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Diseases of Deciduous Fruit Trees Incited by Pseudomonas syringae van Hall

A Review of the Literature With Additional Data

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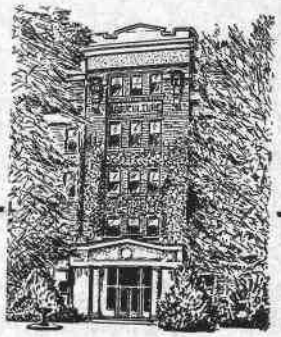

CONTENTS

AUTHOR: H. Ronald Cameron is Associate Professor of Plant Pathology, Oregon State University.

Diseases of Deciduous Fruit Trees Incited by Pseudomonas syringae van Hall

A Review of the Literature With Additional Data

H. Ronald Cameron

HISTORY

The diseases caused by *Pseudomonas syringae* van Hall were investigated in the early nineteen hundreds in several parts of the world (20).[1] Barss (10) suggested that that first work on the disease was done in Europe about 1850. According to Wormald (160), descriptions of the disease were mentioned in the Worburn Fruit Farm reports as early as 1896. The first formal description was apparently made by Sorauer (131). Later Professor Ritzemo Bos worked on the disease (16). His work was continued by Dr. Beijerinck, who in turn gave the project to van Hall. Van Hall's report in 1902 (132) was generally accepted as the first evidence that a bacterium was the cause of cankers and gumming in fruit trees (19, 81).

Papers in 1906 and 1907 by Aderhold and Ruhland (1, 2) established that bacteria caused gum-flow in fruit trees in several of the German fruit-growing areas. These papers were followed in 1911 by Griffin's report of a bacterial bud blight of dormant sweet cherry trees in Oregon (77, 78). Barss (11) later demonstrated that some of the bacteria isolated by Griffin could also cause cankers on limbs. Barss (10) mentioned the occurrence of serious outbreaks of bacterial canker in Oregon as early as 1853.

In 1926, Smith (124) pointed out the close relationship between the organisms causing diseases of avocado, lilac, and citrus. By 1928 Wormald (148) had shown that bacteria were the major cause of cankers in English plum orchards. Rosen and Bleecker in 1933 (117), found difficulty in distinguishing between the bacteria causing bacterial canker in stone fruits, blast on pears, and lilac blight. The synonymy of the organisms reported to cause these diseases was proven independently by Smith and Fawcett in 1930 (126) and Wilson in 1935 (143). Since the organisms causing bacterial canker, pear blast, and avocado blight were now considered to be identical

[1] Numbers in parentheses refer to the Literature Cited, page 57.

with the bacterium causing lilac blight, it became necessary to use *Pseudomonas syringae* van Hall as the correct binomial (133, 126).

Some English workers have been confronted with *Ps. syringae* and a different, although similar, species, *Ps. mors prunorum* Wormald (148, 169). Since the symptoms produced by the two species are almost identical, the two species will be considered as one throughout the following discussion.

In recent years research has been directed toward the disease cycle, methods of infection, classification of strains, and methods of control. Many of these questions are as yet unsolved, and it is hoped that a review of past work will direct attention to areas that can profit by additional investigation.

Host Range

The host range of *Ps. syringae* is wide and extends into many families of plants. Most of the stone fruits have been reported as susceptible to at least one isolate. Peach, *Prunus persica* Batsch. (4, 49, 104, 126); sweet cherry, *P. avium* L. (4, 96, 104, 126); *P. mahaleb* L. (4, 82); sand cherry, *P. pumila* L. (4, 126); Japanese plum, *P. salicina* Lindl. (4, 126); *P. salacina* Lindl. X Wild goose plum, *P. munsoniana* Wight and Hedr. (4); apricot, *P. armeniaca* L. (9, 31, 115, 147); plum, *P. domestica* L. (149, 153); and apricot plum, *P. simonii* Car. (4, 11), are listed as susceptible members of *Prunus*. Other plants that have been reported to be susceptible include: foxtail, *Alopecurus* sp. (146); orach, *Atriplex hortensis* L. (82); *Impatiens balsamina* L. (143); *Lupinus* sp. (82); Natal plum, *Carissa grandiflora* A. DC. (126); sour orange, *Citrus aruantium* L. (126); *Citrus* sp. (30, 35, 48, 50, 57, 65, 66, 67, 68, 69, 95, 125, 127, 128, 129, 146); lemon, *Citrus limon* Burm. (48, 122, 126); sweet orange, *Citrus sinensis* Osbeck (71, 126); *Coprosma baueri* Endl. (126); *Fagopyrum* sp. (82); golden bells, *Forsythia* sp. (48, 146); flowering ash, *Fraxinus ornus* L. (126); Himalayian ash, *Fraxinus floribunda* Wall. (126); rose-mallow, *Hibiscus* sp. (146); jasmine, *Jasminum mesnvi* Hance (126); English Walnut, *Juglans regia* L. (126); tomato, *Lycopersicon esculentum* Mill (126, 135); apple, *Malus sylvestris* Mill (82, 126, 146); stocks, *Matthiola* sp. (48); orange jasmine, *Murraya exotica* L. (126); plantain, *Musa paradisica* L. (126); oleander, *Nerium oleander* L. (126); pearl millet, *Pennisetum glaucum* R. Br. (146); avocado, *Persea americana* Mill. (48, 50, 123, 124, 126, 146); lima bean, *Phaseolus limensis* Macf. (146); dwarf bean and common bean, *Phaseolus vulgaris* L. (48,

146); Eugene poplar, *Populus canadensis* var. *eugenei* Schelle, (48); black poplar, *Populus nigra* L. (82); *Populus* sp. (126); pear, *Pyrus communis* L. (57, 43, 116, 117, 142, 146); coast live oak, *Quercus agrifolia* Nec. (126); interior live oak. *Quercus wislizenii* A. DC. (126); rose, *Rosa odorata* Sweet. (50, 57, 118, 146); sorghum, *Sorghum vulgare* Pers. (146); *Syringae amurensis* var. *japonica* French & Sav. (126); *Syringae laciniata* Mill. (126); common lilac, *Syringae vulgaris* L. (50, 82, 124, 126, 146); clover, *Trifolium* sp. (146); cowpea, *Vigna sinensis* Savi. (48, 146); and corn, *Zea mays* L. (146).

The following species are listed as susceptible to *Ps. morsprunorum:* sweet cherry, *P. avium* L. (38); peach, *P. persica* Batsch. (38, 164); common plum, *P. domestica* L. (38); sour cherry, *P. cerasus* L. (38, 164); apricot, *P. armeniaca* L. (38); flowering almond, *P. triloba* Lindl. (38); myrobalan plum, *P. cerasifera* Enrh. (164); purple leaved plum, *P. cerasifera* var. *pissardii* Kochne (6, 38, 164); almond, *P. amygdalus* Batsch. (6, 38, 164); and *P. sibirica* L. (38).[1]

Because of the rather artificial classification of plant pathogenic bacteria and the apparent existence of numerous strains of *P. syringae,* it is not surprising that most isolates will not go to all of the listed hosts. It is very common for one isolate to be pathogenic on five or six hosts and not cause any symptoms on other plants. A second isolate from the same original host may have either a very different host range, or it may overlap the host range of the first isolate. An example of this phenomenon is reported by Vaughan (134) where two isolates, one from lilac and one from blueberry were both capable of causing symptoms on the other host, but only the isolate from lilac was pathogenic on sweet or sour cherries. The author has had similar experiences when 12 cultures of *Ps. syringae* were inoculated into lemon fruit. Only 60% of the isolates were pathogenic on lemon, even though all of the isolates were pathogenic on sweet cherry and appeared to be identical by morphological and physiological tests.

Because of the observed differences in pathogenicity of strains of *Ps. syringae,* the host range of an isolate cannot be used as a valid criterion for classification according to the present concept of the species.

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[1] Scientific and common names of reported hosts are changed to conform with the classification of L. H. Bailey (8).

Geographic Distribution

Ps. syringae apparently occurs in all major fruit growing areas of the world. On stone fruits it has been reported from Australia, all of the British Isles, Denmark, France, Germany, Holland, New Zealand, northern and western United States (4, 38, 74, 78, 96, 97, 98, 99, 110, 112, 113, 136, 137). Symptoms on citrus, caused by the same bacterium, have been observed in Argentina, California, Cypress, Greece, Italy, Japan, New South Wales, Palestine, Russia, South Africa, South Australia. Tunisia, and Victoria (15, 71, 100, 120, 121). If the countries in which symptoms on lilac have been reported and other hosts were added to the list, distribution would be worldwide.

Common Names

Because of the wide host range, the numerous strains of bacteria, and the variable symptoms on different hosts, the diseases incited by *Ps. syringae* have been given numerous common names. Blossom blast, gumnosis, bacterial canker, blast of stone fruit, die-back, sour-sap, shoot blight, spur blight, wither tip, bacterial sour-sap, cherry gumnosis, and bacteriosis have all been used to describe at least one phase of the symptoms caused by *Ps. syringae* on stone fruits (4, 10, 11, 57, 76, 146, 148, 152). In many cases, the same names have also been used for other diseases, thereby adding to the confusion. Recent usage in both the United States and Europe has favored the common name of bacterial canker, and it is recommended that this terminology be encouraged since it leaves little doubt as to the cause of the most severe symptom. On hosts other than *Prunus,* there are also numerous names such as pear die-back, pear blast, blast of citrus, black pit of citrus, and lilac blight (50, 51, 70, 94, 152).

Economic Significance

The economic importance of *Ps. syringae* is difficult to assess because of serious damage to trees as well as to yields. Hutton considers bacterial canker to be one of the two most important diseases of cherry in New South Wales (96). Crosse and Moore list *Ps. syringae* and *Ps. mors-prunorum* among the most important economic plant diseases of Great Britain (38, 108). Crosse states that *Ps. mors-prunorum* is by far the most serious parasite of prunes in Great Britain (38). Wormald lists tree losses of 10, 25, 32, and 43% in young plum orchards (151, 155). Aderhold and Ruhland observed losses of 30 to 40% of the trees in 3- to 5-year-old grafted cherry

and 5 to 6% loss in 2- to 4-year-old plums. Losses in Oregon cherry orchards vary considerably and are usually related to the quality of the planting stock, the site of the orchard, and cultural practices. Where conditions have been favorable for disease development, tree losses of 75% have been observed in young orchards. Under normal conditions losses between 10 and 20% are not uncommon. In bad years yield losses from dead-bud conditions may be as high as 80%. Large acreages of susceptible varieties free from canker can be grown in Oregon, but orchards in this condition are rarely found.

Symptoms

Most of the symptoms caused by *Ps. syringae* and *Ps. morsprunorum* are similar, and usually consist of a rapid wilting and dying of shoot tips, twigs, or flowers. In other cases, symptoms are associated with canker development and may result in wilting and death of the tree above the canker.

Since it would be almost impossible to describe one syndrome that would apply to all hosts, a description of symptoms on each of the major hosts is included.

Sweet cherry

Cankers on the trunk and scaffold limbs undoubtedly cause the greatest damage to cherry trees. Cankers are sometimes classified as two types, but all shades of differences can be found between the two extremes. The canker characterized by gum exudation is the best known and most easily recognized.

Cankers usually develop at the base of an infected spur and then move up and down the trunk. Spread of the canker is usually much more rapid above the point of infection than below and relatively slow to either side, resulting in a long, narrow canker (Figure 1). Cankers usually develop during the fall and winter and are first noticed in late winter and early spring. Infected areas are slightly sunken and may have a slightly darker brown color than the rest of the bark. When the cankered area is cut, the bark may be any shade from bright orange to brown. The cambium may or may not be affected. At both the upper and lower margins of the canker, narrow brown streaks extend into the normal tissue (Figure 2). As the trees break dormancy in the spring, gum will be formed by the surrounding tissue and may exert enough pressure to break through the bark and run down the outside of the limb (Figure 3). Cankers that do not produce gum are similar, but usually are moister, sunken, and may have a sour smell (103). It should be noted that just as not all cankers gum, it is possible for cherry trees to exude gum from wounds other

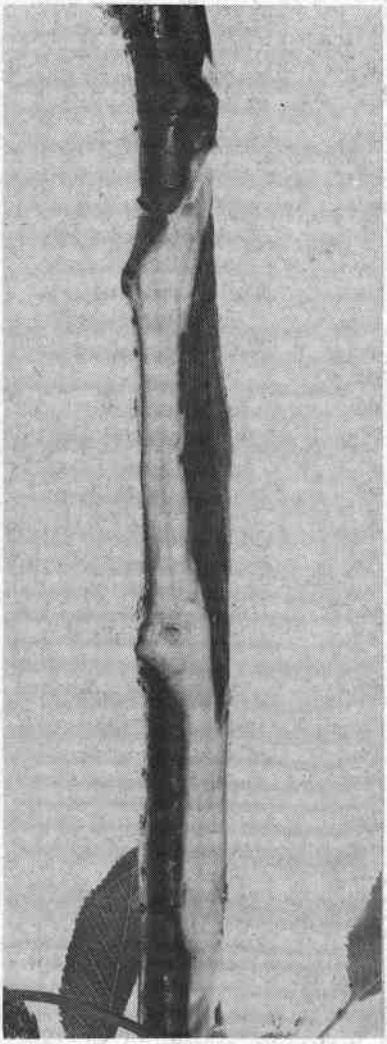

Figure 1. Side view of exposed canker showing typical size and shape on one-year-old sweet cherry.

Figure 2. Longitudinal section above a canker showing narrow brown streaks extending into healthy tissue.

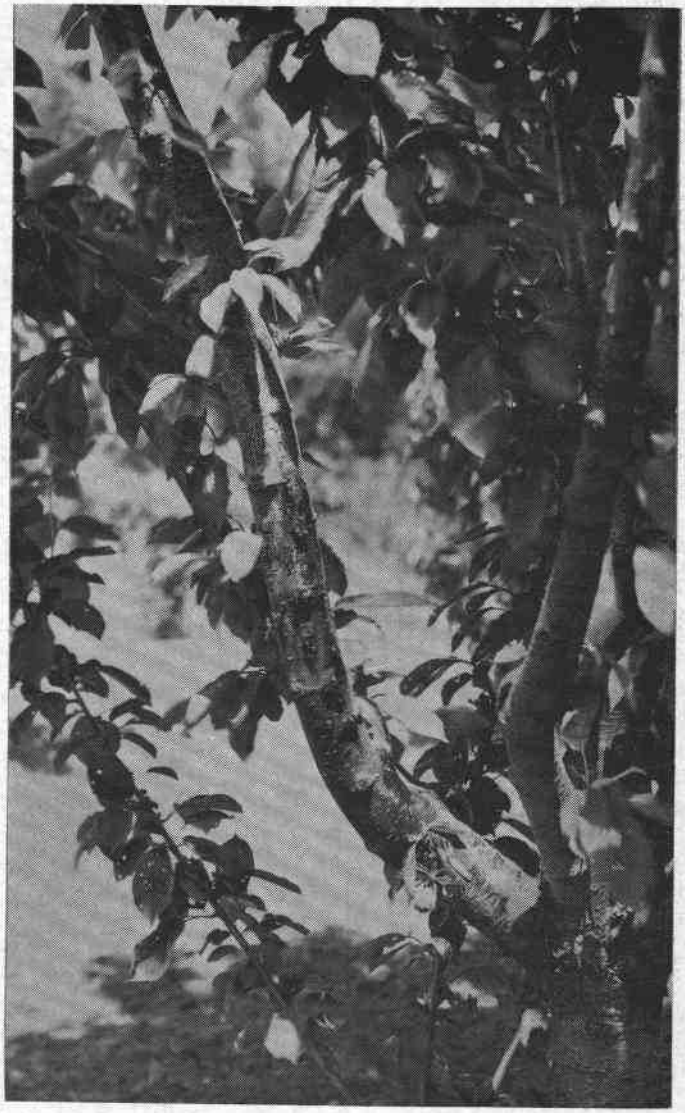

Figure 3. Gum pockets and gum exudate on sweet cherry.

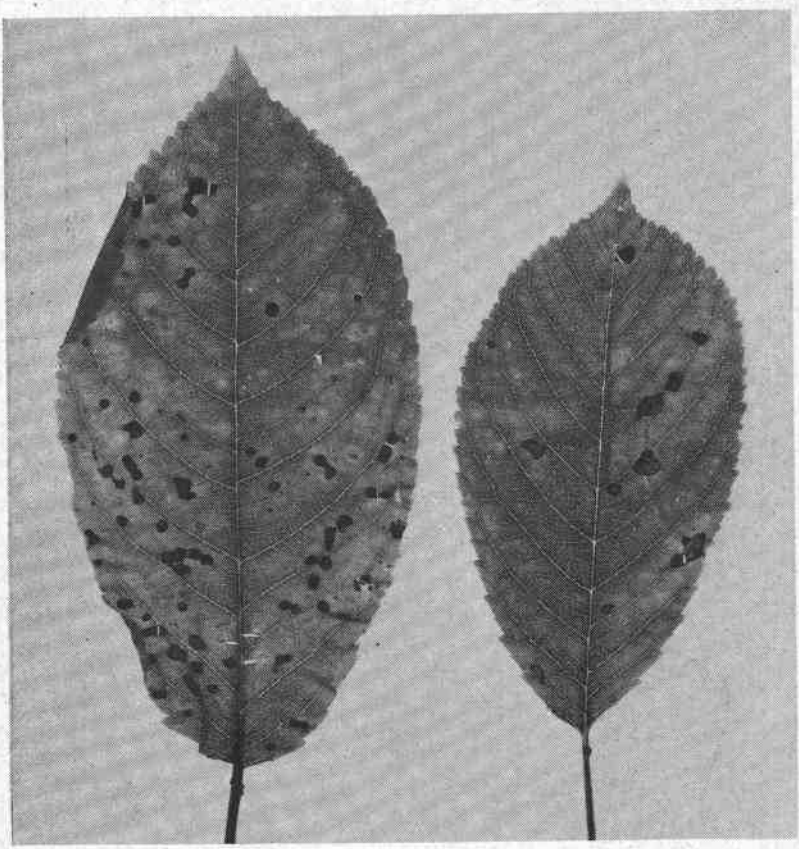

Figure 4. Angular spots on sweet cherry leaves caused by *Ps. syringae*.

than those caused by bacterial canker. Wounds resulting from mechanical damage may easily be confused with bacterial canker symptoms (27).

The first symptoms on cherry leaves are dark green, angular or circular, water-soaked spots about 1-2 mm. in diameter (Figure 4). The spots frequently are surrounded by a yellow halo. As the leaves mature, the water-soaked area becomes brown and eventually the infected area becomes dry and brittle. If these areas fall out, the leaves take on a shot-hole or tattered appearance (Figure 5). In severe cases the entire leaf tip and margin may drop off (50, 119).

Fruit infections are flat, irregular, dark brown to black, and from 2 to 3 mm. in diameter (50). Spots may be depressed and have underlying gum pockets (103).

When the trunk or limb of the tree is girdled by a canker, the area above the girdle will eventually die (Figure 6). The first symptoms above the canker may appear in spring, or they may not be noticed until warm weather in midsummer. A slight inward curling and drooping of the leaves is the first indication that the limb has been girdled (Figure 7). The foliage takes on a light green color, then turns yellow. By this time the leaves are hanging straight down and are strongly rolled (Figure 8). Within a few weeks the affected area is dead (10). If the canker is on the trunk below the scaffold limbs, the entire top of the tree may be killed (Figure 9). When the whole top is killed, several suckers usually arise around the base of the dead trunk (Figure 10). The suckers may be initiated above the graft union, but frequently they come from the seedling rootstock. When growers try to re-form a tree from one of the suckers, they should determine whether it is necessary for the tree to be regrafted to the desired commercial variety.

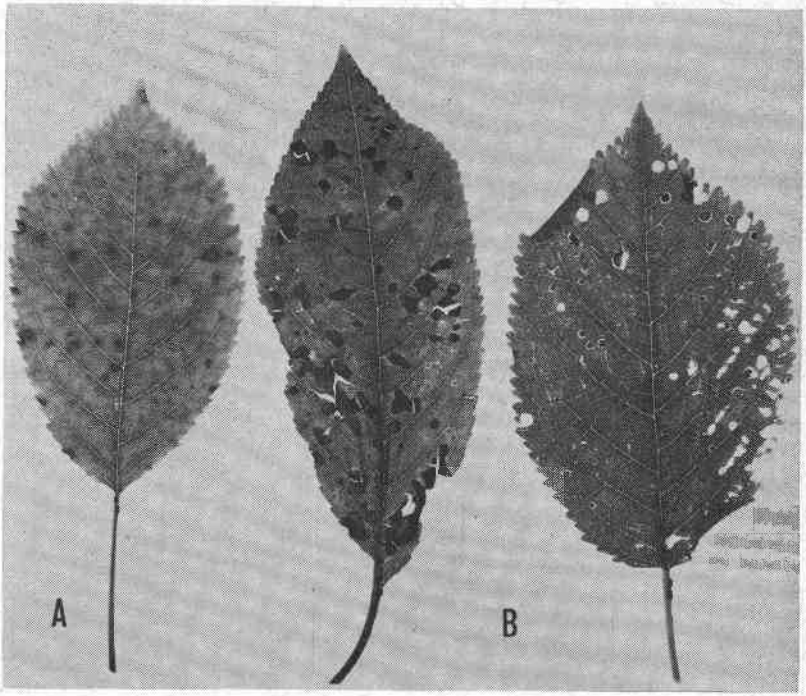

Figure 5. A. Early infection. B. Shot-hole appearance resulting from a severe infection of *Ps. syringae* on sweet cherry leaves.

Figure 6. Dead central leader of sweet cherry tree, resulting from canker.

Figure 7. First symptoms of girdling of limb by *Ps. syringae*. White arrow indicates area of canker. Black arrow indicates inward curling and drooping of leaves.

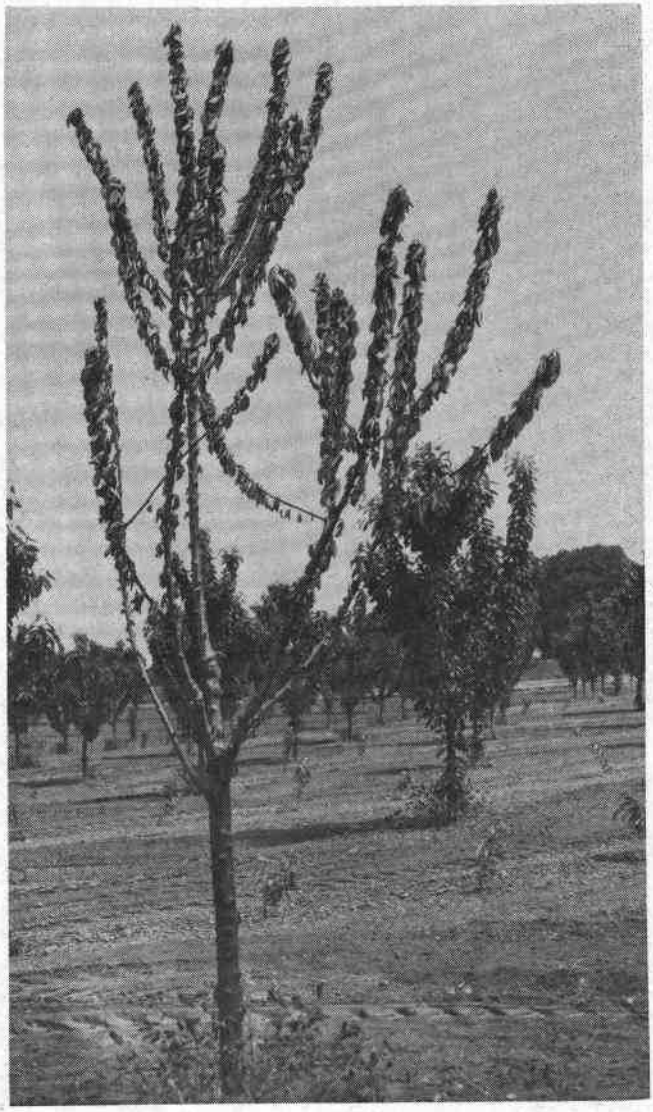

Figure 8. Midsummer symptoms on a sweet cherry tree that has been killed by *Ps. syringae*.

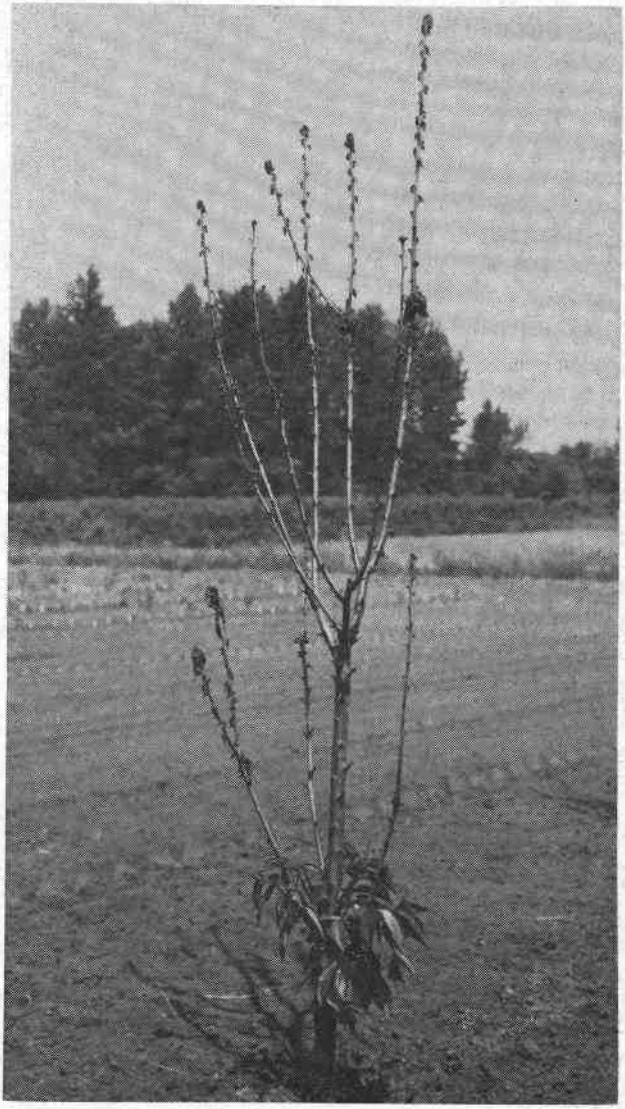

Figure 9. Dead top of sweet cherry tree caused by girdling of the trunk by *Ps. syringae*.

Figure 10. Mazzard suckers arising from around the base of a girdled sweet cherry tree. White arrow indicates the original tree.

Blossom infection in itself is usually not too important on cherry, but it can be very severe under favorable climatic conditions. Infections may go into the twig and cause shoot wilt, or they may spread into the spur and be the start of canker formation. Infected blossoms appear water soaked, turn brown and hang, giving the symptom that prompted the common name "blast" (Figure 11).

Under the conditions prevalent in some areas, a great number of dormant buds are killed. Wormald states, "Isolated buds are often killed and one year old twigs may be seen with one or more buds failing to develop . . ." (161). While the killing of isolated buds is usually not of much importance, the problem can become very severe. In the Willamette Valley of Oregon, 70 to 80% of the buds may be killed. In some cases, the death of dormant buds has made it necessary to remove formerly productive orchards.

First symptoms can be observed by sectioning buds in late February and early March, when a brown discolored area occurs at the base of the bud scales of infected buds (Figure 12 A-B). The brown area extends across the base of the bud and the entire bud eventually dies (Figure 12 C). Both flower and leaf buds are equally affected. The most obvious orchard symptoms are observed during full bloom when the heavy bloom on healthy trees is in sharp contrast with the black skeletons of diseased trees (Figure 13 A-B). Cankers are very rarely found at the base of the buds killed by this isolate of the bacteria (23, 24, 25, 26).

While most of the above symptoms can be found in varying degrees wherever the bacteria are found, certain areas are more prone to a particular phase of the disease. Whether this is due to strain differences of the pathogens or to different climatic conditions has not been determined. Leaf spotting is particularly severe in England (4, 42, 144), almost nonexistent in California (4, 144) and mild in New South Wales (96) and Oregon. Cankers on trunks and limbs are very bad on susceptible varieties in England (38), New South Wales, and New Zealand (50, 96), moderate in western Oregon and California, and mild in eastern Oregon. Blossom and fruit symptoms occur in New Zealand (50), New South Wales (96), Great Britain (166), California (4), and occasionally in Oregon. While death of dormant buds is very severe in western Oregon, it is of minor significance in California and other parts of the world. Shoot and spur withering is common in England (4) but rarely occurs along the Pacific west coast (4). Killing of spurs and buds is usually more severe on mature trees while cankers are more important on young (1- to 8-year-old) trees. Both *Ps. mors-prunorum* and *Ps. syringae* may attack all parts of the cherry (165).

Figure 11. Sweet cherry buds and flowers killed by *Ps. syringae.*

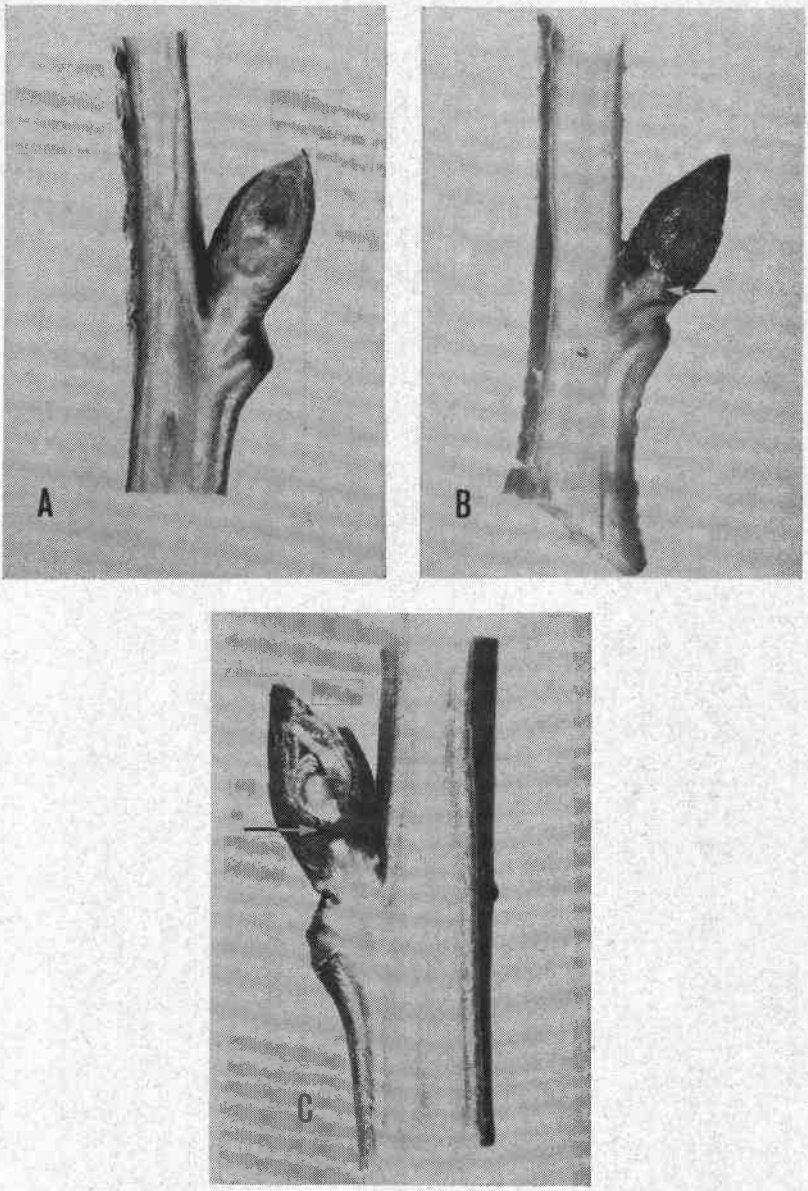

Figure 12. A. Healthy bud. B. Infection at the base of bud scale. C. Entire base of bud killed by *Ps. syringae*.

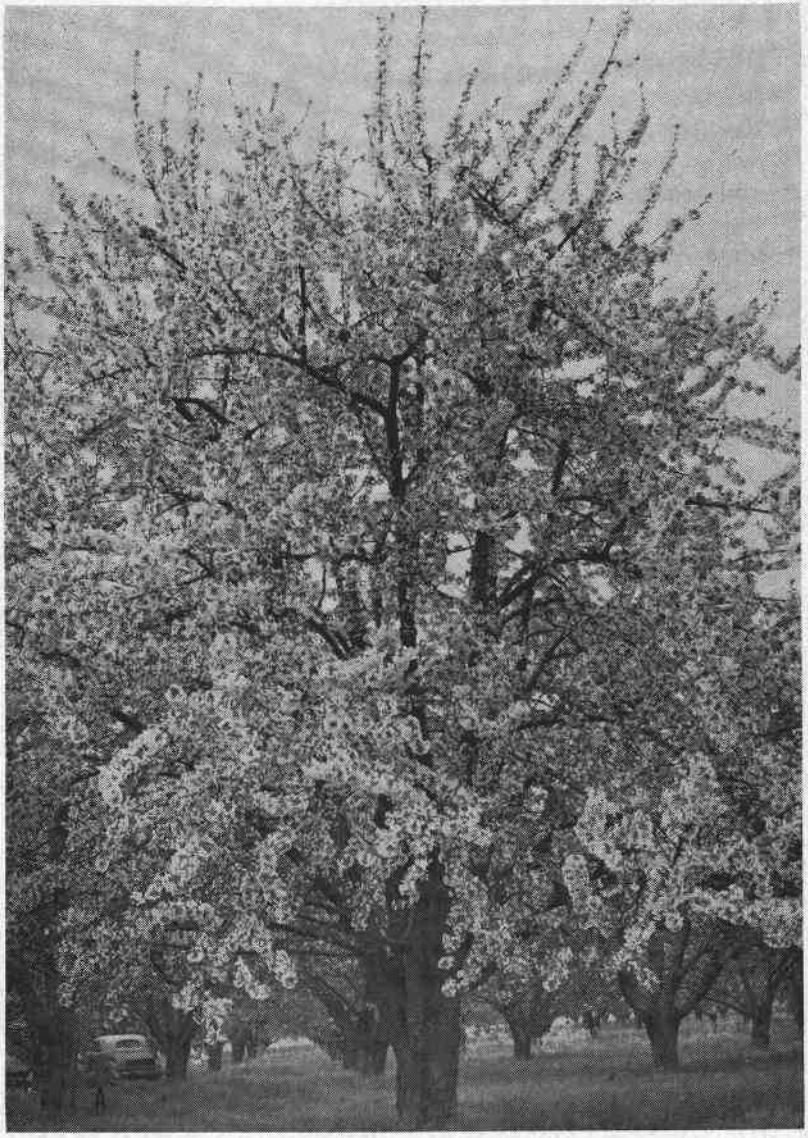

Figure 13A. Healthy sweet cherry tree.

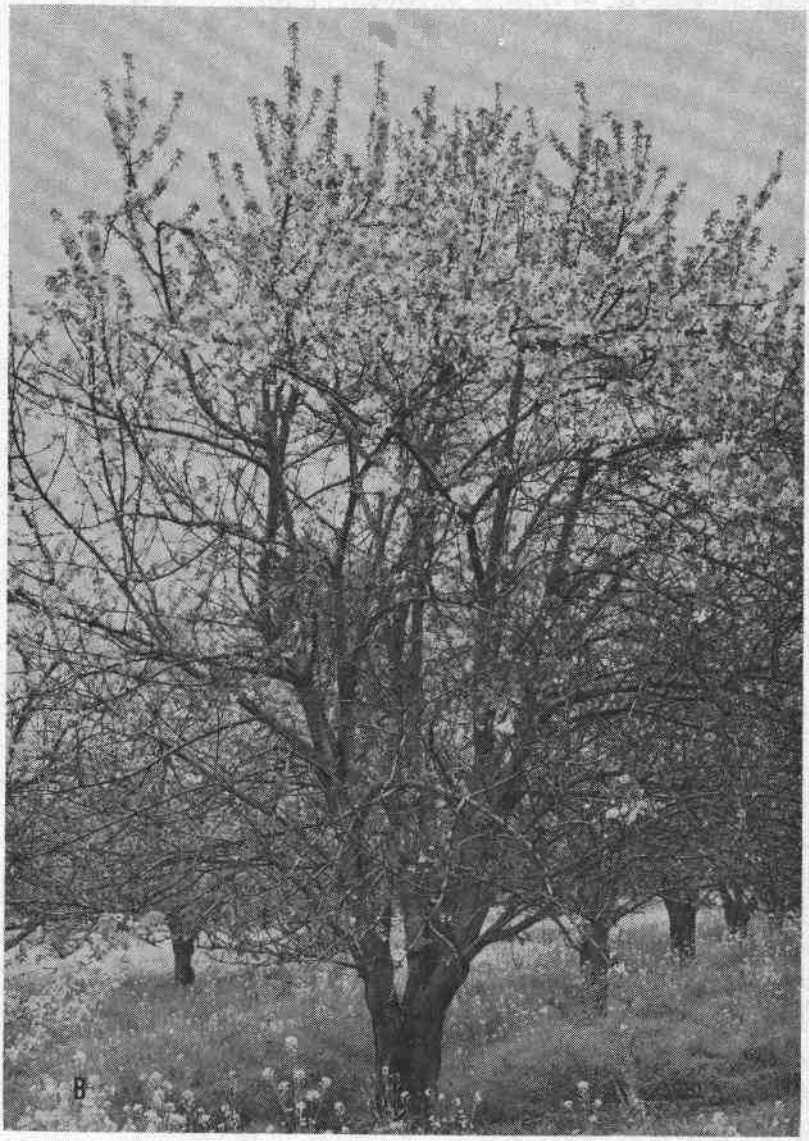

Figure 13B. Cherry tree with most of the buds killed by *Ps. syringae.*

Sour cherry (acid cherries)

Wormald (169) reports canker symptoms on sour cherries similar to those on sweet cherry. The symptoms were caused by two different bacterial isolates, one of which was isolated from branches and stems and appeared identical with *Ps. mors-prunorum.* The other was isolated from flowers, leaves, and fruit and was not described. Cankers caused by *Ps. syringae* have not been observed on sour cherries in California and are rarely found in Oregon. In general the species is considered to be quite resistant.

Leaf symptoms are described as consisting of numerous small and angular spots or a few large round spots. The spots are generally brown, but may have a pale green or yellow halo (169).

Dark sunken spots may be found on the side or distal end of the fruit. Infections may also be found on fruit stalks.

Blossom and twig blight symptoms are reported to be similar to those of brown rot, but no conidia are formed (169).

Peaches

Cankers on peach are very severe in California and are of concern in New Zealand. In Oregon *Ps. syringae* is rarely observed on peach, and the disease is not normally of economic importance. The lack of canker in Oregon peach orchards is peculiar, since the peach acreage is moderate and the disease is very prevalent in nearby cherry orchards. Peach canker is probably the most important phase of bacterial canker in California. Cankers on peach limbs are similar to those described for cherry. On young wood, cankers are light brown with dark margins and often surrounded by reddish zones (50).

Spots on peach leaves are circular, 1 to 3 mm. in diameter and olive green to light brown in color. Lesions occur primarily at the tips and margins of the leaves and at first are somewhat water soaked in appearance. As the leaves mature, the lesions become very noticeable and turn a dark brown to dark red. Eventually infected areas become dry and brittle and drop out, giving a shot-hole or tattered effect (50). Leaf-spot symptoms on peach have not been reported from England (168).

Peach fruit symptoms consist of flat, superficial, circular lesions about 1 to 3 mm. in diameter and brown with a red margin. Older lesions on fruit may be either internal or on the surface. Surface lesions are depressed and irregular, with a dark brown margin; gum may be produced. The lesions may be 2 to 10 mm. in diameter and 2 to 10 mm. deep. The underlying tissue is dark brown to black and is sometimes spongy (50). Internal lesions appear as dark brown to black spherical masses usually located near the surface and sometimes

spongy (50). Blossom and bud infections have seldom been reported on peach.

Plum

Cankers on plums start as small, brown to reddish brown spots that enlarge as water-soaked streaks. In the spring, the area between the streaks turns brown and the area becomes uniformly brown and moist (40, 144, 156). Wilson (144) describes gum formation as follows: "As a rule, little if any, gum is exuded from the affected tissues; but, a watery material may flow from cracks in the bark and cover the limbs. The absence of gum is particularly noticeable in the case of plums." Cracks appear around the margin of the canker as the dead area dries out (Figures 14 and 15). Gum formation is even less evident in infected Japanese plums (144).

Leaf symptoms on plum are similar to those described for peach (50).

Young lesions on plum fruit are slightly raised, circular, 1 to 3 mm. in diameter, and olive green. The older lesions are depressed, irregular, sometimes cracked, dark brown to black in the center, and with a green water-soaked margin. The spots are from 5 to 10 mm. in diameter and penetrate the surface from 2 to 5 mm. (50). Gum may or may not be exuded.

Blossom blighting occurs on plums in California (4) and England (144) but is not usually as serious as on cherry.

Shoot wilt of plum is quite common in England and more frequently incited by *Ps. syringae* than by *Ps. mors-prunorum* (38, 148); however, both can produce similar symptoms (152). The shoot-wilt phase of bacterial canker is also observed in the coastal fruit growing areas of California (75).

Death of dormant buds is not usually of importance in either domestic or Japanese plums.

Apricot

Cankers formed on apricot are more similar to those on cherry than to those found on plums. Most varieties of apricots are very susceptible to bacterial canker and gum profusely (31).

Early leaf symptoms on apricot consist of circular spots, 1 to 2 mm. in diameter, bright red, and often surrounded by narrow yellow halos. The spot first turns dark red or reddish purple, then dark red to brown with a definite yellow halo, and finally almost black with a light brown center (37, 50).

Developing lesions on the fruit are flat, superficial, mainly circular, from 1 to 2 mm. in diameter, and dark red. The established

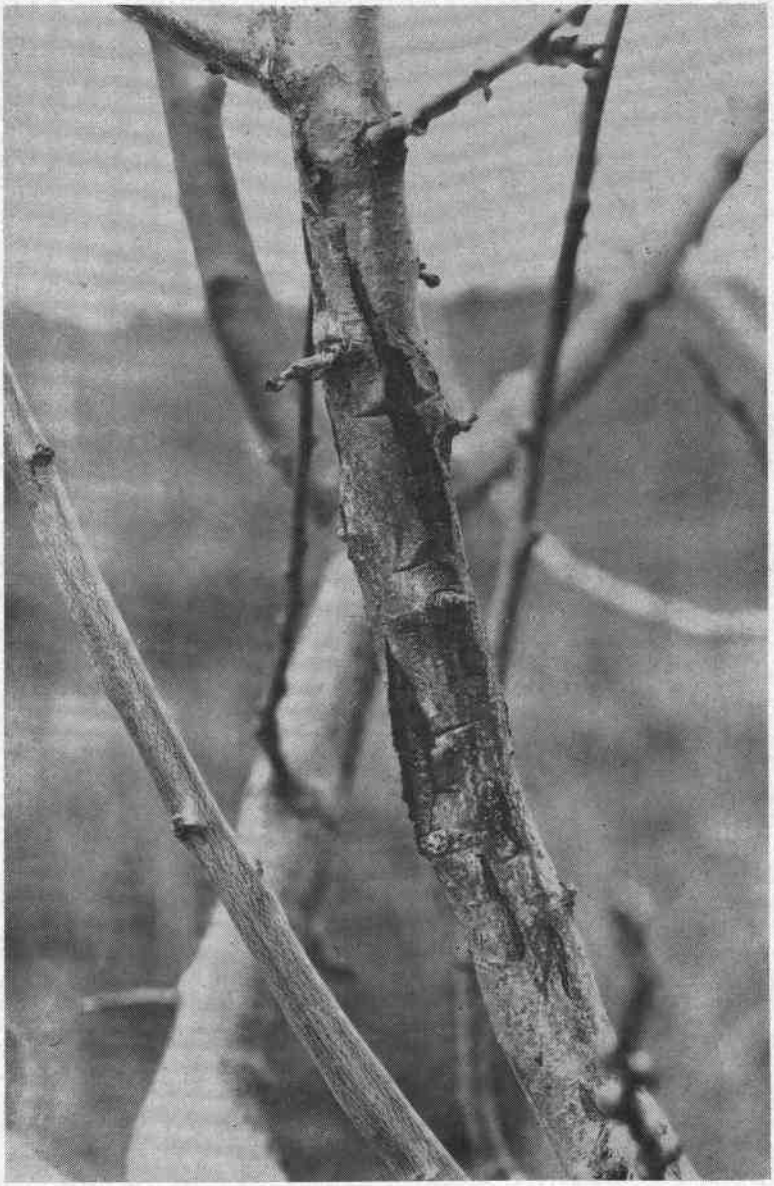

Figure 14. Healing canker on a limb of Italian prune.

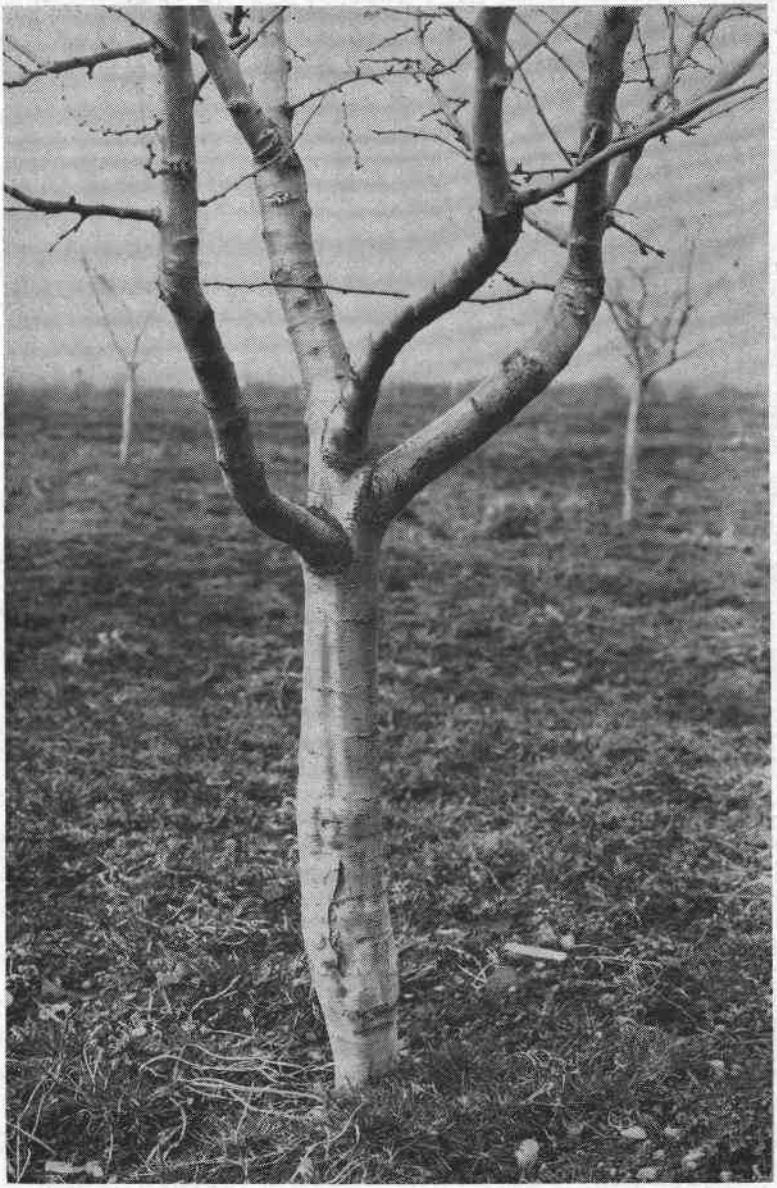

Figure 15. Canker at the base of Italian prune trunk. Note the sunken area in the trunk where the canker extends up to the lower limb.

lesion is raised, scab-like, cracked, light to dark brown, and from 3 to 6 mm. in diameter. The center of the lesion is sometimes depressed (50).

Apricots are rather susceptible to both the bud-blight and shoot-killing phases of bacterial canker (4, 144). Symptoms in both cases are similar to those on cherry.

Almonds and nectarines

Symptoms on almonds and nectarines are very similar to those on peach. These two crops are not grown commercially in many of the areas where bacterial canker is very severe; therefore, the amount of work on them is rather limited. Bacterial canker causes some damage in California almond orchards.

Summary

In general, cherry is the most susceptible host to the blossom and bud-blight stage of bacterial canker, although apricots and plums are also susceptible (144). Apricot is the most likely to gum from cankers, but is closely followed by cherry. Domestic plums seldom produce gum, and gum production is very rare in Japanese plums. Flower infection is not usually a serious problem in stone fruits with the possible exception of sour cherry. Since the organism reported as isolated from sour cherry blossoms was not identified in the literature, it is not known if it is actually part of the bacterial-canker complex. As has already been pointed out, the severity of the canker and leaf-infection phase varies not only with the host susceptibility but also with both geographic area and climatic conditions.

Pears

According to Wormald (152) symptoms on pears are similar to those on the *Prunus* sp. Since the bacterium is not described, it is not known if either *Ps. syringae*, *Ps. mors-prunorum*, or both, are active on pome fruits; however, in other reports *Ps. syringae* is the only listed pathogen (32, 87). Wormald lists twig infection, dormant bud blast, limb canker, blossom blast, and fruit infection as phases of the disease on pear trees. In the Pacific coast states, *Ps syringae* is definitely pathogenic on pear and has caused considerable damage to both mature and 1- to 5-year-old orchards.

The most severe phase of the disease on pear is the blossom-blast symptom stage. Blossoms are attacked when the tree is in full bloom. Small brown water-soaked spots appear at the calyx end of the fruit and then turn dark brown to black. If humid weather continues, the spots spread to the stamens and down the fruit to infect the entire flower. The symptoms then spread to neighboring florets and the

whole spur is killed (57). A severely infected tree looks as if it had been singed by a blow torch. The infection moves into the shoot (167) and may move 6 to 12 inches back into the branch. Symptoms at this time are similar to fire blight, but the twig cankers do not spread as rapidly as those of fire blight and exudation droplets are not observed on the outside of the shoot. Infected parts become dry and brittle and tend to fall from the tree.

Immature fruit infection also occurs in the Pacific Northwest and symptoms consist first of a water-soaked area that becomes sunken and black as the fruit matures. No natural infections by *Pseudomonas syringae* have been reported on mature pear fruit. Artificial infections cause depressed black, circular to angular spots up to one-eighth inch deep (57). Leaves may become infected if the weather remains moist. Dark brown angular spots spread rapidly and the whole leaf turns black (57, 6). Infection of 1- to 5-year-old trees through pruning cuts or wounds can cause considerable tree loss in Oregon.

CAUSAL ORGANISM

In early studies of *Ps. syringae* (Figure 16) there were many independent reports from different parts of the world and on different hosts. This led to numerous names for what is now considered to be one species. Over the years the following names have been reported as synonymous with *Ps. syringae* by various authors:

Pseudomonas syringae van Hall, 1902. (*Bacterium s.* E. F. Smith, 1905, *Phytomonas s.* Bergey et al., 1930.)

Bacillus gummis Comes.

Bacillus spongiosus Aderh. and Ruhl, 1905. (*Pseudomonas s.* Braun, 1927, *Bacterium s.* Elliott, 1930, *Phytomonas s.* Magrow, 1937.)

Pseudomonas cerasus Griffin, 1911. (*Bacillus c.* Holland, 1920. *Bacterium c.* Elliott, 1930, *Phytomonas c.* Bergey et al., 1930.)

Bacterium matthiolae Briosi and Pavarino, 1912. (*Phytomonas m.* Bergey et al., 1930.)

Bacterium citriputeale C. O. Smith, 1913. (*Pseudomonas c.* Stevens, 1925, *Phytomonas c.* Bergey et al., 1930.)

Bacterium citrarefaciens Lee, 1917. (*Pseudomonas c.* Stevens, 1925.)

Pseudomonas hibisci Nakata and Takimoto, 1923. (*Phytomonas h.* Bergey et al., 1930.)

Pseudomonas vignal Gardner and Kendrick, 1923. (*Phytomonas v.* Bergey et al., 1923.)

Pseudomonas viridefaciens Tisdale and Williamson, 1923. (*Phytomonas v.* Bergey et al., 1925.)

Pseudomonas trifoliorum L. R. Jones et al., 1923. (*Phytomonas t.* Burkh., 1926.)

Bacterium holci Kendr., 1926. (*Pseudomonas h.* Kendr., 1926, *Phytomonas h.* Bergey et al., 1930.)

Pseudomonas prunicola Wormald, 1930. (*Phytomonas p.* Wormald, 1930.)

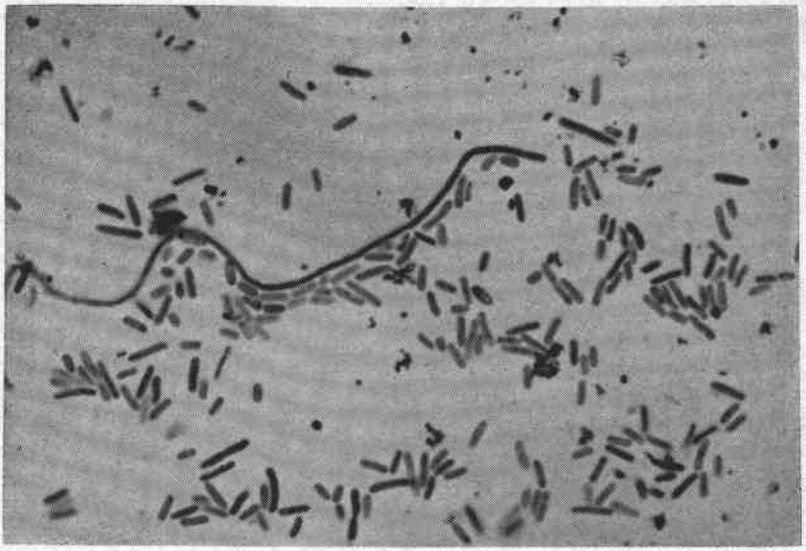

Figure 16. ***Ps. syringae,*** **2000 X.**

Phytomonas vigne var *leguminophila* Burkholder, 1930.
Pseudomonas mors-prunorum Wormald, 1931.
Pseudomonas utiformica Clara, 1932. (*Phytomonas u.* Clara, 1934, *Bacterium u.* Burgwitz, 1935.)
Pseudomonas cerasi var. *prunicola* Wilson, 1933. (*Phytomonas c.* var. *p.* Bergey et al., 1939.)
Pseudomonas nectarophila (Doidge) Rosen and Bleeker, 1933.
Pseudomonas barkeri (Berridge) Clara, 1934.
Pseudomonas rimaefaciens Konig, 1938.
Pseudomonas syringae f. sp. *prunicola* Dowson, 1949.

Descriptions of the causal organism vary to some extent. The original description by van Hall has been translated from his thesis and may be found in Appendix A. Other descriptions of isolates from different hosts by Fawcett, Burkholder, and Dye, as well as the description from the 7th edition of *Bergey's Manual of Determinative Bacteriology* may also be found in the appendices.

Since van Hall's original description of *Ps. syringae,* numerous comparisons have been made in attempts to determine if new isolates were synonymous. Additional information about *Ps. syringae* has resulted from each of these comparisons. Also, as new isolates have been reduced to synonymy, it has been necessary to broaden the description to cover all of the included isolates.

Griffin (77) felt that his isolate closely resembled *Bacillus spongiosus* as reported by Aderhold and Ruhland, but he could not get the

spongy-appearing colonies, and some of the media acquired a green cast. Griffin frequently found an imperfect fungus also associated with the cankers. In Oregon a species of *Pullularia* has frequently been associated with the cankers, but since *Pullularia* is frequently found on healthy trees, it should be considered as part of the natural flora of orchard trees in Oregon. *Cytospora* is often a secondary invader (151, 161). A secondary saprophytic bacterium similar to *Ps. syringae* reduced the number of infections when mixed with *Ps. syringae* in one year, but only reduced the severity of the symptoms the following year (50). All of Griffin's isolates were similar but several of Barss' isolates were distinct strains (10). Type number two did not blight spurs and was a different color on potato sucrose agar. While both of the strains were pathogenic, number two was not as virulent as Griffin's isolates. Barss' strain number two may be similar to the strain that is presently responsible for the dead-bud symptoms in Oregon.

Wormald (148) when he first reported the disease symptoms, considered it as three separate diseases: 1) Bacterial shoot wilt of plum; 2) leaf spot and gummosis of cherry; and 3) bacterial canker and leaf spot of plum and cherry. The organisms causing the first two diseases were similar and were thought to be of the same species. The third disease was attributed to a possible new species. Wormald claimed that these species could always be distinguished by their symptom expression.

Isolates from pear trees were reported to be more vigorous on pear than the cherry isolates and the cherry isolate was, in turn, more vigorous on *Prunus* than the pear isolate, but both were definitely pathogenic on each other's host (152). Both fluorescent and non-fluorescent isolates were obtained from pear and were similar enough to be included in *Ps. syringae* (*Ps. cerasi, Ps. syringae, Ps. citriputeale, Ps. cerasi* var. *prunicola,* and *Ps. utiformica*).

Wormald's isolates showed a tendency to diverge from type when kept in culture (150). He believed this to be a case of dissociation. His isolates had a thermal death point of 40° C. and were in general agreement with the description of *Ps. syringae.*

Goldsworthy (75) believed that more than one organism was responsible for bacterial canker. Two strains of *Ps. syringae* were isolated, only one of which would produce a pigment. The two isolates were specific with no cross agglutination even though the symptoms caused by the two strains were identical.

Smith and Fawcett (126) compared isolates from citrus, *Prunus,* lilac, and avocado. All the cultures showed a very small capsule similar to *Ps. syringae* when stained with Robert's dahlia stain. All of the

isolates caused milk to become alkaline and all showed an absence of indol after 10 days in Dunham solution. The isolates of *Ps. syringae* (*Bacterium cerasi*) from apricot did not produce acid with lactose and maltose as was reported by Griffin (78). Smith and Fawcett concluded that *Bacterium cerasi, Bact. syringae,* and *Bact. citriputeale* are closely related and that their cultural characteristics suggest that they might belong to a single species. They did not combine the species at that time, due to slight differences in pathogenicity on some hosts. Bryan (18) pointed out the effect of pH on fluorescence and growth.

Wilson (139) reported that *Ps. syringae* (*Ps. prunicola*) was slightly gram positive if tested by either the method of Eyre or of the Society of American Bacteriologists. By modifying the procedure to include a washing between steps, both his unknown (from apricot buds in California) and *Ps. syringae* were gram negative. Wilson's isolate from apricot produced a yellow pigment and showed a slight fluorescence. This is in contrast to the green color described by Barss and Griffin. Wilson concluded that his isolate and *Ps. syringae* were almost identical, and were possibly related to Barss' and Griffin's isolate.

In later papers Wilson (140, 141) compared isolates from apricot, cherry, peach, nectarine, almond, plum, and prune with *Ps. syringae* isolates from England. All of the bacteria isolated from the different hosts were similar as were the bacteria obtained from blighted dormant buds, blossom blight, green-shot blight, and leaf spots on the same host. Normally the colonies were white on P.D.A. and a slight lemon-yellow on nutrient agar. A new isolate from cherry was a brilliant green. This new isolate was obtained 10 to 15% of the time; only once were both types found in the same canker. Both types were equally pathogenic and produced similar symptoms when inoculated into the same varieties, except that dormant buds appeared to be blighted only by the white type. The amount of gumming seemed to depend on the host variety and the condition of the host at the time of inoculation. Different locations on the same variety also made a difference in symptom expression. All of the isolates, including *Ps. syringae,* were gram negative. Even though statistically significant differences were noted between the green and white strains of *Ps. syringae,* they were not greater than the differences among different aged cultures of *Ps. syringae.* Many organisms were capsulated in all of the isolates. In Uschinsky's medium, Czapek's agar, and Salicin agar plus sodium nitrate, both the green and white strains produced a yellow-green fluorescence, but the color from the green isolates was more intense. The physical characteristics were similar but pig-

ment production differed on potato dextrose agar, beef extract broth, beef extract agar, and salicin agar with sodium asparaginate.

All of the cultures grew well on gentian-violet bile agar and all cultures liquified gelatin. All of the isolates gave an alkaline reaction in litmus milk, reduced methylene blue, and did not hydrolize starch. They all produced acid on arabinose, mannose, dextrose, levulose, galactose, sucrose, mannitol, and glycerine, but were alkaline on lactose, maltose, trehalose, raffinose, peptone, sodium asparaginate, sodium succinate, sodium citrate, sodium malate, and sodium lactate. There was poor to no growth on rhamnose, sodium tartrate, sodium acetate, sodium benzoate, and sodium nitrate. Optimum temperature for both the green and the white types was between 20 and 30 degrees centigrade. All recent isolations from dead buds in Oregon have been of the white type.

The question of whether *Ps. mors-prunorum* should be maintained as a distinct species has been raised at numerous times. Although differences in degree of pathogenicity have been reported (37, 165), in general, this is not considered a satisfactory basis for determining a species. On the question of species determination on a physiological basis, there are numerous conflicting reports.

Erikson in 1945 (62) compared nine isolates of *Ps. mors-prunorum,* five isolates of *Ps. syringae* (three of *Ps. prunicola* and two of *Ps. syringae* from Wilson), one isolate of *Ps. fluorescence-liquifaciens, Ps. pyocyaneus, Ps. pisi, Ps. cerasi, Ps. tabaci, Ps. phaseolicola, Ps. marginalis,* and *B. pruni,* two isolates of *B. tumefaciens,* plus three unnamed isolates from lilac, one from forsythia, seven from pear, and one pear isolate from the Lister Institute.

According to Erikson, "All of these except *B. tumefaciens, B. pruni,* and the pear isolate from the Lister Institute belong to the gram negative, lophotrichous, green-fluorescence section of *Pseudomonas migula,* which produce a white or colorless growth on ordinary solid media, of which the type species is *Ps. fluorescens*—the Group II of Dawson (1939)."

Lesions were caused on green plums by eight of the nine strains of *Ps. mors-prunorum* and by two of the three strains of *Ps. syringae* (*Ps. prunicola*). The majority of the strains of *Ps. mors-prunorum* and *Ps. syringae* (*Ps. prunicola*), and the apricot, pear, and lilac strains were pathogenic on dwarf bean pods. Proteolytic activities were similar for all strains. On succinic acid, *Ps. syringae* (*Ps. prunicola*), *Ps. syringae* (from apricot), and the pear isolates gave a more rapid fluorescence and alkaline reaction than *Ps. mors-prunorum* and *Ps. syringae* (from lilac). The majority of the strains of *Ps. mors-prunorum* were able to utilize tartaric acid as opposed to other strains.

An acid reaction was recorded when cultures were grown on the mono- and tri-saccharides. In the disaccharide group, the acid production on sucrose was more marked with *Ps. mors-prunorum* and the lilac strains of *Ps. syringae* than with other isolates. From the reports on the use of sucrose by Wormald (154), Clara (33), and Wilson (139), Erikson (62) concludes that there is a continuous variation among the plant pathogenic bacteria. All of the strains, including all of the *Ps. mors-prunorum* isolates, produced a yellow-green fluorescent pigment. The yellow-green fluorescence is most common in a dilute alkaline medium and may be red in a more concentrated alkaline solution. Fluorescence may disappear in an acid medium.

Ps. mors-prunorum, Ps. syringae (*Ps. prunicola*), all of the lilac strains, and some of the pear strains of *Ps. syringae, Ps. pisi, Ps. phaseolicola,* and *Ps. tabaci,* all produced a gummy poysaccharide, similar to that described by Aderhold and Ruhland, from sucrose when furanose was available. Indole production was negative, by Ehrlich's method, for all but one strain of *Ps. syringae.* However, by Galkowski's method, a positive reaction was obtained for two out of three cultures of *Ps. syringae* (*Ps. prunicola*), two out of five cultures of *Ps. mors-prunorum,* three pear strains, and *syringae* strain number three. The indol reaction was found to be due to the production of B-indoleacetic acid from tryptophane.

The longevity of *Ps. mors-prunorum* was compared with that of *Ps. syringae* (*Ps. prunicola*) and was found to be the same on 11 out of 12 different media. Erikson concludes: "On the basis of biochemical characteristics, considered apart from host pathogenicity, there is no justification for erecting to specific rank those various leaven-forming, green fluorescent, phytopathogenic, pseudomonads."

Crosse in 1953 (37) found that most cankers on apricot were caused by *Ps. mors-prunorum* but that *Ps. syringae* (*Ps. prunicola*) could also be isolated. Both *Ps. syringae* and *Ps. mors-prunorum* produced acid on maltose and lactose media, but more acid was produced by *Ps. mors-prunorum.* Both will liquefy gelatine in stab culture, but *Ps. mors-prunorum* only slightly. In 1955 Crosse (40, 42) tested 30 strains of *Ps. mors-prunorum,* and all could use tartrate. From this he proposed a selective medium using sodium tartrate and diochyl-sodium sulphosuccinate to distinguish *Ps. syringae* from *Ps. mors-prunorum.*

Wormald (154) distinguished *Ps. mors-prunorum* from *Ps. syringae* (*Ps. prunicola*) by the following traits: *Ps. mors-prunorum* was white on nutrient broth plus 5% sucrose and usually died in four to six days. The reaction was first alkaline and then acid. Nutrient agar medium containing 2% lactose and brom cresol purple turned

yellow. Yellow coloration was absent or faint in Uschinsky solution. *Ps. syringae* was yellowish on nutrient broth plus 5% sucrose and remained viable for several weeks. The reaction was alkaline and turned the medium blue-purple. On Uschinsky's solution there was a distinct yellow color.

While some of these characteristics may be diagnostic, it should be pointed out that discrepancies exist between Wormald's description of *Ps. syringae* and those of other workers. Fawcett, Dye, and Burger all describe *Ps. syringae* as white or grayish-white in color which would make it the same as *Ps. mors-prunorum.* Regarding the production of an acid or alkaline reaction, van Hall states that the formation of alkali predominates, but that a slight amount of acid may be formed in some media due to the incomplete oxidation of carbohydrates. As has already been mentioned by several authors, the individual isolates vary greatly in their ability to fluoresce. There are considerable differences in both color and fluorescence among isolates of *Ps. syringae* from the Pacific coast states (60).

As early as 1933 Rosen and Bleecker (117) attempted serological studies with isolates of *Ps. syringae* (*Ps. syringae, Ps. citriputeale, Ps. prunicola,* and pear isolates). All of these cultures reacted alike in pathogenicity, cultural, physiological, and serological tests. From this he concluded that all of these isolates should be considered as one species. Crosse (46) in 1959, used phage isolates in an attempt to separate *Ps. mors-prunorum* strains 1 and 2, *Ps. syringae* 1 and 2, and *Ps. phaseolicola. Ps. phaseolicola* could be distinguished by the reaction of the phage, but none of the four phage isolates distinguished between *Ps. syringae* and *Ps. mors-prunorum.*

Fuchs (72, 73) compared isolates from cherry, peach, and plum, and concluded that after several transfers it was impossible to distinguish *Ps. mors-prunorum* isolates from *Ps. syringae* isolates. Fuchs concludes that, "Therefore, it is impossible to separate the two species after prolonged cultivation on peptone agar media either by biochemical or by pathogenic characteristics."

It appears that the only consistent difference between *Ps. mors-prunorum* and *Ps. syringae* is the ability of most isolates of the first organism to utilize sodium tartrate in the medium. In view of the range of differences among the isolates obtained from bacterial cankers and dead buds in Oregon, this difference would not seem to be of sufficient magnitude to warrant maintaining *Ps. mors-prunorum* as an independent species. If further experiments fail to indicate any greater distinction, it would appear that *Ps. mors prunorum* should be reduced to synonomy with, or considered a strain of, *Ps. syringae.*

Another strain of *Ps. syringae* has been reported on blueberry by

Vaughan (134). The isolates from blueberry are the same as those from lilac in their physiological and biochemical reactions, but are somewhat different in pathogenicity and serological reactions.

MODE OF ACTION

Mills (101, 102) made a biochemical study of the mode of action of an enzyme produced by *Ps. syringae*. The prepared enzyme was found to be involved in the splitting of an ester linkage. A pectin esterase was also found. The enzyme was formed adaptively in the presence of pectin, pectate, and galacturonic acid. The enzyme split 75% of the methoxyl groups and approximately 70% of hydrolized tributyrin, tracetin, diacetin, and monoacetin. No action was reported on ethyl acetate, ethyl oxalate, or the methyl ester of x-methyl-galacturonic acid.

DISEASE CYCLE

In the spring of some years, a very severe outbreak of leaf-spot in sweet cherries can be attributed to *Ps. syringae*. These outbreaks occur most frequently in those areas prone to cool, wet springs, and they are associated with periods of high winds and continued moisture (42, 48, 50, 70). The main agent of spread under these conditions seems to be wind-blown rain. The host is most susceptible when growth is succulent and subject to injury by wind-whipping and hail. Fawcett reports that necrotic areas are larger during cool temperatures (17° C.) than at 20.5, 25, or 33.5° C. Griffin (78) felt that symptoms in Oregon were more severe in years of extreme temperature variation; however, spring temperatures above 21° C. or below freezing are uncommon in the cherry growing districts of Oregon. In general, severity of leaf and shoot infection can be correlated with the number of cool, wet spring days.

The leaves become less susceptible as they mature, and the bacteria are difficult to isolate. Acording to Crosse (44), the bacteria ooze out of these leaf infections when they are moist and are then spread around the tree. When the leaves dry, the bacteria are left on the leaf surfaces. It has been estimated that there are as many as one-half million bacteria per leaf on an unsprayed cherry tree by October. While most of these bacteria are not viable in the fall, there are enough to initiate infection (88). Since there has been some evidence that the bacteria may multiply on the leaf surface, it has been thought that controlling the leaf-spot stage might reduce the number of winter cankers (38). However, this theory is not satisfactory, since severe canker-producing infections are also common where leaf-spot symptoms are nonexistent or rare. Also, in

areas where the leaf-spot phase has been controlled, the amount of canker development has not been materially reduced.

During years that are particularly favorable for disease development, new shoot and spur growth may be attacked (38). The shoots wilt and then wither. This symptom is not very common, and it usually does not occur frequently enough to warrant applying control measures.

Two theories have been proposed to explain the infection of the tree during the fall and winter months. Investigations in England have suggested that infection might take place in association with leaf fall (36, 38, 39, 44). Crosse proposed that the summer leaf-spot stage alternates with a winter canker phase (42). The canker infections originate as infections through leaf scars during the autumn leaf-fall period. The bacteria are "sucked" into the xylem vessels of the leaf trace, and the pathogen then spreads into the living tissues. The fact that bacteria could be taken into the xylem was illustrated by placing India ink on the leaf trace as the leaf abscissed. Leaf scars were found to be susceptible from September 6 to the end of October. The ability of leaf scars to be infected was found to decline as the general leaf fall began. This was attributed to either a lowering of the suction in the xylem or to a blocking of the vessels by tyloses. How the bacteria get out of the xylem elements has not been described, but maximum disease development appears in the medullary rays of the phloem (41). A "threshold" number of bacteria were necessary to establish infection and the "threshold" number was higher in the more resistant varieties. Scars formed during dry, windy weather tended to escape infection. On all varieties, leaf scars became immune from infection between 8 and 14 days after they were exposed.

Crosse points out that Wormald had earlier considered that infection originated in the buds or in the perenchymatous tissue around the leaf scars; however, many buds had swollen before they were invaded (39).

Dye also reports cankers being initiated from leaf-scar infections in the fall (50). Trees that were transplanted to induce leaf fall were sprayed with a bacterial suspension. Ninety percent of the infections were at the leaf scars, while those trees that had been defoliated but not inoculated and inoculated but not defoliated remained healthy.

Webb (138), found early infections taking place in the bark and crevices of the spurs rather than in the leaf scars. Attempts to get infections by way of leaf scars gave questionable results. Webb concluded that 90% or more of the cankers arose as spur infections

(36, 71, 138). It is possible to have partially diseased spurs, dead spurs with no cankers, and dead spurs with cankers (39).

The proposed theory of leaf-scar infection has not been compatible with observations on the Pacific coast (Cameron (in press), 146). Data of both Wilson and Cameron show that cankers developed only when inoculations were made well after leaf fall. Cameron was unable to reduce infection of buds even when trees were defoliated in midsummer and leaf scars were allowed ample time to heal before the fall rains. Experiments in the Pacific coast states suggest that infection takes place by the washing of bacteria into cracks at the base of slightly open bud scales. The seriousness of this infection is greatly increased by an early bud swelling followed by a cool moist period. Buds may be infected without wounding (29a, 164).

Canker development varies considerably with the host, the strain of the bacterium, and the weather. In most cases, cankers start to develop in the fall, either at the base of a bud or a spur (36, 39, 138). Other possible points of infection are cracks, wounds, pruning cuts, stomata; and injuries due to wind, rain, hail, frost, sprays, insects, and cultural operations.

Infected areas increase in size during the winter and cankers become visible in early spring. If infection takes place too early in the fall, the area is walled off by callus tissue and cankers are not produced. In general, *Ps. syringae* is a rather weak pathogen and will cause serious canker damage only when the tree is in a dormant condition. Infections during the active growing season are seldom of any consequence and are apparently isolated very quickly by callus tissue. The ability to wall off the infection seems to be correlated with varietal resistance. The more resistant varieties are generally those that are the most vigorous and start growth earliest in the spring (146). However, the English (146) did not find any appreciable differences between resistant and susceptible plum varieties in relation to earliness of growth of the phellogen in the spring.

It has been stated that older trees become resistant to canker. In England, Royal Anne (Napoleon) seldom becomes resistant. Under Oregon conditions, trees are seldom killed after they have been in the orchard for eight years. Some English growers believe that trees grown in sod or under any condition that reduces the amount of soft succulent growth will also reduce gumming (79). While the amount of gum production may be reduced, it is doubtful if the number of infections is materially lower in sod culture.

The effect of the rootstock on the susceptibility of the scion is of considerable interest. In California, apricots on peach are less sus-

ceptible to canker than apricots on Myrobalan. Apricots grafted onto apricot are intermediately susceptible. Plums on peach are more resistant than plums on either Myrobalan or Marianna (146). For some reason, the tops of high-worked trees seem to be less susceptible to *Ps. syringae* than the same varieties budded low. The previous statement appears to apply equally well in England and in Oregon.

At times canker development appears to be associated with temperature and rainfall. Trees held at a constant temperature of 18-21° C. had larger cankers than trees held at 10° C., and cankers were larger at 10° C. than at 2° C. (140). Cankers may be larger on the south side of the tree due to the warming of the sun during the dormant season (146). Optimum temperature for canker development is between 21 and 24° C., but large cankers may still develop at lower temperatures if the infection becomes established early in the winter. Canker size was small if inoculations were made in late summer or autumn, medium in the winter, and large in early spring. The size of the cankers declined again as inoculations were made into the summer (11, 145).

Cankers apparently develop rather rapidly in the fall after the trees have become dormant but before the occurrence of low winter temperatures. Canker development is slow during the cold periods, but is very rapid between the end of cold weather and the start of rapid tree growth of the following spring. Under Oregon conditions, the severity of symptoms will be determined by the length of time between dormancy and cold weather in the fall and the length of time between cold weather and active tree growth in the spring. Of the two periods, the early spring period is the most critical, and is calculated as the number of days with average temperatures between 2° and 7° C. from January 1 until full bloom. In some areas, the total rainfall during this period may be a controlling factor of disease severity (39), but in the Pacific Northwest there is an abundance of rain during the entire dormant period.

Two types of cankers have been described (146). One type is a definite brown with well-defined margins, while the other consists of a watery or gum-soaked area with brownish strands. As the canker matures, the margins grade into a series of reddish-brown streaks. In some cases, bacteria are found only at the centers of the cankers, suggesting that a toxin may be produced. A toxin has been obtained from *Ps. syringae* grown in culture that appears to be an endotoxin of a protein nature. The more resistant varieties are not damaged by the toxin as much as the more susceptible varieties (146). According to Wilson (146) the English have found viable bacteria in the xylem beneath the canker but not in the bark.

In Oregon, Cameron (unpublished data) has demonstrated that viable bacteria may be found in the bark and in the wood. Five-year-old cherry trees were cut off at the ground line and then sectioned into 3-foot lengths. All of the samples were surface sterilized, and isolations were made from both cross and longitudinal sections. Viable bacteria were found in the vascular tissue as far as 8 feet above the nearest visible canker. This semi-systematic ability of the bacterium may in part account for the ineffectiveness of control measures. It would also increase the possibility of spreading the disease by using infected grafting wood that might not show disease symptoms.

There has been considerable discussion as to whether cankers are annual or perennial. Wilson (146) states that the portion that are active for a second year varies greatly from year to year and from location to location. Wormald (157) states that the cankers are not perennial and that the infection does not extend from one canker to other parts of the scaffold system. He reports that "hold-over" cankers are not known to occur in plum and cherry trees. Wilson (141) found some inactive cankers becoming active in October and November on plums. These old infections were thought to be the source of inoculum for new infections. Barss (11) thought that although cankers were perennial in some cases, they were not the source of new cankers. The host usually forms callus tissue around the canker during the late spring and summer. The position of the callus apparently determines whether the canker will be active the following year. If all of the bacteria are surrounded by the callus, then the canker does not continue to spread in succeeding years; but, if the callus layer is formed in such a position that some of the bacteria in the brown streaks are outside the callus, the canker will continue to spread during the following season.

Webb (138) found that bacteria were extruded from infected spurs after they had been wet about one-half hour. Carry-over bacteria have been reported in buds, leaves, and cankers (86). Crosse (50) in England, and Cameron (unpublished data) in Oregon have isolated bacteria from cankers in every month of the year; but the frequency of isolation was greatly reduced during the summer months. This is in contrast to an earlier report from England (86). In California *Ps. syringae* has been found throughout the orchard, on weeds, fruit, limbs, and leaves, and on nonsusceptible hosts (5, 61). The green fluorescent pseudomonads are ubiquitous, and the question is what percentage of the isolates are pathogenic on orchard crops. Because of the diverse habitats of the bacteria and the rapid potential

build-up, it is doubtful if reducing the number of bacteria from one source of inoculum will materially reduce the occurrence of the disease.

Ps. syringae has seldom been isolated from the soil (114), nor have inoculations with soil produced symptoms (157). Cankers have never been reported on roots, and cankers on the trunk seldom extend below soil level (146).

Bacteria are reported to move from winter cankers to the new foliage by wind and rain before the cankers become inactive. Barss (12) and others (103) have suggested that insects may be involved in moving the bacteria. While *Ps. syringae* has been found on the bodies of some orchard insects in Oregon, it has been just as a contaminant on the feet and body, and it is doubtful if insect spread is of much consequence.

The common occurrence of *Ps. syringae* and its wide host range has led to the belief that the bacteria would be present wherever an orchard was established. While this may be true, it is still difficult to account for some of the high percentages of trees killed by *Ps. syringae* on recently cleared land. In some cases, new orchards have been planted as far as 10 miles from the nearest cultivated area, and have lost 80% of the trees due to bacterial canker. Results of this type suggest that the bacteria may have been brought in with the young trees. Distribution of the disease by infected nursery stock is suggested by Crosse and Wormald (38, 39, 164).

Young orchards in England and in Oregon remain healthy for several years and then suddenly develop severe symptoms. One-and two-year-old plantings in Oregon may suddenly break out with a high percentage of the trees infected. It appears that outbreaks of this sort can be traced to nursery practice. Of considerable concern is the practice of tieing trees in tight bundles and then heeling the bundles in under several inches of soil. Open wounds are made during the tieing and ideal infection conditions are maintained by the heeling in.

Treatment of nursery trees with a complete spray schedule is recommended in both New Zealand (56) and England (150) and should be encouraged in Oregon. It is recommended that scion wood be selected from trees that do not have bacterial canker (38); however, Moore (108) reports no clear relationships between the source of scion wood and the amount of canker after eight years.

On pears, infection with *Ps. syringae* appears very early. Wormald and Montgomery (167) suggest that the bacteria may overwinter either in or on the buds. Under the conditions of the

Pacific Northwest, infection is either during the winter with the bacteria entering through pruning wounds or during bloom with the infection subsequently spreading into the twigs. Rosen and Bleecker describe the disease as an intense blackish coloration along the veins and midribs. The symptoms may be considerably ahead of the bacteria, which is in contrast to fire blight. *Ps. syringae* on pear is favored by cool weather (117).

CONTROL MEASURES

Because of high tree mortality, both orchard owners and research workers have tried to discover satisfactory control measures. While it has been possible to control some phases of the disease, the canker phase has not been satisfactorily controlled after 50 years of research. Efforts to reduce losses caused by *Ps. syringae* have been directed at horticultural practices in the orchard, chemical applications to the trees, and the planting of either less susceptible or tolerant varieties.

Horticultural practices

Beard and Wormald (13), in 1936, attempted to correlate the nutritional condition of the trees with canker development. Low available phosphate in the soil apparently reduced the susceptibility of the trees to canker. Trees supplied quadruple phosphate and a complete fertilizer had more cankers than unfertilized trees. The increase in canker severity might be due to an iron deficiency brought on by the excess phosphate. Applications of potash did not appear to reduce susceptibility. In 1938 Wormald and Garner (163) enlarged the original study and applied nitrogen, phosphate, and potash, separately and in all combinations. Applications were made for four years and replicated three times. The trees were inoculated in the second, third, and fourth years. One replication received an additional treatment of 2 tons of lime per acre. The results showed no statistical difference between any of the treatments, although there was some indication of increased susceptibility where lime was added (161, 163).

There does not seem to be any correlation between winter soil conditions or fertilizer applications and the number or size of cankers (146, 158). Wilson (145, 146) reported that fertilized and unfertilized trees were equally attacked, but that subsequent damage was less on fertilized trees. He reported that soil moisture could not be correlated with canker development unless the moisture dropped below the permanent wilting point. When the trees began to wilt,

canker development was usually arrested. No difference in the number of cankers was noted with or without late summer irrigation (140).

Many growers have felt that trees grown in sod were usually not as susceptible to canker, but they were usually not making a comparison with trees handled in a similar manner under clean cultivation. In general, trees that are kept growing vigorously late in the season will tend to have more cankers than trees that have started to harden off in midsummer.

Barss (11) reports wrapping of trunks and limbs with burlap for the first five or six years. This practice has not been either practical or satisfactory and is no longer recommended.

Millthorpe and Vincent (103) suggested that pruning be done as soon as possible after leaf fall in order to reduce infections. Crosse (38) also advocates avoiding wounding the tree during wet periods. Wormald (158) advocates pruning either before October or late in the winter. No evidence of increased canker development has been noted between fall, winter, and summer pruning in the Pacific Northwest, and Moore (109) did not discover any difference between May vs. October pruning. Pruning out old cankers in summer may help reduce the amount of inoculum (146).

The procuring of healthy trees from the nursery is of great importance. Excessive losses are frequently observed in the first two years after planting (34). Dye (56) states that if trees can be kept clean for the first year, further infection is greatly reduced. The New Zealand recommendation is that nursery trees be sprayed with Bordeaux in the fall after budding and with streptomycin in the following spring. During the growing season, trees receive three sprays of streptomycin prior to and during leaf fall followed by Bordeaux during the winter. Young trees are pruned, either heeled in vertically not in bundles, or planted, and then sprayed. The nursery spray schedule is followed for the first year in the orchard.

Whether the tree is high or low worked and the nature of the rootstock-scion combination are both of great importance. As early as 1913, Barss (7) suggested the use of resistant or tolerant trunk stocks. He reported that mazzard as a trunk stock is a "thoroughly practical way of protecting cherry from the disease." The top of the tree seemed to be less susceptible to the disease if the trunk and scaffold limbs were resistant. Grubb (80) tried different rootstocks grafted both high and low. In all cases there was much greater susceptibility on low-worked trees. High-worked F-12/1 was a very consistent stock and only 2.6% of the trees had cankers compared with 27% when mazzard seedlings were used. Considerable variation was

found in all seed sources, but more variation was noted in English trade sources than from seed collected from wild seedlings in Pennsylvania.

In the Pacific Northwest, young mazzards set in the orchard should be allowed to grow until scaffold branches are at least one quarter inch in diameter at a distance of 8 inches from the trunk. Each scaffold branch may then be grafted or budded with the desired commercial variety. Trees high worked in this manner had a much lower incidence of canker than trees grafted near the ground. Using a 0 to 5 rating scale (0 = no infection, 5 = dead tree) the following average ratings were observed: trees worked low on mazzard 3.0, trees worked high on mazzard 1.3, trees worked high on mahaleb 0.7. The use of less susceptible varieties for the trunk and scaffold limbs has also been recommended by Moore and Crosse (38, 108, 109). Four selections of Myrobalan B proved to be highly resistant as rootstocks, but President was incompatible with Myrobalan B (108).

Moore (108) warns that infected scion wood may be regarded as a possible means of infecting the tree. Although results in one commercial orchard in Oregon suggest that it is possible to spread the bacteria by using scion wood from infected trees, this has not been substantiated in controlled experiments. One hundred and fifty trees were bench-grafted with scion wood from trees with severe limb cankers and compared with bench-grafted trees grafted with clean wood. At the end of two years there was no significant difference in the amount of canker. Since canker does not always appear in the first years, the trees will be planted in an orchard and observed for the next four or five years. No definite conclusions may be drawn at this time, but it would be wise to obtain scion wood from healthy trees until it is known if the bacteria may be spread in the grafting wood.

Crosse suggests that scion wood be dipped in Bordeaux before budding or grafting as an extra precaution (38). The Bordeaux may reduce bud take, therefore a 200 ppm solution of streptomycin would probably be preferable.

As has been previously implied, mazzard seedlings are not all resistant to the disease. In a few trees, mazzard trunks will become infected as severely as commercial varieties. In other cases the cankers will develop downward into the resistant trunk from the susceptible scion (161). However, in most cases mazzard seedlings will form a wound layer and quickly close off the infected area (55, 108). It is the ability to wall off the infected area rather than a true resistance that makes mazzard a suitable trunk stock.

Cherry trees worked high on a tolerant rootstock sometimes have less canker in the scion. This may be due to a degree of resistance imparted from the rootstock to the scion or just to a lowering of available inoculum in the tree (80, 10). In light of research in Oregon, the latter theory seems the most likely. Viable bacteria have been found in the vascular elements as far as 8 feet from the nearest canker. If these bacteria are capable of causing infections, then a canker on the trunk could be the origin of many infections in the upper part of the tree. According to this theory, the use of less susceptible trunk stocks could materially reduce the total number of infections. Plum trees with trunks of Warwickshire, Drooper, Utility, and Myrobolan B were reported to be resistant in most years (105). However, according to Amos, Hatton, and McKenzie (3), grafting of plums on peach rootstock is reported to reduce cankers (4).

Chemical control

Numerous attempts have been made to control the different phases of bacterial canker by the application of chemicals. Both leaf-spot and killing of buds have been successfully controlled (22, 45, 46, 48, 108), but control of the canker phase has generally been erratic.

In laboratory tests, several materials are effective bactericides, but these are not always satisfactory in the field. Wormald (159), in 1935, reported the following compounds bacteriostatic at the indicated percentages when tested under laboratory conditions: Phenol 0.1, ethyl alcohol 8.0, formaldehyde .01, 8-hydroxyquinolin potassium sulphate .2, zinc sulphate .005, and mercuric chloride .0005. Cooper sulphate was bactericidal at .01% in 10 minutes, zinc sulphate at .1%, and tar oil at 1.0%. Montgomery and Shaw (104) determined the toxicity of 29 different metals. Mercury was the most toxic followed by gold and silver and uranium and copper. Twelve different forms of copper were tested. Cupric oxide was not toxic while cuprous oxide was. Bordeaux was only slightly more toxic than calcium hydroxide. As a group, the hydroxides were much more toxic than the chlorides. The bacteria were killed in about one hour at pH of 10.4 or one hour at pH of 3.0. Phthalic acid was toxic to the organism in addition to its effect on the acidity. In Oregon, aureomycin, 60 mcg; bacitracin, 20 units; chloromycetin, 60 ccg; dihydrostreptomycin, 100 mcg; penicillin, 10 units; polymycin nitrate, 100 ppm; streptomycin sulphate, 100 ppm; and agrimycin, 100 ppm were tested against *Ps. syringae*. Paper disks with each of the above concentrations were either purchased or prepared and placed in petri dishes inoculated with the bacteria (Figure 17). Streptomycin was

the most effective and was equally effective in either the sulfate or nitrate form. Laboratory tests by Deep[1] have found the experimental chemicals OM-8 and OM-10 effective at 1 ppm and CC-2, CC-1, and HC-1 effective at 10 ppm.

Dye (52, 53) tested 23 fungicides in the greenhouse and reported streptomycin sulfate as giving the only satisfactory control. The chemicals tested were: Bordeaux, copper oxychloride, cuprous oxide, colloidal copper, phenyl mercury chloride, tetramethyl thiuram disulphide, ferric dimethyl dithiocarbamate, 2, 5 dichloro-1, 4-naphtoquinone, N trichloromethylthio-tetrahydro-phthalimide, phenyl mercury salt of methyl methane sulphonic acid, copper zinc chromate, 1, 2, 3-trithio, 5, 8-diazacyclononame, 4, 9-dithione, a-a trithiobis (N dimethylthio-formamide), manganese ethylene bisdithiocarbamate, zinc ethylene bisdithiocarbamate, tetrachloro p-benzoquinone, zinc dimethyl dithiocarbamate, 8-quinolinol, 8-quinolinolate, benzoate, zinc 8-quinolinolate, copper 8-quinolinolate, streptomycin sulfate, aureomycin hydrochloride, terramycin hydrochloride, patulin, and chloromycetin.

The chemicals streptomycin sulfate, N trichloromethylthio-tetrahydro-phthalamide, zinc ethylene bisdithiocarbamate, and Bordeaux were applied in the field to artifically inoculated trees. Streptomycin gave satisfactory control and Bordeaux was satisfactory in some cases. Neither of the other two chemicals was satisfactory (54, 55).

Field trials have shown that some chemicals that rated high in laboratory and greenhouse experiments did not hold up when used under orchard conditions. Harris (87) reported that, in plot trials, autumn sprays of streptomycin produced only slight reduction in the number of viable bacteria, but materially reduced the number of cankers in the following spring. However, the streptomycin was only temporarily effective and autumn sprays of Bordeaux were more effective than those of streptomycin. Streptomycin applied in the spring was more effective in reducing leaf-spot than Bordeaux. Similar results were reported by Crosse (45).

Crosse reports good control of the leaf-spot phase with streptomycin at 200 international units per millimeter applied two or three times during bloom (43, 45). Canker control was not satisfactory. Harris (88) mentions some evidence for the systemic action of streptomycin. He was able to find penetration into the leaves and a slow translocation via the petiole. Translocation of streptomycin has also been reported in peaches by Dye (51). No movement of strepto-

[1] Associate Plant Pathologist, Oregon State University.

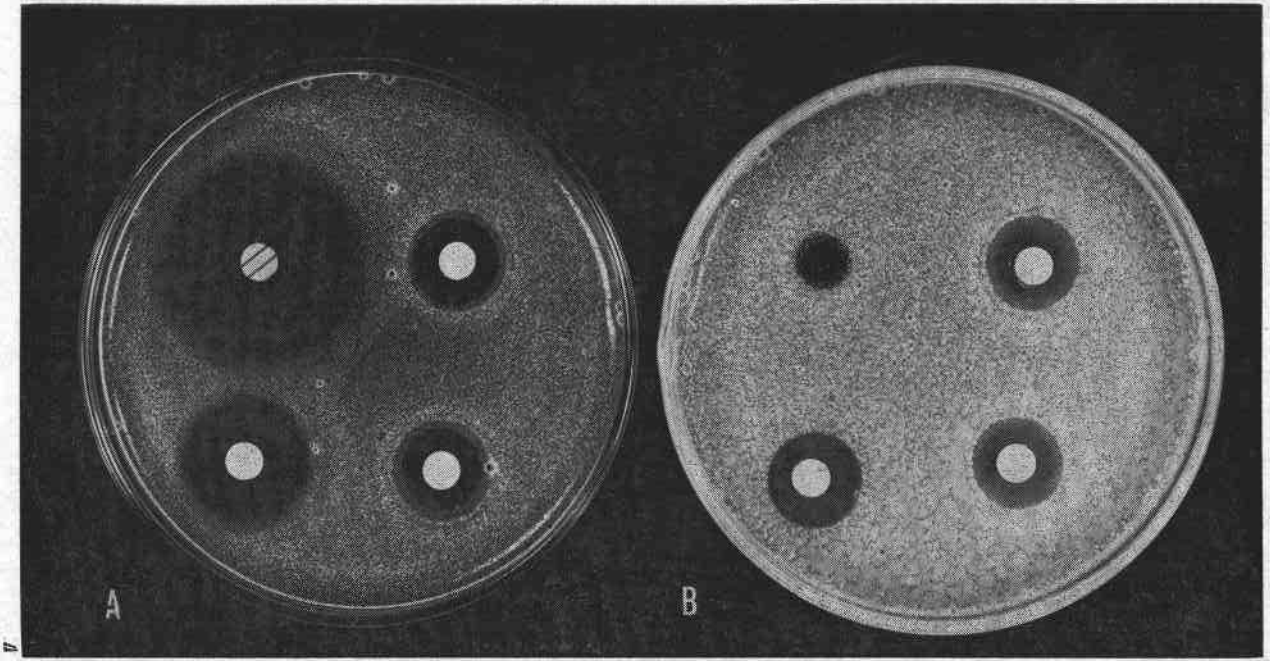

Figure 17. A. Upper right: Difco terramycin. B. Upper right: Difco penicillin. A and B. Upper left: Streptomycin sulfate, 100ppm. Lower right: Terramycin, 100ppm. Lower left: Streptomycin nitrate, 100ppm.

mycin out of cherry leaves has been detected in Oregon even after two applications of 200 ppm to the leaf surface. The life of streptomycin applied in the autumn was usually about six days under Oregon weather conditions and streptomycin was completely ineffective in a commercial control program (28).

The use of Bordeaux in various concentrations and in different seasons has been the most frequently recommended control. Results have been very erratic and have ranged from 85% control to no control. In spite of the fact that Bordeaux usually rates low in laboratory tests, it is generally the most effective field control because of its long residual action. Since initiation of the canker stage is during the fall and winter, it is necessary to apply a material that will protect the tree during the wettest part of the year. It is usually impossible to spray very frequently during the rainy season, since it is difficult to move heavy spray equipment through wet orchards. Because of Bordeaux's slow breakdown over an extended period, it has given the best protection against the canker phase of the disease.

Both Anderson and Moore (4, 108) recommend Bordeaux sprays in spring and autumn. Hutton suggests 15-15-100 Bordeaux in fall, midwinter, and late winter (96). Crosse (38) states that Bordeaux 10-15-100 in mid-October will reduce canker, and used at 6-9-100 in the spring will reduce leaf spot. It is best to use both sprays even though the fall spray may not be as effective. The spray program should be started when the trees are young, and it is usually more effective on cherries than on plums. A similar program used by Montgomery and Moore (106, 107) was effective over a 5-year period. Two sprays in either spring or fall did not significantly improve control.

English et al (61) reported that soil fumigation of sandy soil prior to replanting reduced the amount of tree loss due to *Ps. syringae*. No trees died for three years following soil fumigation with either DD (1, 3 dichloropropene and 1, 2 dichloropropane) or Picfume (chloropicrin) prior to tree planting. Vigor of trees and resistance to artificial inoculation were somewhat greater in trees planted in fumigated soil.

Working with plums, Moore (108) reported that in four seasons fall applications of Bordeaux did not reduce the number of cankers. Bordeaux would control leaf spot, but did not seem to have any effect on the number of stem cankers. Similar results on plums were also reported by Montgomery and Shaw (105). Crosse (38) suggests that poor control results may be due to inadequate coverage, fall sprays applied too late, or the omission of spring sprays. Field observations in Oregon would suggest that inadequate coverage is the

primary cause of poor control. Delayed application reduces the effectiveness of the spray program, but still gives some degree of control. Omission of the spring spray did not influence the effectiveness of the program in Oregon.

Crosse and Shannuganathan reported that spraying trees with either streptomycin or Bordeaux did not appear to increase the pathogenicity of the remaining bacteria. Even clonal lines showed different degrees of resistance (47).

Harris (88), in 1952, reported that liquid injections of 8 hydroxy quinoline potassium sulfate were made into cherry, but no results were given. So far as is known, this is the only attempt to control this disease by chemotherapy.

Sprays of streptomycin during bloom controlled pear blast and increased fruit set, but sprayed trees dropped an excessive number of fruit so that yield differences were barely significant (56). Bordeaux was less effective as a control chemical and also resulted in a large fruit drop.

Host susceptibility

Differences in susceptibility to different parts of the disease cycle of *Ps. syringae* have been reported on many occasions. Growers have noted that certain varieties in mixed plantings were more tolerant of infection than others. Lists have been published by many research centers giving the degree of susceptibility of the varieties in their experimental plantings. These lists have varied widely depending on the area, the phase of the disease being considered, and horticultural practices. The degree of resistance, or tolerance, of a particular variety frequently depends on its stage of development in relation to the time that inoculum is available. Because of differences in time of infection and in climatic conditions, varieties listed as resistant in one part of the world are classed as very susceptible in another part.

Within the *Prunus* species, apricot is listed as most susceptible, sweet cherry and some varieties of plum as second, followed by nectarine. Peach is considered to be relatively resistant and almond is seldom affected (4, 44). Sour and duke cherries are rarely infected (11, 146).

For rootstocks, Wilson (146) reports that Marianna (a hybrid of *P. crasifera* and a native plum) is prone to infection. *P. crasifera* (Myrobalan) is much more resistant as are most mazzard seedlings. English Myrobalan B and Purple Pershore are supposedly resistant, and Mahaleb seems to be resistant. The rootstocks F 1-1, F 5-5, and F 12-1 appeared to induce a degree of resistance to the scion, while F 2-1, F 5-3, F 5-4, and F 12-4 seemed to increase the susceptibility

of the scion (106). Grubb (79) does not feel that there is any direct evidence that rootstocks affect the resistance of the scion. If such an effect does exist, it is thought to be due to an influence on vigor.

Among the apricots, the varieties Tilton, Blenheim, Moorpark, and Royal are all very susceptible (37, 140, 146). Croughton is somewhat more resistant (37).

Susceptibility of sweet cherry varieties varies with the area reporting. Grubb (79) lists Abundance, Bedford Prolific B, Beeve's Heart, Belle de Droures, Bigarreau de Mezel, Bigarreau de Schrechen, Bing, Bloor's Heart, Burbank, Centennial, Chapman, Early Bigarreau, Florence, G. d'Hedelfingen (Bradbourne Black), "Crosse Schwarze Knorpelkirsche," Guigne d'Annonay, Ironsides (Ohio Beauty), Late Amber, Lewelling, West Midlands Bigarreau, Yates' Seedling, Yellow Spanish, August Heart, Beechcroft, Bowyer Heart, Dangler, Leicester, Napoleon Bigarreau, Peggy Rivers, Pointed Heart, Ronald's Heart, Sutton's Prolific, and Werders Early Black as very susceptible. Black Downton, Burr's Seedling, "Early Purple Gean," Goodnestone Black, Great Bigarreau, Guigne trés Précose, Hooker's Black Hoskin, Ken Bigarreau (Amber Heart), Knight's Bigarreau, Ludwig's Bigarreau, Norwegian, Ord, Pontiac, Ramon Oliva, Royal Queen, St. Margaret's (Noble), "Thamenkirscheganz susse Schwarze," and Wellington A. are reported as considerably susceptible.

Grubb (79) also lists Baumann's May A., Bedford Prolific (may be a Roundel), Belle Agathe, Black Eagle, Black Elton, Black Tartarian E., California Advance, Circassian, Cryall's Seedling, Dunn "Mazzard," Early Amber, Early May (Mumford), Early Rivers, Elton Heart, Gov. Wood, Knight's Early Black, Lulsley Early Black (Fruheste der Mark), "Noir de Schmidt," Old Black Heart A, and B., Philpott's Favorite (Big Reverchon), Rockport Bigarreau, "Schneider's Spate Knorpelskirsche," Smoky Dunn, Strawberry Amber, Turkey Heart, Victoria Black, Wellington B., and Windsor as slightly susceptible. The following varieties he lists as very slightly susceptible: Baumann's May B., Bigarreau de Jaboulay (Early Lyons and Fruhe Rote), Black Cluster, Emperor Francis, Frogmore Bigarreau, Ham Green Black, Hollander, Kassin's Fruhe, Lambert, Large Black "Mazzard," Longley's Black Eagle, Maiden's Blush, Newington Black (Carron B.), Noir de Guben, Norbury's Early Black, Nutberry Black, Perserving "Mazzard," "Red Turk," Rodmersham Seedling, Roundel, Small Black "Mazzard," Sutton's Purple, and White Heart. Roundel was the most resistant (42). The varieties not only varied in degree of susceptibility, but also in the amount of infection of various phases of the disease. Thus Early

Rivers was highly susceptible to leaf-spot and tolerant of the canker phase, while Bigarreau de Schrecken was the reverse. Early Rivers has considerable areas of the bark invaded in certain seasons, but the cambium is not damaged and the tree eventually recovers with no sign of injury (79).

Wormald (161) moves Black Eagle, Early Rivers, and Black Tartarian to the very susceptible class and adds Cluster Black Heart, Brandborne Black, Waterloo, and Turk. Frogmore is listed as particularly resistant to canker and leaf-spot while Napoleon (Royal Anne) and Waterloo are very susceptible to killing of young shoots (38, 161). In addition to the above list, Moore (108) moves Emperor Francis, Gov. Wood, and Elton into the susceptible class.

Wilson (140, 146) considers Lambert as the most susceptible variety in California, followed in order of increasing resistancy by Napoleon (Royal Anne), Bing, Chapman, Black Republican, and Black Tartarian. Of the five major varieties grown in Oregon, Van is the most susceptible followed by Napoleon (Royal Anne), Bing, Lambert, and Black Republican (10, 11).

Readings have been taken in variety trials in Oregon from 1956 through 1959. The following varieties are susceptible to *Ps. syringae* under Oregon conditions: Spaulding, Napoleon (Royal Anne) both normal and selections 11, 14, and 24, Lambert, Knight's Early Black, Grose Transparent, Beste Werdersche, Sue, Sam, Delle Isola, Noire de Montreaux, Summer Lapins, Guigne Poupre Native, Wagner, Variety, Planteahole Og Frchandel, Majkors Fran Aonarys, Moretta di Arezzo, Williams Favorite, Rease Farm, Hollender, Inspecteur Lohnis, Kleine Waslae, Schreken Biggareau, Van, Geneva, Vista, Oxheart, Bing, E. F. Palmer, Dickson, Rainbow, Stripe, Centennial, Berger, Hoskins, Merta Favorite, Merton Bigarreau, Merton Bounty, Black Republican, Bigarreau de St. Charnez, Geneva 1491 and 1507, Vic, Belle D'Annonay, McMar, Spanish Yellow, Earnstadter Schwarze Knorpelkirsche, White Heart, Esperon, Rosmarin, Donnaissen's Gelbe Knorpelkirsche, Ramon Oliva, Emperor Francis, Burbank, Dicke Braune Blankenburger, Oliver Long Stem Bing, Fogle, Morasa Nioscate, Allen, Victor, Bigarreau Napoleon (Royal Anne), Early Rivers, and Noble. Magnifique, Griotte du Pays, Persian Cherry, Braunauer, Color de Pigeon, and Meyers were somewhat resistant and Dickson, Emperor Francis, Giant, and Hedelfingen have not shown any symptoms of bacterial canker. The fact that Hedelfingen is listed as very susceptible by Grubb and has not shown any symptoms in Oregon may be due to a failure of an inoculation to take in the Oregon trials.

In 1957 a general survey was made at the United States De-

partment of Agriculture Plant Introduction Garden at Chico, California. In most cases resistance was associated with small-fruited selections and commercially acceptable large-fruited selections were quite susceptible. If, in reality, there is a linkage between resistance and fruit size, the difficulty of obtaining a resistant commercial variety will be greatly increased.

Of the plum varieties, Duarte is considered to be the most susceptible and is followed by President, Clyman, Climax, Giant, Grand Duke, Wickson, Santa Rosa, Tragedy, Burbank, Formosa, Gaviota, California Blue, Earliana, Sugar, Beauty, and Kelsey in decreasing order of susceptibility (4, 146). Other reports would consider Burbank as less susceptible and Beauty as more susceptible (146).

Of the English varieties, Victoria is reported as the most susceptible, followed by Czar, Giant Prune (Burbank), and Early Laxton (84, 108). President, Riber's Early Purple Egg, Gage, and Black Bullance are listed as semi-resistant (158) as is *Prunus demissa* (10).

In peaches the varieties Elberta, Phillips Cling, Halford 2, and J. H. Hale may be severely infected. Alexander and Levy are moderately susceptible and Lovell, Tuskens Yellow, St. John, and Early Crawford are slightly susceptible (140).

Bacterial canker has been reported on Nonpareil, Ne Plus Ultra, and Texas varieties of almond (140).

None of the acid cherries are very susceptible, and in most places they are considered to be completely resistant (10). Grubb lists Flemish Red, Groitte de Portugal, Gros Gobet (Short Stalk Montmorency), and Wye Morello as considerably susceptible; Morello A. as moderately susceptible, and Carnation, Coe's Carnation, Kentish Red, Morello B., Ostheimer Weichsel, and Triaux as very slightly susceptible. (79).

Of the "Duke" cherries (cross between sweet and acid cherries) Planchoury is very susceptible; "Belle de Franconville," May Duke, and Reine Hortense considerably susceptible; Archduke and Ronald's Late Duke (may be "Rote Mai") as moderately susceptible; and "Belle de Chatenay," Belle de Choisy, Empress Eugenie, Nouvelle Royale, Olivet, and Royal Duke as very slightly susceptible.

Actual breeding of cherries for resistance to canker has been reported only from Sweden (111). Progeny of Frogmore and Schreckens were more resistant than progeny of Allman Gulrod, Rivers Early, or Annonay. A breeding program has now been started in England, but results have not been published.[1]

[1] Conversation with Dr. W. G. F. Sewell.

Ps. syringae may cause severe blast on Winter Nellis, William Bon Chretien, Durondaur, Pitmaston, and Catillac varieties of pear (57, 167).

Mode of resistance

In most cases of reported resistance, the host is not actually resistant to the bacteria, but the infected area is quickly walled off and the injury heals over. Two hundred mazzard seedlings were inoculated with *Ps. syringae*, and all of these trees developed cankers; however, all but one of the trees had completely recovered by the next season. Recovery of this type is usually reported as resistance to the bacteria.

Harris (89) reports that much higher concentrations of inoculum were necessary to establish a given level of infection in a resistant variety than in a susceptible variety. More inoculum is available in the fall on the leaves of the more susceptible varieties. Three times as many bacteria were found on leaves of Napoleon as on Roundel (38).

Crosse (38) suggests that resistance to the disease may be both mechanical and chemical. Erikson (63) points out that in susceptible hosts the bacteria were found past the periderm and in the xylem. Isolations of bacteria could be made from the xylem even during the summer. In resistant varieties, the infected area did not progress past the periderm and was usually confined to the phloem. Similar results were obtained in Oregon when 5-year-old Napoleon trees were sectioned and samples taken through the vascular tissue at 2-foot intervals.

Erikson and Montgomery (63, 64) discovered that disease symptoms could be caused by a cell-free filtrate. This filtrate caused the most damage on the more susceptible varieties. Cultures grown on extracts from resistant trees gave more toxic filtrates than those grown on extracts of bark from the more susceptible varieties. Filtrates from strains of *Ps. syringae* varied from noninjurious to very toxic. An acetic acid extract, unheated and unprecipitated, was quite toxic. Evidence suggests that the toxic substance may be of a protein nature (64).

Pectin was the only polysaccharide tested that could be utilized by the pathogen. None of the chemicals, except tannins and phenols, normally found in plum bark, were toxic to the organism. The organism was able to live in limited areas in both the resistant and susceptible varieties, but the periderm was formed earlier in the resistant varieties (64). Very rapid formation of periderm in resistant varieties has been confirmed in Oregon. Since the cells were observed

to disintegrate prior to visible bacteria in the tissue, it was thought that a toxin was released by the pathogen. The toxin was not produced by cultures until they were approximately 5 weeks old (64).

As has been mentioned, resistance is relative, and a tree that may be recorded as resistant under one set of circumstances may be very susceptible if circumstances are changed. Denniston's Gage appears to be resistant in the orchard, but is very susceptible to artificial inoculation (158). Mazzard seedlings are usually resistant in the orchard, but they may have long cankers that heal very quickly when they are artificially inoculated (29). Roundel, which is normally highly resistant to all forms of bacterial canker, was as susceptible, or more so, than Napoleon (Royal Anne) when it was inoculated at petal fall (42). Spur blight was bad on early season and mild on midseason flowering varieties. In California plums are seriously attacked by *Ps. syringae* in the foothill area, but the disease is not serious in plums grown on the valley floor (140).

The degree of disease development is also less in the more resistant varieties. In one case both the resistant and susceptible varieties had an equal number of infected buds, but 11% developed into cankers on the resistant variety as opposed to 63% on the susceptible variety (36). Crosse (39) reports that in the variety Napoleon, infection ultimately spread to the branch tissue in many more cases than in resistant varieties.

Appendix A

Translation of the original description of
Pseudomonas syringae van Hall as taken from
Dr. van Hall's Ph.D. thesis (82)

Translation by AART HARMON HUYSKAMP and H. R. CAMERON

In two-day-old bouillon culture (27°) single and double rods are present, no chains of cells; in 24-hour-old culture in dune-water plus 0.025% K_2HPO_4 plus 0.25% asparagin there are many chains of four to six individuals present. The rods are slender and small. In the bouillon culture the proportions are 1.6μ-3.2μ long, and 0.2μ-0.4μ wide (stained with gentian violet and mounted in Canada balsam). In the asparagin culture the movement is great and the flagella stain easily, both with Pittfield's or with Zettnow's or Leffer's method. The length of the flagella is from 7μ-10μ.

Gelatin is quickly liquified and is clear except on meat gelatin where the liquid is turbid. No precipitate is formed in the first three or four days. The colonies on meat gelatine look like those of *Ps. fluorescens,* but the fluorescence seems to be less. On a solid media containing asparagin or in liquid containing asparagin, the fluorescence is very obvious.

Cultures on cooked potatoes and carrots show no distinguishing characteristics. A vigorous growth is found on malt gelatine. The growth in milk is also vigorous; the casein is precipitated although a slow coagulation occurs.

A temperature of 49° C. for 10 minutes has no harmful influence on 24-hour-old bouillon cultures. – – – – – – – Not all of the specimens are killed after 10-minutes' stay at a temperature of 50° C. – – – – – – – Ten minutes' stay at 51° C. has killed all specimens of the 24-hour-old bouillon culture. Under the before-described circumstances, the maximum temperature lies between 50 and 51 degrees. Accurate determination of the optimum temperature has not been found, but at 27° C. the growth is very rapid.

Ps. syringae is an obligate aerobe. In streak culture in meat agar, meat gelatin, and peptone-cane sugar agar, growth is noticeable only on the surface and, at most, a few millimeters under the surface. No trace of growth is found over the rest of the streak. Completely in accordance with this, there is noticeable sediment on cooked bouillon under oil.

Reduction of methyl blue

For obligate aerobic bacteria, the methods of the results do not give a correct picture of the reduction ability. This is because the growth of such bacteria under oil is very slight and, therefore, the intensity of the reaction is less than if free oxygen were available.

As I have already explained, tests where free oxygen is available are quite variable and, therefore, are practically useless for the description. Taking the slight growth into account and the rate of multiplication of *Ps. syringae* in cooked-out liquids under oil, we have to conclude from the reduction tests that the bacteria has a strong ability to reduce methyl blue. Bouillon plus 0.002% methyl blue: discoloring after two or three days is completed. Bouillon plus 0.004% methyl blue: discoloring completed after three or four days.

The following liquids were all dissolved in dune water plus 0.025% K_2HPO_4:

1% peptone plus 3% mannite plus 0.004% methyl blue: fairly fast discoloring which is usually completed in approximately four days.

0.25% asparagin plus 3% saccharin plus 0.004% methyl blue: slow discoloring, which after 14 days was not yet discolored.

0.1% ammonia sulfate plus 3% glucose plus 0.004% methyl blue: very slow discoloring, after 14 days no distinct discoloring is noticeable.

0.1% glycol plus 3% glycerine plus 0.004% methyl blue: very fast discoloring which is completely completed after four days.

0.1% urea plus 3% glucose plus 0.002% methyl blue: very fast discoloring, completed after approximately four days.

Nitrates are reduced only under certain circumstances. As it seems to me, they are only used when no other better nitrogen source is available. In meat agar plus 0.1% KNO_3, after 24 hours at 27°, no nitrate was formed at the edge of the luxuriant growth, and by continued culture I could not discover nitrate after six days. On 3% cane sugar plus 0.1% KNO_3 and agar, after 24 hours at 27°, there is a vigorous growth and extensive production of nitrite. It is the same in the case of liquid culture using 3% cane sugar and with 0.1% KNO_3 as the only nitrogen source. In growth studies, nitrogen was formed in all carbon sources which supported growth and in which 0.1% KNO_3 was the only nitrogen source available.

Sodium selenite is slowly reduced. Usually, on meat agar plus 0.05% $Na_2Se\ O_3$, the red color starts separating visibly from the S.E.; in stab culture, after about three or four days.

Determination of useful nitrogen and carbon sources by measurement of growth

1. *Compounds which are both nitrate and carbon sources:* Noticeable growth occurs with ammonium citrate, ammonium soccinate, and ammonium acetate; a limited amount with asparagin and gluten; no growth occurs with "Witle" peptone, "Cornelis" peptone, casein, gluten casein, albumin from the white of egg and from the yolk, fibrin, protein, glycocol protein, tyrosine, ammonium tartrate, and ammonium lactate.

2. *Carbon compounds in combination with different nitrogen compounds:* Nitrogen compounds were added to agar-gelatin in the following order: "Witle" peptone 1%, asparagin 0.25%, potassium nitrate 0.1% and glycocol 0.1%. With all of these a strong or noticeable growth occurred by adding: sucrose, glucose, laevulose, galactose, manitol, glycerin, sodium citrate, sodium succinate, sodium acetate, and sodium butyrate. No growth occurred after adding lactose, maltose, dextrine, inulin, and sodium tartrate. The results are put together in the following table. (Table on page 196 is not included in the translation since the information is also in the article.)

Diastase production does not occur by *Ps. syringae,* and the starch is not being converted and, therefore, has no feeding value for the bacteria.

Production of acids and alkalies

On complete aerobis growth (stab culture) on meat agar plus 5% cane sugar and litmus, the formation of alkali predominates. In the region of the stab, after 24 hours at 27°, the purple color is changed to blue. In bouillon plus 5% cane sugar the formation of acid is predominate after two weeks.

When the organism is cultured in different liquids, a slight amount of acid is formed through incomplete oxidation of carbohydrates, although at times a strong alkali is formed. After titration for 14 days in culture of 27°, the following liquids gave the recorded results. (Liquids are listed as A, B, C, D, E, F and are not described; therefore, the table is not included.) Alkali in milk.

Production of indol was not noticed. The indol reaction did not occur in two-week-old cultures in either bouillon or bouillon plus 5% peptone.

Production of hydrogen sulfide did not occur in bouillon. After 14 days the lead-soaked papers were completely white.

Production of gases

P. syringae did not produce gas in the tested liquids. The following medium was tested: dune-water plus 0.025% K_2HPO_4 plus 1% peptone to which, respectively, was added 3% cane sugar, 3% glucose, 3% manitol, 3% galactose, and 3% glycerine (maltose and lactose are not assimilated by these bacteria).

Production of glycogen

Glycogen could still be detected with iodine in two-day-old sucrose-peptone and also in bouillon culture. Production of fat is not noticeable in bouillon cultures, also not in sucrose-peptone culture.

Endurance against drying out

Ps. syringae is resistant against drying out; the dried out material produced a strong turbidity after 24 hours in bouillon.

Influence of acids

After a few days, growth starts in bouillon which is acidified with lemon and apple acids until the reaction is plus 0.5% normal; sometimes growth still occurs in bouillon plus lemon or apple acid of 1% normal. In bouillon with a higher acidity, consistent turbidity has never been noticed.

Acceptance of color

In regard to color susceptibility by *Ps. syringae,* nothing noteworthy was noticed.

Appendix B

The description of the organism isolated from citrus by Fawcett (71) differs in some respects from the original description and also includes some tests that were not run with the original culture by van Hall.

"Cylindrical with round ends, 1.2 to 4 by 0.4 to 1.0 μ (commonly 1.8 by 0.6 μ) motile with one to many polar flagella: No spores or capsules present; growth on standard nutrient agar, pearl gray or grayish white, thin and spreading on sterilized orange and lemon rind, straw color becoming dark fawn. Colonies circular with indistinct entire margins: liquefaction of gelatine, at first crateriform soon becoming stratiform, growth in litmus milk distinctly alkaline without separation of casein; growth in closed arm of fermentation tubes of dextrose and sucrose, but no growth in tubes of lactose or maltose; no reduction of nitrates; indol formation positive. Upper limit of hydrogen ion concentration in nutrient standard bouillon about pH 5.2."

Fawcett's organism is the only isolate to give a positive test for indol formation and is also described as multi-flagellate. No mention is made of green fluorescence.

Appendix C

Burkholder (21) in his "The Genus Phytomonas" describes the bacteria as follows:

"Agar pigmented green. Motile, gram negative, soft curd in litmus milk, alkali in litmus milk, pep. plus in litmus milk, growth on starch weak, dextrose plus, sucrose plus, lactose negative, gelatine plus, nitrite negative, indol negative, hydrogen sulfide negative, faculative aerobe, no capsule production."

Appendix D

Dye (50) gives a complete description of the organism isolated in New Zealand as follows:

"Bacteria exist separately and in pairs as short rods, rounded ends, vary from 0.8 μ to 2.2 μ by 0.5 μ to 0.7 μ with an average of 1.4 μ by 0.6 μ. Gram negative (hucker stain) motile by one to several flagella (gray and Leifson stains). Beef peptone agar colonies show growth at 25° C. in 48 hours as moderate, circular, smooth, entire to undulate, convex later becoming flat, translucent, grayish white, and with no pigment formed in the media.

"On beef peptone agar slant the colonies are moderate, filiform, glistening, grayish white, butyrous, and with a slight odor. The media remains unchanged. In beef peptone broth there was no surface growth after seven days, clouding was slight, no color change, scanty white flocculent sediment, and no odor. After 21 days there was moderate clouding and no surface growth. After seven days' growth on sliced potato, growth was moderate (never abundant), moist, glistening, butyrous, filiform and buff colored. The

potato remained unchanged and no differences were noted after fifty days. Other cultural characteristics were as follows:

Nutrient gelatine stab: 10% at 25° C. infundibuliform liquifaction in 24 hours, later becoming stratiform, 1-2 inch liquification in 10 days with white viscid sediment.

Plain gelatine stab: 10% at 25° C. similar to above.

Purple milk: Clearing in bands shows within seven days, three zones being evident; top, 15-20 mm., translucent, iridescent red-blue; center, 15-20 mm., slightly cleared reddish; bottom 8-10 mm., opaque, grayish white.

Nitrates: No nitrite production in nitrate broth or in beef peptone agar plus nitrate in seven days.

Indole: Not found.

Hydrogen sulfide: No hydrogen sulfide in 21 days.

Starch (beef peptone agar plus 0.2 percent soluble starch): Rapid sometimes irregular spreading growth on fresh plates, no hydrolysis in either 24 hours or 30 days.

M.R.: Negative.

V.P.: Negative.

Synthetic carbohydrate media (1 percent): No gas in any medium, acid in arabinose, xylose, glucose, fructose, galactose, mannose, sucrose, raffinose, glycerol, mannitol and sorbitol in 21 days; lactose and rhamnose, no acid in 28 days, but acid in 50 days; maltose, starch, inulin, dextrin, salicin and melezitose, no acid in 50 days.

Salt (beef peptone broth plus Na Cl): Seven days' growth in 2 percent equal to check, moderate clouding, no pellicle; no growth above 6 percent in 21 days. Sucrose at 5.0 percent included in the above salt solutions caused strong clouding and pellicle formation, but no change in growth range.

Beef peptone agar plus 5 percent sucrose: Colonies circular, smooth, entire, hemispherical, white, opaque, have tendency to become translucent in center after seven days, no loss of viability in seven days.

Beef peptone sucrose agar streak: Abundant filiform, glistening white at first; after seven days, middle of streak clears, becomes less viscous, frequently flows to base of the slope as creamy white fluid which separates into a transparent fluid above a white opaque fluid.

Purple lactose agar stab: Good surface growth, grayish white, no acid formation in 21 days.

Fluorescence: In Erikson's medium a strong yellow green fluorescence observed under ultra violet light.

Pigment production: No definite green pigment observed in 21 days in any of the following medias: Erikson's medium, Erikson's medium having glucose as carbon source and sodium ammonium phosphate as nitrogen either in liquid or agar form, beef liver infusion broth, beef liver infusion broth plus either glucose or sodium fumarate, beef liver infusion agar, beef peptone broth, and beef peptone broth plus 0.5 percent sucrose.

Gentian violet, anilin, and fuchsin were easily absorbed. Gram's method caused a complete discoloring."

Appendix E

With the addition of new information and the decision to list some species as synonymous, the present description given in the 7th edition of Bergey's *Manual of Determinative Bacteriology* (14) is as follows:

"Rods 0.75 to 1.5 to 3.0 microns. Motile with 1 or 2 polar flagella. Gram negative. Green fluorescent pigment produced in culture. Gelatin: Liquified. Beef agar colonies: Circular, grayish white with bluish tinge. Surface smooth. Edges entire or irregular. Broth: Turbid in 36 hours. No pellicle. Milk: Alkaline. Nitrites not produced from nitrates. Indole not produced. Hydrogen sulfide not produced. Not lipolytic. Slight growth in broth plus 4 percent salt. Acid but no gas from glucose, galactose, mannose, arabinose, xylose, sucrose, mannitol and glycerol. Alkaline reaction from salts of citric, malic, succinic and lactic acid. Rhamnose, maltose, lactose, raffinose, salicin, and acetic, formic and tartaric acids not fermented. Starch not hydrolyzed. Aerobic, faculatative."

Other descriptions may be found in Dowson (48), Stapp (132), and Elliott (58).

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