18 Diseases of Apple

Gary G. Grove¹, Kenneth C. Eastwell¹, Alan L. Jones² and Turner B. Sutton³

¹Irrigated Agriculture Research and Extension Center, Washington State University, Prosser, Washington, USA; ²Department of Botany and Plant Pathology, Michigan State University, East Lansing, Michigan, USA; ³Department of Plant Pathology, North Carolina State University, Raleigh, North Carolina, USA

18.1 Introduction 460
18.2 Diseases Caused by Bacteria 460
  18.2.1 Fire blight 460
18.3 Diseases Caused by Fungi 468
  18.3.1 Apple scab 468
  18.3.2 Powdery mildew 470
  18.3.3 Brown-rot diseases 472
  18.3.4 Summer diseases 473
  18.3.5 Phytophthora crown and root rot 476
  18.3.6 European or Nectria canker 477
18.4 Postharvest Diseases 477
  18.4.1 Blue mould 478
18.5 Diseases Caused by Viruses, Viroids, Phytoplasmas and Other Virus-like Agents 478
  18.5.1 Chlorotic leaf spot 479
  18.5.2 Apple decline (on ‘Virginia Crab’) 480
  18.5.3 Flat apple 480
  18.5.4 Apple mosaic 481
  18.5.5 Stem pitting 481
  18.5.6 Union necrosis 481
18.6 Diseases Caused by Phytoplasmas 482
  18.6.1 Proliferation 482
  18.6.2 Chat fruit 482
18.7 Diseases of Apple Caused by Viroids 482
  18.7.1 Blister bark 482
  18.7.2 Dapple apple 483
  18.7.3 Dimple fruit 483
  18.7.4 Fruit crinkle 483
18.8 ‘Virus-like’ or Graft-transmissible Diseases of Apple with No Known Causal Agents 483
  18.8.1 Green crinkle 484
  18.8.2 Rubbery wood 484
  18.8.3 Dead spur 485
18.9 Control Measures 485

18.1 Introduction

Apples are host to over 70 infectious diseases, the vast majority of which are caused by pathogenic fungi (Table 18.1). Apples are also susceptible to diseases caused by bacteria, phytoplasma and virus/virus-like agents. The authors discuss several but not all economically important pre- and postharvest apple diseases in this chapter. Readers should keep in mind that some of the diseases discussed might be major in some areas and minor in others. Others (e.g. fire blight) are of importance or potential importance wherever apples are grown. In most apple-producing regions, disease control is a major annual expense for the grower. For example, in the eastern USA, the management of apple scab can require eight to ten protective fungicide applications annually. In those areas, the apple grower must manage early-season diseases, such as apple scab and cedar apple rust, as well as a group of diseases termed ‘summer diseases’. Conversely, in the arid production regions of the Pacific Northwest, these diseases are non-existent or sporadic in nature and powdery mildew is much more problematic.

Successful disease management usually results from the integration of several methods of disease control. The use of resistant rootstocks and scions, fungicides, bactericides, biological control agents, environmental modification and site selection are some of the means used to control apple diseases. The precise combination and order of control measures are usually disease-specific.

18.2 Diseases Caused by Bacteria

(Table 18.2)

Fire blight, blister spot, blister bark, crown gall and hairy root are diseases of apple caused by bacteria. Fire blight is the most important of these diseases and is described in more detail below. Blister spot, caused by Pseudomonas syringae pv. papulans (Rose) Dhanvantari, occurs in North America and Europe. It causes a fruit spot on ‘Mutsu’/’Crispin’, ‘Fuji’, ‘Redcort’, ‘Sun Crisp’, ‘Smoothee’ and a few other cultivars.

Blister bark has been described from South Africa but the pathogen P. s. pv. syringae van Hall is found as a common epiphyte on apple foliage worldwide. Crown gall and hairy root are soil-borne diseases caused by two species of Agrobacterium; crown gall is the most common of the two problems.

In areas where these bacterial pathogens either are not established or are rare, bacterial diseases are often avoided by planting pathogen-free nursery stock. Where these diseases are established, they are managed through orchard sanitation, wound minimization, the use of resistant rootstocks and cultivars, site selection, cultural practices that promote air movement and drying of plant surfaces and, in some cases, the use of copper and antibiotic sprays.

18.2.1 Fire blight

Fire blight is of major concern in all countries where apples are grown including countries that are currently free of this disease (Vanneste, 2000). When fire blight is epidemic, it can cause serious tree loss in nurseries and orchards (Plate 18.1), even leading to orchard removal. In high-risk areas, fire blight is limiting the planting of some highly susceptible apple cultivars and rootstocks. Strict quarantines and restrictions are maintained in countries where the disease does not currently occur; enforcing quarantine regulations and restrictions and, if necessary, eradicating the disease are very costly.

The fire blight pathogen kills fruit-bearing spur, branches and entire trees. Infected blossoms are initially water-soaked and darker green; spurs with infected blossoms turn brown to dark brown and collapse after 4–5 days. Infected shoots turn brown to black from the tip and bend near the tip to resemble a shepherd’s crook (Plate 18.2). When shoots are invaded from the base, the basal leaves and stem turn brown to black. Leaves may exhibit discoloration of the midrib, followed shortly by a darkening of the lateral veins and surrounding tissues. Bark on infected branches and scaffold limbs is darker than normal. When the outer bark is peeled away, the inner tissues are
<table>
<thead>
<tr>
<th>Disease (distribution)</th>
<th>Fungal pathogen</th>
<th>Reproductive stage</th>
<th>Common symptoms</th>
<th>Main control method</th>
</tr>
</thead>
<tbody>
<tr>
<td>Alternaria blotch (A, AF, NA)</td>
<td><em>Alternaria mali</em> Roberts; = <em>Alternaria alternata</em> (Fr.:Fr) Kessl apple pathotype</td>
<td>Conidia/conidiophores</td>
<td>Leaf spots, defoliation</td>
<td>Fungicides, cultivar selection</td>
</tr>
<tr>
<td>Alternaria rot (W)</td>
<td><em>Alternaria alternata</em> (Fr.:Fr) Keissl</td>
<td>Conidia/conidiophores</td>
<td>Decay of fruit</td>
<td>None</td>
</tr>
<tr>
<td>American brown rot (A, AF, NA, O)</td>
<td><em>Monilinia fructicola</em> (G. Wint.) Honey</td>
<td>Conidia in sporodochia; ascospores in apothecia</td>
<td>Decay of fruit (P)</td>
<td>Avoid preharvest injury to fruit</td>
</tr>
<tr>
<td>Anthracnose canker and bull's-eye rot (E, NA, O)</td>
<td><em>Pezicula malicorticis</em> (H. Jacks.) Nannf</td>
<td>Conidia in acervuli; ascospores in apothecia</td>
<td>Cankers on wood, decay of fruit (P)</td>
<td>Fungicides</td>
</tr>
<tr>
<td>Apple scab (W)</td>
<td><em>Venturia inaequalis</em> (Cooke) G. Wint.</td>
<td>Ascospores in pseudothecia; conidia/conidiophores</td>
<td>Leaf spots, scabs on fruit, defoliation</td>
<td>Sanitation, fungicides, resistant cultivars</td>
</tr>
<tr>
<td>Apple ring rot and canker (A)</td>
<td><em>Botryosphaeria berengeriana</em> De Not. (syn. <em>Physalospora piriola</em> N. C. Gremmen in Boerema &amp; Gremmen [anamorph])</td>
<td>Conidia in pycnidia; ascospores in pseudothecia</td>
<td>Cankers on wood, decay of fruit</td>
<td>Fungicides</td>
</tr>
<tr>
<td>Armillaria (shoestring) root rot (W)</td>
<td><em>Armillaria mellea</em> (Vahl:Fr.) P. Kumm.</td>
<td>Basidiospores in basidiocarps (mushrooms); rhizomorphs</td>
<td>Decay of roots</td>
<td>None, avoid problem sites</td>
</tr>
<tr>
<td>Bitter rot (W)</td>
<td><em>Glomerella cingulata</em> (Stoneman) Spauld. &amp; H. Schrenk</td>
<td>Conidia in acervuli; ascospores in perithecia</td>
<td>Decay of fruit</td>
<td>Fungicides, sanitation</td>
</tr>
<tr>
<td>Black pox (NA)</td>
<td><em>Helminthosporium papulosum</em> Berg.</td>
<td>Conidia/conidiophores</td>
<td>Spots on fruit and leaves</td>
<td>Fungicides</td>
</tr>
<tr>
<td>Black root rot (NA for <em>X. mali</em>; W)</td>
<td><em>Xylaria mali</em> Fromme</td>
<td>Ascospores in perithecia</td>
<td>Decay of roots</td>
<td>None</td>
</tr>
<tr>
<td>Black rot, frog-eye leaf spot and canker (A, E, NA, SA, O)</td>
<td><em>Botryosphaeria obtusa</em> (Schwein.) Shoemaker</td>
<td>Conidia in pycnidia; ascospores in pseudothecia</td>
<td>Spots on leaves and fruit, defoliation, cankers on wood</td>
<td>Sanitation, fungicides</td>
</tr>
<tr>
<td>Blister canker (nail-head canker) (NA)</td>
<td><em>Biscogniauxia marginata</em> (Fr.) Pouzar = <em>Nummularia discreta</em> (Schwein.) Tul. &amp; C. Tul.</td>
<td>Conidia in a sclerotia-like stroma, ascospores in perithecia</td>
<td>Cankers on branches</td>
<td>Removal of infected branches</td>
</tr>
</tbody>
</table>

*Continued*
<table>
<thead>
<tr>
<th>Disease (distribution)</th>
<th>Fungal pathogen</th>
<th>Reproductive stage</th>
<th>Common symptoms</th>
<th>Main control method</th>
</tr>
</thead>
<tbody>
<tr>
<td>Blotch (NA)</td>
<td><em>Phyllosticta solitaria</em> Ellis &amp; Everh.</td>
<td>Conidia in pycnidia</td>
<td>Blotches on fruit, leaf spots, blisters in bark</td>
<td>Fungicides</td>
</tr>
<tr>
<td>Blue mould (W)</td>
<td><em>Penicillium spp.</em> <em>P. expansum</em> Link</td>
<td>Conidia/conidiophores</td>
<td>Decay of fruit (P)</td>
<td>Packing-house sanitation, fungicides</td>
</tr>
<tr>
<td>Brooks fruit spot (NA)</td>
<td><em>Mycosphaerella pomi</em> (Pass.) Lindau <em>Cylindrosporium pomi</em> C. Brooks [anamorph]</td>
<td>Conidia/conidiophores; ascospores in pseudeothecia</td>
<td>Spots on fruit, often at calyx end</td>
<td>Fungicides</td>
</tr>
<tr>
<td>Brown-rot blossom blight (A, AF, E, NA, SA)</td>
<td><em>Monilinia laxa</em> (Aderh. &amp; Ruhl.) Honey</td>
<td>Conidia in sporodochia</td>
<td>Blighting of blossoms, spur dieback</td>
<td>Fungicides</td>
</tr>
<tr>
<td>Calyx-end rot (NA)</td>
<td><em>Sclerotinia sclerotiorum</em> (Lib.) de Bary</td>
<td>Ascosporo in apothecium</td>
<td>Decay of fruit</td>
<td>None</td>
</tr>
<tr>
<td>Clitocybe root rot (NA)</td>
<td><em>Armillaria tabescens</em> (Soop.) Dennis et al = <em>Clitocybe tabescens</em> (Soop.) Bres.</td>
<td>Basidiospores in basidiocarps</td>
<td>Decay of roots</td>
<td>None</td>
</tr>
<tr>
<td>Diaporthe canker (A)</td>
<td><em>Diaporthe tanakae</em> Kobayashi &amp; Sakuma <em>Phomopsis tanakae</em> Kobayashi &amp; Sakuma [anamorph]</td>
<td>Conidia (α and β spores) in pycnidia; ascospores in perithecia</td>
<td>Cankers on 1- and 2-year-old shoots</td>
<td>Sanitation</td>
</tr>
<tr>
<td>Dipodia canker (E, NA, O)</td>
<td><em>Botryosphaeria stevensii</em> Shoemaker = <em>Physalosporula malorum</em> Shear et al. <em>Dipodia mutila</em> (Fr.: Fr.) Mont. [anamorph]</td>
<td>Pycnidia and pseudothecia; often in the same stroma</td>
<td>Cankers on branches</td>
<td>Pruning out of infected branches</td>
</tr>
<tr>
<td>European brown rot (A, E, AF)</td>
<td><em>Monilinia fructigena</em> Honey in Whetzel <em>Monila fructigena</em> Pers.:Fr. [anamorph] <em>Monilinia laxa</em> (Aderhold &amp; Ruhl.) Honey</td>
<td>Conidia in sporodochia; ascospores in apothecia</td>
<td>Blossoms and spur blight, decay of injured fruit</td>
<td>Sanitation, fungicides</td>
</tr>
<tr>
<td>Fish-eye rot (A, E, NA)</td>
<td><em>Butlerella eustacei</em> Weresub &amp; Ilman = <em>Corticium centrifugum</em> (Lév.) Bres.</td>
<td>Basidiomycete, may produce basidiospores in culture</td>
<td>Decay of fruit (P)</td>
<td>None</td>
</tr>
<tr>
<td>Fly-speck (W)</td>
<td><em>Schizothyrium pomi</em> (Mont.:Fr.) Arx <em>Zygothiala jamaicensis</em> E. Mason [anamorph]</td>
<td>Conidia in pycnidia</td>
<td>Small, superficial, dark spots on fruit</td>
<td>Cultural practices, fungicides</td>
</tr>
<tr>
<td>Glomerella leaf spot (NA, SA)</td>
<td><em>Glomerella cingulata</em> (Stoneman) Spauld. &amp; H. Schrenk <em>Colletotrichum gloeosporioides</em> (Penz.) Penz. &amp; Sacc. in Penz. [anamorph]</td>
<td>Conidia in acervuli; ascospores in perithecia</td>
<td>Spots on leaves, defoliation</td>
<td>Fungicides</td>
</tr>
<tr>
<td>Disease</td>
<td>Fungus Name</td>
<td>Host Affected</td>
<td>Symptoms</td>
<td>Control Measures</td>
</tr>
<tr>
<td>----------------------------------------------</td>
<td>--------------------------------------------------</td>
<td>---------------</td>
<td>--------------------------------------------------------------------------</td>
<td>-------------------------------------------------</td>
</tr>
<tr>
<td>Grey-mould rot (W)</td>
<td><em>Botrytis cinerea</em> Pers. Fr.</td>
<td>Apple</td>
<td>Conidia and sclerotia (rare in nature)</td>
<td>Decay often at calyx end of fruit; nest rot of fruit in storage (P)</td>
</tr>
<tr>
<td>= dry-eye rot, blossom-end rot</td>
<td></td>
<td></td>
<td></td>
<td>Leaves</td>
</tr>
<tr>
<td>Leptosphaeria canker and fruit rot (W)</td>
<td><em>Botryotinia fuckeliana</em> (de Bary) Whetzel</td>
<td>Apple</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Marssonina blotch (A, NA, E)</td>
<td><em>Diplocarpon mali</em> Harada &amp; Sawamura <em>Marssonina coronaria</em> (Ellis &amp; J.J. Davis) J.J. Davis [anamorph]</td>
<td>Apple</td>
<td>Ascospores in apothecia; conidia in acervuli</td>
<td>Leaf spots, defoliation, spots on surface of fruit</td>
</tr>
<tr>
<td>Mouldy core and core rot (W)</td>
<td><em>Alternaria</em> spp.; <em>Stemphylium</em> spp.; <em>Cladosporium</em> spp.; <em>Ulocladium</em> spp. <em>Epicrocum</em> spp.; <em>Coniothyrium</em> spp. and <em>Pleospora herbarum</em> (Pers.) Rabenh. wet core rot, mainly <em>Penicillium</em> spp.</td>
<td>Apple</td>
<td>Conidia of various types</td>
<td>Discoloration and decay around the fruit core</td>
</tr>
<tr>
<td>Monilia leaf blight (A)</td>
<td><em>Monilinia mali</em> (Takahashi) Whetzel <em>Monilia</em> spp. [anamorph]</td>
<td>Apple</td>
<td>Ascospores in apothecia; conidia with disjunctors</td>
<td>Blossom and spur blight, decay of young fruit</td>
</tr>
<tr>
<td>Mucor rot (AF, NA, O)</td>
<td><em>Mucor</em> spp. <em>M. pitiformis</em> E. Fischer</td>
<td>Apple</td>
<td>Sporangiospores and zygospores</td>
<td>Soft, watery decay of fruit (P)</td>
</tr>
<tr>
<td>Nectria twig blight = coral spot (E, O, NA)</td>
<td><em>Nectria cinnabarina</em> (Tode:Fr.) Tode:Fr. [anamorph]</td>
<td>Apple</td>
<td>Conidia on sporodochia; ascospores in perithecia</td>
<td>Cankers on branches and trunk</td>
</tr>
</tbody>
</table>

Continued
<table>
<thead>
<tr>
<th>Disease (distribution)</th>
<th>Fungal pathogen</th>
<th>Reproductive stage</th>
<th>Common symptoms (^b)</th>
<th>Main control method</th>
</tr>
</thead>
<tbody>
<tr>
<td>Peniophora root canker (O)</td>
<td><em>Peniophora sacra</em>ta G.H. Cunn.</td>
<td>Basidiomycete with crust-like fruiting-bodies</td>
<td>Infected roots with characteristic cracks</td>
<td>Resistant rootstocks and sanitation</td>
</tr>
<tr>
<td>Perennial canker (NA)</td>
<td><em>Neofabrae perennans</em> Kienholz</td>
<td>Conidia in acervuli; ascospores in apothecia</td>
<td>Cankers on branches, decay of fruit</td>
<td>Sanitation, fungicides</td>
</tr>
<tr>
<td>Phomopsis canker, fruit decay and rough bark (A, E, NA)</td>
<td><em>Phomopsis mal</em> Roberts</td>
<td>Conidia (α and β spores) in pycnidia, ascospores in perithecia</td>
<td>Cankers on wood, fruit decay (P)</td>
<td>None</td>
</tr>
<tr>
<td>Phymatotrichium root rot</td>
<td><em>Phymatotrichopsis omnivora</em> (Duggar) Hennebert</td>
<td>Hyphae with conidia on conidiophores and sclerotia</td>
<td>Decay of roots</td>
<td>None</td>
</tr>
<tr>
<td>Phytophthora crown, collar and root rot; (sprinkler rot) (W)</td>
<td><em>Phytophthora spp.</em></td>
<td>Zoospores produced in sporangia from hyphae or germinating ascospores or chlamydospores</td>
<td>Decay of crown and root tissues</td>
<td>Cultural practices, host resistance, chemical treatment</td>
</tr>
<tr>
<td>Phytophthora fruit rot (E, NA)</td>
<td><em>Phytophthora cactorum</em> (Lebert &amp; Cohn) J. Schrötl.</td>
<td>See above</td>
<td>Rotting of fruit</td>
<td>Late-season fungicide sprays or postharvest treatments</td>
</tr>
<tr>
<td>Pink mould rot (W)</td>
<td><em>Trichothecium roseum</em> (Pers.:Fr.) Link = <em>Cephalothecium roseum</em> Corda</td>
<td>Conidia</td>
<td>Decay of fruit (P)</td>
<td>Prevented by refrigerated cold storage</td>
</tr>
<tr>
<td>Powdery mildew (W)</td>
<td><em>Podosphaera leucotricha</em> (Ellis &amp; Everh.) E.S. Salmon</td>
<td>Conidia</td>
<td>Powdery spots on leaves, fruit russetting</td>
<td>Cultivar selection, fungicides</td>
</tr>
<tr>
<td>Rosellinia root rot</td>
<td><em>Rosellinia  necatrix</em> Prill.</td>
<td>Ascospores in perithecia; pycnidia on synnemata</td>
<td>Decay of roots</td>
<td>Cultural practices, solarization</td>
</tr>
<tr>
<td>= Dematophora root rot</td>
<td><em>Dematophora necatrix</em> R. Hartig [anamorph]</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Rust, American hawthorne (NA)</td>
<td><em>Gymnosporangium globosum</em> (Farl.) Farl.</td>
<td>Pycnia and aecidia on apple; telentospores in telial horns on cedar</td>
<td>Leaves</td>
<td>Removal of alternative host, fungicides</td>
</tr>
<tr>
<td>Rust, cedar apple (NA)</td>
<td><em>Gymnosporangium juniperi-virginianae</em> Schwein</td>
<td>Pycnia and aecidia on apple; telentospores in telial horns on cedar</td>
<td>Spots on leaves, defoliation and distortion of fruit</td>
<td>Removal of alternative host, fungicides</td>
</tr>
<tr>
<td>Disease</td>
<td>Pathogen</td>
<td>Symptoms</td>
<td>Control Measures</td>
<td></td>
</tr>
<tr>
<td>---------------------------------</td>
<td>---------------------------------------------------------------------------</td>
<td>---------------------------------------------------------------------------</td>
<td>-----------------------------------------------</td>
<td></td>
</tr>
<tr>
<td>Rust, Japanese apple (A)</td>
<td>Gymnosporangium yamadae Miyabe ex Yamada</td>
<td>Pycnia and aecidia on apple; telentospores in telia on Juniperus chinensis</td>
<td>Removal of alternate host, fungicides</td>
<td></td>
</tr>
<tr>
<td>Rust, Pacific Coast pear (NA)</td>
<td>Gymnosporangium libocedri (C.Henn.) F. Kern</td>
<td>Aecidia on apple; telentospores in telia on Libocedrus decurrens</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Rust, quince (NA)</td>
<td>Gymnosporangium clavipes (Cooke &amp; Peck) Cooke &amp; Peck in Peck</td>
<td>Pycnia and aecidia on apple; telentospores in telia on cedar</td>
<td>Removal of alternative host, fungicides</td>
<td></td>
</tr>
<tr>
<td>Side rot (E, NA)</td>
<td>Phialophora malorum (M.N. Kidd &amp; A. Beaumont) McColloch</td>
<td>Conidia at the apex of phialides</td>
<td>Cultural practices, postharvest treatments</td>
<td></td>
</tr>
<tr>
<td>Silver leaf (W)</td>
<td>Chondrostereum purpureum (Pers.:Fr.) Pouzar</td>
<td>Basidiospores</td>
<td>Cultural practices</td>
<td></td>
</tr>
<tr>
<td>Sooty-blotch complex (W)</td>
<td>Peltaster fructicola (Johnson, Sutton, Hodges); Geastrumia polystigmatis</td>
<td>Conidia in pycnidia</td>
<td>Cultural practices and fungicides</td>
<td></td>
</tr>
<tr>
<td>Southern blight (AF, NA, SA)</td>
<td>Sclerotium rolfsii Sacc. Athelia rolfsii (Curzi) Tu &amp; Kimbrough [teleomorph]</td>
<td>Sclerotia</td>
<td>Cultural practices</td>
<td></td>
</tr>
<tr>
<td>Thread blight = Hypochnus leaf blight (NA)</td>
<td>Corticium stevensii Burt = Pelticularia koregola Cooke = Hypochnus ochroleucus Noack</td>
<td>Rhizomorphs and sclerotia; basidia with basidiospores</td>
<td>Fungicides</td>
<td></td>
</tr>
<tr>
<td>Valsa canker (A)</td>
<td>Valsa ceratosperma (Tode:Fr.) Maire Cytospora sacculus (Schwein.) Gvritischvili [anamorph]</td>
<td>Pycnia and perithecia in a stroma</td>
<td>Sanitation</td>
<td></td>
</tr>
<tr>
<td>Valsa canker (A)</td>
<td>Valsa ceratosperma (Tode:Fr.) Maire Cytospora sacculus (Schwein.) Gvritischvili [anamorph]</td>
<td>Pycnia and perithecia in a stroma</td>
<td>Sanitation</td>
<td></td>
</tr>
<tr>
<td>Violet root rot (O)</td>
<td>Helicobasidium mompa Tanaka</td>
<td>Basidiospores in basidiocarps</td>
<td>Soil sterilization</td>
<td></td>
</tr>
<tr>
<td>White rot (NA)</td>
<td>Scytinostroma galactinum (Fr.) Donk = Corticium galactinum (Fr.) Burt</td>
<td>Basidia on a resupinate hymenium</td>
<td>None</td>
<td></td>
</tr>
<tr>
<td>White rot (AF, NA, SA, O)</td>
<td>Botryosphaeria dothidea (Moug.) Ces. &amp; De Not. Fusicoccum aesculi Corda [anamorph]</td>
<td>Conidia in pycnidia; ascospores in pseudothecia</td>
<td>Fungicides and sanitation</td>
<td></td>
</tr>
<tr>
<td>Zonate leaf spot (A)</td>
<td>Cristulariella moricola (Hino) Redhead Grovesenia pyramidalis M. Cline et al. [teleomorph]</td>
<td>Ascospores in apothecia; conidia</td>
<td>Sanitation</td>
<td></td>
</tr>
</tbody>
</table>

*Approximate geographical distribution of the disease: A, Asia; AF, Africa; E, Europe; NA, North America; O, Oceania; SA, South America; W, worldwide.

bP, postharvest disease problem.
Fire blight is caused by *Erwinia amylovora* (Burrill) Winslow et al., a Gram-negative, rod-shaped, non-fluorescent bacterium with peritrichous flagella. More than 130 species in 39 genera of the *Rosaceae* are hosts. Important hosts include apple, pear, ornamental *Pyrus* and *Malus* species, quince (*Cydonia oblonga*), loquat (*Eriobotrya japonica*), hawthorn (*Crataegus* spp.), *Cotoneaster* spp., *Sorbus* spp., *Pyracantha* spp. and *Rubus* spp. Most strains of *E. amylovora* from *Rubus* do not infect apple and are therefore considered to be pathologically distinct. Primary isolation of the bacteria is by culturing on Luria–Bertani (LB) agar or some semi-selective medium. They are distinguished from other plant pathogenic bacteria based on colony colour and morphology on MM2Cu and LB media (see Bereswill et al., 1998, for the composition of these media), pathogenicity tests performed on immature fruit, seedlings or vigorous plants, and molecular assays (Bereswill et al., 1995; McManus and Jones, 1995).

The pathogen overwinters in cankers located on branches and tree trunks (Fig. 18.1). Ooze can begin to appear on the surface of these cankers at or just before the onset of bloom. Bacteria are disseminated to flowers by splashing rain and, occasionally, flies and other insects that visit both bacterial ooze and blossoms. Eventually, honey bees visit infected blossoms and pick up pollen or nectar contaminated with bacteria. Spread of bacteria from flower to flower by bees is rapid. Bacteria colonize the stigmatic surface of the pistils of healthy apple and pear flowers, resulting in epiphytic populations of bacteria (Thomson, 1986). Climatic conditions govern the rate of spread and severity of blossom blight and even the infection process itself. Temperatures between 18.3 and 30°C (accompanied by rain or high humidity during the day) favour infection.

Although bacteria invade the flowers primarily through natural openings, storms containing wind-driven rain or hail are important in spreading bacteria during the summer months, which under some condi-
tions lead to sudden and severe outbreaks of disease. Inoculum for secondary infection originates from droplets of ooze produced on infected flowers, fruits and shoots.

Temperature, as measured by the accumulation of degree-days (DD), governs the rate of symptom development. Symptoms are expressed when 55 DD (base 12.5°C) are accumulated following the infection date. The susceptible rootstocks M.9 and M.26 are infected by systemic movement of bacteria through symptomless scion tissue or through infected rootstock shoots (Momol et al., 1998). Although the rootstock Bud.9 is blight-susceptible, rootstock blight has not developed on many Bud.9/scion combinations. Losses from rootstock blight can be avoided by using resistant rootstocks, such as G.16, G.30, G.65 and other blight-resistant rootstocks from the US Department of Agriculture–Agricultural Research Service (USDA-ARS)/Cornell apple-rootstock programme.

Many practices help to prevent or reduce the severity of fire blight. In countries where E. amylovora is not established, only trees produced in fire-blight-free regions should be used when establishing new orchards. These countries may already have strict quarantines to prevent the importation of plant material that may harbour the pathogen.

In countries where fire blight is a problem, sanitation – the removal of infected portions of the tree – is critical to the success of other control measures and is effective provided it is done during the early stages of an epidemic. New orchards should be planted on blight-resistant rootstocks if available. A realistic plan for controlling fire blight is needed before making large plantings of blight-susceptible cultivars. Antibiotics have been highly effective for preventing the blossom-blight stage of fire blight, but in some regions or orchards control with antibiotics is no longer possible, due to the development of streptomycin-resistant strains of E. amylovora. Predictive models, particularly Maryblt and Cougarblight in the USA and Billings’ integrated system and Firescreens in Europe, help growers identify potential infection periods (Billings, 2000). Such information is helpful in timing antibiotic treatment and for avoiding unnecessary treatment. Bacterial antagonists that suppress fire blight may be integrated with antibiotics to control flower infections (Stockwell et al., 1996).
18.3 Diseases Caused by Fungi

Within this diverse group of plant pathogens are the causes of most apple diseases (Table 18.1). Fungi pathogenic to apple can cause root rots, leaf spots, leaf blights, blossom blights, fruit decay, fruit spots, defoliation and trunk, branch and twig cankers. The fungal diseases discussed in this chapter are caused by fungi belonging to the general taxonomic groups represented by the mushrooms (Basidiomycetes) and morels (Ascomycetes). The phylum Oomycota also contains the water moulds, an important group of plant pathogens formerly classified as fungi. Fungi in the ascomycete group have scientific names for the anamorph and teleomorph. The anamorph is the asexual form (also called the imperfect stage) of the fungus and produces asexual spores (such as conidia) or no asexual spores. The teleomorph is the sexual form (also called the perfect or sexual state) and produces sexual spores (ascospores) in various fruiting bodies (pseudothecia, perithecia, apothecia, etc.). These fungi generally reproduce by spores that are dispersed by air currents or splashing water. Fungal diseases are prevented or managed by using resistant cultivars and rootstocks, site selection, sanitation, cultural practices and fungicide practices. Specific management measures vary according to disease and geographical location.

18.3.1 Apple scab

Scab occurs in virtually all apple-producing regions worldwide (MacHardy, 1996). The disease is an annual threat in cool, humid regions with frequent rainfall in spring and early summer. In semi-arid regions, scab is a threat in years with above-normal rainfall or in orchards where artificial wetting periods are created from the improper use of overhead irrigation.

Scab attacks the leaves and fruit throughout most of the growing season; blossoms and bud scales are attacked for short periods in spring and late summer, respectively. Symptoms first appear on the undersides of leaves, the side exposed as buds open. Later, symptoms are found on both sides of leaves. Conidia are produced abundantly in new lesions; therefore, lesions appear as velvety brown to olive spots that turn black with age (Plate 18.4). Severe infection can cause leaves to abscise, resulting in defoliated trees. Return bloom on trees defoliated in midsummer is often reduced due to a lack of flower-bud formation the previous summer. Fruit infections resemble leaf infections when young but turn brown and corky with age (Plate 18.5). Early-season infections are often associated with the blossom end of the fruit; later they can occur anywhere on the surface. Scab infections result in uneven growth of fruit and even cracking of the skin and flesh. Rough, black circular lesions develop in cold storage on fruit infected close to harvest. The latter phase is known as pinpoint scab. Infection of blossoms and bud scales may be observed in high-inoculum orchards when infection periods occur at critical times. Lesions on blossoms and bud scales resemble those on leaves but are seldom observed because these tissues normally drop to the ground before the symptoms are well developed.

Venturia inaequalis (Cooke) G. Wint., anamorph Spilocea pome Fr., causes apple scab. It has one sexual cycle and a series of asexual cycles per year. Flask-shaped, ascus-bearing fruiting bodies (pseudothecia) develop in overwintering infected leaves. Pseudothecia form as a result of the interaction of two strains of opposite mating types (heterothallic fungus). Asci in pseudothecia are eight-spored. Mature ascospores are two-celled, with the upper cell shorter and wider than the lower cell, yellowish green to tan and with smooth walls. Conidia are one-celled, yellowish-olive and pointed.

From late autumn to spring, microscopic, black, pimple-like pseudothecia develop in leaves on the orchard floor (Fig. 18.2). Normally, pseudothecia contain mature ascospores when the blossom buds start to open in spring. Maturation and discharge of ascospores lasts about 5–9 weeks. When leaves on the orchard floor become wet from rain, spores are ejected into the air. Air currents carry them to the emerging tissues, where infection occurs. Young leaves and fruit are highly susceptible to infection, but their
susceptibility declines with maturity. Spore germination begins soon after ascospores land on wet leaves or fruit. Prevailing temperatures govern the infection rate, provided a continuous film of moisture is present for germination of the spores. Depending on the average temperature after penetration, 9–17 days are required before the appearance of the olive-green, velvety scab lesions. In some apple-producing regions and on some cultivars, the fungus also overwinters in lesions on twigs and bud scales (Becker et al., 1992); conidia produced in these lesions are a second form of primary inoculum. However, the number of conidia available from overwintering lesions at bud break is low compared with the number of ascospores potentially available from leaf litter.

Secondary infections are initiated by conidia produced in primary and secondary lesions. Conidia can be produced in new lesions beginning as soon as 7–9 days after infection and these lesions can produce conidia in continuous crops for several weeks. Conidial germination and infection occur under about the same conditions as germination and infection by ascospores. Secondary infection on fruit can occur in the autumn but fails to develop until after the fruit has been held in cold storage for several months. Infections can also build up in leaves after harvest and prior to normal leaf abscission. The fungus overwinters in these leaves and they are often the source of very high levels of inoculum the following spring.

Basic studies on the biology and epidemiology of apple scab from the 1920s to the 1940s established a rational basis for scab control and these concepts continue to be refined. The initial ascosporic inoculum is usually present in large amounts; therefore, estimating the risk of the initial ascosporic inoculum and forecasts of its efficiency are very important for determining when to initiate scab-control programmes and the application frequency. Inoculum risk is determined by monitoring pseudothecia from early spring to midsummer to determine whether ascospores are mature and available for discharge and by detecting ascospore release in orchards during wetting periods. Statistical models have been developed to predict ascospore maturity based on DD accumulations and these models are replacing ascospore-monitoring programmes. Inoculum risk forecasts assume
high inoculum levels, an assumption that is usually incorrect for orchards where scab was well controlled the previous season. Therefore, methods for assessing differences in inoculum among orchards based on a potential ascospore density (PAD) system were developed (Gadoury and MacHardy, 1986). It was found that in low-inoculum orchards spray programmes in spring could be delayed until about the tight-cluster stage of bud development, rather than green tip in the normal spray schedule for high-inoculum orchards. Integrating inoculum risk with environmental risk is accomplished by the identification of 'infection periods'. Rainfall is necessary for the discharge of ascospores – free water for the germination of ascospores and penetration of tissues. The rate of this infection process is temperature-dependent, and the duration of wetness required for successful infection across a wide range of temperatures is well known (Jones, 1998). Therefore, temperature and wetness measurement can be used to forecast the success or failure of each ascospore-discharge event. These events can be monitored with environmental sensors linked to small computers placed in the orchard or with automated weather stations connected directly to computers. Fungicides with post-infection efficacy are applied after predicted but unprotected infection periods.

Apple-breeding programmes aiming for high-quality, disease-resistant cultivars are in progress in New Zealand and some European and North American countries. Over 50 scab-resistant cultivars have been released and are gaining in commercial acceptance as fruit quality and other horticultural characteristics are improved. Guarding against races of the scab pathogen that can overcome the resistance sources used by apple breeders is a high priority in these breeding programmes (Bénaouf and Parisi, 2000).

Prevention of pseudothecia formation in overwintering leaves would probably eliminate scab. Unfortunately, complete elimination of pseudothecia is not possible under orchard conditions using current methods. Spring ascospore production can be reduced by making autumn applications of urea or fungal antagonists to the foliage just prior to leaf fall (Carisse et al., 2000), but this strategy alone is not adequate for season-long scab control. This approach may be more feasible in areas with lower amounts of overwintering inoculum and mild winters.

Scab is controlled primarily with fungicides applied in predetermined schedules, beginning at green tip. The fungicides are applied on a 7–14-day interval with eight to ten applications per season. Several classes of fungicides are available for apple-scab control. They are often rotated during the season or applied as mixtures because of the high potential of the scab fungus to develop fungicide-resistant strains.

### 18.3.2 Powdery mildew


Leaves, flowers and fruit are susceptible to infection by the powdery mildew fungus. The foliage of new terminal growth is extremely susceptible to infection. The initial signs of powdery mildew consist of white to grey felt-like patches on the lower leaf surface. These patches are comprised of masses of fungal mycelia and spores (conidia). As disease progresses, mildew signs may also appear on the upper leaf surface and eventually cover the entire leaf. Infected foliage may curl, blister and eventually
become brittle and necrotic (Plate 18.6). Internodal shortening may occur on severely infected shoots. Infected flower petals are distorted, stunted and light yellow to light green. Fruit infection typically results in stunting accompanied by the presence of a fine network of rough lines (russetting) (Plate 18.7).

Powdery mildew is caused by *Podosphaera leucotricha* (Ell. & Ev.) E.S. Salmon. The fungus is superficial on the host surface but withdraws water and nutrients from host tissue, using a structure called a haustorium. The fungus produces barrel-shaped conidia in chains, which are dispersed by air currents and initiate subsequent infections of foliage and fruit (Fig. 18.3). *P. leucotricha* survives through winter as mycelium in infected buds. This mode of perennation results in the production of ‘flag shoots’ (shoots covered with powdery mildew) when shoots emerge in the spring. Winter temperatures are the most important factor affecting the amount of carry-over inoculum. Temperatures colder than $-12{\degree}C$ kill the fungal mycelium in buds; temperatures lower than $-24{\degree}C$ may kill the infected buds (Spotts et al., 1981). Conidia produced on flag shoots initiate the first spring infections and are therefore primary inocula, which initiate additional new infections. This cycle of sporulation and foliar infection can continue as long as susceptible foliage is being produced. This secondary phase of powdery mildew can cycle many times during the growing season. Temperature is the most important factor affecting disease development. Conidia germinate at temperature between 10 and 25$\degree$C; the optimum temperatures for germination are 20–22$\degree$C. The sexual stage (ascocarps) of *P. leucotricha* occasionally forms on infected twigs. Ascocarps are minute, brown to black, spherical fruiting bodies that contain eight unicellular ascospores. Various researchers have suggested that this stage is insignificant in the epidemiology of the disease. Fruits are especially susceptible to infection during the bloom period (Daines et al., 1984).

Powdery-mildew epidemics are favoured by high humidity; therefore problems with mildew can often be avoided or reduced with cultural practices that promote air movement and light penetration. On susceptible cultivars, effective disease management usually depends on a fungicide-spray programme. Benzimidazole, sulphur, horticultural mineral, oils, demethylation-inhibiting
(DMI) and strobilurin fungicides are effective against powdery mildew. Spray programmes should commence at tight cluster and continue until the production of new foliage ceases. Because powdery mildews can develop resistance to benzimidazole, DMI and strobilurin fungicides, rational resistance-management strategies usually require the inclusion of two or more fungicide classes in a season-long programme. DMI fungicides applied in apple-scab-management programmes generally provide the added benefit of mildew control.

A predictive system has been developed for aid in managing infections caused by *P. leucotricha*. Podem© (Xu, 1999) is a system developed in the UK that simulates epidemics of secondary mildew on vegetative shoots. The effects of weather on conidial production, dispersal and germination are used to calculate a favourability index. The model itself is driven by hourly ambient temperature, relative humidity, shade temperature and the total daily duration of rainfall. The model has been incorporated into an integrated apple disease warning system (ADEM) and successfully used to time fungicide applications and in some cases has resulted in improved disease control, with a 40% reduction in fungicide usage (Berrie and Xu, 1999).

### 18.3.3 Brown-rot diseases

Several brown-rot fungi attack apple in different parts of the world. The species and their distribution are: *Monilinia fructicola* (Wint.) Honey in most regions except Europe, where it is a European Union-listed quarantine pest (Smith *et al.*, 1992); *Monilinia laxa* (Aderh. & Ruhl.) Honey in Asia, Europe, North and South America and South Africa; and *Monilinia fructigena* (Aderh. & Ruhl.) Honey in Europe and Asia (Byrde and Willetts, 1977). A related species, *Monilinia mali* (Takahashi) Whetzel, causes *Monilia* leaf blight of apple in Asia.

The brown-rot fungi cause blossom wilt, spur dieback, cankering and fruit rot (Plate 18.8); the incidence and severity of these symptoms depend on the species of pathogen present. *M. laxa* causes blossom blight, spur dieback and cankering of branches; *M. fructigena* causes fruit rot and sometimes cankers when the fungus spreads into branches from the fruit; and *M. fructicola* causes a fruit rot. *Monilia* leaf blight infects young apple leaves; mycelium invading from leaves kills flower clusters, young fruits and fruiting spurs.

The species of *Monilinia* can be differentiated by microscopic observation of conidia formed in chains in culture or on infected tissue. *M. mali* can be differentiated by disjunctors present within the conidial chains. *M. fructicola*, *M. fructigena* and *M. laxa* can be differentiated based on cultural characteristics, isozyme variation, vegetative interactions and PCR assays.

The life cycles of these pathogens differ only in the role that the perfect stage (apothecia) plays in the overwintering of the fungi. *M. mali* and occasionally *M. fructicola* produce apothecia on mummified fruit on the ground. All species overwinter in infected parts of the tree. Secondary spread is by conidia produced on infected host tissue within the tree and on trees of neighbouring hosts, particularly *Prunus* species.

Losses due to fruit decay caused by *M. fructigena* and *M. fructicola* increase gradually up to harvest time and are usually associated with injuries to the fruit. Cultivars prone to fruit cracking, such as ‘Cox’s Orange Pippin’ and ‘James Grieve’, are especially susceptible to infection. Infection also occurs through wounds caused by birds pecking at fruit, insects infesting fruit and hail. Fruit decay is prevented by avoiding cultivars prone to fruit cracking, by limiting the damage caused by birds and other wounding agents and by orchard sanitation methods aimed at reducing the build-up of inoculum.

Blossom infections from *M. laxa*, *M. fructigena* and *Monilia* leaf blight are controlled by orchard sanitation, combined with the application of fungicides. Blighted spurs and cankers are removed and destroyed during the dormant period and in the growing season. Fungicides applied as the flowers begin to open and one or two times 5–7 days later should prevent blossom blight.
18.3.4 Summer diseases

Summer diseases refer to a collection of nine diseases that tend to be most severe from 2–3 weeks after petal fall until harvest. They are most prevalent in warm and moist growing regions of the world, where they can cause extensive losses if not controlled. They are relatively minor problems in arid and cooler growing regions.

Of all the summer diseases, rot diseases are most destructive and can cause losses of 50% or more if not controlled. These diseases tend to be most severe in the south-eastern USA but cause problems in other areas as well. The life cycles of the pathogens that cause these diseases are similar, as is the overall strategy for managing them.

18.3.4.1 Bitter rot

Bitter rot is the most destructive of the three rot diseases and is the most difficult of the three to control once an epidemic has begun. It is caused by Colletotrichum gloeosporioides (Penz.) & Sacc. in Penz. and Colletotrichum acutatum J.H. Simmons. These pathogens coexist in many orchards, whereas in others one or the other species predominates. Glomerella cingulata (Stoneman) Spauld & H. Shrenk is often listed as the sexual stage of C. gloeosporioides but may be a distinct taxon (Shane and Sutton, 1981a; TeBeest et al., 1997).

The Colletotrichum spp. that cause bitter rot survive from one growing season to another in mummified apples, twig cankers and other dead wood in the tree. In the late spring when temperatures begin to warm, conidia are released, initiating infections on fruit. Fruit is susceptible throughout the season (Shane and Sutton, 1981b; Noe and Starkey, 1982). Lesions begin as small, circular, light tan to brown spots, sometimes surrounded by a red halo. As the lesions enlarge, they become brown and sunken (Plate 18.9) and extend into the flesh of the apple in a V-shaped pattern, which is a good diagnostic technique. Fruiting structures on the surface of the lesion, called aecidia, produce copious amounts of salmon-coloured conidia, which can be splash-dispersed to other fruit, initiating new infections. Consequently, the disease has enormous potential for secondary spread and is very difficult to control when the weather is warm and wet.

G. cingulata also survives from year to year on dead wood and mummified apples. There appear to be several strains, which produce somewhat different symptoms. A serious leaf-spot disease, Glomerella leaf spot, occurs on ‘Gala’ in Brazil, which is associated with G. cingulata (Sutton and Sanhueza, 1998). It causes small irregular lesions 3–12 mm in diameter on leaves and can result in significant defoliation when severe. It also causes small corky lesions on fruit. This strain overwinters in leaves on the orchard floor, much like apple scab, and infection is initiated in the late spring by airborne ascospores, which are discharged during periods of rain. Perennation also occurs in twig cankers and mummified apples. Glomerella leaf spot has been observed in the south-eastern USA (Gonzalez and Sutton, 1999). Another strain of G. cingulata produces a large, firm, brown lesion on the fruit, which is not sunken, and few spores are produced on the surface of the lesion (Shane and Sutton, 1981b). It is not known whether this strain causes a leaf-spot symptom.

Management of bitter rot is based on sanitation and a preventive spray programme. In warm, rainy, growing regions, the disease cannot be successfully managed without pruning to remove inoculum sources and facilitate drying in the tree canopy. Current-season fire blight strikes need to be removed because they can be colonized by the bitter-rot fungi and serve as an inoculum source late in the season. The ethylenebisdithiocarbamate (EBDC) fungicides are most effective, but restrictions on their application after petal fall in the USA have limited their usefulness. Captan, ziram and thiram (applied on a 10–14-day schedule from petal fall to harvest) are the most effective fungicides currently registered in the USA. The benzimidazole fungicides are ineffective.

18.3.4.2. Black rot and bot (white) rot

Black rot and bot rot are caused by Botryosphaeria obtusa (Schwein.) Shoemaker and Botryosphaeria dothidea (Moug.) Ces. &
DeNot., respectively. Although caused by the same fungal genus, the diseases caused by the two species are different in many ways. Both overwinter in dead wood, mummified apples and cankers in the tree canopy and produce ascospores and conidia through most of the growing season. Both cause a fruit rot and cankers, but only *B. obtusa* causes a leaf spot. Infections by *B. obtusa* can begin as early as silver tip and occur throughout the season, whereas infections by *B. dothidea* occur mainly during the warm summer months. *B. obtusa* infections develop into firm brown lesions on the fruit and *B. dothidea* infections develop into light brown, soft, watery rots.

*B. obtusa* infections can occur on the sepals as soon as the buds begin to open. These infections spread into the fruit as they begin to mature, resulting in a firm brown rot on the calyx end (Plate 18.10). Other fruit infections can occur from soon after petal fall until harvest during favourable weather (Arauz and Sutton, 1989). Infections that occur soon after petal fall first appear as small, dark pimples. As lesions enlarge, they become dark and irregular in shape and are often surrounded by a red halo. Eventually the entire fruit may become rotten and shrivel, remaining attached to the tree. Leaf spots (known as frog-eye leaf spot) begin as small purple to brown necrotic lesions, which enlarge to 4–5 mm in diameter and are often surrounded by a purple halo. When infections are numerous, leaves may turn yellow and abscise.

*B. dothidea* infections can occur from soon after petal fall until harvest (Parker and Sutton, 1993). Infections that occur early in the season often remain quiescent and do not begin developing until the soluble solids in the fruit begin increasing. This often leads growers to think that their late-season spray programme is ineffective, when in actuality the disease increase that they are observing is more closely related to their spray programme earlier in the season. As lesions begin to develop on the fruit, they extend in a cylindrical manner towards the core. Once the core is invaded, the entire apple becomes infected. Rotten fruit are often light tan in colour and are very soft and mushy (Plate 18.11). Under cooler temperatures, the rot tends to be darker in colour and firmer, making it more difficult to separate from black rot. Infected fruit may fall or may remain attached to the tree and mummify.

Control of the black rot and bot rot is based on sanitation and preventive fungicide sprays. Colonized dead wood and mummified apples can both serve as inoculum sources. Wood infected by the fire blight pathogen can become colonized by these fungi and produce spores during the current growing season. This wood should be removed during the early summer. Prunings should be chopped by a flail mower or pushed out of the orchard and burned. Fungicides should be applied every 10–14 days from petal fall to harvest. Captan and the benzimidazole fungicides are most effective; the dithiocarbamate fungicides are not especially efficacious.

### 18.3.4.3 Sooty blotch and fly-speck

Sooty blotch and fly-speck are two of the most common diseases in humid growing areas. While they do not reduce yield, affected fruit are usually downgraded from fresh-market to processing or juice grades. Sooty blotch is a disease complex caused by several fungi and is characterized by dusty to dark colonies of fungi growing epiphytically on the surface of the fruit (Williamson and Sutton, 2000; Plate 18.12). They range from small discreet colonies to large, amorphous ones. Each colony has a characteristic appearance, depending on the fungus involved. Fly-speck colonies are characterized by numerous thyrothecia, which are scattered throughout the thallus, giving it an appearance of ‘fly-specks’ (Plate 18.13). Colonies range from several to many millimetres in diameter.

Sooty blotch is a disease complex caused by *Peltaster fructicola* Johnson, Sutton & Hodges, *Leptodontium elatius* (G. Mangenot) De Hoog, *Geastrumia polystigmatis* Batista & M.L. Farr and probably other fungi. These fungi grow superficially on the cuticle of affected fruit. All have numerous reservoir hosts in the wooded areas in close proximity...
to orchards and most inoculum comes from outside the orchard. Conidia of *P. fructicola* and *G. polystigmatis* are primarily water-borne and are spread by wind-blown rain; conidia of *L. elatius* are airborne. Infection can occur as early as several weeks after petal fall. Symptom expression is closely associated with the hours of wetting that occur in the spring. Brown and Sutton (1995) found that the first symptoms of sooty blotch (and fly-speck) appeared after an average of 273 h of leaf wetting of 4 h duration or greater accumulated following the first rain which occurs 10 days after petal fall. *P. fructicola*, *G. polystigmatis* and *L. elatius* all produce conidia on fruit, which serve as secondary inoculum. All apple cultivars are equally susceptible, but the colonies are more visible on light-skinned cultivars. Washing and brushing in the packing line can often remove small colonies with lightly pigmented thalli.

Fly-speck is caused by *Schizothyrium pomi* (Mont.: Fr.) Arx (anamorph *Zygophiala jamaicensis* E. Mason). Ascospores of *S. pomi*, which are produced in thyrothecia on reservoir hosts, provide the primary inoculum for infections. While some infections occur on fruit, the most important infections occur on reservoir hosts, which serve as a source of inoculum (conidia) throughout the growing season. Moisture and temperature requirements for colony development are similar to those of the fungi causing sooty blotch. While the fungi that cause sooty blotch grow superficially on the cuticle, *Z. jamaicensis* metabolizes the cuticle and the incipient thyrothecia become firmly attached to it, making the colonies difficult to remove during washing and brushing in the packing line. There are no differences in susceptibility among cultivars to either fly-speck or sooty blotch.

Control of sooty blotch and fly-speck is based on sanitation to remove reservoir hosts, pruning to open the canopy and facilitate drying, thinning fruit clusters to improve drying, and fungicide sprays. The benzimidazole and dithiocarbamate fungicides are most effective; captan is not very effective. The benzimidazole fungicides have some eradicant activity against the diseases. Brown and Sutton (1995) developed a model for timing sprays of benzimidazole fungicides to manage sooty blotch and fly-speck. They found that sprays of benzimidazoles could be omitted in the cover-spray programme until 225 h of wetting had accumulated. In dry years, using the model could save three to five spray applications. Rosenberger (Agnello *et al.*, 1999) and Hartman and Smigell (Hartman, 1995; Smigell and Hartman, 1998) have modified this model for their growing regions.

18.3.4.4 Brooks fruit spot

Brooks fruit spot caused by *Mycosphaerella pomi* (Pass.) Lindau affects apples primarily in the south-eastern and mid-Atlantic apple-growing regions of the USA (Sutton *et al.*, 1987). Symptoms on fruit appear as small, slightly sunken, superficial lesions on the fruit (Plate 18.14). Lesions resemble cork spot, but there is no corky tissue beneath them. Leaf spots appear as small purple flecks on leaves (Plate 18.15). Affected fruit are often downgraded from fresh-market to processing or juice grades.

*M. pomi* overwinters in apple leaves and ascospores are discharged from pseudothecia during rainfall. The ascospores are produced during a distinct period, about 6 weeks in duration, beginning about 2 weeks after petal fall (Sutton *et al.*, 1987). Symptoms do not appear on fruit and leaves until 6–8 weeks after infection. There is no secondary spread. There are some differences among cultivars; ‘Golden Delicious’, ‘Rome Beauty’, ‘Stayman’ and ‘Idared’ are quite susceptible; ‘Delicious’ is relatively resistant. A preventive fungicide program that includes a dithiocarbamate or benzimidazole fungicide controls the disease.

18.3.4.5 Alternaria blotch

*Alternaria* blotch, caused by *Alternaria mali* Roberts (= *Alternaria alternata* apple pathotype), was first reported in the USA in the late 1980s (Filajdic and Sutton, 1991). The disease affects ‘Delicious’ and cultivars with ‘Delicious’ as a parent (e.g. ‘Empire’) and is characterized by circular, necrotic spots on the leaves (Plate 18.16), which, when abun-
dant, can cause extensive defoliation. Defoliation by the fungus is exacerbated by mite injury. *A. mali* and the European red mite act synergistically to increase defoliation (Filajdic et al., 1995). When defoliation is extensive, fruit size and soluble solids are reduced. Fruit symptoms are usually limited to small, corky, dark lesions, often associated with the lenticels.

The fungus overwinters in leaves on the orchard floor and to a lesser extent in leaf buds. Infection by airborne conidia occurs within a month of petal fall if temperature and moisture conditions are favourable. The numerous secondary cycles that can occur throughout the growing season may result in extensive defoliation.

Control of the disease is based primarily on preventive control of mites to minimize defoliation. Most fungicides currently registered for apples in the USA, with the exception of the strobilurins, have little effect on *Alternaria* blotch.

18.3.4.6 Black pox

Black pox, caused by *Helminthosporium papulosum* Berg., is a problem, especially on ‘Golden Delicious’ in the south-eastern USA. It is characterized by small, black, slightly sunken lesions, 3–9 mm in diameter on the fruit (Plate 18.17). Leaf spots begin as red haloes with light green centres, enlarge to 1.5–11.0 mm and turn tan to brown with purple borders (Taylor, 1963). *H. papulosum* overwinters in twig lesions. Conidia produced in these lesions initiate infections during the summer. Black pox is one of the least-studied summer diseases, and conditions favouring infection are not known. Preventive fungicide sprays of dithiocarbamate or benzimidazole fungicides provide effective control.

18.3.4.7 Necrotic leaf blotch of ‘Golden Delicious’

Necrotic leaf blotch is a physiological disorder that affects primarily ‘Golden Delicious’ and its progeny (Sutton and Sanhueza, 1998) and is a particular problem on the new cultivar ‘Pacific Rose’. It is characterized by the sudden appearance of large, irregular lesions, often bounded by veins. Mid-shoot leaves are most severely affected. Lesions are initially pale green, over a few hours turn chocolate brown and, as they age, often appear tan (Plate 18.18). Severely affected leaves turn yellow in a few days and abscise. The disorder appears following periods of cloudy, cool weather during the summer and can result in 50% or more defoliation. It has been associated with an increase in the production of gibberellins, which is triggered by environmental factors. Necrotic leaf blotch can be controlled by applications of heavy metal-containing fungicides, such as ziram, thiram, mancozeb and Bordeaux, or foliar nutrient sprays (e.g. zinc oxide).

18.3.5 Phytophthora crown and root rot

*Phytophthora* crown and root rot is a potentially serious soil-borne disease wherever apples are grown. The disease is prevalent when susceptible rootstocks are planted in heavy soils or in areas of poor soil drainage. Infected trees exhibit a variety of symptoms. During summer, foliage on infected trees may appear light green. As the season progresses, leaves on infected trees turn a reddish-bronze. Branches on infected trees typically have stunted leaves and poor terminal growth. Fruit on infected trees is smaller than normal and colours prematurely. Accurate diagnosis of *Phytophthora* crown and root rot frequently requires digging in order to expose the upper portions of the root system. Infected tissue is reddish brown and delineated from healthy tissue by a definite margin (Plate 18.19). Diseased trees are often randomly distributed throughout a planting. The disease is sometimes confused with the rootstock phase of fire blight.

The *Phytophthora* species present, soil moisture and temperature, relative resistance of the rootstock and contamination of nursery stock all affect the incidence and severity of the disease (Welsh, 1942; Sewell and Wilson, 1959; Browne, 1984; Jeffers and Aldwinckle, 1986; Ogawa and English, 1991). The disease can result from infection by any of several species in the genus *Phytophthora*: *P. cactorum*,
P. cryptogea, P. cambivora, P. megasperma, P. syringae, P. citricola and P. drechsleri. These fungi can persist in soil for extended periods of time as thick-walled sexual spores, called oospores. When soil becomes saturated and temperatures are conducive for sporulation, oospores germinate to produce sporangia. Within sporangia are numerous unicellular motile spores that are liberated into the soil water. The spores, known as zoospores, swim short distances in the soil pore spaces or can be transported longer distances by runoff or irrigation water. When zoospores contact roots of susceptible hosts, they germinate and establish new infections. Zoospores are liberated only when soil is saturated. Prolonged flooding favours the production of sporangia and dissemination of zoospores and may also predispose the host to infection. The majority of new infections are initiated between the pink stage of blossom development and the beginning of shoot elongation. The optimum temperature for disease development depends upon the Phytophthora species present, but in general soil temperatures between 20 and 30°C favour the disease.

Phytophthora crown and root rot is managed through the integration of chemical and cultural practices. Two of the most important decisions of the producer are to plant pathogen-free nursery stock and to avoid the use of susceptible rootstocks. Rootstocks vary in their susceptibility to the various species of Phytophthora. The vast majority of rootstock-susceptibility evaluations have been conducted using P. cactorum. The MM.104 and MM.106 rootstocks are particularly susceptible to the disease, while M.9 is relatively resistant. During planting, orchard managers should ensure that the graft union is not in contact with soil. Heavy or poorly drained soils should be avoided. In irrigated areas, water should be managed to avoid prolonged flooding and the presence of standing water around the base of trees. Plantings are sometimes established on raised beds in order to facilitate drainage around the base of trees. Acylalanine and ethyl phosphonate fungicides available for control of this disease are applied as soil drenches or foliar sprays, respectively (Jeffers and Wilcox, 1990).

18.3.6 European or Nectria canker

Nectria or European canker is circumglobal in distribution and particularly problematic in northern Europe and parts of South America (Grove, 1990). The disease is considered minor in most of North America, but can be a serious problem in the production areas of coastal California. The disease is characterized by zonate cankers located on the main trunk or scaffold limbs (Plate 18.20). Young infected tissue is darker than surrounding healthy tissue. Limb or tree death can occur from girdling, which results from canker enlargement. A layer of callus tissue is formed annually around each canker. The fungus invades callus tissue and spreads to healthy tissue outside the callus layer. Over a period of years this results in cankers with characteristic zonate appearances. In some areas nurseries are important sources of infected trees.

The disease is caused by Nectria galligena Bres. The fungus produces ascospores and conidia in orange-red fruiting-bodies produced in twig, branch or trunk cankers. Spores are produced in a gelatinous matrix and dispersed by the impaction of water droplets. The disease can be aggravated by over-the-canopy irrigation. Infection occurs through leaf scars and pruning wounds. Nectria canker is favoured by cool, moist weather.

An integrated approach is required for successful disease management. Diseased wood should be removed from the orchard and destroyed. Young infected trees should be severely pruned or removed. In some areas diseased bark is removed to prevent spore production. Copper fungicides (e.g. Bordeaux mixture) are sometimes applied prior to autumn rains in order to protect leaf scars.

18.4 Postharvest Diseases

Apples are also host to a multitude of postharvest diseases and disorders. The former are caused exclusively by pathogenic fungi. Two of the more serious postharvest diseases are blue mould and grey mould, which are the first and second most important postharvest diseases of apple, respec-
tively. Most postharvest pathogens invade fruit wounded during harvest, shipping or handling. Therefore, the management of postharvest diseases begins with very careful fruit harvest, bin sanitation and transport. Fruit bins used for transport should be free of soil, leaves and rotten fruit debris. Fruit should be picked individually and placed very gently into bins. Orchard access roads should be smooth and free of ruts and major bumps. Bins should be transported at speeds that will not result in shifting or bouncing. Additional means of postharvest disease management include packing-house sanitation, rapid immediate postharvest cooling, fungicide drenches and the provision of adequate tree nutrition.

18.4.1 Blue mould

Blue mould, which is caused by fungi in the genus *Penicillium*, is the most common postharvest disease of apple (Rosenberger, 1990; Plate 18.21). Decayed flesh is soft and watery and separates readily from healthy tissue. Infected epidermal tissue is light to dark brown. Infected fruit are malodorous. The causal organism produces blue or blue-green conidia on the surface of infected fruit. *Penicillium* spp. are present in orchard soils and on decayed fruit in the orchard and packing-house and in postharvest drench solutions and flume water. Fruit infection typically occurs through wounds. Wounding should be prevented during harvest, shipping and processing. Infested fruit bins and warehouses should be disinfested before processing fruit.

18.5 Diseases Caused by Viruses, Viroids, Phytoplasmas and Other Virus-like Agents

Viruses or virus-like agents incite over 50 described diseases of apple. These agents include viruses, viroids and phytoplasmas and several graft-transmissible agents that have yet to be identified. Some of these diseases have great economic consequences in that they are associated with fruit deformities or severe tree decline and even death. The degree of affliction depends greatly on the rootstock and scion selection, pathogen isolate and climate. Viruses or virus-like agents that incite severe symptoms on some apple selections will cause no obvious symptoms on others. However, even in the latter case, significant yield reductions have been documented in the few studies undertaken to study such effects (van Oosten, 1983). Thus, infections by viruses and virus-like agents are important to the commercial production of apples.

Although most of these agents do not spread naturally, many are common in commercial orchards. The universal presence of many of these agents is due to collecting grafting material from infected but apparently symptomless trees while preparing for vegetative propagation of new trees. The propagation of trees from infected sources ultimately affects yield and, under certain environmental circumstances or clone combinations, results in orchards that are not economically productive due to fruit-quality issues or tree decline. The only reasonable method for controlling these diseases is the initial use of virus-tested propagation materials.

Quick and reliable diagnostic assays are available for a few of the viruses and virus-like agents that infect apple. However, for the most part, detection of such infections is a cumbersome and laborious proposition. Furthermore, only a few of the agents that incite these diseases have been identified. The use of modern laboratory diagnosis is precluded until such identifications are made. Most assays for these agents are conducted by inoculative assays on sensitive woody indicator selections; these tests require 2 months to 3 years to complete.

Aetiological studies of viruses that infect woody plants are greatly enhanced if the virus is first transmitted to a herbaceous host. Unfortunately, many of the virus-like agents that elicit disease in apple trees have not been successfully transmitted to herbaceous plants and therefore are called ‘non-sap-transmissible’. This may simply mean that the correct combinations of host plants and transfer conditions have not yet been
found. The significance of this limitation is that progress towards characterization of these pathogens has been dramatically slowed. Nevertheless, the non-sap-transmissible viruses of fruit trees present a very interesting and challenging group of viruses, since relatively little is known about them. Contemporary methods in molecular biology have accelerated investigation of these pathogens.

Pathogens are frequently referred to as viruses based on the ability to transmit the pathogen by grafting and/or budding and the absence of any other obvious pathogens. This simplistic distinction is often incorrect. Many disease-causing agents are more correctly referred to as ‘virus-like’ agents since no direct association between a pathogen and disease has been established – that is, Koch’s postulates have not been fulfilled.

The disease names commonly used in the literature are descriptive of the disease and host but provide little information about the pathogen. Consequently, a pathogen or variant thereof may be associated with different disease names, depending on the symptom produced, the latter frequently affected by cultivar and environment. With advances in our ability to isolate and characterize fruit-tree pathogens at the molecular level, this complicated structure of nomenclature is evolving to reflect more accurately the nature of the pathogen, rather than the symptoms that they elicit. This is of practical as well as academic importance. By relating a pathogen to other members of a group that share many important qualities, we may be able to predict the behaviour of that pathogen based on the epidemiology of a few well-studied pathogens in the same group.

The list of apple diseases caused by ‘virus-like’ agents is alarming in its length. However, many of these disease names were applied locally to a particular symptom and thus many of the names are duplicative. Also, many of these diseases have very limited distribution. Only diseases that remain of a more general importance will be discussed in detail. For an expanded list of reported diseases of apple that are induced by virus-like agents, see Németh (1986).

### 18.5.1 Chlorotic leaf spot

Relatively few well-characterized viruses are routinely found in the *Malus* of commerce (Table 18.3). Each of these viruses incites disease with major characteristics on selected host cultivars that help identify the disease agent.

The causal agent apple chlorotic leaf-spot trichovirus (ACLSV) is one of the most widely distributed viruses of fruit trees. Although first described as a virus in apples, it was subsequently found to be very widespread in stone fruits, where it can cause severe losses in production. Although the interaction of ACLSV with many commercial apple cultivars is latent, it was first discovered associated with the

<table>
<thead>
<tr>
<th>Disease</th>
<th>Causal agent</th>
<th>Known means of transmission</th>
</tr>
</thead>
<tbody>
<tr>
<td>Apple chlorotic leaf spot</td>
<td>Apple chlorotic leaf-spot trichovirus</td>
<td>Grafting</td>
</tr>
<tr>
<td>Apple decline</td>
<td>Apple stem-grooving capillovirus</td>
<td>Grafting</td>
</tr>
<tr>
<td>(on Virginia crab)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Apple mosaic</td>
<td>Apple mosaic ilarvirus</td>
<td>Grafting</td>
</tr>
<tr>
<td>Apple mosaic (Tulare)</td>
<td>Tulare apple-mosaic ilarvirus</td>
<td>Grafting</td>
</tr>
<tr>
<td>Apple stem grooving</td>
<td>Apple stem-grooving capillovirus</td>
<td>Grafting</td>
</tr>
<tr>
<td>Apple stem pitting</td>
<td>Apple stem-pitting foveavirus</td>
<td>Grafting</td>
</tr>
<tr>
<td>Apple union necrosis</td>
<td>Tomato ringspot nepovirus</td>
<td>Nematodes and grafting</td>
</tr>
<tr>
<td>Flat apple</td>
<td>Cherry rasp-leaf nepovirus</td>
<td>Nematodes and grafting</td>
</tr>
<tr>
<td>Spy decline</td>
<td>Apple stem pitting foveavirus</td>
<td>Grafting</td>
</tr>
</tbody>
</table>
decline and death of new Malus breeding lines in an apple-scab-resistance breeding programme. Over the past few decades, there have been several examples where the interaction of the virus with specific rootstocks is not latent. The top-working disease of Japan is believed to be a hypersensitive reaction of rootstocks \((\text{Malus prunifolia var. ringo})\) to isolates of ACLSV found in contaminated apple scions from North America. This resulted in significant failure of mature trees (reviewed in Mink, 1989). The death of cells at the graft union results in weakened trees with reduced vigour and productivity and which are very susceptible to wind damage.

ACLSV is diagnosed by serological testing, indexing on herbaceous hosts \((\text{Chenopodium quinoa})\) or budding to Malus indicator species, usually \(\text{Malus sylvestris cv. R12740-7A}\) (also known as ‘Russian’). In the latter case, chlorotic leaf symptoms appear soon after new leaves develop unless the ambient temperature rises above 20°C. Low virus titre and inhibitors in \(\text{Malus}\) often limit the use of serology or indexing on herbaceous plants. Molecular techniques of diagnosis, such as RT-PCR, are becoming more common methods of diagnosis (Kummert et al., 1995; Kinard et al., 1996). The extreme strain variation of ACLSV has limited the widespread acceptance of serological and molecular methods.

18.5.2 Apple decline (on ‘Virginia Crab’)

Infection of most apple trees with apple stem-grooving capillovirus (ASGV) is latent – that is, there are no acute symptoms visible. However, when infected scion material is grafted on to sensitive rootstocks, such as \(\text{M. sylvestris cv. ‘Virginia Crab’}\), growth and development of the ‘Virginia Crab’ wood cylinder is impaired and deep grooves appear under the bark. The vascular tissue of the ‘Virginia Crab’ often becomes necrotic, resulting in weakness and a visible brown line at the graft union. The trees often break at this necrotic union (Németh, 1986). Stem grooving caused by ASGV is not seen on current commercial apple cultivars, but appears on rootstocks with crab apple in their heritage.

ASGV can be diagnosed by herbaceous indexing on \(\text{C. quinoa}\), although this method is not very reliable. Woody indexing on \(\text{Malus micromalus GMAL273.a or M. sylvestris cv. ‘Virginia Crab’ (Howell et al., 1995)}\) or detection by RT-PCR is the preferred method of testing (Kummert et al., 1995; Kinard et al., 1996).

18.5.3 Flat apple

This disease is caused by cherry rasp-leaf nepovirus (CRLV) (Parish, 1977), a virus that is believed to be native to western North America, ranging from Utah to southern British Columbia. CRLV is believed to have originated in one or two species of native vegetation and subsequently been transmitted into horticulturally important crop plants, such as apple and cherry. The symptoms in apple are most striking on the fruit of ‘Red Delicious’ and related cultivars. The length of the fruit is significantly reduced to produce a fruit that is squat, and the stem cavity is almost absent (Plate 18.22). At first, only a few fruit will be affected, but eventually the entire tree will be involved.

CRLV is transmitted by the nematode \(\text{Xiphinema americanum (sensu lato)}\) (Wagnon et al., 1968). Therefore, secondary spread of the disease is relatively slow. Apple trees planted on sites previously planted to rasp-leaf-diseased cherry trees can become infected. In addition to fruit trees, many orchard weeds, such as dandelions and plantains, are hosts, although they exhibit no symptoms of the disease. This makes elimination of the virus from an infected orchard very difficult. The alternative hosts and/or the nematode vectors would have to be eliminated to protect replanted trees.

Since CRLV achieves high concentration in young shoots, it can be readily detected by serology and by mechanical inoculation of \(\text{C. quinoa}\) with extracts from young leaves in early spring. Leaf symptoms and tip necrosis on \(\text{C. quinoa}\) usually develop 3 days after inoculation.
18.5.4 Apple mosaic

Apple mosaic ilarvirus (ApMV) induces dramatic white bands or patterns on leaves of many apple cultivars (Plate 18.23). Affected leaves may have irregular distribution through the tree or even on individual limbs. The severity of the symptoms can vary dramatically from year to year, with symptoms being almost undetectable in seasons with high temperatures during the growing season. This disease can result in significant production losses (ranging up to 40%) depending on the cultivar, virus isolate and environment (Posnette and Cropley, 1956).

ApMV is detected by serological assays but results (when apple tissue is tested) are variable. Therefore, serological testing is best limited to confirming the identity of the pathogen in herbaceous indexing. ApMV can also be detected by inoculating field-grown trees of ‘Golden Delicious’ apple. Herbaceous indexing on C. quinoa is possible but quite unreliable. Molecular methods of detection, such as RT-PCR (Rowhani et al., 1995), are becoming more widely accepted, as the methods are evaluated with more and varied isolates of the virus.

18.5.5 Stem pitting

The viral nature of apple stem pitting was discovered soon after the Second World War in mid-western North America (Guengerich and Millikan, 1956). Production of crab apples had become uneconomic and old crab apple rootstocks were budded over to standard cultivars, such as ‘Delicious’. Two or three years after budding, the crab apple rootstocks became pitted and severely weakened. This is an example where the ‘latent’ virus in the ‘Delicious’ scion resulted in significant economic damage when the variety was budded on to rootstocks with crab apple in their heritage. Apple stem-pitting foveavirus (ASPV), the causal agent of apple stem-pitting disease, is thought to have been distributed worldwide in contaminated apple and pear propagation material.

Woody indexing on any one of a variety of Malus species is the method most frequently used to detect ASPV. Epinasty and decline may be observed after 1 year when ‘Spy227’ is the recipient. ‘Radiant’ crab apple is a more reliable detection host. It is sensitive to a wide range of ASPV isolates and displays severe epinasty and decline within 6 weeks under controlled greenhouse conditions. The development of stem-pitting symptoms on ‘Virginia Crab’ is also reliable, but at least two growing seasons are required for trustworthy results. Fruit with ridges and flutes are produced if the ‘Virginia Crab’ is allowed to bear. Molecular methods are also available for the detection of ASPV. RT-PCR (Nemchinov et al., 1998) is sensitive and offers faster diagnosis relative to woody indexing.

18.5.6 Union necrosis

Union necrosis is caused by tomato ringspot nepovirus (TmRSV) (Stouffer and Uyemoto, 1976). Symptoms of infection generally do not appear until the tree reaches bearing age, at which time the tree assumes an unthrifty growth habit. The leaves and bark are reddish in colour, and bloom and fruit set are abnormally high. Depending on the rootstock/scion combination, the results of infection may range from very mild to a rapid decline in tree health, resulting in death. In severe reactions (‘Red Delicious’ on MM.106), the union of rootstock and scion will reveal a dark necrotic line spanning the union, with soft, spongy, orange bark flanking the union. This disease is common in several locales along the east coast of North America, and has been reported sporadically in the Pacific Northwest of the USA. TmRSV is transmitted by dagger nematodes, X. americanum (sensu lato), which are abundant in eastern fruit-growing areas of North America (Stouffer and Powell, 1989). Other species belonging to this nematode group, Xiphinema revesi and Xiphinema californicum, are also known to be vectors of TmRSV in orchards.

TmRSV is easily transmitted from young leaves to C. quinoa by mechanical inoculation with crude extracts. Prunus tomentosa is a diagnostic woody host for this virus. Alternatively, serological and RT-PCR (Griesbach, 1995) assays can be used to detect it.
18.6 Diseases Caused by Phytoplasmas
(Table 18.4)

18.6.1 Proliferation

Apple proliferation occurs throughout much of Europe, but has not been reported in the western hemisphere. The disease is caused by an apple proliferation phytoplasma. The pattern of occurrence in European orchards suggests that the pathogen is harboured in the native weed population, from which it moves into the orchards. The vector of this pathogen is at least one species of leafhopper, *Fieberiella florii*. This disease reduces fruit size by up to 30–70% and fruit have longer than normal peduncles. However, the most striking symptom, for which the disease is named, is the proliferation of vegetative shoots, otherwise known as witches'-broom. In addition, apple leaves exhibit greatly enlarged stipules. Because the disease is debilitating and insect-vectored, apple proliferation disease has important quarantine significance in the western hemisphere.

The most widely accepted detection method for apple proliferation phytoplasma has been woody indexing in the field. Bark patches from roots of the test plant are budded on to ‘Golden Delicious’ and the tree is observed for 2 years for the appearance of witches'-broom and the enlarged stipules. However, the long observation period and concern about reliable transmission of the pathogen encouraged the development of alternative detection methods. Detection by PCR (Lorenz et al., 1995) is becoming widely accepted and the techniques are becoming increasingly refined to improve the reliability of molecular methods (Carraro et al., 1998; Skrzeczkowski et al., 2001). PCR is now the preferred test method.

18.6.2 Chat fruit

Apple chat fruit is believed to be caused by phytoplasma infection. Sensitive cultivars develop undersized fruit, many of which drop in June. The remaining fruit do not develop colour properly and may exhibit dark water-soaked spots. Many of the current commercial cultivars do not express any symptoms of apple chat fruit. The pathogen is detected by budding on to ‘Lord Lambourne’. An incubation period of up to 3 years is required for reliable detection. Until a firm association of a biological agent with apple chat-fruit disease can be established, the use of biological indicators remains the only means of detection.

18.7 Diseases of Apple Caused by Viroids
(Table 18.5)

Viroids are sub-virus particles that are difficult to detect. Since proteins are not part of their structure, serological methods are not useful. Woody indexing and molecular detection methods are commonly used to diagnose these pathogens.

18.7.1 Blister bark

Apple blister bark is characterized by areas of the bark taking on the appearance of orange tissue-paper. The disease can be induced by apple fruit-crinkle viroid (AFCVd) (Ito et al., 1993). As the underlying tissue becomes desiccated, the bark cracks and peels. Scarring of the underlying tissue occurs. Several mineral deficiencies can mimic infection by AFCVd, so proper diagnosis is important.

<table>
<thead>
<tr>
<th>Disease</th>
<th>Causal agent</th>
<th>Known means of transmission</th>
</tr>
</thead>
<tbody>
<tr>
<td>Apple chat fruit</td>
<td>Phytoplasma</td>
<td>Grafting</td>
</tr>
<tr>
<td>Apple proliferation</td>
<td>Apple-proliferation phytoplasma</td>
<td>Leafhoppers and grafting</td>
</tr>
</tbody>
</table>

Table 18.4. Diseases of apple suspected to be caused by phytoplasma.
18.7.2 Dapple apple

Dapple apple aptly describes one of the diseases caused by apple scar-skin viroid (ASSVd). Affected fruit, especially near the calyx end, bear small circular spots, which enlarge and increasingly contrast with the background colour as the fruit matures (Plate 18.24). Larger spots may coalesce to produce dappling or a broad zone of discoloration. There are no pronounced leaf or bark symptoms. Since many commercial cultivars do not express symptoms, this disease has been particularly problematic when old orchards are top-worked to change cultivars. The original cultivar may not express symptoms, whereas the new cultivar does. Pear also appears to be a symptomless carrier of ASSVd; the role of pear as a symptomless carrier of this pathogen in apple-disease aetiology is unknown. ‘Stark’s Earliest’ (‘Scarlet Pimpernel’) or ‘Delicious’ can be used for woody indexing, and three crops must be observed for accurate assessment of viroid status. ‘Scarlet Pimpernel’ grown at 18ºC under constant light for 2 months will yield diagnostic symptoms on its foliage (Skrzeczkowski et al., 1993). Molecular techniques are preferred for their speed and reliability. Both RT-PCR (Hadidi and Yang, 1990) and hybridization (Hadidi et al., 1991) assays are used for ASSVd.

Apple scar-skin disease is a more severe disease caused by ASSVd, as compared with the milder one referred to as dapple apple. Symptoms usually begin at the distal portion of the fruit and the severity increases as the fruit matures. Small discoloured circles merge to form large green-brown to brown patches (Plate 18.25). Eventually, the brown patches become necrotic and fissures appear on the fruit. Diseased fruit are significantly smaller than fruit from uninfected trees. Diagnosis is performed as described for dapple-apple disease. The dapple and scar-skin diseases evoked by ASSVd appear to be dependent upon climatic and cultivar differences. The original isolates of each produce similar diseases on fruiting trees of ‘Scarlet Pimpernel’ (W.E. Howell, Washington State University, unpublished data).

18.7.3 Dimple fruit

Apple dimple fruit caused by apple dimple-fruit viroid (ADFVd) has been observed in several commercial cultivars of southern Italy (DiSerio et al., 1996). The disease induces depressions on the fruit surface as it matures. Under the depression will be an area of necrotic tissue.

18.7.4 Fruit crinkle

Apple fruit-crinkle disease is caused by isolates of AFCVd (Ito et al., 1993). The viroid was described in Japan, where fruit symptoms are most severe on the cultivar ‘Ohrin’. This pathogen can be detected by woody indexing on the apple cultivars ‘Delicious’ or, preferably, ‘NY5822’, where it induces symptoms of blister bark (Ito et al., 1993).

18.8 ‘Virus-like’ or Graft-transmissible Diseases of Apple with No Known Causal Agents (Table 18.6)

There are many diseases of apple for which no pathogen has been identified. The disease
names are generally descriptive of the symptoms. Some may be caused by pathogens described above but, until more information is obtained about their aetiology, the causal agents remain an enigma. Since the agents for these diseases have not been identified, confirmation of the pathogen depends on symptom expression on susceptible cultivars. All are graft-transmissible. Furthermore, as most of these diseases are spread primarily through the use of infected propagation material, the creation of virus-certification programmes over the past 40 years has essentially eliminated many of these diseases from commercial apple production. Still a few continue to cause concern.

18.8.1 Green crinkle

Apple green-crinkle disease, a severely debilitating disease of some apple cultivars, is always associated with trees infected with ASGV, ASPV and ACLSV. The individual viruses have not been separated to determine if this disease symptom is induced by a mixture of viruses, by a particular isolate of one of the viruses or by another as yet uncharacterized pathogen. Apple green-crinkle disease is diagnosed by woody indexing on the apple indicator ‘Golden Delicious’. The trees are observed for the development of fruit symptoms during the following three crops. Symptoms begin to appear after the fruit reaches 1–2 cm in diameter. Fruits develop deep depressions and distortions, which become more severe as the fruits mature. Cracks may develop in pits and crevices (Plate 18.26). Depressions are linked through the flesh to the vascular system by discoloured tissue. Severe fruit symptoms may appear on only one or two limbs of an infected tree, and there are no acute foliar symptoms associated with the disease (Thomsen, 1989). Budding and grafting of contaminated propagation material is the major mechanism by which apple green-crinkle disease is spread. If field spread occurs, it is very slow and possibly by root grafting only.

18.8.2 Rubbery wood

Apple rubbery wood affects 2-year-old wood of apple and pear trees. On sensitive cultivars, branches develop that are very pliable and droop under their own weights (Waterworth and Fridlund, 1989). Affected limbs are also very sensitive to cold and frost damage. The disease originated in Europe and was later introduced to North America with infected rootstock. Many older rootstocks introduced from Europe were uniformly infected with rubbery wood. In countries with active certification programmes, this disease has become increasingly rare. The degree of symptom development is dependent on climate. The pliable limbs and poor growth are more extreme in moderate climates, relative to warmer environments. No obvious symptoms are associated with many commercial cultivars. Nevertheless, fruit yield and quality can be adversely affected (van Oosten, 1983). The pathogen has been assumed to be a phytoplasma, based on electron-microscopic examination of affected tissues, but attempts to confirm this association by molecular techniques have been contradictory (Poggi Pollini et al., 1995; Bertaccini et al., 1998). The disease is detected by indexing

---

**Table 18.6. Graft-transmissible diseases of apple for which there are no known causal agents.**

<table>
<thead>
<tr>
<th>Disease</th>
<th>Disease</th>
<th>Disease</th>
</tr>
</thead>
<tbody>
<tr>
<td>Apple blister bark</td>
<td>Apple green crinkle</td>
<td>Apple ringspot</td>
</tr>
<tr>
<td>Apple dead spur</td>
<td>Apple horseshoe wound</td>
<td>Apple rosette</td>
</tr>
<tr>
<td>Apple decline</td>
<td>Apple internal bark necrosis</td>
<td>Apple rough skin</td>
</tr>
<tr>
<td>Apple false sting</td>
<td>Apple leaf pucker</td>
<td>Apple rubbery wood</td>
</tr>
<tr>
<td>Apple flat limb</td>
<td>Apple ring russet</td>
<td>Apple pustule canker</td>
</tr>
<tr>
<td>Apple freckle scurf</td>
<td>Apple ring-line pattern</td>
<td></td>
</tr>
</tbody>
</table>

*There are many more described but rare graft-transmissible diseases of apple (Németh, 1986).*
on ‘Lord Lambourne’, followed by 3 years of observation for the development of limbs lacking normal lignification.

Apple flat limb is believed to be caused by the same agent that causes apple rubbery wood (see above). Apple flat limb is only observed in those areas where sensitive cultivars, such as ‘Gravenstein’, are grown. The disease results in flattening of the shoots and branches on limbs that are 2–3 years old and eventually leads to deep furrows that become more severe as the branches become older (Fridlund and Waterworth, 1989). In addition to reduced vigour and production, the weak limbs are susceptible to easy breaking. Severely affected limbs are also more susceptible to winter or frost damage. The disease is detected by woody indexing on ‘Gravenstein’ or ‘Lord Lambourne’, followed by 3 years of observation.

18.8.3 Dead spur

Apple dead spur was originally observed in the western USA, but has since been reported throughout North America and in Europe and Asia (Parish, 1989). The characteristic symptom is death of fruiting spurs, with the resulting development of long segments of blind wood. This is most pronounced on the interior of the tree, so the canopy in the centre of the tree is sparse, with fruiting spurs at the shoot tips. The disease is spread through the use of infected propagation material. No natural spread has been observed. The only means by which the diagnosis based on symptoms can be confirmed is bud-inoculating spur-type ‘Delicious’ trees. Symptoms will develop in the third or fourth year after inoculation. Since this disease is difficult to diagnose, it may be overlooked in virus-testing programmes. Like green-crinkle disease, dead spur has not been observed in the absence of ACLSV, ASPV or ASGV. Therefore, although the dead-spur agent poses a concern for the safe propagation of healthy apple trees, elimination from propagation programmes of trees with these three ‘sentinel’ viruses may well indicate freedom from this and other graft-transmitted diseases of unknown aetiology.

18.9 Control Measures

Very few viruses or virus-like agents of apple disease spread naturally in the orchard. This means that the most efficient and cost-effective control measure for apple virus diseases is the use of propagation material and plants that are certified to be free from viruses. Once the trees are planted in the orchard, they should, for the most part, remain virus-free. Still, some spread of these pathogens occurs in orchards via tree-to-tree root grafts. Such root grafts provide effective means by which pathogens can move from an infected tree to the next. However, this process is relatively slow and involves spread only to adjacent trees. Thus, if a diseased tree is detected in an established orchard, it may be prudent to remove adjacent trees to eliminate the virus.

In those cases where some form of natural field transmission exists, diseases can be much more difficult to control. Again, initial planting of certified virus-free material is the first step in maintaining a healthy orchard. In European countries, where apple proliferation phytoplasma is prevalent, regular insecticide applications to control the leafhopper vector can significantly reduce the incidence of this disease (Kunze, 1976).

The two diseases of apple whose causal agents are known to be transmitted by nematodes, flat apple and union necrosis, can be very difficult to control. Exclusion of the virus initially is crucial since, once the virus is present, it is very difficult to eliminate it from an orchard. Dagger nematodes, *Xiphinema* spp., are nearly universal and difficult to eradicate. Fumigation and soil pretreatment before planting will reduce but not eliminate these nematodes from soil. Leaving the ground planted in plants that are not virus hosts for 1–2 years before replanting will help prevent transmission of these viruses to the young trees. The nematodes lose the ability to transmit these viruses with each moult. Since many weed plants common in the orchard floor, such as dandelion and plantain, act as reservoirs of these two nepoviruses, intense weed control is required during this period of fallow. If these viruses and their nematode vectors are present in an area, it is best to select rootstock/scion combinations that offer protection against the nematode and/or virus disease.


Sewell, E.W.F. and Wilson, J.F. (1959) Resistance trials of some apple rootstock varieties to 


