Stone Fruits

edited by **M. Diekmann and C.A.J. Putter**

in *collaboration* with Istituto Sperimental per la Patologia Vegatale, Roma

Previously published Technical Guidelines for the Safe Movement of Germplasm

Cocoa	1989
Edible Aroids	1989
Musa (1st edition)	1989
Sweet Potato	1989
Yam	1989
Legumes	1990
Cassava	1991
Citrus	1991
Grapevine	1991
Vanilla	1991
Coconut	1993
Sugarcane	1993
Small fruits *(Fragaria, Ribes, Rubus, Vaccinium)*	1994
Small Grain Temperate Cereals	1995
Musa spp. (2nd edition)	1996

CONTENTS

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 pests[1] along with the host plant. In particular, pathogens that are often symptomless, such as viruses, pose a special risk. In order to manage 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 for research purposes, coupled with recent 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 purpose 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 scope of the recommendations in these guidelines is confined to small, specialized consignments used in technical crop improvement programmes, e.g. for research and basic plant breeding programmes. When collecting germplasm, the local plant quarantine procedures, e.g. pest risk assessment, should be considered.

These 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 for whom they work. The guidelines are intended to be the best possible advice to institutions involved in germplasm exchange for research, conservation and basic plant breeding. 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

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

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

The guidelines are written in a short, concise style, in order to keep the volume of the document to a minimum and to facilitate updating. Suggestions for further reading are given at the end, along with the references cited in the text (mostly for geographical distribution, media and other specific information). The guidelines are divided into two parts. The first part makes general recommendations on how best to move stone fruit germplasm. The second part covers the important pests and diseases of quarantine concern. The information given on a particular pest or disease is not exhaustive but concentrates on those aspects that are most relevant to quarantine.

In the present guidelines stone fruits, i.e. almond *(Prunus amygdalus* Batsch, syn. *P. dulcis* (Miller) D.A. Webb), apricot *(Prunus armeniaca* L.), cherry *(Prunus avium* (L.) L., *P. cerasus* L.), peach *(Prunus persica* (L.) Batsch), plum and prune *(Prunus domestica* L., *Prunus salicina* Lindley) are covered. In the text reference is made to common names only. Only pests known to be transmissible with planting material are included. Those attacking fruits only are not included, as are those which are no threat in germplasm movement, such as frosty mildew, cherry scab and rust caused by *Pucciniastrum* species.

The present guidelines were developed at a meeting held in Rome, Italy from 22 to 24 June, 1994. The meeting was hosted by the Istituto Sperimentale per la Patologia Vegetale in Rome.

Input from the following colleagues who could not attend the meeting is gratefully acknowledged: Dr M. Cambra, Instituto Valenciano de Investigaciones Agrarias Moncada (Valencia), Spain; Dr J.C. Desvignes, Centre Technique Interprofesionnel des Fruits et Légumes, Lanxade, France; Dr R. Flores, Instituto de Biología Molecular y Celular de Plantas, Valencia, Spain; Dr W. Jelkmann, Institut für Pflanzenschutz im Obstbau der Biologischen Bundesanstalt, Dossenheim, Germany; Dr E. Pfeilstetter, Braunschweig, Germany; Dr E. Seemüller, Institut für Pflanzenschutz im Obstbau der Biologischen Bundesanstalt, Dossenheim, Germany.

Guideline Update

In order to be useful, the guidelines need to be updated when necessary. We ask our readers to kindly bring to our attention any developments that possibly require a review of the guidelines, such as new records, new detection methods, new control methods, etc. For your convenience, please use the form provided on the last page of this publication.

MEETING PARTICIPANTS

Dr Marina Barba
Virologist
Istituto Sperimentale
per la Patologia Vegetale
Via C. G. Bertero, 22
00156 Rome
ITALY
Tel: +39 6 86890251/2-3-4
Fax: +39 6 86802296

Dr Marlene Diekmann
Germplasm Health Scientist
IPGRI
Via delle Sette Chiese, 142
00145 Rome
ITALY
Tel: +39 6 51892223
Fax: +39 6 5750309
e-mail: m.diekmann@cgnet.com

Dr Luciano Giunchedi
Virologist
Istituto di Patologia Vegetale
Via Filippo Re, 8
40126 Bologna
ITALY
Tel: +39 51 351433
Fax: +39 51 351433

Dr Ahmed Hadidi
Research Plant Pathologist
Molecular Virology Investigations
National Germplasm
Resources Laboratory
USDA, ARS
Bldg. 011A, Rm. 106, BARC-W
Beltsville, MD 20705
USA
Tel: +1 301 504 6460
Fax: +1 301 504 5551
e-mail: ngrlah@ears-grin.gov

Dr Thomas Hasler
Virologist
Swiss Fed. Res. Station for Fruit Growing
8820 Wädenswil
SWITZERLAND
Tel: +41 1 7836111
Fax: +41 1 7806341
e-mail: thomas.hasler@wae.faw.admin.ch

Dr Alan Jones
Plant Pathologist
Michigan State University
Department of Botany and Plant
Pathology
East Lansing, MI 48824
USA
Tel: +1 517 3554573
Fax: +1 517 3535598

Dr Margit Laimer da Camara Machado
Plant Biotechnologist
Institute of Applied Microbiology
University of Agriculture
Nussdorfer Lände 11
1190 Vienna
AUSTRIA
Tel: +43 1 3692924 ext 402
Fax: +43 1 3692924400
e-mail: laimer@mail.boku.ac.at

Dr Gerardo Llácer
Virologist
IVIA-Instituto Valenciano
de Investigaciones Agrarias
Apdo. Oficial
46113 Moncada (Valencia)
SPAIN
Tel: +34 6 1391000
Fax: +34 6 1390240

Dr Tonie Putter
Epidemiologist
FAO-AGPP
Via delle Terme di Caracalla
00100 Rome
ITALY
Tel: +39 6 52254022
Fax: +39 6 52256347
e-mail: tony.putter@fao.org

Prof. Antonio Ragozzino
Virologist
Università degli Studi di Napoli
Facoltà di Agraria
Istituto di Patologia Vegetale
Via Università, 100
80055 Portici (Napoli)
ITALY
Tel: 081 274752/274536
Fax: 081 7753579

Prof. Carmelo Rapisarda
Entomologist
Università degli Studi di Catania
Istituto di Entomologia Agraria
Via Valdisavoia, 5
95123 Catania
ITALY
Tel: +39 95 350721 or 359793
Fax: +39 95 356752

Prof. Dr Wolfgang Zeller
Bacteriologist
BBA
Institut für Pflanzenschutz im Obstbau
Schwabenheimer Strasse 101
69221 Dossenheim
GERMANY
present address:
BBA
Institut für biologischen Pflanzenschutz
Heinrichstr. 243
64287 Darmstadt
GERMANY
Tel: +49 6151 4070
Fax: +49 6151 40790
e-mail: ibp@hrzpub.th-darmstadt.de

GENERAL RECOMMENDATIONS

- Under no circumstances should germplasm be moved as rooted plant material.
- Germplasm should preferably be moved as *in vitro* cultures.
- Pest Risk Assessment methods should be applied when selecting accessions and cultivars for introduction.
- All germplasm should undergo therapy and indexing procedures.
- Germplasm should be accurately labeled to indicate the source and treatments applied.

TECHNICAL RECOMMENDATIONS

Stone fruit germplasm can be moved as seed, dormant or green budwood/budsticks, *in vitro* cultures, or pollen.

A. Collecting and movement of seeds

- Germplasm should be collected from plants free from pests and disease symptoms.
- Seeds should be extracted from the pulp soon after harvest.
- Seeds should be surface-sterilized with a freshly prepared 0.5% sodium hypochlorite solution with 0.1% wetting agent for 10 minutes and then thoroughly rinsed with water
- Seeds should be surface-dried under shaded conditions and dusted with a fungicide.
- Seeds should be vernalized and germinated in sterilized soil mix in an insect-free containment facility.
- Material should be indexed for seed-transmitted pathogens such as Prunus necrotic ringspot virus or prune dwarf virus.

B. Collecting and movement of budwood/budsticks

- Collecting tools (clippers, knives, etc.) should be sterilized by dipping them in a freshly prepared 0.5-1% sodium hypochlorite solution.
- Budwood/budsticks should be collected from plants which have been evaluated for freedom of symptoms on fruit, foliage, branches and trunk. Bark patches and the exposed wood surface should be observed.
- Budwood/budsticks should be collected from 1-year-old branches only, preferably as dormant cuttings, and fumigated before shipment with hydrogen cyanide for 1 hour. They should then be dipped in a solution of organophosphorous insecticide and/or acaricide.
- If budsticks are taken, leaves should be removed.
- In preparation for distribution, collected cuttings should be thoroughly washed. They should then be dipped in a 0.5% sodium hypochlorite solution with 0.1% wetting agent, rinsed thoroughly and towel-dried. The ends of dormant budwood may be dipped in melted low-temperature paraffin wax.
- Material should then be submitted to the indexing procedures described below.

C. Establishment and movement of *in vitro* cultures

- Apical or axillary buds for *in vitro* culture should be collected from plants which have been evaluated for freedom of symptoms on fruit, foliage, branches and trunk. Bark patches and the exposed wood surface should be observed.
- For the movement of *in vitro* cultures, neither antibiotics nor charcoal should be added to the medium.
- Clear plastic culture vessels should be used and the agar concentration should be increased to avoid damage to the plantlets while in transit.
- Special care should be taken to protect the material from extreme temperatures.
- Upon receipt, the material should undergo the indexing procedures described below.

D. Movement of pollen

- Pollen should be collected from pathogen-tested plants.
- Pollinated mother plants and progeny seedlings derived from other pollen sources should be tested for pollen-transmitted viruses.
- Imported pollen found to carry arthropod pests and fungal pathogens of bees should be destroyed.

DISEASE INDEXING AND THERAPY STRATEGY

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

The therapy and indexing procedures required to safely introduce stone fruit germplasm vary with the type of material to be introduced. The risk of introducing pests is reduced by moving seeds rather than budwood/budsticks and by moving in vitro cultures rather than seeds.

To optimize the sensitivity of the biological tests, donor source plants and indicator plants should be actively growing and be free from pests. 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 to 18 hours per day.

Detection of graft-transmitted diseases in stone fruits is largely based on biological indexing, complemented by laboratory tests as listed below:

- observation of symptoms on seedlings or growing plants
- mechanical transmission to herbaceous plants or grafting
- ELISA
- culturing (for bacteria and fungi)
- sPAGE, two-dimensional or return PAGE (for viroids)
- fluorescence microscopy
- dsRNA analysis
- nucleic acid hybridization
- PCR
- immuno tissue printing.

A. Seeds

- Material derived from seeds should be indexed for seed-transmitted viruses before being released.
- If material is found to be infected it should be destroyed or subjected to thermotherapy and/or meristem-tip culture, and retested for freedom from viruses.

B. Budwood/budsticks

- It is recommended that 5-10 budsticks from a single tree be introduced for each accession, to provide material for both propagation and direct indexing.
- As soon as possible after receipt, hot-water therapy at 50°C for 45 minutes should be applied. This treatment should only be applied to cuttings that are fully dormant. If dormant canes show signs of bud break, the procedure for green cuttings should be followed.
- Budsticks should be grafted on virus-tested rootstocks or on virus-free seedlings. Careful observations of these plants for normal growth should be made over 2 years.

C. *In vitro* cultures

- *In vitro* cultures are not necessarily virus-free (Fig. 1).
- *In vitro* cultures should be checked for microbial contamination and contaminated tubes should be discarded.
- Plantlets should be established in sterile potting mix and then indexed.
- If material is found to be infected, it should be destroyed or subjected to thermotherapy and/or meristem-tip culture, and retested for freedom from viruses.

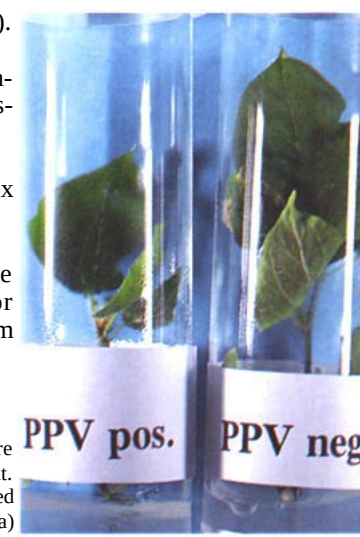

Fig. 1. Symptoms of plum pox virus on an apricot tissue culture plantlet after 2 years *in vitro* (left); a healthy plantlet on the right. (Dr M. Laimer da Camara Machado, Institute of Applied Microbiology, University of Agriculture, Vienna)

DEFINITIONS OF TERMS AS USED IN THIS PUBLICATION

Anamorph
Imperfect state of a fungus, usually producing conidia.

Budstick
A shoot of a plant from which buds are cut for propagation of the plant.

Budwood
Dormant 1-year-old shoots of a plant from which chip buds are cut for propagation of the plant.

Cosmopolitan
This expression is used to describe the distribution of pathogens which are reported to occur in all continents, and in many countries of these continents.

Teleomorph
Perfect state of a fungus, usually producing ascospores or basidiospores.

DESCRIPTIONS OF PESTS

Viruses and viroids

1. American plum line pattern virus (APLPV)

Quasi-isometric ilarvirus; particle size 26-33 nm diameter, sedimenting as four components.

Significance
No significance in stone fruit.

Symptoms
In *Prunus* spp., the symptoms are line pattern as well as chlorotic lines and bands (Fig. 2). Similar symptoms are induced by apple mosaic virus and certain strains of Prunus necrotic ringspot virus, neither of which is serologically related to APLPV.

Hosts
- natural: many cultivars of plum and other *Prunus* spp.
- experimental: wide range of herbaceous hosts infected by sap inoculation.

Geographical distribution
Canada, Mexico, USA (Smith *et al.* 1992).

Biology and transmission
Transmitted by grafting and mechanical inoculation.

Detection
Grafting on peach seedling: fine, wavy, light green or yellowish green band on both leaf surfaces, either running parallel to the main vein or as a line pattern along the veins. Diagnostic herbaceous hosts are *Vigna cylindrica, Crotalaria juncea, Nicotiana megalosiphon.*
Serology (ELISA).

For further reading, see p. 90.

Fig. 2. Chlorotic lines and bands on plum leaves caused by American plum line pattern virus. (Dr A. Hadidi, USDA-ARS, Beltsville)

2. Apple chlorotic leafspot virus (ACLSV)

Trichovirus; elongated, flexuous particles of approximately 720 x 12 nm encapsulating a single-stranded RNA (Martelli *et al.* 1994). Isolates vary in serological relationship and symptomatology.

Significance
Some strains induce serious diseases in stone fruits, such as pseudopox disease of plum and apricot, and plum bark split.

Symptoms
Symptoms appear generally on leaves, fruits and trunk; their severity depends largely on plant species and virus strain. Some strains cause bark splitting on the stem or pseudopox symptoms on fruits of apricot (Fig. 3), peach and plum. Alone or in mixed infection with Prunus necrotic ringspot virus it causes necrotic, sunken spots on fruits of sweet and sour cherry. Most cultivars are latently infected by the virus. In some cultivars of peach, ACLSV causes dark green, sunken spots or wavy lines and light-coloured rings like those induced by plum pox virus on leaves (Fig.. 4). Some strains cause graft incompatibility or severe fruit malformation (apricot pseudopox, apricot fruit blotch) in apricot (Desvignes and Boye 1990).

Fig. 3. Pseudopox symptoms caused by apple chlorotic leafspot virus on apricot in a Spanish local variety. (Dr G. Llácer, Instituto Valenciano de Investigaciones Agrarias, Moncada)

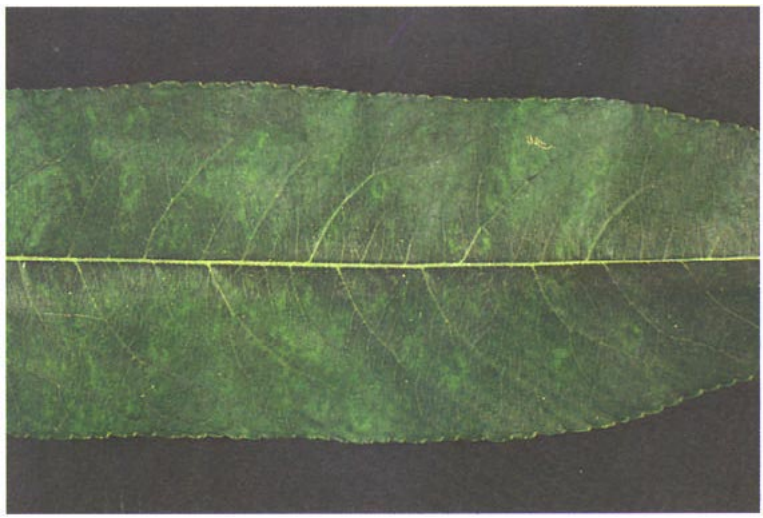

Fig. 4. Wavy lines and light-coloured rings caused by apple chlorotic leafspot virus in peach. (Dr L. Guinchedi, Istituto di Patologia Vegetale, Bologna)

Hosts

- natural: the most important cultivated Rosaceae; in addition to stone fruits also quince, apple and pear.
- experimental: several herbaceous hosts and many Rosaceae (Németh 1986).

Geographical distribution

Cosmopolitan.

Biology and transmission

The virus is transmitted by grafting and sap inoculation.

Detection

ACLSV is detectable by sap transmission on herbaceous hosts. It is also detectable by serological tests (ELISA or immuno tissue printing, Knapp *et al.* 1995) and molecular techniques. The main woody indicators used for ACLSV detection are 'GF305' seedlings, which react with a dark green sunken mottle on leaves, and *Prunus tomentosa.*

For further reading, see p. 90.

3. Apple mosaic virus (ApMV, European plum line pattern)

Isometric or quasi-isometric ilarvirus, particles 25 to 30 nm in diameter. All particles are serologically and electrophoretically homogeneous, but only the larger onesare infective. The same virus causes apple mosaic and rose mosaic. It is serologically distantly related to Prunus necrotic ringspot virus and Danish line pattern virus, but not to Tulare apple mosaic virus and American plum line pattern virus.

Significance

Extent of the damage depends on the virus strain and cultivar. Infection may result in considerable growth and yield reduction.

Symptoms

Light green, yellowish or bright yellow patterns (Fig. 5) develop on plum, apricot, peach and almond leaves; these may form bands, rings or oak-leaf patterns (Fig. 6). Bright yellow vein clearing may also appear. Symptoms are visible mainly in spring or early summer and become masked at higher temperatures. In some cultivars of almond the virus induces the failure of blossom and leaf buds to grow (almond leaf failure).

Hosts

- natural: various *Prunus* spp., such as apricot, cherry, plum and peach show plum line pattern-like symptoms when infected. Not all isolates from *Prunus* induce typical mosaic symptoms in apple. ApMV also occurs naturally in apple, strawberry, *Rubus* spp., *Rosa* spp., birch *(Betula* spp.), hop *(Humulus lupulus),* horse chestnut *(Aesculus hippocastanum)* and filbert *(Corylus maxima).*
- experimental: over 65 herbaceous plant species in 19 families are susceptible to mechanical inoculation. Among these are *Chenopodium quinoa, C. amaranticolor, Cucumis sativus, Cucurbita maxima, Nicotiana clevelandii* and *Petunia hybrida.*

Fig. 5. Bright yellow patterns on a peach leaf caused by apple mosaic virus. (Dr L. Guinchedi, Istituto di Patologia Vegetale, Bologna)

Fig. 6. Oak-leaf pattern caused by apple mosaic virus on apricot. (Dr L. Guinchedi, Istituto di Patologia Vegetale, Bologna)

Geographical distribution
Cosmopolitan.

Biology and transmission
Transmitted by grafting, and to herbaceous plants by mechanical inoculation. No natural vectors are known. Seed transmission has been reported in hazelnut (Cameron and Thompson 1985).

Detection
Graft inoculation on 'GF305' peach seedling or peach cv. Elberta; infected leaves show light green, yellowish green or bright yellow rings, spots, bands or oak-leaf patterns. Mechanical inoculation on *Petunia hybrida* results in lines and grey concentric rings, whereas *Chenopodium quinoa* reacts with systemic irregular chlorotic to necrotic lesions. Also detection serologically by gel-diffusion test and by ELISA. ApMV is detected by ELISA throughout the growing season in individual samples of young leaves or twigs with newly formed buds, and less readily in mature leaves after June.

Treatment
Virus-free plants can be propagated from tips of actively growing shoots maintained at 37°C for 2 to 4 weeks.

For further reading, see p. 91.

4. Cherry green ring mottle virus

Thin, flexuous, rod-shaped closterovirus of 1000 to 2000 nm length and 5 to 6 nm diameter (Zagula *et al.* 1989).

Significance
Affected fruits can not be marketed.

Symptoms
In mature leaves of sour cherry a yellow and green mottle (green island or ringlike bands) appears about 4 to 6 weeks after petals fall. Some virus strains may also induce irregular necrotic spots of varying size. Leaves with symptoms soon drop. Mottle symptoms do not necessarily occur each year.

A constricting chlorosis may appear in areas along the midribs or major lateral veins. Linear areas of chlorotic and distorted tissue appear along these veins (Fig. 7). Some strains induce necrotic pits or rings on fruits (Fig. 8). The affected fruits are bitter and off-flavour.

Infected trees of *Prunus serrulata* (cv. Shirofugen and cv. Kwanzan) show epinasty of the foliage. Portions of the midrib or lateral veins become necrotic, resulting in twisting and curling of affected leaves. Internodes of elongating terminals are shortened. The bark is often roughened by the development of longitudinal fissures.

Fig. 7. Linear areas of chlorotic and distorted tissue on sour cherry leaves, caused by cherry green ring mottle virus. (Dr T. Hasler, Swiss Fed. Res. Station for Fruit Growing, Wädenswil)

Fig. 8. Necrotic pits and rings on cherries caused by cherry green ring mottle virus. (Dr T. Hasler, Swiss Fed. Res. Station for Fruit Growing, Wädenswil)

Hosts
Prunus cerasus, P. serrulata, P. cerasus x P. avium. Latent infections are common in apricot, peach and sweet cherry.

Geographical distribution
Australia, Belgium, Canada, former Czechoslovakia, France, Germany, Hungary, New Zealand, South Africa, Switzerland, USA (Németh 1986).

Biology and transmission
The virus spreads by grafting. In orchards a slow natural spread, mostly to neighbouring trees, has been observed. Transmission experiments to herbaceous plants were unsuccessful. No vector has been reported.

Therapy
Thermotherapy.

Indexing
Prunus serrulata cv. Kwanzan.

For further reading, see p. 91.

5. Cherry leaf roll virus (CLRV)

Cherry leaf roll virus is a nepovirus with isometric particles of 28 nm in diameter. The two RNA species are single-stranded. Isolates from different natural host species or from the same species are serologically distinguishable from each other (Jones 1985).

Significance
Rare in cherry. Economically important in walnut.

Symptoms
Symptoms appear generally on the leaves and on the trunk; their severity depends largely on plant species and virus strain. In cherry, the virus induces delay of flowering, leaf rolling and plant death. In other species chlorotic ringspot, line patterns and/or yellow vein netting may occur.

Hosts
- natural: mainly woody hosts, for example: cherry, walnut, olive, elm *(Ulmus* spp.), birch *(Betula* spp.), ash *(Fraxinus* spp.), elderberry *(Sambucus* spp.), beech *(Fagus* spp.), rhubarb *(Rheum* spp.), dogwood *(Cornus florida),* lilac *(Syringa vulgaris).*
- experimental: numerous herbaceous hosts in more than 36 plant families, and also *Prunus persica* and *P. domestica* (Németh 1986).

Geographical distribution
Europe, North America, former USSR (Németh 1986).

Transmission
The virus is transmitted by grafting, pollen and seeds in some hosts. *Xiphinema coxi, X. diversicaudatum* and *X. vuittenezi* have been reported to be able to transmit CLRV (Fritzsche and Kegler 1964; Flegg 1969). Further evidence for nematode transmission failed when cherry, golden elderberry and rhubarb strains were used with the same or other nematodes (Jones 1985). CLRV is seedborne (0.5-35%) in most natural hosts, up to 100% in many herbaceous hosts. Strong evidence indicates that CLRV is transmitted also by pollen in walnut, birch and elm.

Detection
CLRV is easily detected by sap transmission on herbaceous hosts (mainly *Chenopodium quinoa, Cucumis sativus, Nicotiana* spp.). It can also be detected by serological tests (ELISA) and molecular assays. Woody indicators ('GF305'), when chip-budded with infected *Prunus* spp., show rosetting and slight leaf rolling.

For further reading, see p. 92.

6. Cherry little cherry

Closterovirus-like particles appear to be associated with the disease (Raine *et al.* 1975).

Significance
Particularly important on large-sized, dark-fruited cherry cultivars.

Symptoms
Fruits initially normal but fail to fully ripen. Depending on cultivar, season and location, the fruits are pointed in shape, poorly coloured, small and insipid in taste (Fig. 9). Leaf symptoms like reddening (Fig. 10) or bronzing are usually most apparent in September and October, particularly on sensitive cultivars such as 'Sam', 'Van' and 'Star'.

Hosts
Sweet cherry, also sour and ornamental cherries as well as other *Prunus* spp.

Geographical distribution
Australia, Belgium, Canada, France, Germany, Hungary, Italy, Japan, New Zealand, Norway, Poland, Romania, Spain, Sweden, Switzerland, UK, USA, former USSR (Smith *et al.* 1992).

Biology and transmission
By grafting and the apple mealybug *Phenacoccus aceris.*

Detection
Grafting to the woody indicators 'Canindex' and 'Sam'; molecular hybridization.

For further reading, see p. 92

Fig. 9. Symptom of cherry little cherry disease in sweet cherry 'Sam'; small, poorly colored fruits on the right, healthy plants on the left. (Dr. W. Jelkmann, Institut fur Pflanzenschutz im Ostbau der Biologischen Bundesanstalt, Dossenheim)

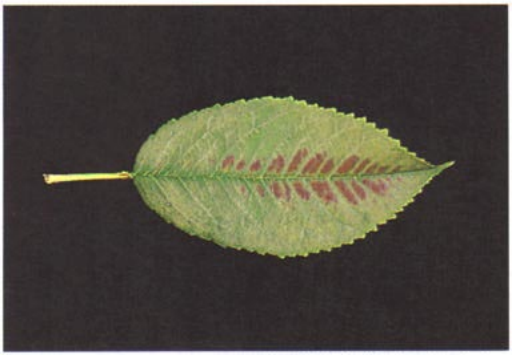

Fig. 10. Reddening symptoms associated with cherry little cherry disease on a leaf of sweet cherry 'Sam'. (Dr. W. Jelkmann, Institut fur Pflanzenschutz im Obstbau der Biologischen Bundesanstalt, Dossenheim)

7. Cherry mottle leaf virus

Striated flexuous closterolike virus; closely related to peach mosaic virus. Monoclonal antibody specific to cherry mottle leaf virus reacts with peach mosaic virus in ELISA assay and Western blot analysis (James and Mukerji 1993).

Significance
In some regions cherry mottle leaf is one of the most severe diseases of sweet cherry. Of much less importance in sour cherry.

Symptoms
Irregular chlorotic mottle and distortion of terminal leaves. Diseased trees exhibit reduced terminal growth and shortened internodes (Fig. 11). Leaf symptoms may be masked by high temperature. The fruits of severely affected trees are small and tasteless with delayed ripening.

Hosts
- natural: sweet cherry and peach, which is symptomless after infection with most strains. Other symptomless hosts include sour cherry and hybrids, as well as *P. serrulata* and *P. yedoensis.*
- experimental: by grafting to several *Prunus* spp. and by mechanical inoculation to *Chenopodium quinoa.*

Geographical distribution
Belgium, Canada, Czech Republic, Italy, Poland, Romania, South Africa, and USA (Németh 1986)

Biology and transmission
Transmitted by grafting, mechanical inoculation and by the leaf mite *Eriophyes inaequalis.*

Detection
Graft or bud inoculation on seedlings of *P. avium* 'Bing' results in irregular chlorotic mottle and distortion of leaves. *Chenopodium quinoa* is a diagnostic herbaceous host, showing stunting and chlorotic spots; *C. amaranticolor* is a local lesion host of the virus. Suitable detection methods are also ELISA and Western blotting using monoclonal antiserum to the virus.

Fig. 11. Irregular chlorotic mottle, distortion of terminal leaves and shortened internodes caused by cherry mottle leaf virus. (Dr A. Ragozzino, Istituto di Patologia Vegetale, Napoli)

For further reading, see p. 92.

8. Cherry rasp leaf virus (CRLV)

Cherry rasp leaf virus is a nepovirus with isometric particles of 30 nm in diameter. The two RNA species are single-stranded.

Significance
Yield losses are caused by dieback of branches or entire trees. In older cherry-growing areas, rasp leaf infection can be as high as 38% (Luepschen *et al.* 1974).

Symptoms
The most characteristic symptoms are the prominent enations on the underside of cherry leaves (Fig. 12). The leaves are more or less deformed, many of them are narrow, puckered or distorted. Newly infected trees usually show symptoms on the lower leaves. The virus spreads slowly in the tree. The affected trees become highly frost sensitive. Many spurs and branches die in the lower part of infected trees, rendering the trees open and bare. In peach it causes small enations, stunted growth and shortened internodes and a general decline. Small enations on the underside of cherry leaves, however, may be induced by other viruses, such as Prunus necrotic ringspot virus, raspberry ringspot virus and Arabis mosaic virus in association with prune dwarf virus.

Hosts
The virus affects sweet cherry and *P. mahaleb* rootstock, peach and apple (flat apple disease).

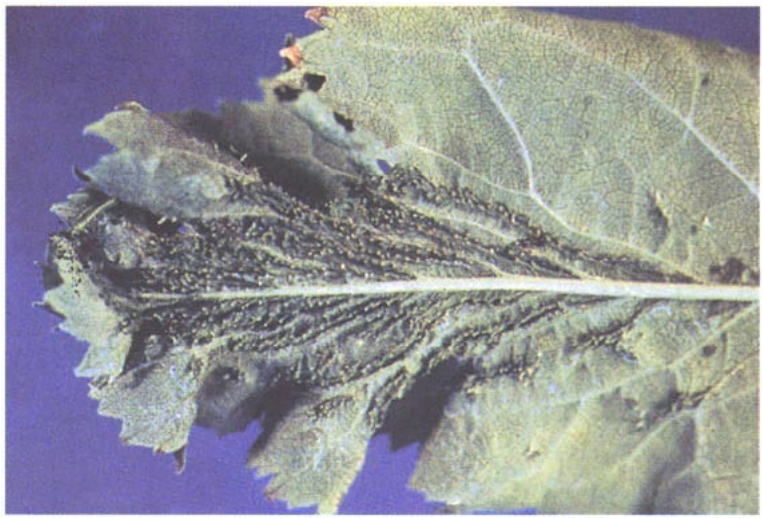

Fig. 12. Enations on the underside of a cherry leaf caused by cherry rasp leaf virus. (Dr A. Hadidi, USDA-ARS, Beltsville)

Geographical distribution
Canada, USA west of Rocky Mountains (Németh 1986).

Biology and transmission
Transmitted by grafting and sap inoculation. The virus does not induce symptoms in naturally infected weedy species of orchards *(Balsamorhiza sagittata, Taraxacum officinale, Plantago major).* Vector is the nematode *Xiphinema americanum.* Secondary spread is generally slow, due to the slow movement of the nematode vector. Sweet cherry or apple trees, planted on the site of earlier infected trees, often become infected.

Therapy
Thermotherapy.

Indexing
Indicator plants *(Prunus avium* cv. Bing, *Cucumis sativus, Cyamopsis tetragonoloba, Chenopodium quinoa, C. amaranticolor)* and ELISA.

For further reading, see p. 93.

9. Cherry twisted leaf

Experiments carried out in Canada about two decades ago showed a relationship between cherry twisted leaf and apricot ring pox diseases. Zhang *et al.* (1992) reported that the causal agents of cherry twisted leaf and apricot ring pox are serologically related to apple stem pitting virus.

Symptoms
The leaves are small and twisted with a tendency to bilateral asymmetry (Fig. 13). The distortion is associated with necrosis of midrib or petiole. In some cases the distal portion of the leaf is abruptly bent downwards (Fig. 13, bottom). Fruit distortion is also present and accompanied by pedicel necrosis.

Hosts
Sweet cherry and chokecherry *(P. virginiana).* Susceptibility varies greatly according to the cultivar; 'Bing' is severely affected.

Geographical distribution
Canada and USA.

Biology and transmission
The disease spreads by grafting. Natural spread was observed in British Columbia in sweet cherry orchards and from chokecherry to sweet cherry.

Detection
Grafting on cv. Bing and molecular assays using RT-PCR (Hadidi *et al.*, unpublished).

For further reading, see p. 93.

Fig. 13. Twisted and distorted cherry leaves caused by cherry twisted leaf virus. Note that the leaf at the bottom is partly bent downwards. (Dr A. Hadidi, USDA-ARS, Beltsville)

10. Hop stunt viroid (HSVd)

Closely related strains of hop stunt viroid (HSVd) cause dapple fruit of plum and peach. HSVd-peach or HSVd-plum is an infectious circular low molecular weight RNA consisting of 297 nucleotides which can form rod-like structure with extensive base pairing.

Significance
Certain HSVd strains cause serious diseases in plum and possibly peach, while others seem to infect grapevine and citrus latently. These plants are believed to play an important role in HSVd epidemiology as a potential source of inoculum.

Symptoms
Plums affected with dapple fruit disease show red blotches or yellowish red colouring. Affected peaches show yellow blotches (Fig. 14).

Hosts
Plum and peach as well as hop, citrus, grapevine and cucumber. HSVd-plum infects, with symptoms, plants in Cucurbitaceae and Compositae such as *Benincasa hispida, Cucumis sativus, C. melo, C. sativus, Lagenaria siceraria, Luffa cylindrica* and *Momordica charantia.* Latent infection occurs on *Citrullus vulgaris, Cucurbita moschata* and *C. maxima.*

Fig. 14. Yellow blotches on a peach affected by hop stunt viroid. (Dr A. Hadidi, USDA-ARS, Beltsville)

Geographical distribution
Dapple fruit disease of plum and peach has been reported only from Japan. However, various isolates of HSVd have been detected from various species of plants in many countries.

Biology and transmission
HSVd-plum or HSVd-peach is transmitted by grafting to stone fruits and by mechanical inoculation to Cucurbitaceae and Compositae.

Detection
Mechanical inoculation to cucumber 'Suyo'. Symptoms appear 17 to 25 days after inoculation and consist of streaking, leaf curling, and vein clearing. Two-dimensional or return gel electrophoresis can be used to separate circular viroid low molecular weight RNA from host nucleic acids. Suitable methods are also dot blot or Northern blot hybridization using labeled HSVd cRNA probe and reverse transcription-polymerase chain reaction using primers for HSVd followed by gel electrophoresis or hybridization analysis.

For further reading, see p. 94.

11. Peach latent mosaic viroid (PLMVd)

A viroid (PLMVd) of 336-338 nucleotides. By molecular hybridization experiments Shamloul *et al.* (1995) found that PLMVd is not related to the agent of peach mosaic disease.

Significance
High incidence worldwide. Reduces economic life of the tree rapidly after the fifth year and affects fruit quality in some cultivars.

Symptoms
Typical mosaic (Fig. 15) is rarely observed on leaves. The opening of buds, blooming and fruit ripeness are delayed 4 to 6 days. Irregular fruits with bumps, chlorotic spots (Fig. 16), cracked suture and stones characteristically deformed and swollen. Open growth habit, bud necrosis and rapid ageing of the tree may also occur. Stem pitting is observed in some cases. The same viroid is reported to cause peach yellow mosaic in Japan (Kishi *et al.* 1973).

Hosts
Peach and peach hybrids (Desvignes 1982 and 1986).

Geographical distribution
Algeria, China, France, Greece, Italy, Japan, Morocco, Spain (Smith *et al.* 1992), Austria, Brazil, Nepal, Pakistan, Romania, South Africa, USA, former Yugoslavia (Shamloul *et al.* 1995).

Fig. 15. Mosaic on peach leaves caused by peach latent mosaic viroid. (Dr L. Guinchedi, Istituto di Patologia Vegetale, Bologna)

Biology and transmission
Transmitted by grafting and mechanically with purified viroid (Desvignes 1980; Flores *et al.* 1990), or by contaminated tools (Shamloul *et al.* 1995). Disease spread is observed in the field. PLMVd can be transmitted experimentally by aphids. Not transmitted experimentally by pollen, seed or mites (Desvignes 1980, 1986; Flores *et al.* 1992).

Detection
Latent strains may be detected in the glasshouse as follows: 'GF305' peach seedlings are inoculated by chip-budding and 2 months later re-inoculated by budding with a severe strain able to produce the foliar mosaic. Absence of the characteristic symptoms of the severe strain on the indicator plant is demonstrative of the presence of a latent strain (Desvignes 1980). PLMVd can also be detected by polyacrylamide gel electrophoresis (Flores *et al.* 1990), by PCR (Shamloul *et al.* 1995) or by molecular hybridization (Ambrós *et al.* 1995).

Therapy
In vitro micrografting (shoot-tip grafting) worked with some cultivars, but not all (Barba *et al.* 1995).

For further reading, see p. 94.

Fig. 16. Chlorotic spots on peach 'Redhaven' caused by peach latent mosaic viroid. (Dr L. Guinchedi, Istituto di Patologia Vegetale, Bologna)

12. Plum pox virus

A single-stranded RNA potyvirus with elongated particles of approximately 764 x 20 nm. Two main groups of strains (D and M) can be discriminated. These two groups have quite different epidemiological properties.

Significance

In Europe, plum pox (sharka) is considered to be the most damaging virus disease of *Prunus* spp., particularly in plum, apricot and peach and their rootstocks.

Symptoms

Symptoms may appear on leaves, flowers or fruits. Severity varies according to species and cultivar, virus strain, season and locality. Symptoms are conspicuous on leaves in spring: chlorotic, sometimes necrotic spots; bands or rings (Fig. 17). Fruits show chlorotic spots or rings (Fig. 18), are deformed and often unmarketable. Stones of apricots and of some plum cultivars show pale rings or spots (Fig. 19).

Hosts

- natural: apricot, plum and peach as well as most *Prunus* rootstocks; almond can be infected without showing symptoms (Németh 1986). The virus also infects most wild or ornamental *Prunus* spp. (Smith *et al.* 1992). The occurrence of PPV in sour cherry has been described by Kalashyan *et al.* (1994), in sweet cherry by Crescenzi *et al.* (1995).
- experimental: some cultivated or weedy annual plants can be infected (EPPO 1974).

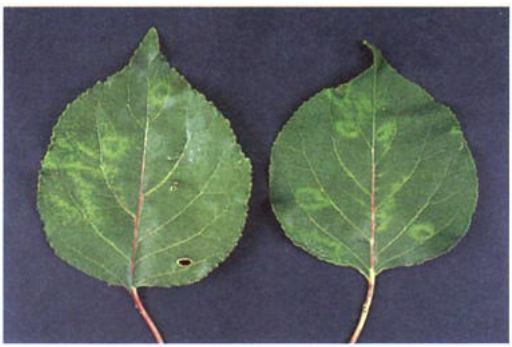

Fig. 17. Apricot leaves showing chlorotic rings caused by plum pox virus. (Dr L. Guinchedi, Istituto di Patologia Vegetale, Bologna)

Fig. 18. Plums showing chlorotic rings and deformation caused by plum pox virus. (Dr L. Guinchedi, Istituto di Patologia Vegetale, Bologna)

Geographical distribution
Europe, Egypt, Syria, Turkey. The first occurrence of PPV in the American continent was reported in Chile (Roy and Smith 1994).

Biology and transmission
PPV is transmitted from infected to uninfected trees either by grafting or by aphids. Several aphid species transmit the virus in a non-persistent manner (Labonne *et al.* 1995). The virus is also sap-transmissible.

Pollen and seed transmission of PPV has been described (Németh 1986). More recently, Eynard *et al.* (1991) and Triolo *et al.* (1993) have found PPV antigens in immature seeds of apricot. However, seedling infection was not reported.

Detection
Grafting on 'GF305' peach seedlings or *Prunus tomentosa*. The ELISA test (using polyclonal or, more recently, specific monoclonal antibodies) and PCR are now widely used. Immuno tissue printing was recently reported (Knapp *et al.* 1995, Fig. 20). Owing to the erratic distribution of the virus in infected trees, several samples from one tree have to be tested.

Treatment
Conventional or *in vitro* micrografting or thermotherapy followed by meristem-tip culture.

For further reading, see p. 95.

Fig. 19. Deformed apricot fruits infected with plum pox virus; the stones are showing pale rings. (Dr G. Llácer, Instituto Valenciano de Investigaciones Agrarias, Moncada)

Fig. 20. Immuno tissue printing in a longitudinal section of an apricot segment with a lateral bud. The purple precipitations correspond to PPV particles. Note that the bud on the heavily infected stem is almost free from virus particles, demonstrating the erratic distribution of the virus in infected trees. (Dr M. Laimer da Camara Machado, Institute of Applied Microbiology, University of Agriculture, Vienna)

13. Prune dwarf virus (PDV)

Ilarvirus, divided genome and five types of particles varying from quasi-isometric (about 22 nm diameter) to bacilliform shape, with different sedimentation coefficients. Strains have been identified based on reaction on experimental hosts.

Significance
High incidence and severity in all areas where stone fruits are grown. It is prevalent in sour cherry and sweet cherry.

Symptoms
The symptoms vary greatly according to species, virus strain and temperature. The diseases caused by PDV have been described under a variety of names:

- on prune: 'prune dwarf', 'prune mosaic', 'willows' and 'shoestring'
- on peach: 'Muir peach dwarf' and 'peach stunt'; some cultivars are insensitive
- on apricot: 'gummosis', 'rosetting of foliage'
- on sour cherry: 'sour cherry yellows', 'yellow leaf', 'chlorotic ringspot' and 'boarder tree'. In association with raspberry ringspot virus, severe rasp leaf symptoms appear, and in association with Prunus necrotic ringspot virus severe stunting and yellows are observed.
- on sweet cherry, 'chlorotic spots and rings' of the foliage; sometimes, mottle and varying degrees of necrosis and shot-holing (Figs. 21, 22) may occur, symptoms identical with those described as 'tatterleaf' and caused by Prunus necrotic ringspot virus. Generally, cultivars of *Prunus salicina* and of plum hybrids as well as the cherry rootstocks 'Mazzard' and 'Mahaleb' and some sweet cherry, apricot and almond cultivars are symptomless. Some virus strains, however, may induce formation of a large amount of gum on apricot stems and branches.

Fig. 21. Chlorotic spots and rings on cherry leaves, caused by prune dwarf virus. (Dr L. Guinchedi, Istituto di Patologia Vegetale, Bologna)

Hosts
- natural: almond, apricot, sweet cherry and sour cherry, peach, plum *sensu lato.*
- experimental: more than 100 *Prunus* spp. and several herbaceous species.

Geographical distribution
Cosmopolitan.

Biology and transmission
PDV is transmitted from infected to uninfected trees either by grafting or by seed or pollen. Planting of infected symptomless rootstocks accounts for the prevalence of this virus in sweet and sour cherry.

Detection
By experimental transmission to 'GF305' peach seedling, *P. tomentosa* and *Cucumis sativus*, and/or by serological tests or molecular techniques (Parakh *et al.* 1995).

Treatment
Thermotherapy at 36-37°C for 15 days or longer.

For further reading, see p. 96.

Fig. 22. Chlorotic spots and rings, as well as necrosis and shot-holing on cherry leaves, caused by prune dwarf virus. (Dr L. Guinchedi, Istituto di Patologia Vegetale, Bologna)

14. prunus nectoic ringspot virus (PNRSV)

Ilarvirus, member of subgroup III, isometric to bacilliform particles about 23 nm in diameter with four molecules of single-stranded RNA. A number of strains cause different symptoms in many hosts (Fulton 1983).

Significance
Considerable economic significance depending on virus strain and fruit species and cultivar.

Symptoms
The virus induces pronounced symptoms in the first to second year after infection, during 'shock' or 'acute' stage of the disease. Subsequently infection becomes symptomless, although some strains are recurrent (Nyland *et al.* 1976; Wells and Kirkpatrick 1986). In the acute stage, symptoms appear in spring as chlorotic or necrotic leaf spots (Fig. 23), rings or irregular lines (Fig. 24); chlorotic and then necrotic areas lead to the shot-hole effect (Fig. 25). In certain hosts there may be delayed bud break, death of leaf and flower buds and terminal dieback. The almond calico strain in some *Prunus* cultivars produces white or bright yellow spots, blotches, lines or oak leaf pattern. In certain almond cultivars, flowers and leaf buds do not grow. After initial symptoms in some peach cultivars, severe infection induces bark necrosis, cankering and splitting of the trunk. In sweet and sour cherry the recurrent strain causes necrotic leaf spots; cherry rugose mosaic strain induces chlorotic blotches with distortion and enations on abaxial leaf surfaces. In some plum rootstock/scion combinations virus induces tree decline. Sometimes the virus occurs with other stone fruit viruses; with prune dwarf virus it induces peach stunt, while in sour cherry it increases the severity of yellow symptoms.

Hosts
- natural: *Prunus* spp. and many *Rosa* spp.
- experimental: moderately wide herbaceous host range.

Geographical distribution
Cosmopolitan.

Fig. 23. Chlorotic and necrotic leaf spots on peach 'Stark Red Gold', caused by Prunus necrotic ringspot virus. (Dr L. Guinchedi, Istituto di Patologia Vegetale, Bologna)

Biology and transmission
Transmitted by grafting and mechanical inoculation. Particles are unstable in crude plant sap. Virus is seedborne and pollenborne in many *Prunus* spp. (Nyland *et al.* 1976). Natural spread is usually slow but it can reach up to 10% per year in peach and cherry (Wells and Kirkpatrick 1986; Howell and Mink 1988). The virus is carried both inside the pollen and on the surface. On the surface it is involved in plant-to-plant transmission while inside it leads to seed infection. In species where pollen is not windborne, such as peach, honeybees may spread infected pollen (Kelley and Cameron 1986).

Detection
Grafting to 'GF305' peach seedling, *P. tomentosa*, *P. serrulata* 'Shirofugen' or *P. avium* 'F12/1'. Mechanical transmission to *Cucumis sativus*. Serology (ELISA of tissues collected early in the vegetation period, Torrance and Dolby 1984, or immuno tissue printing, Knapp *et al.* 1995) and nucleic acid probes (Scott *et al.* 1992).

Treatment
Conventional and *in vitro* thermotherapy; shoot-tip micrografting *in vitro* (Juarez *et al.* 1988).

For further reading, see p. 96.

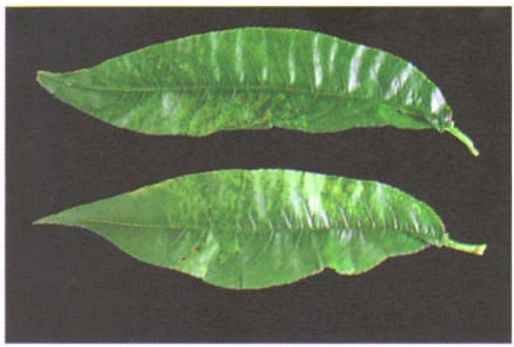

Fig. 24. Irregular lines on leaves of peach 'Stark Red Gold', caused by Prunus necrotic ringspot virus. (Dr L. Guinchedi, Istituto di Patologia Vegetale, Bologna)

Fig. 25. Necrotic spots and 'shot-holes' on sour cherry leaves, caused by Prunus necrotic ringspot virus. (Dr L. Guinchedi, Istituto di Patologia Vegetale, Bologna)

15. Raspberry ringspot virus (RRSV)

Nepovirus with isometric particles of 28 nm in diameter with two single-stranded RNA species. Serologically two strains can be differentiated: Scottish strain RRSV-S and English strain RRSV-E.

Significance
Yield losses are caused by dieback of branches or entire trees.

Symptoms
Leaf symptoms of the raspberry ringspot disease (synonyms: Pfeffinger disease or European cherry rasp leaf) include characteristic large, yellowish-green flecks or 'oil flecks' which are easily seen in transmitted light. Leaves may be smaller than normal, slightly wavy, distorted with deep sinuses (Fig. 26). Often half of the leaf blade is smaller and the leaf becomes characteristically bent and asymmetric.

Secondary symptoms are enations on rasp leaves. The leaves are narrow or deformed, with deep separation, stiff and brittle. Owing to short internodes, the buds form rosettes on which several buds may be found.

Fig. 26. Primary symptoms of Pfeffinger disease (raspberry ringspot virus): large, yellowish green flecks or 'oil flecks', with leaves slightly wavy, distorted with deep sinuses. (Dr T. Hasler, Swiss Fed. Res. Station for Fruit Growing, Wädenswil)

Hosts
- natural: *Prunus avium, P. domestica, Ribes grossularia, R. nigrum, R. rubrum, Rubus idaeus* and *Fragaria* spp.
- experimental: the virus is sap transmittable to herbaceous plants (Murant 1978).

Geographical distribution
Austria, Belgium, Canada, former Czechoslovakia, Denmark, France, Germany, Great Britain, The Netherlands, Norway, Romania, Switzerland, USSR (Németh 1986).

Biology and transmission
Transmitted by grafting, sap inoculation and seeds. Consistent with other nepoviruses, RRSV has been detected in symptomless weed flora. Vectors are the nematodes *Longidorus elongatus* for the Scottish strain and *Longidorus macrosoma* for the English strain (Harrison 1964; Taylor and Murant 1969). Secondary spread is generally slow, due to the slow movement of the nematode vector. Sweet cherry planted on the site of earlier infected trees often becomes infected.

Detection
Prunus avium 'Bing', *P. persica* 'GF305', *Cucumis sativus, Chenopodium quinoa, C. amaranticolor* and ELISA.

Therapy
Thermotherapy.

For further reading, see p. 97.

16. Strawberry latent ringspot virus (SLRSV)

A member of the nepovirus group with polyhedral particles, about 30 nm in diameter, with bipartite single-stranded RNA genome (Francki *et al.* 1985). There are a number of isolates, many serologically similar to the type strain (Murrant 1981).

Significance
In stone fruits, of some economic importance only in peach in certain areas of northern Italy. In other European countries it may be significant in mixed infection with other viruses.

Symptoms
A virulent strain of the virus in Italy induces a disease described as 'willow leaf rosette' (Corte 1968; Belli *et al.* 1986). Symptoms include delayed leafing and flowering, small and narrow leaves often folded upward, malformed and with slight chlorotic spots, stunted shoot internodes with leaves appressed into distinct rosettes (Figs. 27,28). Symptoms may begin on one or a few branches and gradually extend to the whole tree and disappear during the summer. Infected trees are unproductive.

In mixed infection with prune dwarf virus (PDV) growth reduction, rosetting and dieback of peach trees are reported (Scotto La Massese *et al.* 1973).

In apricot, in mixed infection with cucumber green ring mottle virus, bare branches with no short side branches or fruit spurs have been observed. Branches turn upwards, reaching an almost vertical positioned the leaves roll upward along the main veins during summer (Blattny and Janeckova 1980).

In cherry, infection seems to be latent, but in mixed infection with PDV, enations on the underside of the leaves (Fig. 29) and tree decline have been observed (Ragozzino and Alioto 1992).

Fig. 27. Symptoms of small and narrow leaves folded upwards, caused by strawberry latent ringspot virus on peach 'Michelini'. (Dr L. Guinchedi, Istituto di Patologia Vegetale, Bologna)

Hosts

- natural: wide host range including many cultivated and wild perennial plants. Infected plants are often symptomless. In stone fruits, recorded in peach, apricot, sweet cherrv and plum (Németh 1986).
- experimental: wide experimental host range (Murant 1981).

Geographical distribution

Largely confined to Europe (Murant 1981). In addition reported in New Zealand (Fry and Wood 1973), Canada (Allen *et al.* 1970) and USA (Hanson and Campbell 1979). However, the occurrence in North America was on imported material.

Biology and transmission

Transmitted by grafting, mechanical inoculation and seed of some cultivated and weedy host plants. Also transmitted by the nematode *Xiphinema diversicaudatum.* Occurs frequently together with arabis mosaic virus (Murant 1981).

Detection

Graft transmission to *Prunus persicae* 'GF305' and mechanical inoculation. Useful diagnostic herbaceous hosts are *Chenopodium quinoa*, *C. amaranticolor* and *Cucumis sativus.* Serology (ELISA).

For further reading, see p. 97.

Fig. 28. Leaves appressed into distinct rosettes on stunted shoot internodes caused by strawberry latent ringspot virus on peach 'Stark Red Gold'. (Dr L. Guinchedi, Istituto di Patologia Vegetale, Bologna)

Fig. 29. Enations on the underside of sweet cherry leaves caused by mixed infection with strawberry latent ringspot virus and prune dwarf virus. (Dr A. Ragozzino, Istituto di Patologia Vegetale, Napoli)

17. Tobacco ringspot virus (TRSV)

Nepovirus with three types of isometric particles, 28 nm in diameter and a bipartite RNA genome (Stace-Smith 1985).

Significance
No economic significance in stone fruits.

Symptoms
In cherry, delay in bud opening and flowering; irregular light-green leaf blotching.

Hosts
- natural: wide range of annual and perennial crops. In stone fruits only recorded in sweet and ornamental cherries (Uyemoto *et al.* 1977).
- experimental: wide host range.

Geographical distribution
In *Prunus* spp. reported from North America. Virus isolated from various other plant species in different parts of the world.

Biology and transmission
By grafting and mechanical inoculation. Seedborne in some herbaceous hosts. Transmitted to several hosts by *Xiphinema americanum sensu lato* (Stace-Smith 1985); nematode transmission to cherry trees has not been reported.

Detection
Grafting to *P. avium* cv. Bing, mechanical transmission to *Chenopodium quinoa*, and serology (ELISA).

For further reading, see p. 98.

18. Tomato ringspot virus (ToRSV)

Nepovirus, isometric particles, 28 nm in diameter with bipartite genome (Stace-Smith 1984).

Significance

Causes economically important diseases in stone fruits in the eastern and western USA (Stace-Smith and Ramsdell 1987).

Symptoms

Stem pitting (Fig. 30) and decline in peach, cherry, apricot, plum and other *Prunus* spp. (Mircetich and Fogle 1976). Other symptoms include reduced terminal growth, chlorotic leaves curling upwards and turning red in autumn, premature defoliation, enlargement of lower trunk with very thick spongy bark and necrotic areas, longitudinal pitting and grooving on rootstock or scion woody cylinder, or both. Trees die within 3 to 5 years. Yellow bud mosaic strain induces serious disease in peach, nectarine, sweet cherry, almond and other *Prunus* spp. (Schlocker and Taylor 1976). Symptoms include blotches or spots in leaves of newly infected shoots; the following year bud growth severely retarded and leaves small and yellow. Prune brown-line disease in plum trees is characterized by yellow bud mosaic.

Fig. 30. Stem pitting in peach caused by tomato ringspot virus. (Dr L. Guinchedi, Istituto di Patologia Vegetale, Bologna)

Hosts

- natural: wide range of herbaceous and woody plants, including ornamentals, apples, small fruits and legumes.
- experimental: easily transmitted by sap to wide range of herbaceous hosts.

Geographical distribution

Naturally infected *Prunus* are widely distributed throughout the northeastern and western USA, also found in Canada (Stace-Smith 1984) and Chile (Auger 1988). The virus has been isolated from ornamentals and berries in Japan and Europe but apparently not associated with the nematode vectors (Stace-Smith 1984).

Biology and transmission

By infected propagation materials and mechanical inoculation. Seedborne in a range of herbaceous hosts. Transmitted by the dagger nematodes *Xiphinema americanum sensu stricto, X. rivesi* and *X. californicum* (Stace-Smith 1984). The latter is an efficient vector of strains associated with yellow bud mosaic, stem pitting and prune brown-line (Hoy *et al.* 1984).

Detection

Grafting to *P. tomentosa* and *P. persicae* 'GF305', mechanical transmission to *Chenopodium quinoa,* serology (ELISA), and molecular hybridization (Hadidi and Hammond 1988; Hadidi and Powell 1991). The virus is irregularly distributed in some hosts; often tissue samples from the lower trunk are the most reliable (Bitterlin and Gonsalves 1986; Powell *et al.* 1991).

For further reading, see p. 98.

19. Viral twig necrosis of cherry (PeAMV, CIRSV)

Previously, tomato bushy stunt virus (TBSV) or a strain of this virus was regarded as the causal agent of viral twig necrosis. More detailed serological investigations identified the cherry isolates as petunia asteroid mosaic virus (PeAMV), which is a member of the tombusvirus group (Hollings and Stone 1975; Koenig and Kunze 1982; Martelli *et al.* 1989; Gruntzig *et al.* 1989). Recently another tombusvirus, carnation Italian ringspot virus (CIRSV), has been identified in more than 100 sweet cherry trees of northern Bavaria (Lesemann *et al.* 1989; Pfeilstetter *et al.* 1992). CIRSV is clearly distinguishable from PeAMV by means of serology.

This virus disease is not to be confused with 'cherry detrimental canker' as described by Blattny (1962) in Czechoslovakia. Heavily damaged trees show canker-like deformations on the shoots. *Pseudomonas syringae* has been identified as the causal agent of these canker-like alterations (Novák and Lanzová 1980).

Significance
Generally only sporadic occurrence in old trees. In northern Bavaria, however, widely distributed. CIRSV has been detected in six orchards of northern Bavaria in younger trees only. No recent report from Canada.

Symptoms
Shoot symptoms are most conspicuous with bark necrosis at the tips and stunting. Necrosis of the midrib and of some lateral veins causes a twisting of the leaves. Fruits are misshapen with sunken, target-like necrotic spots. Flower symptoms usually include weak necrotic streaks along peduncles (Pfeilstetter 1992). CIRSV causes the same symptoms in cherry trees, however, damage to cherry trees appears to be less than for infections with PeAMV.

Hosts
- natural: PeAMV: Distinct symptoms of viral twig necrosis are reported only in sweet cherry; symptomless infection occurs also in sour cherry, plum, pear and (Kleinhempel *et al.* 1971; Richter *et al.* 1977). Other woody plants and a great number of herbaceous plants belonging to several families may also be infected (Pfeilstetter 1992. CIRSV: *Prunus avium* was the first host in a natural habitat (Lesemann *et al.* 1989). Herbaceous wild plants were also found to be naturally infected (Pfeilstetter 1992).
- experimental: for both viruses a large number of host plants has been reported (Hollings *et al.* 1970; Schmelzer *et al.* 1977; Buttner and Nienhaus 1989).

Geographical distribution
PeAMV: mainly Europe: former Czechoslovakia (Blattny 1962), Switzerland, Germany (Koenig and Kunze 1982; Grüntzig *et al.* 1989), Canada. Only in northern Bavaria has CIRSV been shown to be additionally associated with viral twig necrosis.

Biology and transmission
Transmission by soil (without involvement of a vector) and grafting. Although the virus is seedborne, seed transmission could not be detected (Pfeilstetter 1992; Pfeilstetter *et al.* 1996). Pollen transmission not reported. The fact that CIRSV was also found in propagation stock and only younger trees were infected in northern Bavaria suggests that transmission by grafting may be the main way of virus spread (Pfeilstetter 1992).

Detection
PeAMV is extremely unevenly distributed within diseased trees. The virus seems to be mainly restricted to the symptom-bearing tissue in the different plant parts tested (leaves, fruits, young twig-tips, bark). Trees showing few or no symptoms of the disease reacted rarely positive in the ELISA test. Therefore, reliable indexing for latent infections with PeAMV by means of serology is not possible at the moment. Due to the uneven virus distribution indexing with woody indicators is also unreliable. The same is true for CIRSV.

For further reading, see p. 99.

20. Other European nepoviruses (tomato black ring, arabis mosaic, myrobalan latent ringspot)

Besides raspberry ringspot virus, strawberry latent ringspot virus and cherry leaf roll virus other nepoviruses-namely arabis mosaic virus (ArMV), tomato black ring virus (TBRV) and myrobalan latent ringspot virus (MLRSV)-have been reported in stone fruit species in Europe. These viruses have three types of polyhedral particles, approximately 28 mm in diameter, with a divided genome of two species of single-stranded RNA.

Significance
ArMV is of some economic importance in stone fruits only in restricted European areas where it occurs in mixed infection with other viruses. TBRV and MLRSV are of no economic importance.

Host range
ArMV and TBRV have wide natural and experimental host ranges. In stone fruit species, ArMV has been recorded in sweet cherry and in peach trees. TBRV has been sporadically recorded in peach, almond and sweet cherry trees (Németh 1986). MLRSV has only been reported in *Prunus cerasifera* myrobalan B and in *Prunus* cultivars on this rootstock (Dunez and DuPont 1976).

Symptoms
In sweet cherries ArMV has only a slight effect, causing mottled young leaves and small enations as leaves mature, but in mixed infection with prune dwarf virus or Prunus necrotic ringspot virus it induces rasp leaf. Infected trees are stunted, with narrow leaves, often with large enations on their underside. Almond and peach trees infected by TBRV have very short shoot internodes; in recently infected peach, leaves have irregular chlorotic spots and distorted laminae; almond leaves are narrow with wavy edges and enations on the lower surface (Németh 1986).

There are no data on symptoms induced by ArMV in peach and by TBRV in sweet cherry. In peach, ArMV in association with Prunus necrotic ringspot virus induces growth reduction.

MLRSV is latent in myrobalan while it causes short internodes and rosetting in peach and enations on thelower surface of sweet cherry ‘Bing’.

Transmission
By grafting and mechanical inoculation. ArMV and TBRV are transmitted to a large proportion of seeds of many host plants including many crops and weeds (Murant 1981). Both viruses are transmitted to several hosts by nematodes; ArMV by *Xiphinema diversicaudatum*, TBRV by *Longidorus attenuatus* and *L. elungatus.* The nematode vectors of MLRSV have not been established.

Geographical distribution
ArMV and TBRV occur mainly in Europe. Both viruses have also been reported in other parts of the world on imported materials (Murant 1987a, 1987b). MLRSV has only been reported in southwestern France (Dunez and DuPont 1976).

Indexing
Transmission by grafting to *Prunus persicae* 'GF305' and mechanical inoculation. Diagnostic herbaceous hosts are *Chenopodium quinoa* and *C. amaranticolor*. Serology (ELISA). ArMV and TBRV can also be detected by molecular hybridization (Hadidi and Hammond 1988; Bretout *et al.* 1988).

For further reading, see p. 100.

Diseases of unknown etiology

1. Cherry necrotic rusty mottle

Cause
Unknown etiology, suspected to be a virus.

Significance
Cherry necrotic rusty mottle is a serious disease of cherry, leading to significant yield losses and to an early death of trees.

Symptoms
Symptoms vary considerably according to cultivar, virus strain and temperature. The first leaf symptoms become visible 3 to 6 weeks after full bloom. Angular necrotic spots are formed on the leaves (Fig. 31). Strongly necrotized leaves fall off. On leaves with scattered necrotic spots, shot-holes may develop, but the leaves remain on the tree. The autumnal colouring of the leaves occurs earlier than usual. Green rings and line pattern are produced on a yellow, brown or red background. As the disease develops, part of the buds may be killed, resulting in bare branches with terminal tufts of foliage. Bark symptoms consist of shallow, necrotic areas, discrete shallow gum blisters, or deep gum pockets (cherry bark blister). Affected trees are frost sensitive.

Hosts
Prunus avium, P. cerasus, P. persica, P. armeniaca, P. mahaleb rootstock; symptoms may not be evident in some.

Geographical distribution
Canada, Chile, France, Great Britain, New Zealand, USA (Németh 1986).

Biology and transmission
The agent spreads by grafting. Natural spread was observed by Cameron and Moore (1985). No vector has been reported.

Therapy
Thermotherapy.

Indexing
Prunus avium cv. Sam.

For further reading, see p. 100.

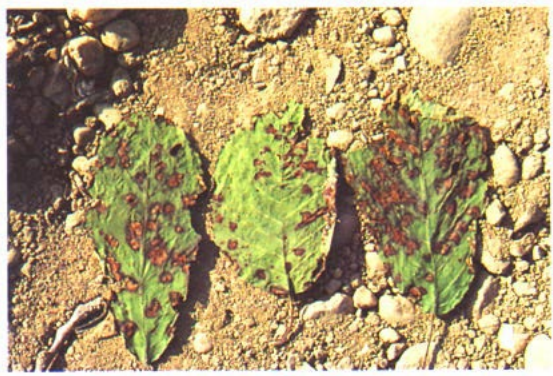

Fig. 31. Symptoms of cherry necrotic rusty mottle: angular necrotic spots. (Dr A. Ragozzino, Istituto di Patologia Vegetale, Napoli)

2. Cherry rusty mottle (American)

Cause
Unknown etiology, suspected to be a virus. Two agents of the disease are described: mild rusty mottle and severe rusty mottle.

Significance
The infected trees decline and the main branches and limbs die back. Trees affected by mild rusty mottle decline more slowly and the fruits are less reduced in size than those affected by severe rusty mottle.

Symptoms
The first leaf symptoms appear 4 to 5 weeks after full bloom. The light green or yellow mottling appears first on the small basal leaves. The chlorotic areas retain their light green colour, while the remaining part of the leaf develops bright yellow, brown or red late-season colouring. The leaves are soon abscised. Thereafter, chlorotic mottling appears again on most of the foliage (Fig. 32). In trees infected with severe rusty mottle, autumnal colours develop very early, so that 30 to 70% of the foliage drops before fruit ripening. Fruits are small, have an insipid flavour and ripen late.

Hosts
- natural: cherry.
- experimental: apricot, peach, *P. mahaleb*, *P. serrulata*, *P. virginiana* (Zeller and Milbrath 1947; Reeves 1951).

Geographical distribution
USA; severe rusty mottle is more widely distributed in the states of Washington, Idaho and Montana, while mild rusty mottle is limited to Oregon (Németh 1986).

Biology and transmission
The agent spreads by grafting. In orchards a slow natural spread, mostly to neighbouring trees, has been observed.

Therapy
Thermotherapy.

Indexing
Prunus avium cv. Bing.

For further reading, see p. 101.

Fig. 32. Symptoms of American cherry rusty mottle: chlorotic mottling.
(Dr A. Hadidi, USDA-ARS, Beltsville)

3. Cherry rusty mottle (European)

Cause
Unknown etiology, suspected to be a virus.

Significance
Average decrease in growth of Mazzard F12/1 cherry rootstock is 23% and in fruit yield of some sweet cherry cultivars is 25% (Posnette and Cropley 1956, 1960; Posnette *et al.* 1968).

Symptoms
First symptoms appear on mature leaves in July. Small groups of tertiary and quaternary veins are first clear, then become yellow (Fig. 33). Affected leaves gradually develop a pale green colour, in contrast to the bright green colour of healthy leaves. By the end of August rusty red mottling appears on the yellowish-green leaf surface, mostly along the yellow veinlets (Fig. 34). Some sweet cherry cultivars are latent carriers (Posnette 1951; Németh 1986).

Hosts
Sweet cherry. Transmission experiments to herbaceous plants were unsuccessful.

Geographical distribution
Belgium, Germany, Great Britain, Hungary, Romania, South Africa, Switzerland (Németh 1986).

Transmission
The agent spreads by grafting.

Therapy
Thermotherapy.

Indexing
Prunus avium cv. Sam.

For further reading, see p. 101.

Fig. 33. Symptoms of European cherry rusty mottle in July: small groups of tertiary and quaternary veins turn yellow. (Dr T. Hasler, Swiss Fed. Res. Station for Fruit Growing, Wädenswil)

Fig. 34. Symptoms of European cherry rusty mottle in August: rusty red mottling. (Dr T. Hasler, Swiss Fed. Res. Station for Fruit Growing, Wädenswil)

Diseases caused by phytoplasms (formerly mycoplasma-like organisms, MLO)

1. Cherry lethal yellows

Cause
A phytoplasma that is closely related to the phytoplasma causing Jujube witches' broom (Lee *et al.* 1995). The two pathogens form a new subgroup within the elm yellows group (S. Zhu *et al.* unpublished).

Significance
Very important on Chinese cherry *(Prunus pseudocerasus)* in China (Zhu and Shu 1992). Young trees die within 1 to 3 years after showing symptoms; 20-year-old trees die within 3 to 5 years.

Symptoms
Infected trees develop diffused yellow discolouration of foliage in late spring, defoliate prematurely, produce small size fruit which do not mature or no fruit at all. Dieback symptoms (Fig. 35) are followed by death within 3 to 5 years. Witches' broom grows on the trunk of the dying tree in spring, serving as a source for further pathogen spread.

Hosts
Chinese cherry *(Prunus pseudocerasus* Lindl.), possibly sweet and sour cherry.

Geographical distribution
The cherry lethal yellows disease has been reported from China (Zhu and Shu 1992). It may also be present in other Asian countries.

Biology and transmission
Transmitted by grafting and probably by an insect vector. Pathogen spread in the field is reported to be rapid in China.

Detection
Nonspecifically by fluorescence microscopy and electron microscopy. Specifically by nested PCR utilizing DNA primers specific for phytoplasma cherry lethal yellows subgroup.

Fig. 35. Dieback symptoms in a cherry tree in China, caused by cherry lethal yellows phytoplasma. (Dr A. Hadidi, USDA-ARS, Beltsville)

For further reading, see p. 101.

2. European stone fruit yellows

Cause
A phytoplasma that is closely related to the phytoplasmas causing apple proliferation and pear decline (Lorenz *et al.* 1994).

Significance
Apricot chlorotic leaf roll and Japanese plum leptonecrosis severely affect the respective crops in most areas where they are grown in Europe. The same pathogen also induces diseases of peach, called peach yellows, European peach yellows, or peach decline, as well as diseases of nectarine and almond. The significance of these latter diseases is less well known.

Symptoms
Symptom intensity is greatly influenced by the susceptibility of the rootstock.

Apricot: interveinal foliar yellowing, leaves smaller than normal with stiff and brittle texture, conical rolling of the leaves along the longitudinal axis (Fig. 36). Trees usually show premature budbreak and extended growth in late summer and fall. Extended phloem necroses (Fig. 37) may cause quick death (apoplexy) or gradual decline over a period of 1 to 4 years.

Fig. 36. Rolling of apricot leaves along the longitudinal axis, associated with European stone fruit yellows. (Dr G. Llácer, Instituto Valenciano de Investigaciones Agrarias, Moncada)

Fig. 37. Phloem necrosis in apricot, associated with European stone fruit yellows. (Dr L. Guinchedi, Istituto di Patologia Vegetale, Bologna)

Japanese plum: leaves smaller than normal, cylindrically rolled, slightly chlorotic, later brownish-red and brittle (Fig. 38). Premature budbreak and off-season growth in fall and winter. Extended phloem necroses and decline of branches or entire trees. The root-stocks usually survive.

European plum: infection is usually latent; however, trees grafted on *Prunus marianna* may show similar symptoms to those reported for Japanese plum (Fig. 39).

Peach and nectarine: foliar yellowing or reddening (Fig. 40), rolling and curling of leaves (Fig. 41), premature leaf drop. Veinal swelling and development of corky tissue along the veins may occur. Reduced vigour and productivity and gradual decline within a few or several years.

Almond: slightlv chlorotic and severelv rolled leaves which drop prematurelv. Reduced vigour and gradual decline of the trees.

Fig. 38. Cylindrically rolled and brownish-red leaves of Japanese plum, associated with European stone fruit yellows. (Dr L. Guinchedi, Istituto di Patologia Vegetale, Bologna)

Fig. 39. Symptoms of European stone fruit yellows on *Prunus domestica:* smaller, slightly chlorotic and brownish-red leaves after a premature budbreak. (Dr L. Guinchedi, Istituto di Patologia Vegetale, Bologna)

Hosts
Almond, apricot, nectarine, peach, plum, Japanese plum *(Prunus salicina), P. serrulata.*

Geographical distribution
Mediterranean countries of Europe, Germany, Hungary, Romania, Switzerland (Sanchez-Capuchino *et al.* 1976; Morvan 1977; Rumbos and Bosabalidis 1985; Ahrens *et al.* 1993).

Biology and transmission
Transmitted by grafting and probably by leafhopper vectors.

Detection
Non-specifically by fluorescence microscopy and electron microscopy. Specifically by nucleic acid hybridization and PCR. For indexing, a susceptible Japanese plum cultivar such as 'Ozark Premier', 'Red Heart' or 'GF305' peach seedlings is recommended as indicator. Grafting should be performed in August or September.

For further reading, see p. 102.

Fig. 40. Reddening of a peach leaf cv. Super Crimson Gold, associated with European stone fruit yellows. (Dr L. Guinchedi, Istituto di Patologia Vegetale, Bologna)

Fig. 41. Rolling and curling of peach leaves, associated with European stone fruit yellows. (Dr L. Guinchedi, Istituto di Patologia Vegetale, Bologna)

3. Peach rosette

Cause
Phytoplasma occurring in different strains (Kirkpatrick *et al.* 1975).

Significance
Low incidence, with sporadic outbreaks in the past in several areas of the USA.

Symptoms
First symptoms appear in spring on part or all of the canopy. Initial symptoms are yellowing and inward folding of leaves. Infected dormant shoots show premature budbreak. The laterals are short (a few centimetres) and the few buds formed on these immediately produce leaves. Compact tufts of more than 200 small leaves (rosette) develop (Fig. 42). Blossoms rarely develop and the fruit, if formed at all, falls from the tree before reaching maturity. Diseased trees leaf out the second year following infection and die.

Hosts
- natural: peach, Japanese plum *(P. salicina)*, plum hybrids, chickasaw plum *(P. angustifolia)*, *P. injucunda*, *Acer rubrum.*
- experimental: wide range of *Prunus* spp. and hybrids, as well as herbaceous hosts.

Fig. 42. Compact tuft of peach leaves caused by peach rosette phytoplasma. (Dr A. Hadidi, USDA-ARS, Beltsville)

Geographical distribution
Peach rosette has been recognized mainly in the southern USA.

Biology and transmission
Although a natural spread of the disease has been observed, no vector could be identified. The causal agent survives in the field mostly on wild plums. The disease is graft-transmissible to several Prunoideae and through dodder *(Cuscuta campestris)* to periwinkle, tomato and *Nicotiana glutinosa.*

Detection
Non-specifically by fluorescence microscopy and electron microscopy. Specifically by nucleic acid hybridization and PCR. For indexing, 'GF305' peach seedling or peach cv. Elberta are recommended as indicator.

Treatment
The pathogen is heat-labile and was inactivated in budsticks by hot water treatment for 8 to 10 minutes at 50°C.

For further reading, see p. 102.

4. Peach X disease

Cause
Phytoplasma (Nasu *et al.* 1970; Granett and Gilmer 1971).

Significance
Very important in peach and sweet and sour cherry in eastern and western USA and Ontario, Canada. Young trees usually die within 1 to 3 years after the first symptoms appear. Chronically infected trees set few or no fruit.

Symptoms
Water-soaked spots which develop into red or yellow blotches on leaf blades, while the remainder turns chlorotic. Leaf margins roll upwards (Fig. 43). Affected leaves drop prematurely, leaving only small rosetted leaf tufts at the shoot tips. Fruits are misshapen and insipid or bitter and usually drop prematurely or fail to ripen.

Hosts
- natural: peach, sweet cherry and sour cherry are the main hosts; *P. virginiana* (chokecherry) acts as a natural reservoir for the pathogen.
- experimental: various herbaceous plants were infected through transmission with dodder or by leafhopper vectors.

Geographical distribution
Canada, USA.

Biology and transmission
Transmitted by grafting, and mainly by the leafhopper vectors *Scaphytopius acutus, Colladonus montanus, Paraphlepsius irroratus* and others.

Detection
Grafting on peach or chokecherry. Serology, fluorescence microscopy, molecular methods.

Treatment
The pathogen is heat-labile and was inactivated in budsticks by hot water treatment for 8 to 10 minutes at 50°C.

For further reading, see p. 102.

Fig. 43. Peach leaves showing rolling, red blotches, and tattering due to X disease. Defoliation starts at the base of the shoot. (Dr A. L. Jones, Michigan State University, East Lansing)

5. Peach yellows

Cause
Phytoplasma occurring as different strains causing respectively 'peach yellows', 'little peach' and 'red suture' (Jones *et al.* 1974a, 1974b).

Significance
It was a disease of high incidence during the 18th century and the beginning of 19th century. Due to eradication of diseased trees the presence of peach yellows in the principal peach-growing areas is low.

Symptoms
Peach yellows. It may be easily confused with nutritional disorders. Usually, the symptoms do not appear in the first year after infection. In early stages, the diseased tree may be slightly off-colour and on close examination bunches of slender, willowy shoots may be seen. Dormant buds often start growing immediately. Leaves developed on these shoots are narrow, yellowish and red-spotted; they roll inward and tend to droop downward (Fig. 44). The willow-like growth commonly occurs on the larger branches arising from adventitious buds, but they may develop in a terminal position anywhere on the tree. Terminal dieback of twigs and branches occurs in advanced stages of the disease; the tree dies within 2 or 4 years. Fruits are frequently larger than normal and insipid. The flesh shows reddish streaks and a decided deep-red colour around the pit.

Fig. 44. Willow-like growth with narrow, yellowish, rolled and drooping leaves caused by peach yellows phytoplasma. (Dr A. Hadidi, USDA-ARS, Beltsville)

Little peach. Fruits are reduced in size and ripen several days to 3 weeks later than normal. The willowy growth of the laterals is less conspicuous.

Red suture. Fruits ripen and soften on the suture prematurely. On red-fruited cultivars the suture is blotched with dark red or purple, while the other side is green and hard. Yellow-fruited cultivars show a deeper yellow on the suture side. Leaves have a yellowish-green to greenish-bronze appearance starting a few weeks after petal fall or just before harvest.

Hosts
Peach, almond, apricot and some ornamental *Prunus* spp. The plum cultivars 'Abundance', 'Chalco', 'Chabot' and *P. cerasifera* are symptomless carriers of the 'little peach' strain.

Geographical distribution
Peach yellows is confined to the eastern USA and Canada.

Biology and transmission
The disease is graft-transmissible to several *Prunus* spp. and is spread in the field by the plum leafhopper *(Macropsis trimaculata).*

Detection
The disease can be identified by grafting buds to woody indicators. The incubation period in the field is 1 to 3 years; in greenhouse about 60 days. Fluorescence microscopy (DAN) and molecular techniques are also suitable.

Treatment
The phytoplasma is inactivated by hot-water-treatment at 50°C for 10 minutes.

For further reading, see p. 103.

Bacterial diseases

1. Bacterial canker

Cause
Pseudomonas syringae pv. *morsprunorum* (Wormald) Young *et al.*, a gram-negative bacterium with fluorescent pigment.

Significance
As the bacterium often occurs with *P. syringae* pv. *syringae* van Hall, losses due to the individual bacteria are difficult to estimate. Collectively, these bacteria reduce yields by killing buds, fruiting spurs, small branches and trees.

Symptoms
Blossom and spur blight appear soon after the bloom period. Infected leaves develop slightly angular necrotic spots (Fig. 45), become tattered, or turn yellow and fall prematurely. Deep, black depressions with water-soaked margins develop in the flesh and on stems of young cherry fruit. Infected leaf and flower buds fail to open in spring. Depressed cankers with gummosis develop at the base of dead buds, blighted spurs, and along scaffold branches (Fig. 46). Girdling from expanding cankers results in a dieback of shoots and branches.

Fig. 45. Angular necrotic spots on sour cherry, caused by *Pseudomonas syringae* pv. *morsprunorum.* (Dr W. Zeller, Institut für biologischenPflanzenschutz der Biologischenm Bundesanstalt, Darmstadt)

Hosts
Prunus spp., predominantly sour cherry, sweet cherry, plum and prune.

Geographical distribution
Europe (widespread), eastern North America, South Africa, Lebanon and Australia (CMI 1980; Dhanvantari 1969). Reports from Lebanon and Australia have not been confirmed.

Biology and transmission
The occurrence of abundant epiphytic populations of the pathogen on leaf and shoot surfaces, of autumn leaf-scar infection, of spring bud-scar infection, and systemic invasion through the tree increase the likelihood of selecting budwood with latent bacteria.

Detection
King's B medium is best for isolation. Colonies that produce a fluorescent pigment when grown on King's B are identified based on biochemical and physiological tests, the reaction of immature cherry fruit to inoculation, the hypersensitive reaction of *Nicotiana tabacum* to infiltration of suspensions of the bacterium, and more recently by a DNA probe and PCR (Latorre and Jones 1979; Louws et *al.* 1994; Paterson and Jones 1991; Roos and Hattingh 1987).

For further reading, see p. 103.

Fig. 46. Leaf and flower buds infected with *Pseudomonas syringae* pv. *morsprunorum* failed to open. Dr W. Zeller, Institut für biologischen Pflanzenschutz der Biologischen Bundesanstalt, Darmstadt)

2. Bacterial canker of almond

Cause
Pseudomonas amygdali Psallidas and Panagopoulus, a nonfluorescent bacterium.

Significance
Affected trees have dead branches and twigs as a result of girdling; if the trunk is girdled by a canker the whole tree dies, so the economic damage can be considerable.

Symptoms
Cankers on branches, twigs and trunks; swellings of the bark, later cracking open and surrounded by swollen cortex.

Hosts
Host specific to almond trees.

Geographical distribution
Greece, Turkey (CMI 1978).

Biology and transmission
The bacterium overwinters in a tree canker and is disseminated by rain and wind. Infection takes place during leaf fall from leaf scars and wounds. Transmission with infected budwood is common.

Detection
Culturing on nutrient agar with 5% sucrose will result in typical white coloured colonies (Psallidas and Panagopoulos 1975).

For further reading, see p. 104.

3. Bacterial dieback of peach

Cause
Pseudomonas syringae pv. *persicae* (Prunier *et al.)* Young *et al.,* a gram-negative bacterium with a fluorescent pigment.

Significance
A serious disease, which destroys numerous trees every year in the central Rhone Valley of France.

Svmptoms
On young shoots, characteristic olive-green discolouration and browning around dormant buds resulting in dead buds or dieback of shoots in spring. On the trunk, brownish-red lesions. On young leaves, necrotic spots with a chlorotic halo, later causing a shot-hole effect. Fruits with necrotic spots, covered by a transparent gum.

Hosts
Peach and nectarine are the only hosts.

Geographical distribution
First described in France (Rhone Valley); Yugoslavia and New Zealand.

Biology and transmission
The bacterium enters shoots in autumn and winter through leaf scars. In spring the pathogen spreads to young shoots and remains in an epiphytic phase, which constitutes the inoculum reservoir.

Detection
Culturing on a selective medium (Luisetti *et al.* 1972).

For further reading, see p. 104.

4. Bacterial leaf spot

Cause
Xanthomonas campestris pv. *pruni* (Smith) Dye, a gram-negative bacterium (Hayward and Waterston 1965).

Significance
High incidence reduces economic life of trees and affects fruit quality.

Symptoms
Small, circular to irregular water-soaked leaf spots, which turn yellow, later purple or brown (Fig. 47). Affected areas drop out giving a shot-hole effect, particularly on plum. Bacterial ooze may be associated with the spots. On fruits small, circular brown spots, often with wet dark green halos. On twigs dark to black, slightly sunken circular lesions, resulting in dark purplish summer cankers or sunken elliptical lesions. If produced in late summer they are perennial.

Hosts
Only *Prunus* spp., particularly almond, peach, cherry, plum and apricot.

Geographical distribution
Nearly cosmopolitan.

Fig. 47. Yellow peach leaves with angular necrotic spots and tip necrosis caused by *Xanthomonas campestris* pv. *pruni*.
(Dr A. L. Jones, Michigan State University, East Lansing)

Biology and transmission

The bacterium overwinters primarily in twig lesions, and also in buds and fallen leaves of plum. In spring bacteria spread by rain, wind and insects to young leaves, fruits and twigs, which are infected through natural openings, leaf scars and wounds. Summer cankers are produced and perennation cankers develop after a winter incubation period. The pathogen can persist year-round on surfaces of peach and plum trees even in the absence of symptoms (Shepard and Zehr 1994). Material collected at any time could be infected without showing obvious symptoms.

Detection

With a detached-leaf bioassay (Randhawa and Civerolo 1985) and by isolation on a selective medium, which clearly shows the typical water-soluble yellow pigment (Gitaitis *et al.* 1988).

For further reading, see p. 104.

5. Crown gall

Cause
Agrobacterium turnefaciens (Smith and Townsend) Conn., mainly biovar 1, a gram-negative bacterium.

Significance
Serious disease in nurseries. Affected plants are unmarketable and cannot be exported.

Symptoms
Galls on all woody parts, particularly near the soil surface (crown), graft union and roots (Fig. 48).

Hosts
Many dicotyledons (643 species from 331 genera). *Prunus* spp., roses and small fruits *(Rubus* spp.) are most seriously affected. Also pome fruits.

Geographical distribution
Cosmopolitan.

Biology and transmission
Wound pathogen with saprophytic survival in vascular tissue and in infested soil. The pathogen stimulates the parenchyma cells of the host plant to abnormal growth with the production of galls. Transmission occurs with contaminated tools and grafting material or soil (Smith *et al.* 1988).

Detection
Culturing on selective media and pathogenicity test on indicator plants (tomato, *Datura,* sunflower or *Bryophyllum)* (Anderson and Moore 1979; Brisbane and Kerr 1983). New methods are based on molecular techniques with PCR (Dong *et al.* 1992).

For further reading, see p. 104.

Fig. 48. Galls on sweet cherry, caused by *Agrobacterium tumefaciens.* (Dr W. Zeller, Institut für biologischen Pflanzenschutz der Biologischen Bundesanstalt, Darmstadt)

6. Phony peach, plum leaf scald, almond leaf scorch

Cause
Xylella fastidiosa Wells *et al.*, a xylem-limited, fastidious, gram-negative bacterium. The diseases were considered of virus etiology until the bacterium causing Pierce's disease was visualized and cultured. Strains of *X. fastidiosa* can be grouped into two or more pathotypes. The Pierce's disease strain causes almond leaf scorch and differs pathogenically from the strain that causes phony peach and plum leaf scald.

Significance
Effective eradication programmes reduced the diseases to insignificance.

Symptoms
Peach trees with phony peach are compact, flattened, umbrella-like with shortened stem internodes, dense dark green foliage and fruit about half normal size. Marginal leaf scorching is the most diagnostic characteristic of almond leaf scorch and plum leaf scald. It is followed by decreased productivity, general decline and subsequent death of affected trees.

Hosts
Grapevine, to a lesser extent peach, almond, plum and apricot and a wide range of annual and perennial wild and cultivated plants. Many hosts are symptomless.

Geographic distribution
Phony peach, southeastern USA; plum leaf scald, southeastern USA and South America (Argentina, Brazil and Paraguay); almond leaf scorch, western USA (California) and India (Smith *et al.* 1992).

Biology and transmission
Transmitted in a persistent manner by various leafhopper vectors and by grafting. The bacteria are sensitive to low temperatures and this appears to restrict their geographic distribution to areas with moderate climate.

Detection
Culturing on selective media, electron-, phase contrast and fluorescence microscopy; ELISA, and PCR (French *et al.* 1977; Minsavage *et al.* 1994).

Therapy
Hot water treatment of dormant wood at 45°C for 3 hours (Goheen *et al.* 1973).

For further reading, see p. 105.

Fungal diseases

1. Black knot

Cause
Teleomorph: *Apiosporina morbosa* (Schwein.:Fr) Arx, formerly *Dibotryon morbosum* (Schwein.:Fr) Theiss. & Syd., anamorph: *Fusicladium* sp., formerly *Cladosporium* and *Hormodendrum.*

Significance
Destructive on wild plum and on plum, prune and sour cherry in gardens near wild hosts. Economically important in commercial plantings of plum, prune, sour cherry, and occasionally on other *Prunus* spp. including ornamentals.

Symptoms
Longitudinal swellings or corky outgrowths on shoots, spurs, branches and occasionally on trunks. Initially knots are greenish and soft, later hard and black but often with new swellings developing at the ends (Fig. 49). Limbs or entire trees may be killed from girdling as knots expand. See biology and transmission concerning symptomless budsticks.

Fig. 49. Prune branch es with young (top) and old (bottom) knots by *Apiosporina morbosa.* (Dr A. L. Jones, Michigan State University, East Lansing)

Hosts
Prunus spp. Common on plum and prune, occasional on sour cherry, rare on apricot, sweet cherry, peach and other *Prunus* spp.

Geographical distribution
North America (CMI 1994).

Biology and transmission
Local spread by ascospores discharged during rain from perithecia in 2-year-old knot tissue. Infection occurs after bud break while shoots are actively growing. Symptoms of infection visible in autumn or the following spring. Long-distance spread possible from latent infections on bud sticks.

Detection
Fungus can be confirmed by culturing on media or by microscopic observation of ascospores in perithecia immersed in knot tissue.

Quarantine measures
Infected wild hosts should be removed from hedges surrounding nurseries and budwood orchards. Inspect trees propagated with budwood obtained from problem countries for two growing seasons for knots.

For further reading, see p. 105.

2. Brown rot of stone fruits

Cause
Teleomorph: *Monilinia fructicola* (G. Wint.) Honey, formerly *Sclerotinia fructicola* (G. Wint.) Rehm, anamorph: *Monilia* sp.

Significance
Greatest loss is from decay of fruit in the orchard, in transit, in the market and prior to consumption. Losses from blossom blight are minor compared with those caused by *M. laxa* (Aderh. & Ruhl.) Honey.

Symptoms
Infected blossoms wilt and turn brown. Infected fruit develops circular, light brown spots that rapidly expand to decay the flesh (Fig. 50). Spurs on peach, nectarine and apricot trees may be blighted near harvest following systemic fungal invasion from infected fruit including 'mummies' (shrivelled fruit).

Hosts
All species of *Prunus*. Occasionally infects fruit of other genera only as a fruit wound pathogen.

Geographical distribution
Temperate regions including Argentina, Australia, Bolivia, Canada, Central America, Egypt, Japan, New Zealand, Peru, South Africa, USA, Venezuela (Mordue 1979).

Biology and transmission
Transfer of ascospores and conidia from mummified fruits, pedicles and small cankers is unlikely when nonbearing shoot growth is collected as budwood.

Detection
Fungus can be confirmed by microscopic observation of conidia formed in chains in culture or on infected tissue. *M. fructicola* can be differentiated from *M. laxa* based on cultural characteristics, isozyme variation and vegetative interactions (Penrose *et al.* 1976; Sonoda *et al.* 1982).

For further reading, see p. 106.

Fig. 50. Sour cherry fruits with conidia of *Monilinia fructicola*, the cause of brown rot. (Dr A. L. Jones, Michigan State University, East Lansing)

3. Eutypa dieback

Cause
Teleomorph: *Eutypa lata* (Pers.:Fr.) Tul. & C. Tul., formerly *E. armeniacae* Hansf. & Carter, anamorph *Libertella blepharis* A. L. Smith, formerly *Cytosporina* sp.

Significance
Infection develops in apricot trees of all ages, but increases from year to year as trees become older. Dieback of limbs and trees reduces yields and economic viability of orchards.

Svmptoms
Cankering around wounds exhibiting sapwood; gumming; dieback of branches above cankers in summer with collapsed leaves remaining; attached for several months on affected, sometimes swollen, limbs. Light brown to dark brown sapwood extends upwards and downwards from the canker.

Hosts
Common on apricot and grapevine *(Vitis* spp.), also on almond, plum, sweet cherry and chokecherry *(Prunus virginiana* var. *demissa).* Fungus has wide host range including approximately 90 species distributed across 28 botanical families (Carter *et al.* 1983).

Geographical distribution
Cosmopolitan (CMI 1982).

Biology and transmission
Perithecia develop in a stroma on dead branches and are most common in regions where apricots and grapevines are grown with more than 600 mm of annual precipitation (sprinkler irrigation in orchards in semi-arid regions may also favour development of perithecia (Munkvold and Marois 1994)). Airborne ascospores, or secondary dispersal of ascospores by splashing rainwater, infect through vascular tissue exposed at fresh pruning or mechanical wounds.

Detection
This pathogen should not occur on budwood material transported as recommended.

For further reading, see p. 106.

4. Fusicoccum canker (blight of almond and peach, constriction disease)

Cause
Anamorph: *Phomopsis amygdali* (Delacr.) Tuset & Portilla, comb. nov., formerly *Fusicoccum amygdali* Delacr. and *Phomopsis amygdalina* Canonaco (Tuset and Portilla 1989).

Significance
Losses from dieback or defoliation may be quite severe in the Mediterranean region on almond and peach trees in commercial orchards and budwood blocks. In the Mid-Atlantic region of the USA, devastating losses occurred on highly susceptible peach cultivars in the 1940s and early 1950s (Zehr 1995). Of negligible economic importance in orchards of New Zealand (Atkinson 1971).

Symptoms
Elongate, brown, sunken cankers, often with a zonate pattern, form at the base of infected buds or nodes of l-year-old shoots. Twigs are blighted from girdling and from the action of a toxin secreted by the fungus (Fig. 51). Circular or irregular, zonate, large brown spots develop in leaves.

Hosts
Prunus spp., particularly peach and almond, occasionally apricot and rarely plum.

Fig. 51. Dieback of peach due to constriction by a canker, caused by *Phomopsis amygdali*, at the base of the shoots. (Dr A. L. Jones, Michigan State University, East Lansing)

Geographical distribution
USA, particularly eastern and southeastern states; Europe, particularly Bulgaria, France, Greece, Italy, Spain, Portugal and United Kingdom; Japan; South America, particularly Argentina and Brazil; Tunisia; New Zealand (Atkinson 1971; Zehr 1995).

Biology and transmission
Pycnidia exude conidia in white tendrils or cirri during wet weather. Conidia spread by rain infect through leaf scars in autumn and through buds, bud scale scars, stipule scars, fruit scars, or directly through young shoots during the growing season. Symptoms of infection visible in spring and infections become increasingly evident as more blighted shoots appear through late summer.

Detection
The fungus can be confirmed by culturing on media or by microscopic observation of pycnidia in spherical or flattened, subepidermal, erumpent, dark stroma in cankered tissue. This pathogen should not occur on material collected from trees free of disease symptoms and treated for transport as recommended.

For further reading, see p. 106.

5. Leaf scorch of apricot and cherry

Cause
Teleomorph: *Apiognomonia erythrostoma* (Pers.) Hahn., formerly *Gnomonia erythrostoma* (Pers.) Auersw., anamorph: *Phomopsis stipata* (Lib.) Sutton, formerly *Libertina stipata* (Lib.) Hahn.

Significance
More serious on apricot than on cherry in Eastern Europe (Smith *et al.* 1988).

Symptoms
Irregular, yellow to reddish leaf spots that become brown as lesions expand to several centimeters in diameter (Fig. 52). Severely infected leaves fall prematurely, although leaf fall patterns vary according to climate. Premature fruit drop is also a feature of the disease if defoliation is severe before harvest.

Hosts
Prunus spp., particularly apricot, sweet cherry and sour cherry.

Geographical distribution
Europe, particularly Austria, Balkan Peninsula, Czech Republic, France, Italy and Slovakia (Zehr 1995).

Biology and transmission
Inoculum confined to ascospores discharged during spring from perithecia formed in winter in fallen leaves. Fruits and shoots are not infected.

Detection
This pathogen should not occur on material transported as recommended.

For further reading, see p. 107.

Fig. 52. Apricot leaves with leaf scorch caused by *Apiognomonia erythrostoma.* Note the necrosis extending along the leaf vein. (Dr W. Wittmann, Bundesanstalt für Pflanzenschutz, Vienna)

6. Leucostoma canker

Cause
Teleomorph: *Leucostoma cincta* (Fr.) Hahn., formerly *Valsa cincta* (Fr.ex Fr.) Fr., anamorph: *Leucocytospora cincta* (Sacc.) Hahn., formerly *Cytospora cincta* Sacc. and teleomorph: *Leucostoma persoonii* Hahn., formerly *V. leucostoma* (Pers. ex Fr.) Fr., anamorph: *Leucocytospora leucostoma* (Pers.) Hahn., formerly *Cytospora leucostoma* Sacc.

Significance
The disease is part of the 'apoplexy' disease complex in Europe and the 'peach-tree short-life syndrome' in the southern USA (Biggs 1995). It reduces the bearing surface and tree longevity.

Symptoms
Elongated cankers, often with copious amber-coloured gum, typically on the trunk and scaffold limbs and in branch crotches (Fig. 53). Dieback of short shoots and twigs in the interior of trees, especially following winter injury. Limb dieback, particularly on plum, prune, apricot and sweet cherry.

Hosts
Found on cultivated and wild hosts, mostly in the family Rosaceae. Both *Leucostoma* spp. occur frequently on *Prunus* spp., particularly on peach and apricot, less frequently on plum, prune and sweet cherry, and infrequently on sour cherry.

Fig. 53. Leucostoma canker on peach, caused by *Leucostoma cincta*.
(Dr A. L. Jones, Michigan State University, East Lansing)

Geographical distribution
Cosmopolitan (Atkinson 1971; Biggs 1995).

Biology and transmission
Both *Leucostoma* spp. attack host tissues damaged or weakened by low temperatures, mechanical damage, pruning wounds and other stress factors. Pycnidia and later perithecia are formed in a stroma and erupt through dead bark. *Leucostoma* is distinguishable from the genus *Valsa,* which may be saprophytic on *Prunus,* based on the presence of a black delimiting zone line or conceptacle surrounding a well-developed ascocarp.

Detection
This pathogen is unlikely to occur on budwood transported as recommended. *L. cincta* can be differentiated from *L. persoonii* based on isozyme variation and cultural characteristics (Surve-Iyes *et al.* 1995).

For further reading, see p. 107.

7. Peach leaf curl and related diseases

Cause

Several *Taphrina* spp. attack *Prunus* spp.

The most common is *Taphrina deformans* (Berk.) Tul. (peach leaf curl). Others include: *T. armeniacae* Georgescu & Badea (witches' broom); *T. communis* (Sadeb.) Giesenh. (bladder plum or plum pocket); *T. confusa* (Atk.) Giesenh.; *T. farlowii* Sadeb. (leaf curl); *T. flavorubra* W.W. Ray; *T. flectans* Mix (leaf curl and witches' bloom); *T. jenkinsoniana* Mix; *T. pruni* Tul. (bladder or pocket plums); *T. pruni-subcordatae* (Zeller) Mix; *T. thomasii* Mix; *T. wiesneri* (Rathay) Mix, formerly *T. cerasi* (Fuckel) Sadeb. (witches' broom and leaf curl of cherry and apricot) (Farr *et al.* 1989).

Significance

Potentially destructive in commercial plantings without regular chemical control. These diseases can be very severe in wet, humid regions where bud burst extends for 2 to 7 weeks.

Symptoms

Parts of peach and nectarine leaves are swollen, distorted and curled downwards (Fig. 54). Affected leaves first appear reddish, then turn yellowish. Also blossoms, fruits and the current year's twigs may be affected. In plum, the disease first appears as small white blisters on the fruit.

Fig. 54. Peach leaf curl on leaves and fruit, caused by *Taphrina deformans.* (Dr A. L. Jones, Michigan State University, East Lansing)

Hosts
Prunus spp.; each *Taphrina* spp. is limited to one or two *Prunus* spp. only.

Geographical distribution
Taphrina deformans and T. wiesneri are widely distributed, other species are limited to specific regions:
T. armeniacae: Romania
T. communis: central and eastern North America
T. confusa: temperate North America
T. farlowii: eastern North America
T. flavorubra: temperate North America
T. flectans: western North America
T. jenkinsoniana: western USA
T. pruni: temperate northern hemisphere
T. pruni-subcordatae: western North America
T. thomasii: California.

Biology and transmission
Most *Taphrina* spp. have a yeast-like saprophytic phase on host surfaces and a parasitic phase inside the vegetative growth and/or fruit. Intercellular growth of the fungus causes cell division and cell enlargement (hyperplasia and hypertrophy) that result in thickened, curled to convoluted, or blistered leaves, shoots, or fruits.

Detection
This pathogen is unlikely to occur on budwood transported as recommended.

For further reading, see p. 107.

8. Peach scab (freckle)

Cause
Teleomorph: *Venturia carpophila* E.E. Fisher, anamorph *Cladosporium carpophilum* Thüm., formerly *Fusicladium carpophilum* (Thüm.) Oudem.

Significance
Economically important in regions with high rainfall, high humidity and warm temperatures between bloom and harvest. Fruit lesions reduce the appearance, quality and market value of the fruit. Usually rare in semi-arid stone fruit producing regions.

Symptoms
Circular, olivaceous to black, velvety spots produced on fruit and twigs (Fig. 55), less frequently on leaves. Lesions on fruit coalesce when numerous, followed by cracking of the fruit. Shoot and twig infections are slightly raised, circular to oval, becoming brown with slightly raised purple margins later in the season.

Hosts
Prunus, largely apricot, plum, peach and almond. A notable exception is cherry.

Geographical distribution
Cosmopolitan (CMI 1979).

Biology and transmission
Overwintering occurs in lesions on twigs with conidial production beginning about when shucks covering the fruit split. When selecting budwood, note that infections are latent for 40 to 60 days.

Detection
Microscopic observation of fungus in lesions. Isolation by single-spore techniques only when actively sporulating.

Quarantine measures
Since incipient infection may occur, tissue culture is the only way to avoid this problem. Trees propagated with budwood from areas where the disease is a problem should be observed for symptoms for 1 year.

For further reading, see p. 107.

Fig. 55. Peach scab lesions on the fruit, caused by *Venturia carpophila.* (Dr A. L. Jones, Michigan State University, East Lansing)

9. Powdery mildew

Cause
Teleomorph: *Sphaerotheca pannosa* (Wallr.:Fr.) Lév., anamorph: *Oidium* spp.

Significance
Most important on peach, apricot, almond and nectarine. Fruit infections cause the greatest economic loss, infections to leaves and shoots can reduce vegetative growth, particularly on nursery trees.

Symptoms
Leaves and shoots superficially covered with white felt-like mycelium, later becoming distorted and stunted. White circular spots on young fruit expand in size, later the mycelium may slough off, leaving a russeted patch with dead epidermal cells (Fig. 56). The russeted area expands as the fruit enlarges.

Hosts
Prunus spp. (peach as well as apricot, almond, nectarine). *Rosa* spp. are also hosts of *S. pannosa*. The mildew on each host is considered pathologically distinct (Yarwood 1939); however, Kable *et al.* (1980) suggest the existence of a strain of *Oidium* pathogenic on rose and peach.

Fig. 56. Peaches with superficial, white, powdery mildew lesions, caused by *Sphaerotheca pannosa.* (Dr A. L. Jones, Michigan State University, East Lansing)

Geographical distribution
Appears to be cosmopolitan but exact distribution on *Prunus* often unclear due to possible confusion with *Podosphaera clandestina* (Wallr.:Fr.) Lev. (formerly *P. oxyacanthae* (DC.) de Bary).

Biology and transmission
The fungus overwinters as mycelium under bud scales. Disease spread is by airborne conidia.

Detection
The identification of *S. pannosa* on *Prunus* is difficult because ascocarps are rarely present. The imperfect stage is distinguished by the shape and dimensions of the conidiophore and location of the basal septum. This pathogen should not occur on budwood collected from trees free of mildew and transported as recommenced.

For further reading, see p. 108.

10. Stone fruit rust diseases

Cause
Tranzschelia discolor (Fuckel) Tranzschel & Litv., formerly *T. pruni-spinosae* (Pers.:Pers.) Dietel var. *discolor* (Fuckel) Dunegan and *T. pruni-spinosae* (Pers.:Pers.) Dietel.

Significance
Economically important in stone fruit regions with mild winters; of minor or no economic importance in regions with severe winters. Losses potentially severe where orchards and nurseries are not sprayed regularly.

Symptoms
Small, yellow, irregular spots appear on the upper and lower surfaces of leaves (Fig. 57). Those on the lower surface later turn rusty brown (Fig. 58). Leaves with numerous lesions turn yellow and fall. Small, superficial, pale brown, slightly raised spots, which eventually form longitudinal splits or cankers, develop on shoots. Rarely, small circular dark green spots formed on fruit of peach and apricot. Lesions become depressed with reddish margins as the fruit matures.

Hosts
T. discolor is common on almond, peach, nectarine, apricot, plum and wild *Prunus* spp.; infrequent on cherry. *T. pruni-spinosae* is common on wild *P. spinosa,* infrequent on commercial hosts. *Anemone,* especially *A. coronaria* and *A. ranunculoides,* is the alternate host.

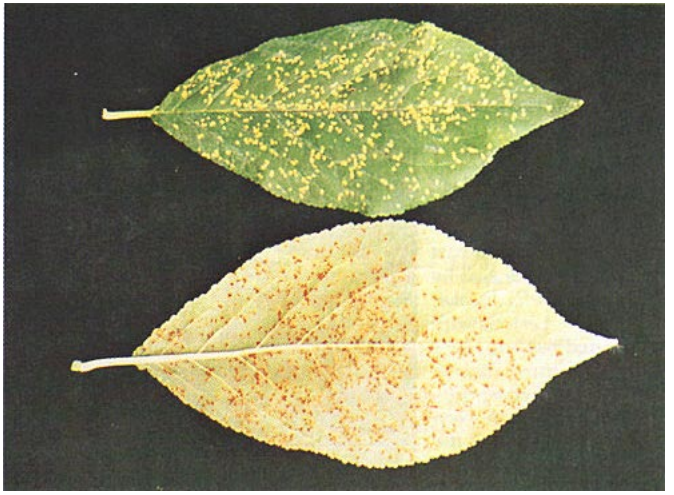

Fig. 57. Stone fruit rust, caused by *Tranzschelia discolor,* on the top and bottom side of leaves of Japanese plum. (Dr A. Sztejnberg, The Hebrew University of Jerusalem, Rehovot, Israel)

Geographic distribution
The distribution of these fungi is not entirely clear because both species were classified as *T. pruni-spinosae* in the early literature. *T. discolor* is cosmopolitan, while *T. pruni-spinosae* exists primarily in Europe and the USA (Laundon and Rainbow 1971a, 1971b).

Biology and transmission
Shoots infected in the autumn or early winter. Infections visible in late autumn or in spring. Disease spreads by urediospores produced in lesions on shoots. Although anemone plants help perpetuate the rusts where *Prunus* and *Anemone* are growing together, cankers on shoots are sufficient for perpetuation in the absence of anemone.

Detection
Infections on budwood detected by microscopic examination for yellowish-orange spots. *T. discolor* distinguished from *T. pruni-spinosae* based on differences in morphology of the teliospores (Bloomer 1960). Rust pathogens should not occur on material collected from trees free of disease symptoms and treated for transport as recommended.

For further reading, see p. 108.

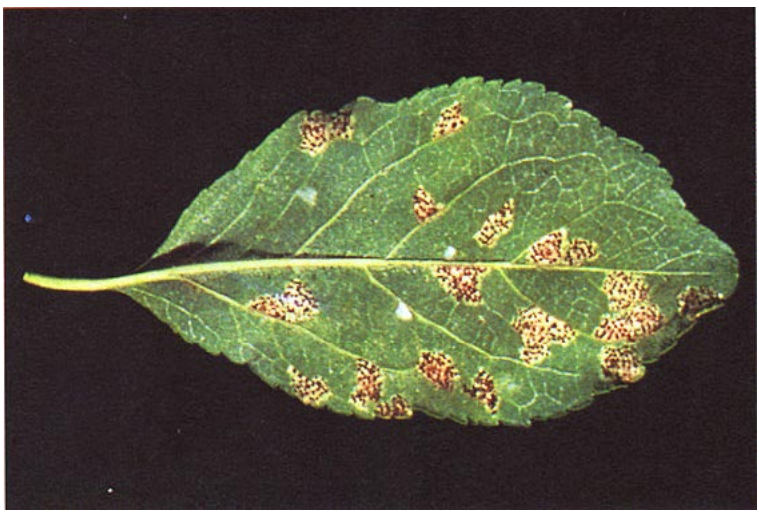

Fig. 58. Stone fruit rust on prune, caused by *Tranzschelia discolor*. Lesions with urediospores; dark structures may be teliospores. (Dr R. C. Seem, New York State Agricultural Experiment Station, Geneva)

Arthropods

The risk of introducing new arthropod pests with stone fruit germplasm is mainly due to the occurrence of eggs or other stages not easily detected on green or especially dormant budwood. The following groups may be found:

1. Aphids

Numerous species, such as *Brachycaudus* spp., *Hyalopterus* spp. and *Myzus* spp., belonging mostly to the family Aphididae.

Significance
Depending on aphid and host plant species, ranging from almost unimportant to highly injurious. Some species may transmit viruses.

Damage
During feeding activity toxic saliva or virus particles are injected into the plants. Also honeydew production may occur.

Hosts
Monoecious species live exclusively on the primary host plant, such as *Pterochloroides persicae* (Cholodkovsky) or *Brachycaudus schwartzi* (Borner) on peach. Dioecious species live on stone fruit trees only for a part of their life cycle and migrate in summer to secondary, herbaceous host plants (examples: *Brachycaudus helichrysi* (Kaltenbach), *Hyalopterus* spp. and *Myzus* spp.).

Geographical distribution
Some are cosmopolitan, such as *Brachycaudus helichrysi* and *Myzus persicae* Sulz., others are more restricted in distribution. *Hysteroneura setariae* (Thomas) occurs in America, small areas in South Africa, India, Far Eastern Asia and Oceania; *Asiphonaphis pruni* Wilson & Davis in North America; *Brachycaudus prunicola* (Kaltenbach) in Europe; *Tuberocephalus* spp. in East Asia (Blackman and Eastop 1984).

Biology
Holocyclic species, such as *Hyalopterus* spp. and *Myzus* spp., overwinter as eggs on stems and buds of stone fruit trees.

Treatment
Infested material should be dipped in currently used aphicides or fumigated with methyl bromide (32 g/m^3 for 3 hours).

For further reading, see p. 108.

2. Armoured scale insects

Important species include *Quadraspidiotus perniciosus* (Cornstock), *Q. ostreaeformis* (Curtis), *Pseudaulacaspis pentagona* (Targioni-Tozzetti), *Lepidosaphes ulmi* (L.) and *Parlatoria oleae* (Colvée).

Significance
Varies, from fruits losing market value owing to the occurrence of scales on the epicarp, to plant death.

Damage
Females and male larvae mainly settle on stems and branches of their host plants (Figs. 59, 60), attacking also fruits and leaves (Fig. 61). No honeydew is produced. When occurring on fruits, some species, such as *Quadraspidiotus perniciosus* or *Parlatoria oleae*, cause round, red spots at the points of inserting their stylets.

Hosts
Generally polyphagous on various fruit trees and other plants. *Quadraspidiotus perniciosus* is reported to occur on about 700 hosts.

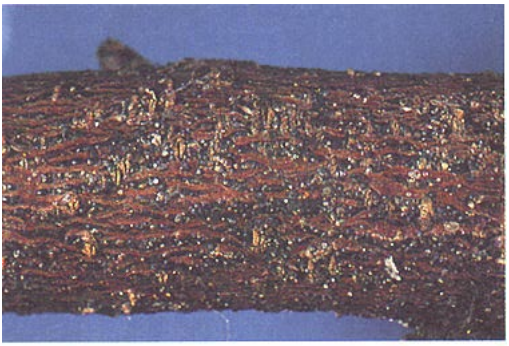

Fig. 59. First instar larvae ('black cap' phase) of the San José scale *Quadraspidiotus perniciosus* on the bark of a peach tree. (Dr C. Rapisarda, Istituto di Entomologia Agraria, Catania)

Fig. 60. Peach tree infested with the white peach scale *Pseudaulacaspis pentagona*. (Dr C. Rapisarda, Istituto di Entomologia Agraria, Catania)

Geographical distribution

Some species almost cosmopolitan, such as *Pseudaulacaspis pentagona* or *Quadraspidiotus perniciosus* (Kosztarab and Kozar 1988; Kozar 1990). Others are restricted: *Lepidosaphes ulmi* to Central and South Europe, Central and East Asia, North and South America, as well as some areas of Oceania, or *Parlatoria oleae* to the Mediterranean region, Central India and South America (Kozar 1990).

Biology

Most species with winged adult males and apterous females and young males. Apterous forms have their body covered by a protective 'scale' whose shape, dimensions, colour and location of ecdysis are all important characters for identification. Most of the species are polyvoltine with up to 4 to 5 generations per year, according to climatic conditions. Some species are strictly viviparous (e.g. *Quadraspidiotus perniciosus)* but most are oviparous. Winter diapause is facultative for some species, obligate for others and spent in different stages (egg for *Lepidosaphes ulmi,* larva for *Parlatoria oleae, Q. perniciosus, Q. ostreaefoumis,* adult female for *P. oleae, Pseudaulacaspis pentagona).*

Treatment

Fumigation of material with methyl bromide (32 g/m^3 for 3 hours).

For further reading, see p. 109.

Fig. 61. Young larvae of the brown scale *Parthenolecanium corni* on a bud. (Dr C. Rapisarda, Istituto di Entolnologia Agraria, Catania)

3. Mites

Different mite species occur on stone fruit trees. Most important are Tetranychidae, such as the brown mite *Bryobia rubrioculus* (Scheuten) and the European red mite *Panonychus ulmi* (Koch) as well as Eriophyidae, such as the plum rust mite *Aculus fockeui* (Nalepa and Trouessart), and the peach silver mite *A. cornutus* (Banks).

Significance
Highly variable, according to the species. *Panonychus ulmi* is the most important mite and one of the main pests in stone fruit orchards.

Damage
Mites injure plants directly by feeding on the leaves. Withdrawal of lymph and chlorophyll initially causes discolouration in infested leaf areas. Later, leaves may turn bronze and, in some cases, they may be rolled or distorted. Early leaf fall may result. Mites in the family Eriophyidae may transmit viruses (Oldfield 1970).

Hosts
Most species are polyphagous on deciduous fruit trees.

Geographical distribution
Brown and European red mites are cosmopolitan (CIE 1972, 1984). Other species have a limited distribution (Jeppson *et al.* 1975).

Biology
Nearly all mites feeding on stone fruits are polyvoltine. Some of them, such as *Bryobia rubrioculus* or *Panonychus ulmi*, overwinter as red and onion-shaped eggs on budwood, whereas Eriophyidae hibernate as adult females in bark crevices or under loose bud scales.

Treatment
Budwood infested by mites should be dipped in acaricides, preferably ovicides, such as clofentezine, or larvo-adulticides, such as dicofol or fenbutatin oxide.

For further reading, see p. 109.

4. Planthoppers (sensu lato)

A number of species belonging to various families of Homoptera Auchenorrhyncha, such as *Stictocephala bisonia* Koppe & Yonk (Membracidae), *Metcalfa pruinosa* (Say) (Flatidae), *Cicadella viridis* (L.), *Edwardsiana rosae* (L.), *Empoasca decedens* Paoli and *Zygina* spp. (Cicadellidae).

Significance
Some species are important as virus vectors.

Damage
Injuries occur by feeding and by eggs laid in wounds created with the ovipositor along twigs and stems. Infested leaves show discoloured areas.

Hosts
Widely polyphagous on cultivated and wild plants.

Geographical distribution
Widespread in temperate regions. Some species are spreading, for example the flatid planthopper *Metcalfa pruinosa,* recently introduced into southern France and Italy (CIE 1992).

Biology
Species overwinter in the egg stage. Up to four generations may occur in spring and summer.

For further reading, see p. 109.

5. Soft scale insects

This group includes several species of the family Coccidae; the most important ones are *Eulecanium tiliae* (L.), *Parthenolecanium corni* (Bouché) and *P. persicae* (Fabricius).

Significance
Ranges from almost unimportant to highly injurious. *Parthenolecanium corni* may become a serious pest in stone fruits.

Damage
Soft scale insects weaken infested plants. Infested leaves and fruits are covered with sooty moulds, which develop on honeydew.

Hosts
Most species are polyphagous.

Geographical distribution
The mentioned *Parthenolecanium* spp. are diffused almost worldwide, both occurring nearly throughout the Palaearctic region, Australasia, North and South America, Indian region (Ben-Dov 1993); *Eulecanium tiliae* is reported only from Palaearctic and Indian region, North America and Tasmania (Kosztarab and Kozar 1988). Several other, secondary species have a more restricted diffusion (Kosztarab and Kozar 1988; Ben-Dov 1993).

Biology
The three species mentioned above have only one generation per year and overwinter as second-stage larvae on bark.

Treatment
Fumigation of infested material with methyl bromide (32 g/m^3 for 3 hours).

For further reading, see p. 109.

Nematodes

Many nematode species may be found associated with soil surrounding plant roots or in the roots. Some of them, such as *Longidorus* spp. or *Xiphinema* spp., may transmit viruses. The risk of introducing these pests with stone fruit germplasm is low when material is handled as recommended in these guidelines and no germplasm is moved as rooted plant material.

BIBLIOGRAPHY

General references on stone fruit diseases

Desvignes, J.C., R. Boye, D. Cornaggia and N. Grasseau. 1990. Maladies Virus des Arbres Fruitiers (Maladies à virus, à Mycoplasmes et à Viroïdes). Centre Technique Interprofessionel des Fruits et Legumes (CTIFL), Paris, France.

Gilmer, R.M., J.D. Moore, G. Nyland, M.F. Welsh and T.S. Pine (eds.). 1976. Virus Diseases and Non-infectious Disorders of Stone Fruits in North America. Agriculture Handbook No. 437, USDA, Washington, D.C., USA.

Németh, M. 1986. Virus, Mycoplasma and Rickettsia Diseases of Fruit Trees. Martinus Nijhoff Publishers, Dordrecht, The Netherlands.

Virus indexing

Knapp E., A. da Câmara Machado, H. Pühringer, Q. Wang,V. Hanzer, H. Weiss, B. Weiss, H. Katinger and M. Laimer da Câmara Machado. 1995. Localization of fruit tree viruses by immuno tissue printing in infected shoots of *Malus* and *Prunus* sp. J. Virol. Meth. 55:157-173.

Virus therapy

Knapp E., V. Hanzer, H. Weiss, A. da Câmara Machado, Q. Wang, B. Weiss, H. Katinger and M. Laimer da Câmara Machado. 1995. New aspects of virus elimination in fruit trees. Acta Hort. 386:409-418.

American plum line pattern virus

Fulton, R.W. 1982. Ilar-like characteristics of American plum line pattern virus and its serological detection in *Prunus*. Phytopathology 72:1345-1348.

Fulton, R.W. 1984. American plum line pattern virus. CMI/AAB Descriptions of Plant Viruses No. 280. Commonwealth Agricultural Bureaux, Kew, UK.

Kirkpatrick, H.C. and R.W. Fulton. 1976. Plum line pattern. Pp. 166-175 *in* Virus Diseases and Non-infectious Disorders of Stone Fruits in North America (R.M. Gilmer, J.D. Moore, G. Nyland, M.F. Welsh and T.S. Pine, eds.). Agriculture Handbook No. 437, USDA, Washington, DC, USA.

Paulsen, A.Q. and R.W. Fulton. 1968. Hosts and properties of a plum line pattern virus. Phytopathology 58:766-772.

Smith, I.M., D.G. McNamara, P.R. Scott and K.M. Harris (eds.). 1992. Quarantine pests for Europe. Data sheets on quarantine pests for the European Communities and for the European and Mediterranean Plant Protection Organization. CAB International, Wallingford, UK, and European and mediterranean Plant Protection Organization, Paris, France.

Apple chlorotic leafspot virus

Candresse, T., M. Lanneau, F. Revers, N. Grasseau, G. Macquaire, S. German, T. Malinovsky and J. Dunez. 1995. An immunocapture PCR assay adapted to detection and the analysis of the molecular variability of the apple chlorotic leafspot virus. Acta Hort. 386:136-147.

Desvignes, J.C. and R. Boye. 1989. Different diseases caused by the chlorotic leaf spot virus on the fruit trees. Acta Hort. 235:31-38.

Knapp, E., V. Hanzer, H. Weiss, A. da Câmara Machado, Q. Wang, B. Weiss, H. Katinger and M. Laimer da Câmara Machado. 1995. Distribution of apple chlorotic leafspot virus in apple shoots cultivated *in vitro*. Acta Hort. 386:187-194.

Martelli, G.P., T. Candresse and S. Namba. 1994. Trichovirus, a new genus of plant viruses. Arch. Virol. 134:451-455.

Nemchinov, L., A. Hadidi, T. Candresse, J.A. Foster and T. Verderevskaya. 1995. Sensitive detection of apple chlorotic leaf spot virus from infected apple or peach tissue using RT-PCR, IC-RT-PCR or multiplex IC-RT-PCR. Acta Hort. 386: 51-62.

Németh, M. 1986. Virus, Mycoplasma and Rickettsia Diseases of Fruit Trees. Martinus Nijhoff Publishers, Dordrecht, The Netherlands.

Paunovic, S. 1988. Properties of two apple chlorotic leaf spot virus isolates. Acta Hort. 235:39-47.

Apple mosaic virus (European plum line pattern)

Cameron, H.R. and M. Thompson. 1985. Seed transmission of apple mosaic virus in hazelnut. Acta Hort. 193:131.

De Sequeira, O.A. 1967. Purification and serology of an apple mosaic virus. Virology 31:314-322.

Digiaro, M., V. Savino and B. Di Terlizzi. 1992. Ilarviruses in apricot and plum pollen. Acta Hort. 309:93-98.

Fulton, R.W. 1972. Apple mosaic virus. CMI/AAB Descriptions of Plant Viruses No. 83. Commonwealth Agricultural Bureaux, Kew, UK.

Mink, G.I. 1989. Apple mosaic virus. Pp. 34-39 *in:* Virus and Viruslike Diseases of Pome Fruits and Simulating Noninfectious Disorders (P.R. Fridlund, ed.). Washington State University, Pullman, WA.

Posnette, A.F. and C.E. Ellenberger. 1957. The line pattern virus disease of plums. Ann. Appl. Biol. 45:74-80.

Seneviratne, S.N. de S. and A.F. Posnette. 1970. Identification of viruses isolated from plum trees affected by decline, line pattern and ringspot diseases. Ann. Appl. Biol. 68:115-125.

Torrance, L. and C.A. Dolby. 1984. Sampling conditions for reliable detection by enzyme-linked immunosorbent assay of three ilarviruses in fruit trees. Ann. Appl. Biol. 104:267-276.

Cherry green ring mottle virus

Barksdale, T.H. 1959. Green ring mottle virus as a entity distinct from sour cherry ring spot and yellows viruses. Phytopathology 49:777-784.

Gilmer, M. 1961. The frequency of necrotic ring spot, sour cherry yellows and green ring mottle viruses in naturally infected sweet and sour cherry orchard cherry trees. Plant. Dis. Reptr. 45:608-611.

Milbrath, J.A. 1966. Severe fruit necrosis of sour cherry caused by strains of green ring mottle virus. Plant. Dis. Reptr. 50:59-60.

Nemeth, M. 1986. Virus, Mycoplasma and Rickettsia Diseases of Fruit Trees. Martinus Nijhoff Publishers, Dordrecht, The Netherlands.

Parker, K.G., P.R. Fridlund and R.M. Gilmer. 1976. Green ring mottle. Pp. 193-199 *in* Virus Diseases and Non-infectious Disorders of Stone Fruits in North America (R.M. Gilmer, J.D. Moore, G. Nyland, M.F. Welsh and T.S. Pine, eds.). Agriculture Handbook No. 437, USDA, Washington, DC, USA.

Zagula, K.R., N.M. Aref and DC. Ramsdell. 1989. Purification, serology, and some properties of a mechanical transmissible virus associated with green ring mottle disease in peach and cherry. Phytopathology 79:451-456.

Cherry leaf roll virus

Flegg, J.M. 1969. Tests with potential nematode vectors of cherry leaf roll virus. Ann. Rep. East Malling Res. Sta., A52,155-157.

Fritzsche, R. and N. Kegler. 1964. Die öbertragung des Blattrollvirus der Kirsche durch Nematoden. Naturwissenschaften 51:299.

Jones, A.T. 1985. Cherry leafroll virus. AAB Descriptions of Plant Viruses No. 306. Association of Applied Biologists, Wellesbourne, Warwick, UK.

Mircetich, SM. and A. Rowhani. 1984. The relationship of cherry leaf roll virus and blackline disease of English walnut trees. Phytopathology 74:423-428.

Nemeth, M. 1986. Virus, Mycoplasma and Rickettsia Diseases of Fruit Trees. Martinus Nijhoff Publishers, Dordrecht, The Netherlands.

Quacquarelli, A. and V. Savino. 1977. Cherry leaf roll virus in walnut *(Juglans regia):* II. Distribution in Apulia and transmission through seed. Phytopathol. Medit. 16:154-156.

Cherry little cherry

Hansen, A.J. and L. Green. 1985. Canindex I, a superior indicator cultivar for little cherry disease. Plant Dis. 69:11-12.

Raine, J., R.D. McMullen and A.R. Forbes. 1986. Transmission of the agent causing little cherry disease by the apple mealybug *Phenacoccus aceris* and the dodder *Cuscuta lupuliformis.* Can. J. Plant Pathol. 8:6-l 1.

Raine, J., M. Weintraub and B. Schroeder. 1975. Flexuous rods and vesicles in leaf and petiole phloem of little-cherry diseased *Prunus* spp. Phytopathology 65:1181-1186.

Smith, I.M., D.G. McNamara, P.R. Scott and K.M. Harris (eds.) 1992. Quarantine pests for Europe. Data sheets on quarantine pests for the European Communities and for the European and Mediterranean Plant Protection Organization. CAB International, Wallingford, UK, and European and Mediterranean Plant Protection Organization, Paris, France.

Welsh, M.F. and P.W. Cheney. 1976. Little cherry. Pp. 231-237 *in* Virus Diseases and Non-infectious Disorders of Stone Fruits in North America (R.M. Gilmer, J.D. Moore, G. Nyland, M.F. Welsh and T.S. Pine, eds.). Agriculture Handbook No. 437, USDA, Washington, DC, USA.

Cherry mottle leaf virus

Cheney, P.W. and CL. Parish. 1976. Cherry mottle leaf. Pp.. 216-218. *in* Virus Diseases and Noninfectious Disorders of Stone Fruits in North America (R.M. Gilmer, J.D. Moore, G. Nyland, M.F. Welsh and T.S. Pine, eds.). Agriculture Handbook No. 437. USDA, Washington, DC, USA.

James, D. 1992. Partial characterization of a closterovirus-like particle transmitted from cherry *(Prunus avium)* infected with several viral. diseases. Acta Hort. 309:39-44.

James, D. and S. Mukerji. 1993. Mechanical transmission, identification, and characterization of a virus associated with cherry mottle leaf. Plant Dis. 77:271-275.

Németh, M. 1986. Virus, Mycoplasma and Rickettsia Diseases of Fruit Trees. Martinus Nijhoff Publishers, Dordrecht, The Netherlands.

Oldfield, G.N., R.G. Creamer, C. Gispert, F. Osorio, R. Rodriguez and T.M. Perring. 1995. Incidence and distribution of peach mosaic and its vector *Eriophyes insidiosus* (Atari: Eriophyidae) in Mexico. Plant Dis. 79:186-189.

Cherry rasp leaf virus

Bodine, E.W. and J.H. Newton. 1942. The raspleaf of cherry. Phytopathology 32:333-335.

Hansen, A.J., G. Nyland, F.D. McElroy and R. Stace-Smith. 1974. Origin, cause, host range and spread of cherry rasp leaf disease in North America. Phytopathology 641721-727.

Luepschen, N.S., H.H. Harder, K.G. Rohrbach and K.G. Sisson. 1974. Sweet cherry rasp leaf incidence in Colorado. Plant Dis. Reptr. 58:26-27.

Nemeth, M. 1986. Virus, Mycoplasma and Rickettsia Diseases of Fruit Trees. Martinus Nijhoff Publishers, Dordrecht, The Netherlands.

Nyland, G. 1976. Cherry rasp leaf. Pp. 219-221 *in* Virus Diseases and Non-infectious Disorders of Stone Fruits in North America. (R.M. Gilmer, J.D. Moore, G. Nyland, M.F. Welsh and T.S. Pine, eds.). Agriculture Handbook No. 437. USDA, Washington, D.C., USA.

Parish, CD. 1976. A comparison between the causal agents of cherry rasp leaf and flat apple disease. Acta Hort. 67:199-202.

Uyemoto, J.K. and S.W. Scott. 1992. Important diseases of *Prunus* caused by viruses and other graft-transmissible pathogens in California and South Carolina. Plant Dis. 76:5-11.

Wagnon, H.K., J.A. Taylor, H.E. Williams and AC. Weiner. 1968. Investigations of cherry rasp leaf disease in California. Plant Dis. Reptr. 52:618-622.

Williams, H.E., J.A. Taylor and H.K. Wagnon. 1967. Evidence of the retention of cherry rasp leaf virus in soil in California. Phytopathology 57:103.

Cherry twisted leaf

Desvignes, J.C., R. Boye, D. Cornaggia and N. Grasseau. 1990. Maladies Virus des Arbres Fruitiers (Maladies à virus, à Mycoplasmes et à Viroïdes). Centre Technique Interprofessionel des Fruits et Legumes (CTIFL), Paris, France.

Hansen, A.J. and P.W. Cheney. 1976. Cherry Twisted Leaf. Pp. 222-225 *in* Virus Diseases and Noninfectious Disorders of Stone Fruits in North America (R.M. Gilmer, J.D. Moore, G. Nyland, M.F. Welsh and T.S. Pine, eds.). Agriculture Handbook No. 437. USDA, Washington, DC., USA.

Zhang, Y.P., G.I. Mink, M.G. Tiffany and W.E. Howell. 1992. Isolation of viruses associated with cherry twisted leaf, apricot ring pox, and apricot pit pox diseases and their relationship to apple stem pitting virus. Abstract A803 of the Annu. Meeting Am. Phytopathol. Soc., Portland, OR, USA.

Hop stunt viroid

Hadidi, A., Y. Terai, CA. Powell, SW. Scott, J.C. Desvignes, L.M. Ibrahim and L. Levy. 1992. Enzymatic cDNA amplification of hop stunt viroid variants from naturally infected fruit crops. Acta Hort. 309:339-344.

Sano, T., T. Hataya, Y. Terai and E. Shikata. 1989. Hop stunt viroid strains from dapple fruit disease of plum and peach in Japan. J. Gen. Virol. 70:1311-1319.

Sasaki, M. and E. Shikata. 1977. Studies on the host range of hop stunt disease in Japan. Proc. Jpn. Acad. 53B:103-108.

Terai, Y. 1985. Symptoms and graft transmission of plum dapple fruit disease. Ann. Phytopathol. Soc. Jpn. 51:363.

Terai, Y., T. Sano, T. Hataya, and E. Shikata. 1987. Graft-transmissible relationship of PDFD and SYFD. Ann. Phytopathol. Soc. Jpn. 53:423.

Peach latent mosaic viroid

Ambrós, S., J.C. Desvignes, G. Llácer and R. Flores. 1995. Peach latent mosaic and pear blister canker viroids: detection by molecular hybridization and relationships with specific maladies affecting peach and pear trees. Acta Hort. 386:515-521.

Barba, M., A. Cupidi, S. Loreti, F. Faggioli and L. Martino. 1995. *In vitro* micrografting: a technique to eliminate peach latent mosaic viroid from peach. Acta Hort. 386:531-535.

Desvignes. J.C. 1980. Different symptoms of the peach latent mosaic. Acta Phyto-pathol. Acad. Sci. Hung. 15:183-190.

Desvignes, J.C. 1986. Peach latent mosaic and its relation to peach mosaic and peach yellow mosaic virus diseases. Acta Hort. 193:51-57.

Flores, R., C. Hernández, J.C. Desvignes and G. Llácer. 1990. Some properties of the viroid inducing peach latent mosaic disease. Res. Virol. 141:109-118.

Flores, R., C. Hernández, L. Avinent, A. Hermoso, G. Llácer, J. Juárez, J.M. Arregui, L. Navarro and J.C. Desvignes. 1992. Studies on the detection, transmission and distribution of peach latent mosaic viroid in peach trees. Acta Hort. 309:325-330.

Kishi, K., K. Takanashi and K. Abiko. 1973. New virus diseases of peach, yellow mosaic, oil blotch and star mosaic. Bull. Hort. Res. Sta. Jpn., Ser. A 12:197-208.

Shamloul, A.M., A. Minafra, A. Hadidi, L. Guinchedi, H.E. Waterworth and E.K. Allam. 1995. Peach latent mosaic viroid: nucleotide sequence of an Italian isolate, sensitive detection using RT-PCR and geographic distribution. Acta Hort. 386:522-530.

Smith, I.M., D.G. McNamara, P.R. Scott and K.M. Harris (eds.). 1992. Quarantine pests for Europe. Data sheets on quarantine pests for the European Communities and for the European and Mediterranean Plant Protection Organization. CAB International, Wallingford, UK, and European and Mediterranean Plant Protection Organization, Paris, France.

Plum pox virus

Crescenzi, A., M. Nuzzaci, L. Levy, P. Piazzolla and A. Hadidi. 1995. Plum pox virus (PPV) in sweet cherry. Acta Hort. 386:219-225.

da Câmara Machado A., E. Knapp, H. Pühringer, V. Hanzer, H. Weiss, Q. Wang, H. Katinger and M. Laimer da Câmara Machado. 1995. Progress in pathogen-mediated resistance breeding against plum pox virus. Acta Hort. 386:318-326.

Dosba, F., M. Lansac and J.P. Eyquard. 1994. Résistance des *Prunus* à la sharka. EPPO Bull. 24:691-696.

EPPO. 1974. Progrès réalisés dans la connaissance de la sharka. EPPO Bull. 4:1-125.

Eynard, A., P. Roggero, R. Lenzi, M. Conti and R.G. Milne. 1991. Test for pollen and seed transmission of plum pox virus (sharka) in two apricot cultivars. Adv. Hort. Sci. 5:104-106.

Hadidi, A. and L. Levy. 1994. Accurate identification of plum pox potyvirus and its differentiation from Asian Prunus latent potyvirus in *Prunus* germplasm. EPPO Bull. 24:633-643

Kalashyan, Y.A., N.D. Bilkey, T.D. Verderevskaya and E.V. Rubina. 1994. Plum pox potyvirus on sour cherry in Moldova. EPPO Bull. 24:645-649.

Knapp E., A. da Câmara Machado, H. Pühringer, Q. Wang, V. Hanzer, H. Weiss, B. Weiss, H. Katinger and M. Laimer da Câmara Machado. 1995. Localization of fruit tree viruses by immuno tissue printing in infected shoots of *Malus* and *Prunus* sp. J. Virol. Meth. 55:157-173.

Labonne, G., M. Yvon, J.B. Quiot, L. Avinent and G. Llácer. 1995. Aphids as potential vectors of plum pox virus: comparison of methods of testing and epidemiological consequences. Acta Hort. 386:207-218.

Levy, L. and A. Hadidi. 1994. A simple and rapid method for processing tissue infected with plum pox potyvirus for use with specific 3' non-coding region RT-PCR assays. EPPO Bull. 24:595-604.

Llácer, G., M. Cambra and A. Laviña. 1985. Detection of plum pox virus in Spain. EPPO Bull. 15: 325-329.

Nemchinov, L., A. Hadidi and T. Verderevskaya. 1995. Detection and partial characterization of a plum pox virus isolate from infected sour cherry. Acta Hort. 386:226-236.

Németh, M. 1986. Virus, Mycoplasma and Rickettsia Diseases of Fruit Trees. Martinus Nijhoff Publishers, Dordrecht, The Netherlands.

Németh, M. and M. Kolber. 1983. Additional evidence on seed transmission of plum pox virus in apricot, peach and plum, proved by ELISA. Acta Hort. 130:293-300.

Roy, A.S. and I.M. Smith. 1994. Plum pox situation in Europe. EPPO Bull. 24:515-523.

Smith, I.M., D.G. McNamara, P.R. Scott and K.M. Harris (eds,). 1992. Quarantine pests for Europe. Data sheets on quarantine pests for the European Communities and for the European and Mediterranean Plant Protection Organization. CAB International, Wallingford, UK, and European and Mediterranean Plant Protection Organization, Paris, France.

Triolo, E., M. Ginanni, A. Materazzi and A. Paolucci. 1993. Further evidence of the non-transmission through seed of plum pox virus in apricot. Adv. Hort. Sci. 7:109-110.

Wetzel, T., T. Candresse, G. Macquaire, M. Ravelonandro and J. Dunez. 1992. A highly sensitive immunocapture polymerase chain reaction method for plum pox potyvirus detection. J. Virol. Methods 39:27-37.

Prune dwarf virus

Fulton, R.W. 1981. Ilarviruses. Pp. 377-413 *in* Handbook of Plant Virus Infections and Comparative Diagnosis (E. Kurstak, ed.). Elsevier, Amsterdam, The Netherlands.

Parakh, D.R., A.M. Shamloul, A. Hadidi, SW. Scott, H.E. Waterworth, W.E. Howell and G.I. Mink. 1995. Detection of prune dwarf ilarvirus from infected stone fruits using reverse transcription - polymerase chain reaction. Acta Hort. 386:421-430.

Prunus necrotic ringspot virus

Fulton, R.W. 1981. Ilarviruses. Pp. 377-413 *in* Handbook of Plant Virus Infections and Comparative Diagnosis (E. Kurstak, ed.). Elsevier, Amsterdam, The Netherlands.

Fulton, R.W. 1983. Ilarvirus group. CMI/AAB Descriptions of Plant Viruses No. 275. Commonwealth Agricultural Bureaux, Kew, UK.

Greber, R.S., D.S. Teakle and G.I. Mink. 1992. Thrips-facilitated transmission of prune dwarf and Prunus necrotic ringspot viruses from cherry pollen to cucumber. Plant Dis. 76: 1039-1041.

Howell, W.E. and G.I. Mink. 1988. Natural spread of cherry rugose mosaic disease and two Prunus necrotic ringspot virus biotypes in a central Washington sweet cherry orchard. Plant Dis. 72:636-640.

Juárez, J. J.M. Arregui, E. Camarasa, M. Cambra, G. Llácer, C. Ortega, V. Ortega and L. Navarro. 1988. Recovery of virus-free peach trees from selected clones by shoot-tip grafting *in vitro*. Acta Hort. 235:177-82.

Juárez, J., E. Camarasa, C. Ortega, V. Ortega, J.M. Arregui, M. Cambra, G. Llácer and L. Navarro. 1992. Recovery of virus-free almond plants by shoot-tip grafting *in vitro*. Acta Hort. 309:393-400.

Kelley, R.D. and H.R. Cameron. 1986. Localization of prune dwarf and Prunus necrotic ringspot viruses associated with sweet cherry pollen and seed. Phytopathology 76:317-322.

Knapp E., A. da Câmara Machado, H. Pühringer, Q. Wang, V. Hanzer, H. Weiss, B. Weiss, H. Katinger and M. Laimer da Câmara Machado. 1995. Localization of fruit tree viruses by immuno tissue printing in infected shoots of *Malus* and *Prunus* sp. J. Virol. Meth. 55:157-173.

Nyland, G., R.M. Gilmer and J.D. Moore. 1976. "Prunus" ring spot group. Pp. 104-132 *in* Virus Diseases and Non-infectious Disorders of Stone Fruits in North America (R.M. Gilmer, J.D. Moore, G. Nyland, M.F. Welsh and T.S. Pine, eds.). Agriculture Handbook No. 437, USDA, Washington, DC, USA.

Scott, S.W., V. Bowman-Vance and E.J. Buchman. 1992. The use of nucleic acid probes for the detection of Prunus necrotic ringspot virus and prune dwarf virus. Acta Hort. 309:79-83.

Torrance, L. and C.A. Dolby. 1984. Sampling conditions for reliable routine detection by enzyme-linked immunosorbent assay of three ilarviruses in fruit trees. Ann. Appl. Biol. 104:267-276.

Wells, J.M. and H.C. Kirkpatrick. 1986. Symptomatology and incidence of Prunus necrotic ringspot virus in peach orchards in Georgia. Plant Dis. 70:444-447.

Raspberry ringspot virus

Harrison, B.D. 1964. Specific nematode vectors for serologically distinct forms of raspberry ringspot and tomato black ring viruses. Virology 22:544-550.

Murant, A.F., M.A. Mayo, B.D. Harrison and R.A. Goold. 1972. Properties of virus and RNA components of raspberry ringspot virus. J. Gen. Virol. 16:327-338.

Murant, A.F. 1978. Raspberry Ringspot Virus. CMI/AAB Descriptions of Plant Viruses No. 198. Commonwealth Agricultural Bureaux, Kew, UK.

Németh, M. 1986. Virus, Mycoplasma and Rickettsia Diseases of Fruit Trees. Martinus Nijhoff Publishers, Dordrecht, The Netherlands.

Taylor, C.E. and A.F. Murant. 1969. Transmission of strains of raspberry ringspot and tomato black ring viruses by *Longidorus elongatus* (de Man). Ann. Appl. Biol. 64: 43-48.

Strawberry latent ringspot virus

Allen, W.R., T.R. Davidson and M.R. Bristol. 1970. Properties of a strain of strawberry latent ringspot virus isolated from sweet cherry growing in Ontario. Phytopathology 60:1262-1265.

Belli, G., A. Fortusini and P.A. Bianco. 1986. Etiology of peach willow leaf rosette. Acta Hort. 193:63-65.

Blattny, C. and M. Janeckova. 1980. Apricot bare twig and unfruitfulness. Acta Hort. 94:383-390.

Corte, A. 1968. Soilborne viruses associated with a peach disease occurring in northern Italy. Tagungsber. Deutsche Akad. Landwirtschaftswiss. DDR, Berlin 97:187-194.

Francki, R.I.B., R.G. Milne and T. Hatta. 1985. Atlas of Plant Viruses, Vol. II. CRC Press Rota Raton, Florida, USA.

Fry, P.R. and G.A. Wood. 1973. Further viruses of *Prunus* in New Zealand. N. Z. J. Agric. Res. 16:131-142.

Hanson, C.M. and R.N. Campbell. 1979. Strawberry latent ringspot virus from 'Plain' parsley in California. Plant Dis. Reptr. 63:142-146.

Murrant, A.F. 1981. Nepoviruses. Pp. 197-238 *in* Handbook of Plant Virus Infections and Comparative Diagnosis (E. Kurstak, ed.). Elsevier, Amsterdam, The Netherlands.

Nemeth, M. 1986. Virus, Mycoplasma and Rickettsia Diseases of Fruit Trees. Martinus Nijhoff Publishers, Dordrecht, The Netherlands.

Ragozzino, A. and D. Alioto. 1992. Cherry rasp leaf etiology in Campania. Acta Hort. 309:115-118.

Scotto La Massese, C., C. Marenaud and J. Dunez. 1973. Analyse d'un phénomène de dégénérescence du pêcher dans la vallée de 1'Eyrieux. C. R. Seances Acad. Agric. Fr. 59:327-339.

Tobacco ringspot virus

Stace-Smith, R. 1985. Tobacco ringspot virus. AAB Descriptions of Plant Viruses No. 309. Association of Applied Biologists, Wellesbourne, Warwick, UK.

Stace-Smith, R. and A.J. Hansen. 1974. Occurrence of tobacco ringspot virus in sweet cherry. Can. J. Bot. 52:1647-1651.

Uyemoto, J.K., M.F. Welsh and E. Williams. 1977. Pathogenicity of tobacco ringspot virus in cherry. Phytopathology 67:439-441.

Tomato ringspot virus

Auger, J. 1988. Tomato ringspot virus (Tom-RSV) associated with brownline disease of prune trees in Chile. Acta Hort. 235:197-204.

Bitterlin, M.W. and D. Gonsalves. 1986. Serological and sampling techniques for detecting tomato ringspot virus in peach trees. Acta Hort. 193:291-296.

Cummins, J.N. and D. Gonsalves. 1986. Constriction and decline of 'Stanley' prune associated with tomato ringspot virus. J. Am. Soc. Hort. Sci. 111:315-318.

Hadidi, A. and R.W. Hammond. 1988. Construction of molecular clones for identification and detection of tomato ringspot and arabis mosaic viruses. Acta Hort. 235:223-230.

Hadidi, A. and CA. Powell. 1991. Complementary DNA cloning and analysis od RNAs of a Prunus stem-pitting isolate of tomato ringspot virus. Mol. Cell. Probes 5:337-344.

Hoy, J.W., SM. Mircetich and B.F. Lownsberry. 1984. Differential transmission of Prunus tomato ringspot virus by *Xiphinema californicum*. Phytopathology 74:332-335.

Mircetich, SM. and H.W. Fogle. 1976. Peach stem pitting. Pp. 77-87 *in* Virus Diseases and Non-infectious Disorders of Stone Fruits in North America (R.M. Gilmer, J.D. Moore, G. Nyland, M.F. Welsh and T.S. Pine, eds.). Agriculture Handbook No. 437, USDA, Washington, DC, USA.

Powell, CA., A. Hadidi and J.M. Halbrandt. 1991. Detection and distribution of tomato ringspot virus in infected nectarine trees using ELISA and transscribed RNA probes. Hort. Sci. 26:1290-1292.

Schlocker, A. and J.A. Taylor. 1976. Yellow bud mosaic. Pp. 156-165. *in* Virus Diseases and Non-infectious Disorders of Stone Fruits in North America (R.M. Gilmer, J.D. Moore, G. Nyland, M.F. Welsh and T.S. Pine, eds.). Agriculture Handbook No. 437, USDA, Washington, DC, USA Stace-Smith, R. 1984. Tomato ringspot virus. CMI/AAB Descriptions of Plant Viruses No. 290. Commonwealth Agricultural Bureaux, Kew, UK.

Stace-Smith, R. and D. Ramsdell. 1987. Nepoviruses of the Americas. Pp. 131-166 *in* Current Topics in Vector Research, Vol. 3 (K.F. Harris, ed.). Springer-Verlag, New York, USA.

Viral twig necrosis of cherry (PeAMV, CIRSV)

Blattny, C. 1962. Detrimental canker, a virus disease of cherry [in Czech, with English summary]. Rostl. Vyroba 8:577-588.

Grüntzig, M., E. Fuchs, J. Kraková, H. Kegler, H. Kleinhempel and J. Richter. 1989. Zum Erreger der Virösen Zweignekrose an Süß-und Vogelkirschen (cherry detrimental canker) in der DDR Arch. Phytopathol. Pflanzenschutz 25:203-207.

Hollings, M. and O.M. Stone. 1975. Serological and immunoelectrophoretic relationships among viruses in the tombusvirus group. Ann. Appl. Biol. 80:37-48.

Kleinhempel, H., H. Kegler, H.-H. Schimanski and H.B. Schmidt. 1971. Charakterisierung eines Virus aus Kern- und Steinobstarten. Zentralbl. Bakt. II Abt. 126:659-667.

Koenig, R. and L. Kunze. 1982. Identification of tombusvirus isolates from cherry in southern Germany as Petunia asteroid mosaic virus. Phytopathol. Z. 103:361-368.

Lesemann, D-E., L. Kunze, G. Krischke and R. Koenig. 1989. Natural occurrence of carnation Italian ringspot virus in a cherry tree. J. Phytopathol. 124:171-174.

Martelli, G.P., D. Gallitelli and M. Russo. 1988. Tombusviruses. Pp. 13-72 *in* The Plant Viruses, Vol. 3. PolyhedralVirions with Monopartite RNA Genomes (R. Koenig, ed.). Plenum Press, New York and London.

Martelli, G.P., M. Russo and D. Gallitelli. 1989. Tombusvirus group. AAB Descriptions of Plant Viruses No. 352. Association of Applied Biologists, Wellesbourne, Warwick, UK.

Novák, J.B. and J. Lanzová. 1980. Some diseases of fruit trees in which the tomato bushy stunt virus occurs and new natural hosts of this virus. Acta Phytopathol. Acad. Sci. Hung. 15:323-327.

Pfeilstetter, E. 1992. Untersuchungen zur virösen Zweignekrose an Süsskirschen Oberfrankens: Vorkommen, Ausbreitung und Möglichkeiten. des Nachweises der Erreger Petunia asteroid mosaic virus (PAMV) und carnation Italian ringspot virus (CIRV). Dissertation, Technische Universität München.

Pfeilstetter, E., V. Zinkernagel and L. Kunze. 1992. Occurrence of petunia asteroid mosaic (PAMV) and carnation Italian ringspot (CIRV) viruses in cherry orchards in northern Bavaria. Acta Hort. 309:345-353.

Pfeilstetter, E., L. Kunze and V. Zinkernagel. 1994. Some epidemiological aspects of the viral twig necrosis in northern Bavaria. Transmission with scions and seeds, new host plants and virus detection in soil. In preparation.

Pfeilstetter, E., L. Kunze and V. Zinkernagel. 1996. Viral twig necrosis of sweet cherry. Modes of transmission and spread of Petunia asteroid mosaic virus (PeAMV). Ann. Appl. Biol. (in press).

Richter, J., H. Kleinhempel, H.-H. Schimanski and H. Kegler. 1977. Nachweis des Tomatenzwergbusch-Virus (tomato bushy stunt virus) in Obstgehölzen. Arch. Phytopathol. Pflanzenschutz. 13:367-368.

Other European nepoviruses (tomato black ring, arabis mosaic, myrobalan latent ringspot)

Bercks, R. 1963. Serological cross-reactions between isolates of the tomato black ringspot virus. Phytopathol. Z. 6:97-100.

Bretout, C., T. Candresse, O. Le Gall, V. Brault, M. Revelonandro and J. Dunez. 1988. Virus and RNA - specific molecular hybridization probes for two nepoviruses. Acta Hort. 235:231-235.

Cropley, R. 1964. Further studies of Euro-pean rasp leaf and leaf roll diseases of cherry trees. Ann. Appl. Biol. 53:333-341.

Dunez, J. and Delbos, R. 1976. Myrobalan latent ring spot, a bipartite genome virus and strain of toma-to black ring virus. Mitt. Biol. Bundesanst. Land Fortwirtsch. Berlin-Dahlem. 170:9-16.

Dunez, J. and G. DuPont 1976. Myrobalan latent ringspot virus. CMI/AAB Descrip-tions of Plant Viruses No. 160. Common-wealth Agricultural Bureaux, Kew, UK.

Francki, R.I.B., R.G. Milne and T. Hatta. 1985. Atlas of plant viruses, Vol. II. CRC Press, Boca Raton, FL, USA.

Fritsch, C., I. Koenig, A.F. Murant, J.H. Raschke and M.A. Mayo. 1984. Comparison among satellite RNA species from five isolates of tomato black ring virus and one isolate of myrobalan latent ringspot virus. J. Gen. Virol. 65:289-294.

Gallitelli, D., I?. Piazzolla, V. Savino, A. Quacquarelli and G.P. Martelli. 1981. A comparison of myrobalan latent ringspot virus with other nepoviruses. J. Gen. Virol. 53:57-65.

Hadidi, A. and R.W. Hammond. 1988. Construction of molecular clones for identification and detection of tomato ringspot and arabis mosaic viruses. Acta Hort. 253:223.

Murant, A.F. 1981. Nepoviruses. Pp. 197-238 *in* Handbook of Plant Virus Infections and Comparative Diagnosis (E. Kurstak, ed.). Elsevier, Amsterdam, The Netherlands.

Murant, A.F. 1987a. Raspberry yellow dwarf and associated diseases of *Rubus* caused by arabis mosaic and strawberry latent ringspot viruses. Pp. 204-211 *in* Virus Diseases of Small Fruits (R.H. Converse, ed.). Agriculture Handbook No. 631, USDA, Washington, DC, USA.

Murant, A.F. 198713. Raspberry ringspot and associated diseases of *Rubus* caused by raspberry ringspot and tomato black ring viruses. Pp. 211-220 *in* Virus Diseases of Small Fruits (R.H. Converse, ed.). Agriculture Handbook No. 631, USDA, Washington, DC, USA.

Németh, M. 1986. Virus, Mycoplasma and Rickettsia Diseases of Fruit Trees. Martinus Nijhoff Publishers, Dordrecht, The Netherlands.

DISEASES OF UNKNOWN ETIOLOGY

Cherry necrotic rusty mottle

Cameron, H.R. and D.L. Moore. 1985. Reduction in spread of necrotic rusty mottle with removal of affected trees. Phytopathology 75:1311 (Abstr.).

Németh, M. 1986. Virus, Mycoplasma and Rickettsia Diseases of Fruit Trees. Martinus Nijhoff Publishers, Dordrecht, The Netherlands.

Posnette, A.F., and R. Cropley. 1964. Necrotic rusty mottle disease of sweet cherry in Britain. Plant Pathol. 13:1-3.

Posnette, A.F., R. Cropley and A.A.J. Swait. 1968. The incidence of virus diseases in English sweet cherry orchards and their effect on yield. Ann. Appl. Biol. 61:351-360.

Wadley, B.N. 1966. Variants of necrotic rusty mottle virus in Utah orchards. Utah State Hort. Soc. Proc. (1966):73-78.

Wadley, B.N. and G. Nyland. 1976. Rusty mottle group. Pp. 242-249 *in* Virus Diseases and Noninfectious Disorders of Stone Fruits in North America (R.M. Gilmer, J.D. Moore, G. Nyland, M.F. Welsh and T.S. Pine, eds.). Agriculture Handbook No. 437, USDA, Washington, DC, USA.

Cherry rusty mottle (American)

Németh, M. 1986. Virus, Mycoplasma and Rickettsia Diseases of Fruit Trees. Martinus Nijhoff Publishers, Dordrecht, The Netherlands.

Reeves, E.L. 1940. Rusty mottle, a new virosis of cherry. Phytopathology 30:789

Reeves, E.L. 1951. Rusty mottle. Pp. 112-115 *in* Virus Diseases and Other Disorders with Viruslike Symptoms of Stone Fruits in North America. Agriculture Handbook No. 10, USDA, Washington, DC, USA.

Wadley, B.N. and G. Nyland. 1976. Rusty mottle group. Pp. 242-249 *in* Virus Diseases and Noninfectious Disorders of Stone Fruits in North America (R.M. Gilmer, J.D. Moore, G. Nyland, M.F. Welsh and T.S. Pine, eds.). Agriculture Handbook No. 437, USDA, Washington, DC, USA.

Zeller, S.M. and J.A. Milbrath. 1947. Mild rusty mottle of sweet cherry *(Prunus avium)*. Phytopathology 37177-84.

Cherry rusty mottle (European)

Németh, M. 1986. Virus, Mycoplasma and Rickettsia Diseases of Fruit Trees. Martinus Nijhoff Publishers, Dordrecht, The Netherlands.

Posnette, A.F. 1951. Virus diseases of sweet cherry. Ann. Rep. East Malling Res. Sta. 1950. A:209-210.

Posnette, A.F. and R. Cropley. 1956. Virus diseases of cherry trees in England. II. The suppression of growth caused by some viruses. J. Hort. Sci. 31:298-302.

Posnette, A.F. and R. Cropley. 1960. Virus diseases of cherry trees in England. III. Crop reduction caused by some viruses. Ann. Rep. East Malling Res. Sta. 1959. A:92-95.

Posnette, A.F., R. Cropley and A.A.J. Swait. 1968. The incidence of virus diseases in English sweet cherry orchards and their effect on yield. Ann. Appl. Biol. 61:351-360.

DISEASES CAUSED BY PHYTOPLASMAS FORMERLY MYCOPLASMA-LIKE ORGANISMS, MLO)

Cherry lethal yellows

Lee, I.-M., S. Zhu, D.E. Gundersen, C. Zhang and A. Hadidi. 1995. Detection and identification of a new phytoplasma associated with cherry lethal yellows in China (abstract). Phytopathology 85:1179.

Zhu, S. and X. Shu. 1992. A mycoplasmalike organism on cherry [Chinese with English abstract]. Acta Phytopath. Sinica 22:25-28.

European stone fruit yellows

Ahrens, U., K.-H. Lorenz and E. Seemüller. 1993. Genetic diversity among mycoplasmalike organisms associated with stone fruit diseases. Mol. Plant-Micro. Interact. 6:686-691.

Dosba, F., M. Lansac, K. Mazy, M. Garnier and J.P. Eyquard. 1991. Incidence of different diseases associated with mycoplasma-like organisms in different species of *Prunus*. Acta. Hortic. 283:311-320.

Giunchedi, L., F. Marani and R. Credi. 1978. Mycoplasma-like bodies associated with plum decline (leptonecrosis). Phytopathol. Medit. 17:205-209.

Giunchedi, L., C. Poggi-Pollini and R. Credi. 1982. Susceptibility of stone fruit trees to the Japanese plum-tree decline causal agent. Acta Hort. 130:285-290.

Lederer, W. and E. Seemüller. 1992. Demon-stration of mycoplasmas in *Prunus* species in Germany. J. Phytopathol. 134:89-96.

Lorenz, K.H., F. Dosba, C. Poggi-Pollini, G. Llácer and E. Seemüller. 1994. Phytoplasma diseases of *Prunus* species in Europe are caused by genetically similar organisms. Z. Pflanzenkrankr. Pflanzenschutz. 101:567-575.

Morvan, G. 1977. Apricot chlorotic leaf roll. EPPO Bull. 7:37-55.

Poggi-Pollini, C., R. Bissani, L. Guinchedi and E. Vindimian. 1995. Occurrence of phytoplasma infection in European plums *(Prunus domestica)*. J. Phytopathol. (in press).

Rumbos, I.C. and A.M. Bosabalidis. 1985. Mycoplasmalike organisms associated with declined plum trees in Greece. Z. Pflanzenkrankr. Pflanzenschutz. 92:47-54.

Sanchez-Capuchino, J.A., G. Llácer, R. Casanova, J.B. Forner and R. Bono. 1976. Epidemiological studies on fruit tree mycoplasma diseases in the eastern region of Spain. Acta Hort. 67:129-136.

Seemüller, E. 1976. Investigations to demonstrate mycoplasmalike organisms in diseased plants by fluorescence microscopy. Acta Hortic. 67:109-111.

Seemüller, E., B. Schneider, R. Mäurer, U. Ahrens, X. Daire, H. Kison, K.-H. Lorenz, G. Firrao, L. Avinent, B.B. Sears and E. Stackebrandt. 1994. Phylogenetic classification of phytopathogenic mollicutes by sequence analysis of 16S ribosomal DNA. Int. J. Syst. Bacterial. 44:440-446.

Peach rosette

KenKnight, G. 1976. Peach rosette. Pp. 73-76 *in* Virus Diseases and Noninfectious Disorders of Stone Fruits in North America (R.M. Gilmer, J.D. Moore, G. Nyland, M.F. Welsh and T.S. Pine, eds.). Agriculture Handbook No. 437. USDA, Washington, DC, USA.

Kirkpatrick, H. C., S.H. Lowe and G. Nyland. 1975. Peach rosette: the morphology of an associated mycoplasmalike organism and the chemotherapy of the disease. Phytopathology 65:864-870.

Peach X disease

Granett, A.L. and R.M. Gilmer. 1971. Mycoplasmas associated with X-disease in various *Prunus* species. Phytopathology 61:1036-1037,

MacBeath, J.H., G. Nyland and A.R. Spurr. 1972. Morphology of mycoplasmalike bodies associated with peach X-disease in *Prunus persica*. Phytopathology 62:935-937.

Nasu, S., D.D. Jensen and J. Richardson. 1970. Electron microscopy of mycoplasma-like bodies associated with insect and plant hosts of peach western X-disease. Virology 41:583-595.

Rosenberger, D.A. and A.L. Jones. 1978. Leafhopper vectors of the peach X-disease pathogen and its seasonal transmission from chokecherry *(Prunus virginiana)*. Phytopathology 68:782-790.

Gilmer, R.M. and E.C. Blodgett. 1976. X disease. Pp. 145-155 *in* Virus Diseases and Non-infectious Disorders of Stone Fruits in North America (R.M. Gilmer, J.D. Moore, G. Nyland, M.F. Welsh and T.S. Pine, eds.). Agriculture Handbook No. 437, USDA, Washington, DC, USA.

Peach yellows

Jones, A.L., G.R. Hooper and D. A. Rosenberger. 1974a. Association of mycoplasmalike bodies with little peach and X-disease. Phytopathology 64:755-756.

Jones, A.L., G.R. Hooper, D.A. Rosenberger and J. Chevalier. 1974b. Mycoplasma-like bodies associated with peach and periwinkle *(Vinca rosea)* exhibiting symptoms of peach yellows. Phytopathology 64:1154-1156.

Pine, T.S. and R.M. Gilmer. 1977. Peach yellows. Pp. 91-95 *in* Virus Diseases and Noninfectious Disorders of Stone Fruits in North America (R.M. Gilmer, J.D. Moore, G. Nyland, M.F. Welsh and T.S. Pine, eds.). Agriculture Handbook No. 437. USDA, Washington, DC, USA.

BACTERIAL DISEASES

Bacterial canker

CMI. 1980. Distribution Maps of Plant Diseases. Map No. 132, edition *3. Pseudomonas mors-prunorum* Wormald. CAB International, Wallingford, UK.

Dhanvantari, B.N. 1969. Occurrence of bacterial canker of sweet cherry and plum in Ontario. Can. Plant Dis. Surv. 49:5-7.

Hattingh, M.J., I.M.M. Roos, and E.L. Mansvelt. 1989. Infection and systemic invasion of deciduous fruit trees by *Pseudomonas syringae* in South Africa. Plant Dis. 73:784-789.

Latorre, B.A. and A.L. Jones. 1979. *Pseudomonas mar-sprunorum*, the cause of bacterial canker of sour cherry in Michigan, and its epiphytic association with *P. syringae*. Phytopathology 69:335-339.

Louws, F.L., D.W. Fulbright, CT. Stephens and F. J. de Bruijn. 1994. Specific genomic fingerprints of phytopathogenic *Xanthomonas* and *Pseudomonas* pathovars and strains generated with repetitive sequences and PCR. Appl. Environ. Microbial. 60:2286-2295.

Paterson, J.M. and A.L. Jones. 1991. Detection of *Pseudomonas syringae* pv. *morsprunorum* on cherries in Michigan with a DNA hybridization probe. Plant Dis. 75:893-896.

Roos, I.M.M. and M.J. Hattingh. 1987. Pathogenicity and numerical analysis of phenotypic features of *Pseudomonas syringae* strains isolated from deciduous fruit trees. Phytopathology 77:900-908.

Bacterial canker of almond

CMI. 1978. Distribution Maps of Plant Diseases. Map No. 525. *Pseudomonas amygdali* Psallidas & Panagopoulos. CAB International, Wallingford, UK.

Psallidas, P.G. and C.G. Panagopoulos. 1975. A new bacteriosis of almond caused by *Pseudomonas amygdali* sp. nov. Ann. Inst. Phytopathol. Benaki 11:94-108.

Smith, I.M., J. Dunez, R.A. Lelliot, D.H. Philips and S.A. Archer (eds.). 1988. European Handbook of Plant Diseases. Blackwell Scientific Publications, Oxford, UK.

Bacterial dieback of peach

Gardan, L., J.P. Prunier and J. Luisetti. 1972. Etudes sur les bactérioses des arbres fruitiers. IV. Recherche et étude des variations de *Pseudomonas mors-prunorum* f.sp. *persicae* à la surface des feuilles de pêcher. Ann. Phytopathol. 4:229-244.

Luisetti, J., J.P Prunier and L. Gardan. 1972. A medium for the proof of the production of a fluorescent pigment by *Pseudomonas mors-prunorum* f.sp. *persicae*. Ann. Phytopathol. 4(3):295-296.

Vigouroux, A., J.F. Berger and C. Bussi. 1987. La sensibilité du pêcher au dépérissement bactérien en France: incidence de certaines caractéristiques du sol et de l'irrigation. Relations avec la nutrition. Agronomie 7:483-495.

Vigouroux, A. and M. Blache. 1967. Un nouveau dépérissement de pêcher dans 1'Ardèche. Phytoma 192:34-45.

Young, J.M. 1988. *Pseudomonas syringae* pv. *persicae* from nectarine, peach and Japanese plum in New Zealand. EPPO Bull. 18:141-151.

Bacterial leaf spot

CMI. 1987. Distribution Maps of Plant Diseases. Map No. 340, edition 4. *Xanthomonas pruni* (E.F. Smith) Dowson. CAB International, Wallingford, UK.

Gitaitis, R.D., J.D. Hamm and P.F. Bertrand. 1988. Differentiation of *Xanthomonas campestris* pv. *pruni* from other yellow-pigmented bacteria by the refractive quality of bacterial colonies on an agar medium. Plant Dis. 72:416-417.

Goodman, C.A. and M.J. Hattingh. 1988. Differentiation of *Xanthomonas campestris* pv. *pruni* in plum nursery trees. Plant Dis. 72:643.

Hayward, A.C. and J.M. Waterson. 1965. *Xanthomonas pruni*. CMI Descriptions of Pathogenic Fungi and Bacteria No. 50. CAB International, Wallingford, UK.

Randhawa, P.S. and E.L. Civerolo. 1985. A detached-leaf bioassay for *Xanthomonas campestris* pv. *pruni*. Phytopathology 75:1060-1063.

Shepard, D.P. and E.I. Zehr. 1994. Epiphytic persistence of *Xanthomonas campestris* pv. *pruni* on peach and plum. Plant Dis. 78:627-629.

Crown gall

Anderson, A.R. and L.W. Moore. 1979. Host specificity in the genus *Agrobacterium*, cause of crown gall tumors or hairy root of plants. Phytopathology 69:320-323.

Brisbane, P.F. and A. Kerr. 1983. Selective media for three biovars of *Agrobacteri-um*. J. Appl. Bacterial. 54:425-431.

Dong, L.C., C.W. Sun, K.L. Thies, D.S. Luthe and C.H. Graves. 1992. Use of polymerase chain reaction to detect pathogenic strains of *Agrobacterium*. Phytopathology 82:434-439.

Kado, C.I. and S.T. Liu. 1981. Rapid procedure for detection and isolation of large and small plasmids. J. Bacterial. 145:1365-1373.

Smith, I.M., J. Dunez, R.A. Lelliot, D.H. Philips and S.A. Archer (eds.). 1988. European Handbook of Plant Diseases. Blackwell Scientific Publications, Oxford, UK.

Phony peach, plum leaf scald, almond leaf scorch

French, W.J., R.G. Christie and D.L. Stassi. 1977. Recovery of rickettsialike bacteria by vacuum infiltration of peach tissues affected with phony disease. Phytopathology 67:945-948.

Goheen, AC., G. Nyland and S.K. Lowe. 1973. Association of a rickettsialike organism with Pierce's disease of grapevines and alfalfa dwarf and heat therapy of the disease in grapevines. Phytopathology 63(3):341-345.

Hutchins, L.M. and J.L. Rue. 1939. Promising results of heat treatment for inactivation of phony disease virus in dormant peach nursery trees. Phytopathology 29:12 (Abstr.).

Jindal, K. K. and R. C. Sharma. 1987. Almond leaf scorch, a new disease from India. FAO Plant Prot. Bull. 35:64-65.

Minsavage, G.V., C.M. Thompson, D.L. Hopkins, R.M.V.B.C. Leite and R.E. Stall. 1994. Development of a polymerase chain reaction protocol for detection of *Xylella fastidiosa* in plant tissue. Phytopathology 84:456-461.

Mircetich, S.M., S.K. Lowe, W.J. Moller and G. Nyland. 1976. Etiology of almond leaf scorch disease and transmission of the causal agent. Phytopathology 66:17-24.

Raju, B.C., J.M. Wells, G. Nyland, R.H. Brlansky and S.K. Lowe. 1982. Plum leaf scald. Isolation, culture, and pathogenicity of the causal agent. Phytopathology 72:1460-1466.

Smith, I.M., D.G. McNamara, P.R. Scott and K.M. Harris (eds.) 1992. Quarantine pests for Europe. Data sheets on quarantine pests for the European Communities and for the European and Mediterranean Plant Protection Organization. CAB International, Wallingford, UK, and European and Mediterranean Plant Protection Organization, Paris, France.

Wells, J.M., B.C. Raju, H.-Y. Hung, W.G. Weisburg, L. Mandelco-Paul and D.J. Brenner. 1987. *Xylella fastidiosa* gen. nov., sp. nov. Gram negative, xylem-limited, fastidious plant bacteria related to *Xanthomonas* spp. Int. J. System. Bacterial. 37:136-143.

FUNGAL DISEASES

Black knot

CMI. 1994. Distribution Maps of Plant Diseases. Map No. 48, edition 3. *Dibotryon morbosum* (Schwein.) Theiss. & Syd. CAB International, Wallingford, UK.

Gourley, C.O. 1962. A comparison of growth, life cycle and control of *Dibotryon morbosum* (Sch.) Th. & Syd. on peach and plum in Nova Scotia. Can. J. Plant Sci. 42:122-129.

Koch, L.W. 1935. Investigations on black knot of plums and cherries. III. Symptomatology, life history, and cultural studies of *Dibotryon morbosum* (Sch.) T. & S. Sci. Agric. 15:80-95.

Smith, D.H., F.H. Lewis and S.H. Wainwright. 1970. Epidemiology of the black knot disease of plums. Phytopathology 60:1441-1444.

Smith, I.M., D.G. McNamara, P.R. Scott and K.M. Harris (eds.) 1992. Quarantine pests for Europe. Data sheets on quarantine pests for the European Communities and for the European and Mediterranean Plant Protection Organization. CAB International, Wallingford, UK, and European and Mediterranean Plant Protection Organization, Paris, France.

Sutton, B.C. and J.M. Waterston. 1970. *Dibotryon morbosum.* CMI Descriptions of Pathogenic Fungi and Bacteria, No. 224. CAB International, Wallingford, UK.

Wainwright, S.H. and F.H. Lewis. 1970. Developmental morphology of the black knot pathogen on plum. Phytopathology 60:1238-1244.

Brown rot of stone fruits

Byrde, R. J.W. and H.J. Willetts. 1977. The brown rot fungi of fruit: their biology and control. Pergamon Press Ltd., Oxford, UK.

Mordue, J.E.M. 1979. *Sclerotinia fructicola.* CMI Descriptions of Pathogenic Fungi and Bacteria, No. 616. CAB International, Wallingford, UK.

Penrose, L. J., J. Tarran and A.L. Wong. 1976. First record of *Sclerotinia laxa* Aderh. & Ruhl. in New South Wales: differentiation from *Sclerotinia fructicola* (Wint.) Rehm by cultural characteristics and electrophoresis. Aust. J. Agric. Res. 27:547-556.

Sonoda, R.M., J.M. Ogawa and B.T. Manji. 1982. Use of interactions of cultures to distinguish *Monilinia laxa* from *M. fructicola.* Plant Dis. 66:325-326.

Eutypa dieback

Carter, M.V. 1995. Eutypa dieback. Pp. 32-33 *in* Compendium of Stone Fruit Diseases (J.M. Ogawa, E.I. Zehr, G.W. Bird, D.F. Ritchie, K. Uriu and J.K. Uyemoto, eds.). APS Press, St. Paul, MN.

Carter, M.V., A. Bolay and F. Rappaz. 1983. An annotated host list and bibliography of *Eutypa armeniacae.* Rev. Plant Pathol. 62:251-258.

CMI. 1982. Distribution Maps of Plant Diseases. Map No. 385, edition *3. Eutypa armeniacae* Hansf. & Carter. CAB International, Wallingford, UK.

Munkvold, G.P. and J.J. Marois. 1994. Eutypa dieback of sweet cherry and occurrence of *Eutypa lata* perithecia in the Central Valley of California. Plant Dis. 78:200-207.

Smith, I.M., J. Dunez, R.A. Lelliot, D.H. Philips and S.A. Archer (eds.). 1988. European Handbook of Plant Diseases. Blackwell Scientific Publications, Oxford, UK.

Fusicoccum canker (blight of almond and peach, constriction disease)

Atkinson, J.D. 1971. *Fusicoccum amygdali* Delacr. P. 262 in Diseases of Tree Fruits in New Zealand. New Zealand Department of Scientific and Industrial Research Information Series No. 81. A.R. Shearer, Government Printer, Wellington, New Zealand.

Grosclaude, C. 1956. Recherches sur le chancre du pêcher causé par *Fusicoccum amygdali* Delacr. Ann. Inst. Rech. Agron. Ser. C. (Ann. Epiphyt.) 7:397-419.

Guba, E.F. 1953. Large leaf spot and canker of peach caused by the fungus *Fusicoccum amygdali* Delacr, Plant Dis. Reptr. 37:560-564.

Tuset, J.J. and M.T. Portilla. 1989. Taxonomic status of *Fusicoccum amygdali* and *Phomopsis amygdalina.* Can. J. Bot. 67:1275-1280.

Zehr, E.I. 1995. Constriction canker. Pp. 31-32 *in* Compendium of Stone Fruit Diseases (J.M. Ogawa, E.I. Zehr, G.W. Bird, D.F. Ritchie, K. Uriu and J.K. Uyemoto, eds.). APS Press, St. Paul, MN.

Leaf scorch of apricot and cherry

Monod, M. 1983. Monographie taxonomique des Gnomoniaceae. Sydowia Beih. 9:1-315.

Smith, I.M., J. Dunez, R.A. Lelliot, D.H. Philips and S.A. Archer (eds.). 1988. European Handbook of Plant Diseases. Blackwell Scientific Publications, Oxford, UK.

Vukovits, G. and W. Wittmann. 1990. Identification, biology and control of the fungus *Apiognomonia erythrostoma* (Pers.) v. Höhnel; causing the leaf scorch (leaf browning) in apricots [in German]. Pflanzenschutzberichte51:78-90.

Zehr, E.I. 1995. Gnomoniosis. P. 24 *in* Compendium of Stone Fruit Diseases. (J.M. Ogawa, E.I. Zehr, G.W. Bird, D.F. Ritchie, K Uriuand J.K. Uyemoto, eds.). APS Press, St. Paul, MN.

Leucostoma canker

Atkinson, J.D. 1971. Valsa injury. Pp. 255-257 *in* Diseases of Tree Fruits in New Zealand. New Zealand Department of Scientific and Industrial Research Information Series No. 81. A.R. Shearer, Government Printer, Wellington, New Zealand.

Biggs, A.R. 1995. Leucostoma canker. Pp. 28-30 *in* Compendium of Stone Fruit Diseases (J.M. Ogawa, E.I. Zehr, G.W. Bird, D.F. Ritchie, K. Uriu and J.K. Uyemoto, eds.). APS Press, St. Paul, MN.

Surve-Iyer, R.S., G.C. Adams, A.F. Iezzoni and A.L. Jones. 1995. Isozyme detection and variation in *Leucostoma* species from *Prunus* and *Malus.* Mycologia 87:471-482.

Peach leaf curl and related diseases

Booth, C. 1981. *Taphrina deformans.* CMI Descriptions of Pathogenic Fungi and Bacteria, No. 711. CAB International, Wallingford, UK.

Booth, *C.* 1981. *Taphrina wiesneri.* CM1 Descriptions of Pathogenic Fungi and Bacteria, No. 712. CAB International, Wallingford, UK.

Booth, C. 1981. *Taphrina pruni*. CMI Descriptions of Pathogenic Fungi and Bacteria, No. 713. CAB International, Wallingford, UK.

Farr, D.F., G.F. Bills, G.P. Chamuris and A.Y. Rossman. 1989. *Taphrina* Fr. Hemiascomycetes, Taphrinales. Pp. 986-987 *in* Fungi on Plants and Plant Products in the United States. The American Phytopathological Society, St. Paul, MN.

Peach scab (freckle)

CMI. 1979. Distribution Maps of Plant Diseases. Map *No.* 198. *Venturia carpophila* Fisher. CAB International, Wallingford, UK.

Gottwald, T.R. 1983. Factors affecting spore liberation by *Cladosporium carpophilum.* Phytopathology 73:1500-1505.

Lawrence, E.G., Jr. and E.I. Zehr. 1982. Environmental effects on the development and dissemination of *Cladosporium carpophilum* on peach. Phytopathology 72:773-776.

Raymondaud, H., R. Pineau and M. Martin. 1985. Contribution à la connaissance de la biologie et de l'épidémiologie de *Cladosporium carpophilum* agent de la tavelure du mirabellier en Lorraine. Agronomie 5:563.

Sivanesan, A. 1974. *Venturia carpophila*. CMI Descriptions of Pathogenic Fungi and Bacteria, No. 402. CAB International, Wallingford, UK.

Powdery Mildew

Boeswinkel, H.J. 1979. Differences between the conidial states of *Podosphaera clandestina* and *Sphaerotheca pannosa*. Ann. Phytopathol. 11:525-527.

Grove, G.G. 1995. Powdery mildew. Pp. 12-15 *in* Compendium of Stone Fruit Diseases (J.M. Ogawa, E.I. Zehr, G.W. Bird, D.F. Ritchie, K. Uriu and J.K. Uyemoto, eds.). APS Press, St. Paul, MN.

Kable, P.F, P.M. Fried and D.R. MacKenzie. 1980. The spread of a powdery mildew *Oidium* sp. of peach. Phytopathology 70:601-604.

Mukerji, K.G. 1968. *Sphaerotheca pannosa*. CMI Descriptions of Pathogenic Fungi and Bacteria, No. 189. CAB International, Wallingford, UK.

Weinhold, A.R. 1961. The orchard development of peach powdery mildew. Phytopathology 51:478-481.

Weinhold, A.R. 1961. Temperature and moisture requirements for germination of conidia of *Sphaerotheca pannosa* from peach. Phytopathology 51:699-703.

Yarwood, C.E. 1939. Powdery mildews of peach and rose. Phytopathology 29:282-284.

Stone fruit rust diseases

Bloomer, S. 1960. Studies on the morphology and biology of *Tranzschelia pruni-spinosae* (Pers.) Dietel and *T. discolor* (Fuck.) Tranz. & Litv. [in German]. Phytopathol. Z. 38:355-383).

Laundon, G.F. and A.F. Rainbow. 1971a. *Tranzschelia pruni-spinosae* var. *discolor*. CMI Descriptions of Pathogenic Fungi and Bacteria, No. 287. CAB International, Wallingford, UK.

Laundon, G.F. and A.F. Rainbow. 1971b. *Tranzschelia pruni-spinosae* var. *pruni-spinosae*. CMI Descriptions of Pathogenic Fungi and Bacteria, No. 288. CAB International, Wallingford, UK.

Linfield, C.A. and D. Price. 1983. Host range of plum anemone rust, *Tranzschelia discolor*. Trans. Br. Mycol. Soc. 80:19-21.

Smith, I.M., J. Dunez, R.A. Lelliot, D.H. Philips and S.A. Archer (eds.). 1988. European Handbook of Plant Diseases. Blackwell Scientific Publications, Oxford, UK.

ARTHROPODS

Aphids

Blackman, R.L. and V.F. Eastop. 1984. Aphids on the World's Crops. An Iden-tification Guide. John Wiley & Sons, Chichester, UK.

Blackman, R.L. and V.F. Eastop. 1994. Aphids on the World's Trees. An Identification and Information Guide. CAB International, Wallingford, UK.

Armoured scale insects

CIE. 1986. Distribution Maps of Pests, Series A (Agricultural). Map No. 7. *Quadraspidiotus perniciosus* (Cornstock). CAB International, Wallingford, UK.

Kosztarab, M. and F. Kozar. 1988. Scale Insects of Central Europe. Dr W. Junk Publishers, Dordrecht, The Netherlands.

Kozar, F. 1990. Deciduous fruit trees. Pp. 593-602 *in* Armored Scale Insects - their Biology, Natural Enemies and Control (D. Rosen, ed.). World Crop Pests, Vol 4B. Elsevier, Amsterdam, The Netherlands.

Mites

CIE. 1972. Distribution Maps of Pests, Series A (Agricultural). Map No. 31. *Panonychus ulmi* (Koch). CAB International, Wallingford, UK.

CIE. 1984. Distribution Maps of Pests, Series A (Agricultural). Map No. 461. *Bryobia rubrioculus* (Scheuten). CAB International, Wallingford, UK.

Jeppson, L.R., H.H. Keifer and E.W. Baker. 1975. Mites Injurious to Economic Plants. Univ. of California Press, Berkeley, USA.

Oldfield, G.N. 1970. Mite transmission of plant viruses. Ann. Rev. Entomol. 15: 343-380.

Planthoppers

CIE. 1992. Distribution Maps of Pests, Series A (Agricultural). Map No. 529. *Metcalfa pruinosa* (Say). CAB International, Wallingford, UK.

Soft scale insects

Ben-Dov, Y. 1993. A Systematic Catalogue of the Soft Scale Insects of the World (Homoptera: Coccoidea: Coccidae) with Data on Geographical Distribution, Host Plants, Biology, and Economic Importance. Flora and Fauna Handbook No. 9. Sandhill Crane Press, Inc., Gainesville, Florida.

Kosztarab, M. and F. Kozar. 1988. Scale Insects of Central Europe. Dr W. Junk Publishers, Dordrecht, The Netherlands.

Comments on Technical Guidelines for the Safe Movement of Stone Fruits

Please send to:

Germplasm Health Scientist
IPGRI
Via delle Sette Chiese 142
00145 Rome, Italy
Fax: +39-6-5750309

and

Chief, Plant Protection Service
FAO
Via delle Terme di Caracalla
00100 Rome, Italy
Fax: +39-6-5225-6347

I would like to bring the following

[ ] inaccuracy (ies)
[ ] new development (s)
[ ] omission (s)
[ ] concerns

to the attention of the editors:

Disease

Comments

From:

Name

Address

Date

Signature

No. 16. Stone Fruits

FAO/IPGRI Technical Guidelines for the Safe Movement of Germplasm are published under the joint auspices of the Plant Production and Protection Division of the Food and Agriculture Organization of the United Nations (FAO) and the International Plant Genetic Resources Institute (IPGRI).

The International Plant Genetic Resources Institute (IPGRI) is an autonomous international scientific organization operating under the aegis of the Consultative Group on International Agricultural Research (CGIAR). IPGRI's mandate is to advance the conservation and use of plant genetic resources for the benefit of present and future generations. IPGRI works in partnership with other organizations, undertaking research, training and the provision of scientific and technical advice and information, and has a particularly strong programme link with the Food and Agriculture Organization of the United Nations. Financial support for the agreed research agenda of IPGRI is provided by the Governments of Australia, Austria, Belgium, Canada, China, Denmark, France, Germany, India, Italy, Japan, the Republic of Korea, the Netherlands, Norway, Spain, Sweden, Switzerland, the UK and the USA, and by the Asian Development Bank, IDRC, UNDP and the World Bank.

Citation: Diekmann, M. and C.A.J. Putter. 1996. FAO/IPGRI Technical Guidelines for the Safe Movement of Germplasm. No. 16. Stone Fruits. Food and Agriculture Organization of the United Nations, Rome/International Plant Genetic Resources Institute, Rome.

ISBN 92-9043-l60-l