scorch virus. (Dr. J. Postman, USDA-ARS-NCGR, Corvallis)

Therapy

No information is available about the elimination of BBScV from *Vaccinium*.

Indexing

ELISA detects BBScV and SPHD-related virus in blueberry.

References

- MacDonald, S.G., Martin, R.R. & Bristow, P.R. 1989. Viruses capable of causing a scorch disease of highbush blueberry. *Acta Hort.* **241**:295-300.
- Martin, R.R. & Bristow, P.R. 1988. A carlavirus associated with blueberry scorch disease. *Phytopathology* **78**:1636-1640.
- Martin, R.R. & Bristow, P.R. 1994. Blueberry scorch. In: *Compendium of Blueberry and Cranberry Diseases*. Eds. F.L. Caruso & D.C. Ramsdell. APS Press (in press).
- Podleckis, E.V. & Davis, R.F. 1989. Infection of highbush blueberries with a putative carlavirus. *Acta Hort.* **241**:337-342.

3. Blueberry shock ilarvirus (BSIV)

A virus with quasi-isometric particles 26-29 nm in diameter.

Symptoms

Affected young leaves wilt and have blackened veins and black streaks down the petiole, or they blight to an orange colour. Both blighted blossoms and leaves drop, so by early summer the affected bushes can be completely defoliated. As the season progresses the bushes appear to recover as a second flush of leaves is produced. By late summer, these plants appear normal except there is little fruit produced. On occasion, the affected bushes are stunted, especially when symptoms persist for 3 or 4 years.

Host range

Highbush blueberry
(*Vaccinium corymbosum*).

Geographical distribution

Northwest USA.



Fig. 41. Blighted blossoms and leaves in highbush blueberry infected with blueberry shock ilarvirus. (Dr. P.R. Bristow, Washington State University, Puyallup)

Transmission

BSIV is graft-transmissible. It can be transmitted mechanically to *Nicotiana clevelandii* from blossoms of infected bushes. It has not been possible to transmit the virus mechanically from other blueberry tissues. The virus can be transmitted to a number of species of *Nicotiana* and maintained in these plants in a glasshouse at temperatures below 22°C, but not at higher temperatures. Also pollen-borne in blueberry. Natural infection only occurs during the bloom period. There is a one year latent period between infection and symptom development.

Therapy

No information is available about the elimination of BSIV from *Vaccinium*.

Indexing

ELISA is reliable for the detection of BSIV in blueberry tissues. Graft-inoculation to 'Berkeley'.

References

- MacDonald, S.G., Martin, R.R. & Bristow, P.R. 1991. Characterization of an ilarvirus associated with a necrotic shock reaction in blueberry. *Phytopathology* **81**:210-214.
- Martin, R.R. & Bristow, P.R. 1994. Blueberry shock. In: *Compendium of Blueberry and Cranberry Diseases*. Eds. F.L. Caruso & D.C. Ramsdell. APS Press (in press).

4. Blueberry shoestring virus (BSSV)

An isometric virus, about 28 nm in diameter.

Symptoms

The most prominent symptoms consist of elongated reddish streaks approximately 0.4 x 1.25 to 2.5 cm on current year and 1-year-old stems. Blossoms may have a pinkish cast on infected bushes. Infected leaves are often strap-shaped or may be crescent shaped. Some leaves may show a reddish oak-leaf pattern on the lamina. Ripe fruit on infected bushes usually has a reddish to purplish cast rather than a deep blue colour. The terminal one third of stems may be crooked on infected bushes. There is a 4-year latent period between infection and onset of symptoms.

Host range

Host range of BSSV is limited to some cultivars of highbush blueberry (*Vaccinium corymbosum*).

Fig. 42. Strap-shaped leaves infected with blueberry shoestring virus (in foreground left) compared to normal leaves. (Dr. J. Postman, USDA-ARS-NCGR, Corvallis)



Geographical distribution

Northeast USA.

Transmission

BSSV is transmitted by the blueberry aphid *Illinoia pepperi*. The virus is not mechanically transmissible.

Therapy

No information.

Indexing

Indexing by ELISA is routinely used to detect BSSV in suspect plants. Polyclonal antisera work extremely well. Commercial ELISA kits are available in the USA.

References

- Morimoto, K.M., Ramsdell, D.C., Gillett, J.M. & Chaney, W.G. 1985. Acquisition and transmission of blueberry shoestring virus by its aphid vector *Illinoia pepperi*. *Phytopathology* **75**:709-712.
- Ramsdell, D.C. 1987. Blueberry shoestring. pp. 103-405. In: *Virus Diseases of Small Fruits*. Ed. R.H. Converse. United States Department of Agriculture, Agriculture Handbook No. 631, Washington, D.C.
- Ramsdell, D.C. 1994. Shoestring. In: *Compendium of Blueberry and Cranberry Diseases*. Eds. F.L. Caruso & D.C. Ramsdell. APS Press (in press).

5. Nepoviruses

Four nepoviruses have been reported from blueberry, namely tobacco ringspot virus (TRSV), tomato ringspot virus (ToRSV), blueberry leaf mottle virus (BLMV), and peach rosette mosaic virus (PRMV). Virus particles are isometric, about 30 nm in diameter with angular profiles.

Symptoms

Symptoms vary with the individual virus, cultivar and time of the year. Symptoms are usually more conspicuous in spring, when new growth may show chlorotic and necrotic spots. Stem dieback and stunting frequently occur when susceptible cultivars are infected.

Host range

Within *Vaccinium*, the viruses have only been found in the highbush blueberry (*V. corymbosum*). Nepoviruses that have been isolated from blueberry have a wide natural host range.

Geographical distribution

In highbush blueberry, the viruses are confined to the USA. ToRSV occurs in the northern states, TRSV in the eastern states, BLMV and PRMV in Michigan.

Transmission

In nature, transmitted by vectors belonging to the Dorylaimidae. Natural spread of TRSV, ToRSV and PRMY is thought to be by *Xiphinema americanum*. Natural transmission of BLMV is by honey bees (*Apis mellifera*). Pollen contains a high level of virus.

Therapy

No information available for *Vaccinium*.

Indexing

Serological procedures (usually ELISA) may be used to identify nepoviruses either directly from blueberry or from herbaceous test plants. Nepoviruses can be mechanically transmitted from blueberry to herbaceous hosts, which are satisfactory for detection, but not for identification.

References

- Boylan-Pett, W., Ramsdell, D.C., Hoopingarner, R.A. & Hancock, J.F. 1991. Honeybee foraging behavior, in-hive survival of infectious, pollen-borne blueberry leaf mottle virus and transmission of the virus in highbush blueberry. *Phytopathology* **81**:1407-1412.
- and sections on nepoviruses in: *Compendium of Blueberry and Cranberry Diseases*. Eds. F.L. Caruso & D.C. Ramsdell. APS Press (in press).

6. Ringspot of cranberry

The causal agent has not been positively identified, but is probably a caulimovirus. Virus-like particles and inclusion bodies have been observed in diseased leaf tissue.

Symptoms

Fruit on affected plants is often misshapen with pale or whitish rings when ripe. There is necrosis at the blossom end of many berries and in severe cases whole berries are necrotic. Ringspot symptoms are also produced on the leaves. Rings become visible in the autumn when leaves turn colour; the rings stay green as the remainder of the leaf turns reddish. The disease appears to be systemic. It adversely affects the keeping quality of fruit.

Host range

American cranberry (*Vaccinium macrocarpon*). There is no information on experimental hosts.

Geographical distribution

Eastern USA.



Fig. 43. Ringspot of cranberry: fruits with whitish rings (top) and ringspot symptoms on leaves (bottom). (Dr. P.R. Bristow, Washington State University, Puyallup)

Transmission

At present there is no information on either natural or experimental transmission.

Therapy

No information available.

Indexing

No information available.

Reference

- Boone, D.M. 1994. Ringspot. In: *Compendium of Blueberry and Cranberry Diseases*. Eds. F.L. Caruso & D.C. Ramsdell. APS Press (in press).
- Stretch, A.W. 1987. Ringspot disease of cranberry. pp. 123-124. In: *Virus Diseases of Small Fruits*. Ed. R.H. Converse. United States Department of Agriculture, Agriculture Handbook No. 631, Washington, D.C.

Disease of unknown etiology

Blueberry mosaic

Cause

Unknown.

Symptoms

Patterns of yellow, light green or white mottle and mosaic that vary in intensity. Pink areas sometimes appear in the mosaic or mottled tissues. Symptoms may be uneven on an infected bush, infecting all or only a few branches. Over several years, symptoms may appear, disappear, and reappear on the same bush. Fruit yield and quality are reduced. The light and dark green mosaic pattern on leaves of the cultivar 'Coville' may be of genetic origin.

Host range

Vaccinium corymbosum and *V. vacillans*.

Geographical distribution

Canada and USA.

Transmission

Blueberry mosaic disease is not sap-transmitted or dodder-transmitted. It spreads slowly in the field by unknown means, and is graft-transmissible.

Fig. 44. Blueberry mosaic disease. (Dr. J. Postman, USDA-ARS-NCGR, Corvallis)



Therapy

No information.

Indexing

The cultivars 'Rubel' and 'Bluecrop', chip-bud graft-inoculated at bud-break, can be used as indicators. True latency of blueberry mosaic disease in highbush blueberry cultivars is not known to occur. However, because of the irregular timing of the appearance of symptoms, a bush being used to obtain softwood or hardwood cuttings for propagation should be observed for several years before being declared free from this disease.

Reference

Ramsdell, D.C. & Stretch, A.W. 1987. Blueberry mosaic. pp. 119-120. In: *Virus Diseases of Small Fruits*. Ed. R.H. Converse. United States Department of Agriculture, Agriculture Handbook No. 631, Washington, D.C.

Prokaryotic diseases - 'MLOs'

1. Blueberry stunt (BBS)

Cause

A phloem-limited non-cultivable mollicute, often referred to as mycoplasma-like organism (MLO), with a diameter ranging from 160 to 700 nm.

Symptoms

On highbush blueberry, overall dwarfing of the bush is the primary symptom, hence the name stunt. Symptomatic leaves may be spoon-shaped 'rather than lanceolate; they are usually cupped slightly downward, and exhibit chlorosis of the margins. Chlorosis of the leaves may occur between lateral veins. The midribs and lateral veins usually retain normal green coloration. Chlorotic areas often turn a brilliant red in the late summer and early autumn. Stem internodes become shortened, and growth of normally dormant buds causes twiggy branching.

Host range

All cultivars of highbush blueberry are susceptible. 'Rancocas' is the only cultivar with a high degree of resistance. *V. australe*, *V. vacillans*, *V. atrocuccum*, *V. stamineum* and *V. myrtilloides* are also susceptible to natural infection. Graft transmission to *V. amoenum*, *V. altomontanum*, *V. elliotii* and *V. ashei* has been achieved. Dodder (*Cuscuta campestris* and *C. subinclusa*) has been successfully used to transmit the causal organism to periwinkle (*Catharanthus roseus*).

Geographical distribution

Stunt disease has been reported from eastern Canada, and eastern and southeastern USA.

Transmission

The leafhoppers *Scaphytopius magdalensis*, *S. acutus* and *S. frontalis* have been shown experimentally to be vectors. The causal organism is not mechanically transmitted.

Therapy

Thermotherapy is effective in eradicating infected blueberry of the causal organism.

Indexing

Indexing on cultivar 'Cabot'.

References

Chen, T.A. 1971. Mycoplasma-like organisms in sieve tube elements of plants infected with blueberry stunt and cranberry false blossom. *Phytopathology* **61**:233-236.

- Maramorosch, K. 1955. Transmission of blueberry stunt virus by *Scaphytopius magdalensis*. *J. Econ. Entom.* **48**:106.
- Ramsdell, D.C. 1987. Blueberry stunt. pp. 106-108. In: *Virus Diseases of Small Fruits*. Ed. R.H. Converse. United States Department of Agriculture, Agriculture Handbook No. 631, Washington, D.C.
- Tomlinson, W.E. Jr., Marucci, P.E., & Doehlert, C.A. 1950. Leafhopper transmission of blueberry stunt disease. *J. Econ. Entom.* **43**:658-662.
- Tozzi, D.C.M., Ramsdell, D.C., Taboada, O., Lee, I.M., & Davis, R.E. 1993. Epidemiological studies on the stunt disease of highbush blueberry. *Ann. Appl. Biol.* **123**:579-599.

2. Cranberry false blossom

Cause

The causal agent appears to be a non-cultivable mollicute, often referred to as a mycoplasma-like organism (MLO).

Symptoms

This disease is most easily recognized at bloom. Flowers on infected plants are upright because the pedicels are straight rather than arched. The petals of diseased flowers are short and streaked with green and red. The lobes of the calyx are enlarged compared to those on a healthy flower. Diseased flowers are usually sterile as both-the stamens and pistil are abnormal. Axillary buds which normally remain dormant are stimulated to produce branches giving a witches'-broom effect to infected uprights. Leaves on these branches are usually closely appressed to the stem. In autumn infected branches prematurely redden. Terminal flower buds on infected uprights are enlarged.

Host range

In nature restricted to American cranberry (*Vaccinium macrocarpon*) and European cranberry (*V. oxycoccus*), which reflects vector feeding preference.

Geographical distribution

Appears to be indigenous in Wisconsin, USA and was probably distributed to other production areas in North America.

Transmission

Natural transmission is by the blunt-nosed leafhopper *Scleroracus vaccinii*. This insect is the only known vector.

Fig. 45. Cranberry false blossom: upright flowers with straight pedicels and short petals (right) compared to normal flowers (left). (Dr. R.H. Converse, Horticultural Crops Research Laboratory, Corvallis)



Therapy

Hold potted plants at 42-43°C for 8 days. Treat a large number of plants as about one-third will be killed by the treatment. High temperatures for periods up to 10 days caused less injury than treatments at somewhat lower temperatures for longer periods. The pathogen is eliminated from the vines but it is unknown if the roots are pathogen-free following thermotherapy.

Indexing

Transmission to tomato or periwinkle by dodder or by grafting.

References

- Chen, T.A. 1971. Mycoplasma-like organisms in sieve tube elements of plants infected with blueberry stunt and cranberry false blossom. *Phytopathology* **61**:233-236.
- Dobrosky, I.D. 1929. Cranberry false blossom spread by a leafhopper. *Science* **70**:635.
- Kunkel, L.O. 1945. Studies of cranberry false blossom. *Phytopathology* **35**:805-821.
- Stretch, A.W. 1987. Cranberry false blossom. pp. 110-111. In: *Virus Diseases of Small Fruits*. Ed. R.H. Converse. United States Department of Agriculture, Agriculture Handbook No. 631, Washington, DC.

3. Witches'-broom

Cause

Electron microscopy of ultra-thin sections has revealed polymorphous cell-wall-less non-cultivable mollicutes, often referred to as mycoplasma-like organisms (MLO), in phloem sieve tubes. No dimensions of the causal agent are reported.

Symptoms

Infected *V. myrtillus* plants show a very dense, bushy growth. This is due to the erect position of the excessively formed new branches. This phenomenon is associated with a striking reduction in size of branches and leaves. Branches of plants affected in a later stage of growth have only an erect position instead of the plagiotropic position of normal, healthy plants. Plants affected earlier in their development remain small, are heavily branched and have smaller leaves. Diseased plants drop their leaves later in autumn than healthy plants. Sometimes leaves on infected plants show some reddening due to increased anthocyanin formation. Diseased plants do not flower at all.

Host range

Vaccinium myrtillus, *V. vitis-idaea*, *V. uliginosum* and *V. oxycoccus*.

Geographical distribution

Czechoslovakia, France, Germany, The Netherlands, Scotland and Yugoslavia.

Transmission

The leafhopper *Idiodunus cruentatus* can transmit the disease to *V. myrtillus*, however, the disease also occurs where *I. cruentatus* has not been found. Experiments have indicated that the leafhoppers *Empoasca solani*, *Neophilaenus exclamationis*, *Aphrodes bicinctus*, *Euscelis* spp., and *Macropsis fuscula* do not transmit the disease.



Fig. 46. Symptoms of witches' broom (right) compared to healthy *Vaccinium myrtillus* (left). (Dr. R.H. Converse, Horticultural Crops Research Laboratory, Corvallis)

Indexing

The disease may be detected by graft or vector transmission to *V. myrtillus*, *V. vitis-idaea*, *V. uliginosum* or *V. oxycoccus*.

References

deLeeuw, G.T.N. 1987. Witches'-broom of *Vaccinium*. pp. 108-110. In: *Virus Diseases of Small Fruits*. Ed. R.H. Converse. United States Department of Agriculture, Agriculture Handbook No. 631, Washington, DC.

Prokaryotic disease - bacteria

Crown gall

Cause

The soil-borne bacterium *Agrobacterium tumefaciens* (E.F. Smith & Towns.) Conn, belonging to the family *Rhizobiaceae*. The causal agent of the disease in eastern North America was attributed to *Agrobacterium tumefaciens* var. *rubi* (Hildebrand) Starr & Weiss, a pathogen initially isolated from galls on floricanes of *Rubus* spp.

Symptoms

Galls are most common at the base of canes or on the major roots but rarely on the smaller roots. Occasionally galls form on branches higher in the bush, especially after flooding. Young galls are cream-coloured to light brown while mature galls are dark brown to black. Older galls are rough and hard. Galls may be elongate as a result of several smaller adjacent galls coalescing, but are usually spherical. They vary in size, with a few becoming large (1 to 16 cm in diameter). Infected bushes may be stunted or weak compared to healthy ones. When older bushes (2 to 5 years) are affected the foliage discolours prematurely in the summer. Initially the foliage on an affected branch or bush takes on a reddish hue and then becomes yellowish-brown as the disease worsens.

Host range

Isolates of *A. tumefaciens* from other crops, including apple, dahlia, hop and peach; did not induce gall formation on blueberry. Similarly, isolates from blueberry were unable to infect apple and peach.

Geographical distribution

Crown gall is present in all blueberry growing areas of North America. The disease has also been seen in Chile, but it may have come in on planting stocks from North America.

Biology and transmission

The pathogen requires a wound to enter the host and initiate infection. Wounds can result from natural causes, i.e. lateral root formation or bud scale and leaf scars, or from mechanical causes, i.e. pruning, cultivating, harvesting, insect feeding, root injury at planting, or frost damage. Wounds generally remain susceptible for 2 to 4 days during the warm temperatures of summer; at temperatures below 13°C they can remain susceptible for weeks. During this period infection is also influenced by soil moisture and population levels of the pathogen.

References

- Demaree, J.B. & Smith, N.R. 1952. Blueberry galls caused by a strain of *Agrobacterium tumefaciens*. *Phytopathology* **42**:88-90.
- Moore, L.W. 1980. Controlling crown gall with biological antagonists. *Amer. Nurseryman* **151**:40-44.

Fungal diseases**1. Botryosphaeria stem canker****Cause**

Botryosphaeria corticis (Demaree & Wilcox) von Arx & Müll.

Symptoms

Symptoms first appear as small red lesions on succulent stems 7 days after infection. Lesions develop slowly, becoming swollen and broadly conical within 6 months in susceptible cultivars. Symptoms vary with the susceptibility of the plant. Large swollen cankers with deep fissures and cracks develop on susceptible cultivars after 2 to 3 years, while cankers are restricted in size on the more resistant cultivars. On very susceptible cultivars, e.g. 'Weymouth' and 'Wolcott', cankers enlarge and may girdle and kill the stem.

Host range

The causal fungus infects highbush blueberry (*Vaccinium corymbosum*) and rabbit&eye blueberry (*V. ashei*).

Geographical distribution

Southeastern USA, including New Jersey.

Biology and transmission

Stem infection of current season's growth occurs in late spring when ascospores and conidia are released from perithecia and pycnidia, respectively, during rain and are disseminated throughout the planting. Temperature influences the number and type of lesions. The optimal temperature for fungal growth, sporulation and spore germination is 25-28°C. Disease development is limited to small red flecks at 16°C. However, at optimal temperatures large cankers form and stems become girdled and die.

Reference

Milholland, R.D. & Galletta, G. J., 1969. Pathogenic variation among isolates of *Botryosphaeria corticis*. *Phytopathology* **59**:1540-1543.

2. Cottonball (Hard rot, Tip blight)

Cause

Monilinia oxycocci (Woronin) Honey.

Symptoms

The tip blight stage of the disease is visible in late spring when tips of uprights suddenly wilt. The stem curves over forming a crazier. Masses of greyish-white powdery conidia cover the crazier. Conidia infect flowers via the stigma and style but flowers remain symptomless. Infected fruit ripen abnormally; rarely showing any red colour. Yellowish-brown bands appear lengthwise on infected berries and they expand until the berry is uniformly yellowish-brown in colour. The interior of the berry is filled with the cottony white mycelium of the pathogen. Sclerotia form in infected fruit in the fall (hard rot stage of the disease).



Fig. 47. Tip blight stage of cottonball disease caused by *Monilinia oxycocci*. (Dr. P.R. Bristow, Washington State University, Puyallup)

Host range

American cranberry (*Vaccinium macrocarpon*) and possibly European cranberry (*V. oxycoccus*).

Geographical distribution

North America.

Biology and transmission

The pathogen overwinters as sclerotia on fruits. New vegetative growth is infected by ascospores during early spring. Conidia only infect flowers, leading to the hard rot stage.

References

- Boone, D.M. 1982. Hard rot and tip blight of cranberry. University of Wisconsin Extension A 3194.
- Sanderson, P.G. & Jeffers, S.N. 1992. Cranberry cottonball: Dispersal periods of primary and secondary inocula of *Monilinia oxycocci* host susceptibility and disease development. *Phytopathology* **82**:384-392.
- Sanderson, P.G. & Jeffers, S.N. 1994. Cottonball. In: *Compendium of Blueberry and Cranberry Diseases*. Eds. F.L. Caruso & D.C. Ramsdell. APS Press (in press).

3. Fusicoccum Canker (Godronia canker)**Cause**

Godronia cassandrae Peck (anamorph = *Fusicoccum putrefaciens* Shear).

Symptoms

Reddish-brown elliptical cankers 1 to 10 cm in length form on 1- and 2-year-old stems. These cankers are often covered with black pycnidia that are 1 to 2 mm in diameter. Usually cankers are centered on a leaf scar. Most cankers are in the bottom 1/3 of the bush or in the crown area. During the summer when a fruit load is on the bush, the entire stem may wilt. Leaves turn a reddish-brown colour and remain attached. On older wood (especially pruning stubs), apothecia may form. They are 1 to 3 mm in diameter, hard, black and somewhat difficult to find.

Host range

Found on highbush blueberry (*Vaccinium corymbosum*), *V. angustifolium*, *V. caespitosum*, and *Chamaedaphne calyculata*. The causal organism is similar to that causing end rot of cranberry.

Geographical distribution

Canada, Finland, northern Germany, UK and northern USA.

Biology and transmission

The causal fungus is spread by rain-splashed conidia from pycnidia on the canker surface during the growing season from bud break in spring until leaf drop in autumn. Direct infection occurs on current year as well as 1- and 2-year-old stems.

References

- Lockhart, C.L. & Craig, D.L. 1962. Fusicocum canker of highbush blueberry in Nova Scotia, Canada. *Plant Dis. Survey* **47**:17-20.
- Lockhart, C.L. 1972. Occurrence and pathogenicity of *Godronia cassandrae* f. *vaccinii* on lowbush blueberry in Nova Scotia, Canada. *Plant Dis. Survey* **52**:49-121.
- Parker, P.B. & Ramsdell, DC. 1977. Epidemiology and chemical control of *Godronia (Fusicocum)* canker of highbush blueberry. *Phytopathology* **67** :1475-1480.
- Weingartner, D. P. & Klos, E. J. 1975. Etiology and symptomatology of canker and dieback diseases of highbush blueberries caused by *Godronia (Fusicocum) cassandrae* and *Diaporthe (Phomopsis) vaccinii*. *Phytopathology* **65**:105-110.

4. Mummy berry disease

Cause

Monilinia vaccinii-corymbosi (Reade) Honey.

Symptoms

A shoot blight occurs in early spring when shoots are 4-10 cm long. Symptoms appear about two weeks after ascospore infection of newly emerging leaf buds. Blighted shoots and blossom clusters turn brown. Tan-coloured sporulation forms in tufts on infected leaves. Conidia infect the blossoms as they open. No symptoms occur until fruit ripening, at which point the fruit turns soft and pinkish. The fruits fall to the ground, become shriveled, brown, and resemble a miniature pumpkin.

Host range

Highbush (*Vaccinium corymbosum*), lowbush (*Vaccinium angustifolium*) and rabbiteye (*Vaccinium ashei*) blueberry.

Geographical distribution

North America.

Fig. 48. Mummy berry disease caused by *Monilinia vaccinii-corymbosi*: shoot blight occurring in early spring (top) and pinkish fruits (bottom). (Dr. D.C. Ramsdell, Department of Botany and Plant Pathology, Michigan State University, East Lansing)



Biology and transmission

The fungus overwinters in mummified fruits. With sufficient moisture, apothecia are formed in spring, which liberate ascospores and infect the newly emerging green tissue.

References

- Ramsdell, D.C., Nelson, J.W. & Myers, R.L. 1974. An epidemiological study of mummy berry disease of highbush blueberry. *Phytopathology* **64**:222-228.
- Ramsdell, D.C., Nelson, J.W. & Myers, R.L. 1975. Mummy berry disease of highbush blueberry: Epidemiology and control. *Phytopathology* **65**:229-232.

5. Phomopsis canker of blueberry (twig blight)

Cause

Phomopsis vaccinii Shear.

Symptoms

Cankers may be formed on 1, 2 and 3-year-old stems. They are usually found from the soil-line up to 1 to 1.5 m above ground. Newly formed cankers become evident in early to mid-summer. They are brownish and are on 1-year-old stems with a length of 2 to 10 cm or more. A canker may encircle the entire stem. Cankers on older stems turn a greyish colour and become somewhat flattened. The surface of older cankers is usually covered with pycnidia that are about 0.5 mm in diameter. The cankers usually progress downward and become 10 to 20 cm long, covering the whole shoot. During the summer when a load of fruit is on the bush, cankered stems wilt. Leaves are reddish in colour and will remain on the stem. This symptom is very similar to that associated with *Fusicoccum* canker.

Host range

Limited to highbush blueberry (*Vaccinium corymbosum*).

Geographical distribution

Northern Germany and northern USA.

Biology and transmission

The causal fungus is spread by conidia in pycnidia on cankers as the result of splashing rain. Disease spread occurs from bud break in the spring until late summer when inoculum is depleted. Infection of current year and 1- and 2-year-old stems occurs through wounds caused by mechanical abrasion and frost injury.

References

- Parker, P.E. & Ramsdell, D.G. 1977. Epidemiology and chemical control of *Phomopsis* canker of highbush blueberry. *Phytopathology* **67**:1481-1484.
- Weingartner, D.P. & Klos, E.J. 1975. Etiology and symptomatology of canker and dieback diseases on highbush blueberries caused by *Godronia* (*Fusicoccum*) *cassandrae* and *Diaporthe* (*Phomopsis*) *vaccinii*. *Phytopathology* **65**:105-110.

6. *Phytophthora* root rot

Cause

Phytophthora cinnamomi Rands.

Symptoms

Foliar symptoms depend on the extent of root damage. When severe, early spring growth may wilt and die suddenly. Less severe root damage may result in stunting

of terminal growth, yellowing of leaves, and possibly defoliation. Leaves on canes which die suddenly usually turn orange. The fine fibrous roots are destroyed. Major roots and the crowns show a reddish-brown vascular discoloration when infected.

Host range

Highbush blueberry (*Vaccinium corymbosum*). Rabbiteye blueberry (*V. ashei*) is tolerant to the disease. Fungus reported on some 900 host plants.

Geographical distribution

Worldwide.

Biology and transmission

The fungus is a common soil inhabitant with a wide host range outside of *Vaccinium*. The disease is favoured by warm soil temperatures combined with high soil moisture. The pathogen may be introduced via irrigation water from lakes and rivers. The fungus survives for a number of years in soil or decomposing plant debris as spores, and for shorter periods as chlamydospores. Zoospores released from germinating sporangia are attracted to roots.

References

- Milholland, R.D. 1975. Pathogenicity and histopathology of *Phytophthora cinnamomi* on highbush and rabbiteye blueberry. *Phytopathology* **65**:789-793
- Milholland, R.D. 1994. *Phytophthora* root rot. In: *Compendium Of Blueberry and Cranberry Diseases*. Eds. F.L. Caruso & D.C. Ramsdell. APS Press, St. Paul (in press).

7. Rose bloom

Cause

Exobasidium oxycocci Rostr. ex Shear.

Symptoms

Infected dormant axillary buds begin to grow in the early spring, producing fleshy pink abnormal branches. Affected buds are usually on the previous year's growth (one year-old wood). Leaves on the abnormal branch are swollen and close together. The branch looks like a miniature rose blossom, hence the common name for the disease. Prior to bloom the entire surface of the abnormal branch becomes

covered with whitish masses of basidia and basidiospores. Basidiospores infect axillary buds on the current year's growth which give rise to the abnormal branches the following spring. By late June the abnormal branches wither and turn black.

Host range

American cranberry (*Vaccinium macrocarpon*), European cranberry (*V. oxycoccus*) and *V. palustris*.

Geographical distribution

Primarily in Oregon, Washington and British Columbia, but also reported from other cranberry growing regions of North America. Originally reported from northern Europe.

Transmission

Primarily in infected buds of one-year old wood.

Reference

Bristow, P.R. 1994. Rose bloom. In: *Compendium of Blueberry and Cranberry Diseases*. Eds. F.L. Caruso & D.C. Ramsdell. APS Press (in press).



Fig. 49. Rose bloom of cranberry caused by *Exobasidium oxycocci* fleshy pink branches. (Dr. R.S. Byther, Washington State University, Puyallup)

8. Twig blight

Cause

Lophodermium oxycocci (Fr.) P. Karst. and *L. hypophyllum* (Dearn. & House) Shear.

Symptoms

Infection of new vine growth occurs during the summer but symptoms do not appear until winter or more commonly the following spring. Hence, symptomless dormant vines maybe infected. The fungus kills only 1-year-old wood and does not progress into older wood. Leaves on 1-year-old wood first turn light brown but fade to a bleached tan and eventually silvery grey. Infected leaves are dull. Elliptical black apothecia form on the underside of infected leaves. Mature apothecia open by a median slit to expose the hymenial layer.

Host range

Limited to the American cranberry (*Vaccinium macrocarpon*) and possibly the European cranberry (*V. oxycoccus*) and Lingonberry (*V. vitis-idaea*).

Geographical distribution

Cranberry growing regions in North America, but primarily in the states of Oregon and Washington and Province of British Columbia, as well as Northern Europe.

Biology and transmission

Transmitted by vegetative propagation material and ascospores.



Fig. 50. Apothecia of the twig blight fungus *Lophodermium oxycocci* on cranberry leaves. (Dr. P.R. Bristow, Washington State University, Puyallup)

References

- Bristow, P.R. 1983. *Lophodermium* twig blight of cranberry; relationship between inoculum density, period of susceptibility and disease severity. *Phytopathology* **73**:957 (abstr.).
- Bristow P.R. 1994. Twig blight. In: *Compendium of Blueberry and Cranberry Diseases*. Eds. F.L. Caruso & D.C. Ramsdell. APS Press (in press).

9. Upright dieback (Phomopsis canker of cranberry)

Cause

Diaporthe vaccinii Shear
(anamorph: *Phomopsis vaccinii* Shear).

Symptoms

Diseased upright branches have a yellowish cast in early spring that later may become orange or bronze. When the branch dies it turns brown. At first, individual leaves may exhibit a yellow mottling. The pathogen infects fruits causing a berry rot called “viscid rot”. Infected leaves are soft and pale in colour. The most diagnostic symptom for viscid rot is the stringing out of a viscous substance when a finger is touched to and then withdrawn from the cut surface of the rotted berry.

Host range

American cranberry (*Vaccinium macrocarpon*), European cranberry (*V. oxycoccus*).

Geographical distribution

North America.

Biology and transmission

The pathogen can be isolated from symptomless cranberry tissues, suggesting that incipient disease exists. While little is known about the disease cycle, the disease appears to be favoured by warm temperatures. The pathogen may overwinter in infected berries left in beds. Both pseudothecia and pycnidia have been observed on rotted berries. Both types of fruiting bodies are occasionally found on uprights killed during the previous crop year.

Reference

- Boone, D.M. 1982. Viscid rot and upright dieback of cranberry. University of Wisconsin. Extension Bulletin 3195.

Pests of small fruit

Arthropods

Many insects and mites are associated with small fruit germplasm. Some vector viruses, while others are economic pests in their own right. Since information is incomplete with respect to the rather large number of arthropods that are found on the plant material that could be transported for breeding purposes, the application of sound phytosanitary procedures to prevent the movement of pests is necessary.

Arthropods that are feeding on the roots should be eliminated by propagating runners or stem cuttings. If it is necessary to move whole plants, the soil must be washed from the roots. Vegetative material should be treated with a fumigant that has ovicidal activity, or dipped in an appropriate insecticide prior to potting in sterilized media. Placement of a systemic insecticide (e.g. aldicarb or oxamyl) in the root zone of the plant will kill the remaining insects and mites that are feeding within the plant tissues. Since some arthropod pests may emerge from the plant material during treatment, the plants should be isolated within a quarantine facility for at least 10 months. Seed should be free of fruiting material, and fumigated to eliminate stored products pests if observed or suspected. Pollen may be contaminated with mites and insects. It may also carry fungi and bacteria that cause diseases of bees. Although storage of pollen at -20 C will be lethal to many arthropods, it may not kill all of them. There is no technique available to eliminate fungi or bacteria that cause bee diseases without destroying the viability of the pollen. Tissue culture is the best way of ensuring pest-free material.



Fig. 51. Damage of eriophyid mite on *Ribes curvatum*. (Dr. J. Postman, USDA-ARS-NCGR, Corvallis)

Eriophyid mites could present special problems for the transfer of *Ribes* germplasm. In particular, the black currant gall mite *Cecidophyopsis ribis* is a serious pest of black currant, inducing galls of buds. It is also a vector of currant reversion disease, which occurs in *Ribes* worldwide with the exception of the Americas. Gall mites can be eradicated from buds by immersing them in water at 46 C for 10-20 minutes. However, vegetative material should be treated with a systemic insecticide that has proven acaricidal value, to eliminate any of the 14 known species of eriophyids on *Ribes*.

References

- Alford, D.V. 1984. *A Colour Atlas of Fruit Pests: Their Recognition, Biology and Control*. Wolfe, London.
- Hill, D.S. 1987. *Agricultural Insect Pests of Temperate Regions and their Control*. Cambridge University Press, Cambridge.
- Jeppson, L.R., Keifer, H.H. & Baker, E.W. 1975. *Mites Injurious to Economic Plants*. University of California Press, Berkeley.

Nematodes

Nematodes may be found associated with soil surrounding plant roots, within the roots themselves, or within the above-ground plant tissues. Symptoms of infection often go undetected and precautions should be taken to eliminate these organisms. Nematodes that feed on the roots can be eliminated by propagating runners or stem cuttings. If it is necessary to move whole plants, all soil must be washed from the roots. All vegetative material should be potted in sterilized media and treated with a systemic nematicide (e.g. aldicarb or fenamiphos) on a regular basis for at least 10 months to kill the remaining nematodes. During this time, the plants should be isolated in a quarantine facility. Seed and pollen may be moved without concern for nematodes. Tissue culture is the best way of ensuring nematode-free material.

Bud and leaf nematodes of the genera *Aphelenchoides* and *Ditylenchus* can present a difficult problem for the transfer of germplasm. In particular, the strawberry eelworm, *Aphelenchoides fragariae*, deserves special mention. It is a serious pest in Europe, causing severely distorted strawberry leaves and reduction of plant growth. The nematodes inhabit the folded young leaves of crowns and runner shoots. Field populations normally increase in the early spring and autumn, but plant damage is not manifested until late spring and summer, when populations have already declined. Under glasshouse conditions suitable for

runner production, they may survive in low numbers, causing no symptoms until they are transferred to the field where they experience a period of increasing temperatures. They also survive for long periods on plants maintained *in vitro*. No combination of conventional treatments, nematicide, hot-water treatment (46 C for 10 min.) or meristem tip culture is capable of ensuring freedom from these nematodes. However, nematode infestation can be entirely eliminated from high-value strawberry plants (e.g. nuclear stock) by cutting away the upper part of the crown containing the leaves, thus removing all green tissue from the plant, and then allowing regeneration from previously dormant buds below.

References

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- McNamara, D.G., Stickles, J.E. & Flegg, J.J.M. 1986. Control of leaf nematodes in nuclear stock strawberries. pp. 217-219. In: British Crop Protection Council Monograph No. 33. Symposium on Healthy Planting Material.

APPENDIX 1: INSTITUTIONS MAINTAINING SMALL FRUIT GERmplasm ***

Centre de Recherches Agronomiques
Station des Cultures Fruitières et
Maraîchères
234 chaussee de Charleroi
B-5030 Gembloux
BELGIUM

Fragaria, *in-vitro*
virus-tested

Plant Gene Resources of Canada
Canadian Clonal Genebank
Agriculture Canada
P.O. Box 340
Trenton, Ontario K8V 5R5
CANADA

Fragaria and *Rubus*
some material is virus-tested;
also virus-tested tissue cultures
available

Agricultural Research Centre of Finland
Laukaa Research and Elite Plant Unit
Juntula
FIN-41340 Laukaa
FINLAND

Fragaria, *Ribes*, *Rubus*, *Vaccinium*
also virus-tested tissue cultures
available

Centre Interrégional de Recherche et
d'Expérimentation de la Fraise
C.I.R.E.F.
Lanxade
24130 Prignonieux
FRANCE

Fragaria
some material is virus-tested;
also virus-tested tissue cultures
available

Centre Technique Interprofessionel des
Fruits et Légumes (C.T.I.F.L.)
B.P. 32
Centre de Balandran
30127 Bellegarde
FRANCE

Fragaria
some material is virus-tested;
also virus-tested tissue cultures
available

G.E.V.E.S. (Dr. F. Boulineau)
49250 Brion
FRANCE

Fragaria
virus-tested

Institute for Fruit and Ornamentals
Dept. of Fruit Breeding
Fertod - H 9431
HUNGARY

Holt Research Station
P.O. Box 2502
N 9002 Tromso
NORWAY

Research Institute of Pomology and
Floriculture
P.O. Box 105
96100 Skierniewice
POLAND

N.I. Vavilov Institute of Plant Industry
Bolshaya Morskaya 4244
190000 St. Petersburg
RUSSIA

Centro de Investigación y Desarrollo
Agrario
C.I.D.A.
Finca Cortijo de la Cruz
29140 Churriana
Malaga
SPAIN

Horticulture Research International
East Malling
Kent, ME 196BJ
UNITED KINGDOM

Fragaria, Rubus, Ribes
some material is virus-tested;
also virus-tested tissue cultures
available

Rubus chamaemorus,
Ribes spicatum 'Atlas',
wild *Ribes spicatum*.
The material is not virus-tested.

Fragaria, virus-tested: Senga
Sengana', 'Cama', 'Dukat'
Rubus, virus-tested: 'Veten',
'Norna', 'Canby', 'Mailing Seedling',
'Polana', 'Beskid', 'Mailing Jewel'

Fragaria, Rubus, Ribes
part of the collection has undergone
meristem tip culture, but no virus
indexing has been conducted.
Accessions received from other
genebanks are supplied with
information on the virus status.

Fragaria
some material is virus-tested

Fragaria, in vitro
some material is virus-tested
Rubus
untested root cuttings

Scottish Crop Research Institute
 Invergowrie
 Dundee DD25DA
 Scotland
 UK

Fragaria, Ribes, Rubus. virus
 indicator species

Prof. Dr. Donald C. Ramsdell
 Dept. of Botany and Plant Pathology
 166 Plant Biology Building
 Michigan State University
 East Lansing, MI 48824
 USA

virus-tested highbush blueberry

USDA-ARS
 National Plant Germplasm Quarantine
 Center
 Building 508 BARC-E
 Beltsville, MD 20705
 USA

Fragaria
 seeds of virus indicator 'Alpine'
Ribes, Rubus
 cuttings of virus indicators 'Lloyd
 George', 'Norfolk Giant', 'Munger',
 'Baldwin', 'Amos Black'

USDA-ARS-NCGR
 33447 Peoria Road
 Corvallis, OR 97333
 USA

Fragaria, Ribes, Rubus, Vaccinium
 Some accessions are untested or
 virus-infected;

*** This is not an exhaustive list; it contains information given by the contributors to these guidelines. The details given in the table were supplied by the institutions listed. FAO and IPGRI do not guarantee the accuracy of this information. Institutions and individuals wishing to import material on this list should negotiate directly with the supplier. FAO and IPGRI cannot be held responsible for problems resulting from the introduction of material from the sources listed here.

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Edible Aroids	1989
<i>Musa</i>	1989
Sweet Potato	1989
Yam	1989
Legumes	1990
Cassava	1991
Citrus	1991
Grapevine	1991
Vanilla	1991
Coconut	1993
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