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Diseases of fruit trees caused by pathovars of *Pseudomonas syringae* van Hall are of major concern in fruit-producing areas worldwide, are exceedingly difficult to control, and result in significant economic losses. The pathogen has the ability to kill both young and older trees. Systemic infection and death of young trees is a perennial problem in nurseries, and canker development leading to the girdling and death of scaffold limbs and entire trees is a common event that can lead to the rapid demise of older orchards. For example, bacterial canker of plum caused by *P. syringae* pv. *syringae* causes annual tree mortality rates as high as 30% in Germany (43), and bacterial canker of hazelnut has resulted in the mortality of large numbers of trees in Italy and other European countries (77). Yield losses due to lesions on fruit are typically more sporadic in occurrence and variety dependent, but significant yearly losses have been reported in some instances. Finally, the

pathogen's trait of ice nucleation activity also could exacerbate the importance of frost injury as a predisposing factor for infection. Frost damage in commercial orchards is a sporadic problem that facilitates colonization and the initiation of pathogenesis for these opportunistic pathogens.

P. syringae and its close relatives cause diseases of monocots, herbaceous dicots, and woody dicots, and these pathogens utilize an impressive array of virulence factors such as effectors, toxins, and phytohormones to incite disease symptoms. Aspects of the systematics, ecology, and genetics of *P. syringae* have been reviewed and discussed by many authors (cited literature examples: 6,27,44,47,52). Extensive recent research efforts have focused on *Pseudomonas* diseases of herbaceous plants, and our understanding of *P. syringae*-host interactions has been facilitated by genome sequencing, comparative genomics, and functional studies (cited literature examples: 21,31,96).

Our understanding of and ability to manage *P. syringae* diseases of fruit trees is relevant to our understanding of *P. syringae* diseases of herbaceous hosts, and vice versa. The exciting new insights into host-pathogen interactions from the model systems can give us clues on how to pro-

ceed with the less genetically pliable fruit tree systems. Studies of the fruit tree systems, and comparisons with the model systems, can provide insights into the interactions of *P. syringae* strains with woody hosts and address relevant questions, such as: What is the role of type III secretion in infection of woody tissues? What virulence traits enable the colonization of woody tissue by *P. syringae*? Are there differences in the roles of toxins in diseases of woody hosts and herbaceous hosts? How did some pathogens evolve the ability to induce galls in woody hosts? Can resistance mechanisms characterized in the model-system herbaceous hosts be exploited in fruit trees?

The objectives of this article are (i) to provide an overview of several *Pseudomonas* diseases of fruit trees, (ii) to discuss the current and emerging understanding of virulence factors such as toxins, phytohormones, and effectors in these diseases, and (iii) to discuss current and future management strategies. We will highlight a few specific diseases in which our laboratories have expertise: bacterial canker of stone fruit, caused by *P. syringae* pv. *syringae* and *P. syringae* pv. *morsprunorum*; olive knot, caused by *P. savastanoi*; and apical necrosis of mango, caused by *P. syringae* pv. *syringae*.

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Taxonomic Relationships

Members of six pathovars of *P. syringae* are fruit tree pathogens, as are other related species within the genus *Pseudomonas* (Table 1). The analysis of *P. syringae* strains from fruit trees is somewhat complicated by the fact that a given host can support multiple pathogens (Table 1). The taxonomic relationships of these fruit tree strains have been assessed recently in several genetic studies, leading to the designation of new pathovars, such as *P. syringae* pv. *avii* (63) and *P. syringae* pv. *coryli* (81), as well as new species such as *P. avellanae* (formerly *P. syringae* pv. *avellanae*; 46). All of the *P. syringae* pathovars that are fruit tree strains appear to be relatively narrow host range strains, except for those classified as pv. *syringae*.

The *syringae* pathovar contains members pathogenic to over 180 plant species, including stone fruits, pome fruits, other woody hosts such as willow, crop plants such as bean, and grasses such as wheat (7). An analysis of strains at the pathovar level using polymerase chain reaction (PCR) fingerprinting indicated a high overall level of variation among strains and yet clustering of the majority of strains, regardless of host, within a few groups (80). There is evidence for host specificity among the bean strains and the grass-infecting pv. *syringae* strains (20,37), and a genetic diversity analysis confirmed that bean strains clustered as a separate group compared to other pv. *syringae* strains (51). Likewise, PCR fingerprinting studies done at the population level differentiated populations from stone fruit in California and ornamental pear in Oklahoma from pv. *syringae* strains from other hosts (55,90). Most pv. *syringae* strains share only a few common characteristics, including carriage of genes such as *syrB* involved in the biosynthesis of the toxin syringomycin and pathogenicity to lemon fruit (80). Thus, it is likely that this pathovar represents a collection of strains, some of which have a truly broad host range, but with most representing pathovars awaiting further classification and delineation from the larger group. With the genome sequence of the bean pathogen *P. syringae* pv. *syringae* B728a now available (31), comparative genomic analyses could also be utilized to address this problem.

In addition to studies of strains from individual pathovars or species, two research groups have addressed taxonomic issues of a broad group of phytopathogenic *P. syringae* strains using different genetic techniques. Gardan et al. (34) grouped 56 pathovars of *P. syringae* and relatives into six genomospecies based on total DNA-DNA hybridization, and Stead et al. (87) classified 500 strains using repetitive extragenic palindromic (REP)-PCR fingerprints. In both of these studies, *P. syringae* pathogenic to woody hosts were distributed among different groups and did not

cluster together even when strains were isolated from the same species. Thus, the lack of strain relatedness with host is a common theme in the overall analysis of *P. syringae*. This observation was confirmed in a phylogenetic analysis of the *P. syringae* species that was performed using multilocus sequencing typing (73). In summary, work from a number of researchers indicates that *P. syringae* pathogens of fruit trees are not necessarily distinct from *P. syringae* pathogens of herbaceous dicots and monocots, and this knowledge will be useful along the path to understanding mechanisms of pathogenesis of the fruit tree strains.

Symptoms of Key Diseases

The different *Pseudomonas* pathovars and species from fruit trees exhibit commonalities and differences in life cycles and the symptoms they incite. Like many of their relatives that infect herbaceous hosts, *Pseudomonas* pathogens of fruit trees infect leaves, shoot tips, and fruit. However, fruit trees also provide infection and overwintering sites that are unique to woody perennials. Infection of woody tissue and the formation of cankers is important on fruit trees because cankers eventually girdle and kill branches, thereby resulting in loss of fruiting surface and tree death. In this section, we will describe the symptomatology of three key fruit tree diseases caused by *P. syringae* pathogens.

Bacterial canker of stone fruit. Bacterial canker is caused by *P. syringae* pvs. *syringae* and *morsprunorum*, and is an important disease of sweet cherry (*Prunus avium* L.), tart cherry (*Prunus cerasus* L.), apricot (*Prunus armeniaca*), and other stone fruit. Disease symptoms include blossom blast and spur dieback, leaf and fruit lesions, cankers with associated gummosis of woody tissue, loss of scaffold limbs, and overall decreased fruit yields. The epidemiology of bacterial canker has been a topic of study for many years, and the versatility of the *P. syringae* pv. *syringae* and *P. syringae* pv. *morsprunorum*

pathogens in colonizing trees both epiphytically and endophytically plays a prominent role in limiting effective disease management. While strains of both pathovars can cause bacterial canker symptoms and coexist in cherry orchards, strains of *P. syringae* pv. *syringae* are the more virulent pathogen on sweet cherry and other stone fruit hosts except for tart cherry (28,48). On tart cherry, *P. syringae* pv. *morsprunorum* appears to be the more important pathogen (50).

Early in the growing season, pathogen populations develop on apparently healthy blossoms. During periods of cool, wet weather or after frost events, blossom blast symptoms can occur (Fig. 1A). Blossom blast symptoms on stone fruit are similar to those of blossom blast on pear (60,98) (Fig. 1B and C), except the damage to pear is not accompanied by further wood infection. Frost damage is recognized as an important predisposing injury for bacterial canker infection, and cankers initiated from blossom infections are a common symptom. Frost-associated damage resulting in tip dieback symptoms also occurs in some situations (Fig. 1D).

During the growing season, fruit spot symptoms are common on susceptible varieties and can appear as water-soaked lesions that develop a chocolate-brown color (Fig. 2A). Leaf spot symptoms also occur sporadically; these spots are surrounded by chlorotic rings early in development, and the spots expand and eventually drop out of leaves, causing the shot-hole symptom. On sweet cherry, leaf spot symptoms also typically occur on the margins of leaves, resulting in a curling effect (Fig. 2B).

Canker formation is facilitated by stress events such as exposure to freezing conditions and injury from frost damage, causing a weakening and predisposition of trees to infection. Inoculation experiments have consistently shown that increased incidence of infection and canker length are associated with freeze injury (84,97). Canker formation in tissue that is not in-

Table 1. *Pseudomonas* species and *P. syringae* pathovars that are pathogens of fruit trees

Organism	Disease	Host(s)	Reference ^a
<i>Pseudomonas avellanae</i>	Bacterial canker	European hazelnut	77
<i>Pseudomonas savastanoi</i>			
pv. <i>savastanoi</i>	Olive knot	Olive	101
<i>Pseudomonas syringae</i>			
pv. <i>avii</i>	Bacterial canker	Wild cherry	63
pv. <i>coryli</i>	Twig dieback	European hazelnut	81
pv. <i>morsprunorum</i>	Bacterial canker	Stone fruit	50
pv. <i>papulans</i>	Blister spot	Apple	5
pv. <i>persicae</i>	Bacterial canker	Peach	3
pv. <i>syringae</i>	Apical necrosis	Mango	17
	Bacterial canker	Stone fruit	48, 50
	Blister bark	Apple	59
	Blossom blast	Pear	60, 98

^a Cited references are articles that provide general overviews of the disease and/or descriptions of the pathogen.

jured by freezing is associated with factors such as tissue water-soaking, stem water content, and larger stem diameter (12,95). Cankers formed on scaffold branches and trunks of sweet cherry are typically sunken in appearance and associated with gummosis (Fig. 3A and B). These cankers can become quite large, girdling branches and

trunks leading to death of limbs or the entire tree (Fig. 3B). Cankers are commonly initiated when the pathogen invades woody tissue following infection of blossoms or dormant buds. Wounds are another entry point for *P. syringae* strains, and pruning wounds represent a particularly troublesome entry point for the pathogen

in commercial orchards (Fig. 3C). Dormant buds can sometimes also be killed by the pathogen as a result of direct infection or systemic spread from a nearby canker, resulting in the “dead-bud” symptom (Fig. 3D). Trees that have accumulated large numbers of cankers and dead fruit spurs do not produce many flowers, thereby limit-

Fig. 1. Blossom blast symptoms caused by *Pseudomonas syringae* pv. *syringae* on: A, sweet cherry; and B and C, pear. D, Blossom blast and tip dieback symptoms on plum. B and C courtesy of M. Danilovic.

Fig. 2. Bacterial canker fruit lesions (A) and leaf spot symptoms (B) caused by *Pseudomonas syringae* pv. *syringae* on sweet cherry.

ing the fruiting potential of the trees over time (Fig. 3E). In addition, as with other bacterial fruit tree diseases such as fire blight, young trees are more susceptible to bacterial canker; endophytic infection of these trees is particularly important and contributes to rapid decline and death (Fig. 3F).

Olive knot. Olive (*Olea europaea* L.) knot disease is caused by *P. savastanoi*, an organism formerly regarded as a subspecies of *P. syringae* that has since been reclassified (33). Symptoms on trees infected by *P. savastanoi* include hyperplasia formation (galls or knots of variable size) on the stems and branches of the host plant

and, rarely, on the leaves and fruits (Fig. 4A). The accumulation and expansion of knots can be severe in some cases (Fig. 4B). Sections of young knots disclose pale, apparently healthy tissue surrounding a branching dark core; older knots can expand to form rough-surfaced galls of several centimeters in diameter (Fig. 5).

Fig. 3. Bacterial canker symptoms caused by *Pseudomonas syringae* pv. *syringae* on: A, sweet cherry including a sunken trunk canker; B, canker and associated gummosis spreading from a branch to the main trunk; C, canker formed following infection through a pruning wound (arrow); and D, cankers and associated dead bud symptoms (arrows). E, Effect of extensive tree cankers and systemic infection causing a severe reduction on return bloom and death of some young sweet cherry trees; and F, closer view of a young sweet cherry tree killed following systemic infection.

Fig. 4. A, Hyperplasia outgrowths (knots) on olive stems caused by *Pseudomonas savastanoi*, and B, extensive knot production on an older olive tree.

Fig. 5. Progressive dissection of an olive knot showing external necrosis and necrosis associated with infection of the stem.

Shoots with multiple galls often die, reducing the fruiting potential of the tree. Knots that occur early in tree development can weaken branches, leading to long-term reductions in yield (101). Trees with olive knot can also produce “off”-tasting fruit (76).

Apical necrosis of mango. Bacterial apical necrosis of mango (*Mangifera in-*

dica L.) is caused by *P. syringae* pv. *syringae* (17). This disease is characterized by a rapid expansion of necrotic lesions on buds and leaves. Disease symptoms include necrosis of vegetative and flower buds and bud failure before budbreak, with necrotic lesions on buds sometimes extending through the leaf petiole to the stem (Fig. 6A to C). Fruit lesions have not been ob-

served. Flower panicles are also affected, which can result in severe economic losses due to decreased fruit set (Fig. 6D). Lesions on leaves start as interveinal, angular, water-soaked spots which may coalesce, becoming black and slightly raised (Fig. 6E). Lesions can extend down stems and ultimately kill or severely debilitate trees (Fig. 6F). When disease incidence is high, the vegetative growth of trees is delayed, a factor that ultimately results in trees becoming alternate bearing.

Disease Cycles

Bacterial canker. The disease cycle of bacterial canker is initiated in spring by the colonization and development of large populations on blossoms (Fig. 7). The primary inoculum originates from bacteria located in dormant buds or in cankers. Blossom populations can become quite large, on the order of 10^4 to 10^6 CFU per blossom, and blast symptoms can occur following extended periods of cool, wet weather or after a frost event. Blossom infection leading to wood invasion and canker formation also can occur but typically requires extensive frost injury to predispose the tree to wood infection. Canker formation also can occur following pathogen colonization of pruning wounds. For example, in a study performed in Georgia, inoculation of pruning wounds of peach trees with a virulent *P. syringae* isolate resulted in tree mortality of 57 to 100% when trees were pruned in October or December, but tree mortality was not observed when the pruning and wound inoculation was performed in April (18). In a study performed in California, increased *P. syringae* infection was observed on French prune following pruning in December or March as compared to in November (68). These studies show that the timing of pruning plays an important role in disease severity and that winter pruning is particularly problematic, most likely due to the role of tissue freezing in wood infection by *P. syringae* (84,95,97).

Fig. 6. Symptoms of apical necrosis of mango caused by *Pseudomonas syringae* pv. *syringae* including: A, bud necrosis accompanied by a drop of exudate; B, necrosis extending outward from a bud along the central vein of mango leaves; C, longitudinal view of necrosis extending downward from a bud through stem tissue; D, initial symptoms of necrosis and associated exudate on flower panicles; E, closer view of raised lesions along the central vein on mango leaves; and F, heavy attack affecting nearly all of the branches of an infected mango tree. X indicates that this tree received no treatments in a disease control trial.

Epidemiological studies of bacterial canker indicate that *P. syringae* pvs. *syringae* and *morsprunorum* have prominent epiphytic and endophytic phases. In fact, the concept of a plant pathogen surviving as an epiphyte on healthy tissues was first proposed in studies of *P. syringae* pv. *morsprunorum* on sweet cherry (26,44). In his studies, Crosse consistently recovered resident populations of *P. syringae* pv.

morsprunorum from leaf washings of healthy tissue and concluded that these populations were the source of inoculum for new cankers (26). The epiphytic phase of *P. syringae* pvs. *syringae* and *morsprunorum* includes growth and survival on apparently healthy blossoms and on leaves throughout the entire growing season (92). During the summer months, the bacterial canker pathogens will cause fruit

and leaf spot lesions and/or survive epiphytically on apparently healthy leaf surfaces. Epiphytic populations typically dwindle to undetectable levels during the summer months, but these populations rebound in autumn as temperatures decrease and rainfall increases (25,27,92) (Fig. 7).

Dormant buds are an overwintering site for the bacterial canker pathogens. In

cherry trees, *P. syringae* pv. *morsprunorum* has been shown to overwinter in buds in the United Kingdom (24), and both *P. syringae* pvs. *syringae* and *morsprunorum* were detected in dormant buds in South Africa and in Michigan (71,92). Apple buds are likewise an overwintering site for *P. syringae* pv. *syringae* and the blister spot pathogen *P. syringae* pv. *papulans*, with larger bacterial populations recovered from the innermost tissues (5,9). Dormant buds of cherry are colonized via infection of leaf scars during autumn months at leaf drop. Environmental conditions at leaf drop typically include cooler temperatures, wind, and driving rain. At this time, *P. syringae* pvs. *syringae* and *morsprunorum* are transported from leaf surfaces to leaf scars, where systemic colonization occurs (24,92). Bacteria colonizing leaf scars move systemically and colonize and overwinter in dormant buds (92). In most

cases, the buds remain apparently healthy, but buds can sometimes be killed by the pathogen, resulting in the “dead-bud” symptom (Fig. 3D) and canker initiation. Bacterial populations in apparently healthy dormant buds provide the inoculum for blossom colonization the following spring. It should be noted that the leaf scar is not universally utilized as an avenue of infection of cherry, particularly in warmer climates. For example, in South Africa, the *P. syringae* pathogens are thought to migrate systemically within trees, reaching buds before leaf fall (41). Because *P. syringae* is not heat tolerant, it seems likely that epiphytic populations of the pathogen would not survive as well as endophytic populations, thus favoring organisms that colonize systemically.

Olive knot and apical necrosis of mango. As with other fruit tree diseases caused by Pseudomonads, the olive knot

pathogen is most active in spring and autumn. The formation of knots is most prevalent during the period of active tree growth in spring; the pathogen also survives as an epiphyte on olive leaf surfaces (30,101). In addition, several authors have suggested that *P. savastanoi* is able to migrate within the plant tissues and produce secondary knots in new wounds in olive plants (69).

Symptom development in mango buds inoculated with isolates of *P. syringae* pv. *syringae* is strongly influenced by environmental conditions, with a higher proportion of symptomatic buds observed following cool rainy weather. Apical necrosis in mango trees is frequently observed from late October to March and reaches its maximum development during January and February (17), which is the coolest, wettest season in the Mediterranean area. Incidence of bacterial apical

Fig. 7. Disease cycle of the bacterial canker pathogen *Pseudomonas syringae* pv. *syringae*.

necrosis in the field was highest when temperatures reached values below 0°C for a few nights. As happens with other tropical trees, mango trees become dormant under 15°C, and with temperatures below 0°C, tissue damage becomes important, a fact that could be increased by the bacterial ice-nucleation activity of the pathogen *P. syringae* pv. *syringae* (16). However, other factors such as the establishment of large epiphytic population sizes of *P. syringae* pv. *syringae* are also involved in disease initiation (17). Thus, the disease cycle of apical necrosis of mango seems very similar to that of bacterial canker of stone fruits, with the main exception being the absence of fruit spots.

Virulence Factors

Ice nucleation activity. The role of freezing temperatures in the development of *P. syringae* diseases of fruit trees has been documented many times in controlled studies. Freezing temperatures increase the severity of *P. syringae* pv. *syringae* infection of leaves of sour cherry and branches of apricot and peach (49,88,97). The exact temperature required for canker formation varies. For example, necrosis of sweet cherry was more severe and pathogen populations were higher when shoots inoculated with *P. syringae* pv. *syringae* or *P. syringae* pv. *morsprunorum* were exposed to -10°C, but not -5°C (84). In addition to the controlled studies, cold temperatures in the field have often been correlated with increased disease severity of bacterial canker and apical necrosis (17,86).

The phenotype of ice nucleation activity (INA) is shared by many pathovars of *P. syringae* and results in the ability of bacterial cells to act as nuclei capable of catalyzing ice formation at temperatures only slightly below freezing (54). INA *P. syringae* growing epiphytically on frost-sensitive leaves are implicated in causing frost damage of herbaceous crops such as potatoes and strawberries. However, the *P. syringae* strains involved do not cause disease symptoms after the frost event. Thus, ice nucleation has not traditionally been viewed by plant bacteriologists as a virulence factor in *P. syringae* pathosystems. In fruit tree systems, however, the role of frost damage as a predisposing factor of infection (65), particularly in the epidemiology of bacterial canker, suggests that the INA phenotype is a virulence factor. However, this has been a controversial topic, as it is known that stone fruit trees also produce an indigenous ice nucleating factor (38), and the results of one field study indicated that there were no significant differences in frost injury to blossoms of various fruit trees that varied in carriage of INA bacteria following exposure to temperatures of -2.8°C (70).

It should be noted that the role of INA in virulence of *P. syringae* pv. *syringae* is linked not with frost injury to blossoms but

with subsequent invasion and tissue infection. Frost events causing severe damage are typically accompanied by systemic infection and canker formation in susceptible varieties (Fig. 8A and B). For example, in 2002 in Michigan, a particularly damaging hard radiational frost event occurred during bloom. This freeze event resulted in extensive injury to sweet cherry

blossoms and was followed by widespread wood invasion, canker formation, and death of trees. Thus, a critical factor in the linkage between INA and infection appears to be the intimate association of the INA bacteria and ice formation with sites of injured host tissue that is predisposed to infection. The observation that pathogen cells must be present within 20 minutes of

Fig. 8. Canker formation and enlarging cankers on sweet cherry caused by *Pseudomonas syringae* pv. *syringae* as a result of blossom blast and internal infection the previous spring. A, Note canker formation at each fruiting spur. B, Close-up view of a canker formed after infection showing the remains of tissue killed by blossom blast, gummosis, canker formation, and wood invasion.

thawing of frost-injured tissue for successful infection to occur (88) provides correlative evidence for the importance of the co-occurrence of INA bacteria, ice, and injured tissue in infection. Ultimately, the question of the involvement of INA in pathogen virulence will have to be answered using a defined mutant in inoculation studies on many hosts exposed to a number of different freezing temperatures.

Effectors and the type III secretion system. The type III secretion system (T3SS) of *P. syringae* has been well characterized, and work is underway to determine the function of effectors in host-pathogen interactions. The T3SS functions as a delivery system in an intimate interaction between the pathogen and host plant cell whereby the pathogen introduces effector proteins directly into plant cells (47). A varying number of effectors are secreted depending on the *P. syringae* pathosystem, with the effectors functioning either to suppress a host defense response or to effect disease through currently unknown mechanisms (21). Genetic analyses have shown that a functioning T3SS is required for *P. syringae* strains to cause disease; however, individual effectors typically have only a small effect on virulence. The repertoire of effectors is thought to influence host range or host specificity of individual *P. syringae* pathovar strains (32).

Analyses of the T3SS and effector complement of *P. syringae* fruit tree pathogens have been initiated. Genes associated with the *hrp* pathogenicity island, including genes encoding structural components of the T3SS apparatus and effectors such as *hrpZ* and *hrpW*, have been identified in *P. avellanae* and *P. syringae* pv. *morsprunorum* (53,56). A significant advance has been the recent demonstration that knot formation caused by *P. savastanoi* pv. *savastanoi* on olive plants is dependent on a functional T3SS, as a mutant containing a disruption of the *hrcC* gene did not cause disease symptoms on olive plants although it was still able to produce phytohormones (82). Distinct combinations of effector genes found in the exchangeable effector locus, which is a sequence region adjacent to the *hrp* pathogenicity island, have been detected in fruit tree strains of *P. syringae* pv. *syringae* (19), and plasmid-encoded effector genes have been detected by PCR or hybridization in strains of *P. avellanae* and oleander strains of *P. savastanoi* (56,102).

Genetic studies of host-pathogen interactions with *Pseudomonas* strains and woody host plants will always be slowed by the difficulties of working with the woody plant systems. It is hoped that critical knowledge of the effector complement necessary for woody tissue invasion, canker formation, and systemic movement can be gained through comparative genomic analyses and subsequent genetic studies.

Phytotoxins. *Pseudomonas* pathogens of fruit trees produce both pore-forming toxins (such as syringomycin) that target the host plasma membrane, and antimetabolites (such as tabtoxin and phaseolotoxin) that inhibit biosynthesis of amino acids. The presence of these toxins and others has been documented in pathogens of fruit trees, but the function of these toxins in virulence often remains unclear. Further study in this area should involve the use of genetically defined mutants and evaluations of the role of the toxins in infection of woody tissue.

Syringomycin, produced by *P. syringae* pv. *syringae*, is in the cyclic lipodepsinonapeptide phytotoxin class (6). This toxin induces necrosis in host tissues, and expression of genes involved in syringomycin biosynthesis is induced by plant signal molecules, including compounds present in sweet cherry leaves (64). Syringomycin production affected virulence in a laboratory sweet cherry fruit infection assay, but the role of this toxin in canker formation in woody tissues is unknown (64). Coronatine is a phytotoxin produced by *P. syringae* pv. *morsprunorum*, the weaker of the two *P. syringae* pathovars that cause bacterial canker (53). The role of coronatine as a virulence factor has not been evaluated in the *P. syringae* pv. *morsprunorum*-cherry pathosystem. Persicomyacin, a group of 3-(3'-hydroxy)hydroxy fatty acids, was identified as a necrosis-inducing substance and virulence factor from *P. syringae* pv. *persicae* strains causing disease on peach (3).

A new antimetabolite toxin, mangotoxin, produced by *P. syringae* pv. *syringae* strains mainly isolated from mango has recently been reported (2,13). Mangotoxin is a small oligopeptide that targets the enzyme ornithine *N*-acetyltransferase, a key enzyme in the biosynthetic pathways of arginine and ornithine. A clear relationship between mangotoxin production and the virulence of the *P. syringae* pv. *syringae* UMAF0158 has been demonstrated. Tomato leaflets were inoculated with the wild type, some mangotoxin-defective mutants, and the complemented strains (1,13). Symptoms caused by the mangotoxin-defective mutants were clearly less severe than those produced by the mangotoxin-producing strains. The mutants also displayed a delay in the initiation of the necrotic symptoms. Furthermore, ED₅₀ values were clearly higher for the mutants defective in mangotoxin production, in comparison with the wild type and with the complemented strains with mangotoxin production restored (1).

Phytohormones. The knot-forming *P. savastanoi* pathogens differ from almost all other bacterial plant pathogens in the production of substantial quantities of the phytohormones indole-acetic acid (IAA) and cytokinins (CK). Gall development in plants infected with *P. savastanoi* is de-

pendent on bacterial production of IAA, which is delivered to the plant tissues. However, IAA production is not necessary for *P. savastanoi* cells to grow within the host tissue (83). In *P. savastanoi*, IAA is synthesized from tryptophan in two steps catalyzed by the products of the *iaaM* (tryptophan monooxygenase) and *iaaH* (indoleacetamide hydrolase) genes. *P. savastanoi* strains also convert IAA to IAA-Lysine through the action of the *iaaL* gene (IAA-Lysine synthetase). Conjugates are regarded as storage forms of IAA in plants, which contain the necessary enzyme to hydrolyze the conjugate and release free IAA. However, inactivation of the *iaaL* gene in a *P. savastanoi* pv. *nerii* strain by Tn5 mutagenesis resulted in reduced virulence of the mutant on oleander plants, and replication within the host tissue was different between the mutant strain and the wild type (35).

Cytokinin production is usually regarded as contributing to virulence in *P. savastanoi* and all other gall-forming bacteria. Virulent strains of *P. savastanoi* and *E. herbicola* produce substantially greater quantities of cytokinin in vitro than do avirulent strains of those bacteria. In contrast, virulent and avirulent strains of *A. tumefaciens* and *R. fascians* produce similar quantities of these hormones in vitro (45). An oleander strain of *P. savastanoi* has been reported to secrete zeatin and ribosylzeatin (57). A higher level of zeatin secretion was shown in the olive isolate EW1006, which also produces dihydrozeatin, ribosyl dihydrozeatin, and ribosyl-1"-methylzeatin (57).

Disease Control

Management of most fruit tree diseases caused by *Pseudomonas* spp. currently is almost unattainable, due to the lack of effective chemical or biological control measures, lack (and little available knowledge) of host resistance, and the endophytic nature of the pathogen during some phases of the disease cycle. Copper compounds are the standard bactericides for controlling many bacterial diseases, but there are several limitations to their use, including lack of systemic activity, difficulty in timing, emergence of resistant strains, and phytotoxicity. Copper is a contact material and does not penetrate to control bacterial populations that are internal, such as inside dormant buds, knots, or cankers. Thus, applications of copper must be timed to coincide with periods when the host is susceptible, when the pathogen is accessible, and when conditions are favorable to disease. Such timings may not be possible; for example, copper is highly phytotoxic to sweet cherry trees and, during bloom, can only be used at very low levels that are essentially ineffective in lowering pathogen populations on blossoms.

An understanding of the windows of susceptibility for the host at different

phenological stages can help optimize bactericide application timing. Autumn copper sprays targeting epiphytic populations and protecting leaf scars from infection have been effective in reducing bacterial canker inoculum in some situations (67,99). Combination treatments including copper sprays and biannual fertilization controlled bacterial canker on French prune better than copper sprays alone (74). In California, although olive trees shed leaves all year long, most leaves drop in spring, and applications of cupric hydroxide at this time significantly reduced leaf scar infection (93). In blister spot of 'Mutsu' apple, caused by *P. syringae* pv. *papulans*, fruit are highly susceptible for only about 4 to 5 weeks starting from about 2 weeks postbloom (8). This short period of susceptibility in 'Mutsu' apple has effectively reduced the number of bactericide applications utilized for disease control.

Copper resistance is a widespread phenomenon in *P. syringae* pathogens of many hosts in which copper is utilized for disease control (22,61,75,89), and has been detected in *P. syringae* pv. *syringae* strains from cherry in Michigan (91) and mango in Spain (15). The lack of alternative control measures to copper exacerbates the problems associated with bactericide resistance. Antibiotics such as streptomycin and oxytetracycline, which are registered in some countries for use on other bacterial diseases of fruit trees such as fire blight and bacterial spot of peach, are typically not registered for control of bacterial canker or other *Pseudomonas* spp. diseases. Even if these materials were available, it is likely their usefulness would quickly be compromised by resistance, as the occurrence of antibiotic resistance in *P. syringae* also is common (62).

What other options are available for controlling *Pseudomonas* diseases of fruit trees? Acibenzolar-S-methyl (ASM, trade name Actigard in the United States and Bion in Europe) is a compound that enhances the natural defenses of plants by inducing systemic acquired resistance (SAR). Plants must be treated with ASM prior to infection so that resistance is induced prior to pathogen attack. Monthly applications of ASM moderately reduced bacterial decline of hazelnut caused by *P. syringae* pv. *syringae* in Italy (79). Effective reduction of the percentage of necrotic buds of mango and of leaves with necrotic lesions was achieved with six to seven monthly applications of ASM; control with ASM was similar to that achieved with the same number of applications of Bordeaux mixture (14).

The growth regulator prohexadione-calcium (trade name Apogee in the United States and Regalis in Europe, BASF) has been shown to inhibit fire blight of apple, and this inhibition is associated with a reduction of succulent shoot growth,

thereby limiting the availability of highly disease-susceptible shoots (23,66,100). Several reports suggest that prohexadione-calcium stimulates host resistance by triggering production of the antimicrobial compound luteoflorol (85). If these observations are correct, and prohexadione-calcium triggers a general resistance to multiple pathogens, then it could prove useful in management of *P. syringae* diseases of fruit trees. While prohexadione-calcium did not reduce shoot growth in peaches (10), it did induce peach trees to produce luteoflavan, a compound associated with luteoflorol (36). Luteoflavan, however, was not detected in plum or cherry trees treated with prohexadione-calcium. While Bazzi et al. (4) did not investigate a *P. syringae* disease of fruit trees, they studied the effects of prohexadione-calcium on development of leaf spots on plum caused by another bacterial pathogen, *Xanthomonas arboricola* pv. *pruni*. A high dose of prohexadione-calcium reduced shoot length, but had no effect on incidence or severity of bacterial spot. Thus, the potential of prohexadione-calcium in *P. syringae* diseases of fruit trees remains unclear.

Breeding for fruit tree resistance to *P. syringae* diseases presents the same challenges as breeding for fruit tree resistance in general. Challenges include the length of time involved, difficulties in screening for resistance, the acceptance by growers of new varieties, and the establishment of new orchards. In addition, sources of resistance are not readily available, and studies concerning naturally occurring resistance have been sporadic. Recently, several research groups have screened wild cherry clones for resistance to *P. syringae* pvs. *morsprunorum*, *syringae*, *persicae*, and *avii* in greenhouse- and field-grown plants (72,78,94). Unfortunately, the best clones displayed only moderate resistance. Hassani et al. (40) screened 25 cultivars of olive, including several established cultivars, crosses among them, and open-pollinated progeny, for olive knot sensitivity. All cultivars developed olive knot, although the knots were smaller on some genotypes. Penyalver et al. (69) evaluated the proportion and weight of primary knots and the presence of secondary knots in 29 Spanish olive cultivars inoculated with two different *P. savastanoi* strains. Using this method, most of the tested cultivars were placed into three categories of susceptibility. However, none of them were found to be highly resistant to the disease (69). Somaclonal variation has also been tested as a method to increase resistance in peach to *P. syringae* pv. *syringae* (39). At least one resistant somaclone was recovered, although reversion to susceptibility was proposed to be a problem with this method (39).

The use of biotechnology and the production of transgenic trees with resistance

to *Pseudomonas* diseases represents another possible future control method. Trees with transgenic resistance are an attractive option from a disease control perspective because the resistance would likely be effective in controlling endophytic populations and limiting cankers, which are the main symptoms resulting in tree death. Genetic engineering of stone fruit trees is possible, and transgenic plum trees with resistance to *Plum pox virus* have been produced and tested in the field (42). The success of a similar project in the production of transgenic resistance against *Pseudomonas* diseases would require a detailed understanding of the interaction between the *Pseudomonas* pathogen and its tree host, such that appropriate genes conferring resistance can be identified and transferred into susceptible varieties. This approach would greatly benefit from work done with model systems and comparative genomics approaches in the identification and functional understanding of effectors and other virulence factors in the *Pseudomonas* pathogens of fruit trees.

The use of transgenic approaches for the development of apple and pear cultivars with resistance to fire blight has been ongoing in several laboratories. These studies initially focused on inserting genes from other species encoding toxins, but have more recently focused on using genes from the target host that are specifically involved in disease resistance or involved in a generalized resistance response. One approach that might be directly applicable to fruit tree-*Pseudomonas* systems is the overexpression of the general disease resistance gene *MpNPR1* in apple leading to increased fire blight resistance (58). The *NPR1* gene plays a critical role in defense against pathogens in *Arabidopsis thaliana* and other host plants (29); elevated expression of *NPR1* is associated with increased resistance to bacterial and/or fungal pathogens (11). These studies suggest that it is possible that overexpression of the *Prunus avium NPR1* gene, for example, may increase resistance to bacterial canker.

Conclusions

The species *P. syringae*, and related species *P. avellanae*, *P. avii*, and *P. savastanoi*, collectively cause important diseases on numerous plant hosts including fruit trees. The significance of damage to fruit trees includes effects on crop yield due to blossom blast, fruit lesions, killing of scaffold branches, and cankers that can ultimately kill trees. Several aspects of fruit tree diseases are distinguished from *P. syringae* diseases of herbaceous plants, including wood infection, overwintering sites within the perennial hosts, and systemic movement through hosts. Phylogenetic analyses indicate that *P. syringae* pathogens of fruit trees typically cluster with *P. syringae* pathogens of herbaceous hosts. This suggests that many strategies of

pathogenesis may overlap among these pathogen groups. Genetic studies of host-pathogen interactions with *Pseudomonas* strains and woody host plants will always be slowed by the difficulties of working with these plant systems. However, we hope that critical knowledge of the effector complement necessary for woody tissue invasion, canker formation, and systemic movement will be gained through comparative genomic analyses and subsequent genetic studies. Such studies leading to improved host resistance may ultimately represent the best means for control of diseases such as bacterial canker that cannot be adequately controlled through conventional chemical methods.

The successful development of transgenic fruit trees with resistance to *Pseudomonas* diseases will require a detailed knowledge of the interaction of these pathogens with their woody hosts. Such studies will be especially enlightening to the overall knowledge of *Pseudomonas* pathogenesis when framed against the knowledge gained from work done with model systems.

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