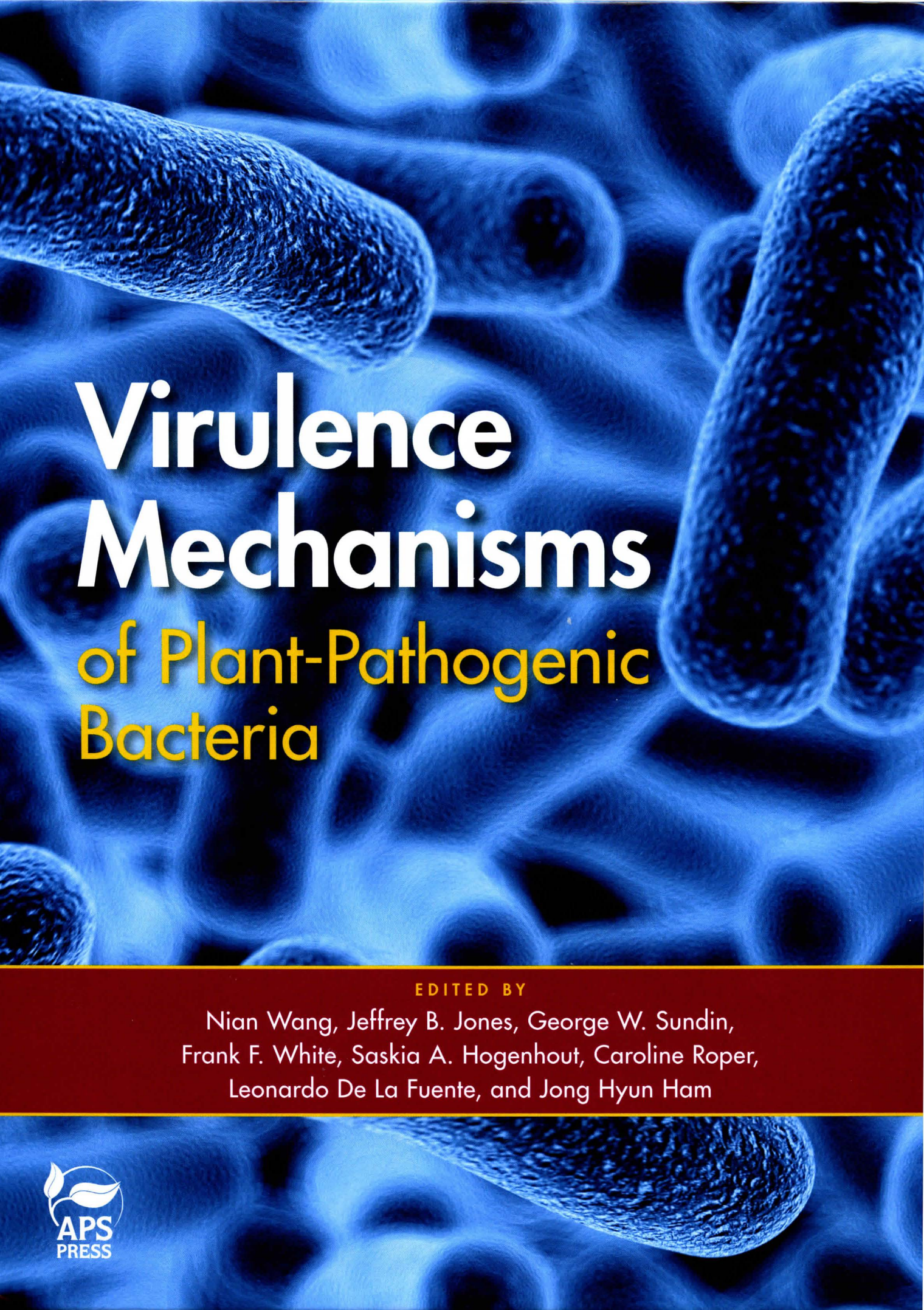

# Virulence Mechanisms of Plant-Pathogenic Bacteria

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CHAPTER 21

# The Dynamic World of the Genus *Xanthomonas*

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The genus *Xanthomonas* represents a diverse group of mostly plant-associated bacterial species in the gamma proteobacteria (Parkinson et al., 2009). The bacteria are the causal agents of an assortment of persistent and periodic plant diseases, some of which have high agronomic and economic importance—particularly in regions of subsistence agriculture and those with poor crop management resources (Hayward, 1993; Leyns et al., 1984). *Xanthomonas* spp. infect hosts from the major groups of monocotyledonous and dicotyledonous plants and

cause diseases that include vascular wilts, cankers, leaf and fruit spots, and leaf blights (Table 21.1).

Yet despite the wide host range of the xanthomonads, individual strains are generally restricted to particular host plants. At the same time, some diseases incited by the xanthomonads are caused by several species. Diseases on citrus and solanaceous species, for example, can involve a variety of *Xanthomonas* spp. (Moreira et al., 2010; Potnis et al., 2011). Persistent diseases such as bacterial blight of rice, bacterial canker of citrus, leaf scald of sugarcane, leaf blight of onion, and bacterial spot of pepper remain important targets for improved management strategies. Other diseases involving *Xanthomonas* spp. are becoming increasingly severe, including bacterial spot of pumpkin, bacterial blight of hazelnut, bacterial blight of wheat, and bacterial wilt of banana (Adhikari et al., 2012; Badadoost and Ravanlou, 2012; Biruma et al., 2007; Lamichhane et al., 2012). Studies of various diseases and strains of *Xanthomonas* have revealed new information about and insights into bacterial diseases, in general, and disease complexes, in particular. This chapter presents research about *Xanthomonas* spp. and reviews of several specific disease complexes.

**TABLE 21.1.** Representative diseases caused by *Xanthomonas* spp.

| Pathogen(s) | Disease |
|---|---|
| *X. vasicola* pv. *musacearum* | Banana wilt |
| *X. citri* pv. *citri*, *X. fuscans* subsp. *aurantifolii* | Citrus canker |
| *X. axonopodis* pv. *dieffenbachiae* | Bacterial blight of anthurium |
| *X. euvesicatoria*, *X. perforans*, *X. gardneri* | Bacterial spot of pepper and tomato |
| *X. oryzae* pv. *oryzae* | Bacterial blight of rice |
| *X. oryzae* pv. *oryzicola* | Leaf streak of rice |
| *X. albilineans* | Leaf scald of sugarcane |
| *X. arboricola* pv. *corylina* | Bacterial blight of hazelnut |
| *X. cucurbitae* | Bacterial spot of pumpkin |
| *X. axonopodis* pv. *allii* | Leaf blight of onion |

## • *Xanthomonas albilineans*

*Xanthomonas albilineans* (Ashby) Dowson is known to invade the xylem of its host and to cause leaf scald of sugarcane (Birch, 2001; Rott and Davis, 2000). *X. albilineans* is transmitted mainly by mechanical means and is not known to be insect transmitted. This pathogen causes symptoms that vary from pencil-line symptoms to death of the plant, including chlorosis and necrosis of leaves. The disease occurs in more than 60 sugarcane-growing locations worldwide, and screening sugarcane for resistance to leaf scald is essential for disease control.

The first virulence factor described in *X. albilineans* is a small, secreted molecule called "albicidin," which exhibits phytotoxic and antibiotic properties (Birch, 2001). Albicidin is a potent DNA gyrase inhibitor with a novel mode of action (Hashimi et al., 2007). It targets chloroplastic DNA gyrase A, inhibits chloroplast DNA replication, and blocks chloroplast differentiation, resulting in white, foliar-stripe symptoms (Birch and Patil, 1987a, 1987b).

Albicidin also targets bacterial DNA gyrase A and, as a consequence, exhibits a potent antibiotic activity against a wide range of gram-positive and gram-negative bacteria (Birch and Patil, 1985a). This antibiotic activity may help *X. albilineans* to combat rival microorganisms during sugarcane invasion. In the mid-1980s, researchers observed that mutants of strains of the pathogen from Australia that were deficient in toxin production were no longer able to produce disease symptoms, but they were still able to invade the sugarcane xylem (Birch and Patil, 1987a). A 1999 study reported that sugarcane plants transformed with a gene producing an esterase that detoxifies albicidin became resistant to leaf scald disease (Zhang et al., 1999). However, a 2011 study demonstrated that albicidin-deficient mutant isolates of a strain of *X. albilineans* from the U.S. state of Florida were still able to produce severe disease symptoms, suggesting that engineered detoxification of albicidin may not confer resistance to all strains of the pathogen or under all environmental conditions (Rott et al., 2011).

Three genomic regions involved in albicidin biosynthesis—XALB1, XALB2, and XALB3—have been cloned and sequenced (Rott et al., 1996; Royer et al., 2004; Vivien et al., 2005). XALB1 contains 20 open reading frames (ORFs), including three large nonribosomal peptide synthetase (NRPS) genes, as well as several putative resistance, regulatory, and tailoring genes. NRPSs belong to the family of megasynthetases, which are among the largest known enzymes, having

molecular weights up to approximately 2.3 megadaltons (MDa) (i.e., approximately 21,000 residues). NRPSs are multimodular enzymes that catalyze the nonribosomal assembly of peptides from proteinogenic and nonproteinogenic amino acids (Finking and Marahiel, 2004). XALB2 encodes a phosphopantetheinyl transferase required for posttranslational activation of NRPSs, and XALB3 encodes the heat-shock protein HtpG (Vivien et al., 2005).

Preliminary analyses by nuclear magnetic resonance (NMR) spectroscopy and mass spectrometry (MS) did not allow determination of the structure of albicidin (Birch and Patil, 1985a, 1985b). Nonetheless, these early studies showed that albicidin contains approximately 38 carbon atoms and has an estimated molecular mass of 842 daltons (Da). In 2004, in silico analyses of XALB1 provided further insights into the structure of this pathotoxin and suggested that NRPSs involved in the biosynthesis of albicidin incorporate nonproteinogenic substrates that are as yet unknown (Royer et al., 2004). Thus, the data that have accumulated about albicidin and its biosynthesis gene cluster suggest that it is a potent DNA gyrase inhibitor with a novel mode of action and therefore might constitute a lead for the development of new antibiotics.

Sequencing of the genome of *X. albilineans* revealed that this pathogen experienced genome reduction during its speciation (Pieretti et al., 2009). Additionally, this xanthomonad is notably missing the hypersensitive response (HR) and pathogenicity (Hrp) type III secretion system (T3SS) (see Chapters 7 and 9 ) and the xanthan gene cluster (see Chapter 4), which are commonly found in pathogenic *Xanthomonas* spp. However, *X. albilineans* possesses other secretion systems that may deliver virulence determinants into the plant cell or to the bacterial environment, such as type I, II, and V secretion systems (Pieretti et al., 2012). Surprisingly, this pathogen does possess a T3SS of the SPI-1 injectisome family, which is usually found in animal-associated pathogens. The role of this SPI-1 T3SS is unknown, however, and mutants in this secretion system are still able to spread efficiently within the sugarcane stalk and to cause leaf symptoms (Marguerettaz et al., 2011). An in-depth analysis of the genome of *X. albilineans* strain GPE PC73 from Guadeloupe (France) produced a description of all of the genomic features specific to this bacterium (Pieretti et al., 2012).

During 2009–2010, researchers produced a Tn*5* mutant library of strain XaFL07-1 from Florida and inoculated 1,200 mutants into sugarcane plants; 61 mutants were affected in symptom production and/or colonization of the sugarcane stalk (Rott et al., 2011). Characterization of the mutated regions in these pathogenicity-defective mutants resulted in the identification of 33 pathogenicity-related loci, which are purported to encode proteins involved in a variety of functions: namely, exopolysaccharide (EPS) and lipopolysaccharide (LPS) biosynthesis (six loci), fatty acid biosynthesis (two loci), amino acid biosynthesis (one locus), purine biosynthesis (one locus), carbohydrate metabolism (one locus), nitrogen metabolism (one locus), regulatory and cell signaling (two loci), secretion systems (one locus), transport (five loci), catalytic activity (one locus), DNA binding (two loci), and drug resistance (one locus), as well as several unknown functions (nine loci). One of the proteins with an unknown function encodes an outer-membrane protein (XaOmpA1) that appears to be essential for several cellular processes, such as motility and resistance to sodium dodecyl sulfate (Fleites et al., 2012; Rott et al., 2011). Researchers found that although the XaOmpA1 mutants were no longer able to produce disease symptoms, they were still able to produce the toxin albicidin, similar to the wild-type parental strain.

Several processes that contribute to virulence are regulated in plant-pathogenic bacteria by quorum sensing (QS), a form of cell–cell communication that is mediated by diffusible signal molecules (Ryan and Dow, 2011). *X. albilineans* (like other *Xanthomonas* spp.) and *Xylella fastidiosa* Wells et al. each have an *rpf* (regulation of pathogenicity factors) cluster of genes that is involved in QS and that contributes to disease expression (Rott et al., 2013). This system—and more specifically, the transduction system of the diffusible signal factor (DSF)—negatively regulates the spread of *X. albilineans* in the xylem of sugarcane stalks. Mutants of the DSF sensor (RpfC) and/or regulator (RpfG) of *X. albilineans* strain XaFL07-1 were found able to colonize sugarcane stalks more efficiently than the wild-type strain of the pathogen. Furthermore, mutants affected in both *rpfG* and *rpfC* were also severely impaired in swimming motility, suggesting that this type of motility is not involved in the spread of the pathogen in the xylem of the host plant (Rott et al., 2013).

*X. albilineans* has traditionally been considered to be limited to the xylem of sugarcane, but several studies published in 2011 and 2014 have shown that this pathogen is able to invade tissues other than the xylem of sugarcane leaves and stalks (Legaz et al., 2011; Mensi et al., 2014). The mechanisms used by the pathogen to invade the vascular system of sugarcane and other tissues are unknown, however.

The reduced genome and the absence of a T3SS of the Hrp1 and Hrp2 injectisome families suggest that the virulence of *X. albilineans* relies both on unique features from yet-to-be identified virulence effectors and on features that allow the bacterium to spread in a nutrient-poor environment, to evade sugarcane surveillance systems, and to resist sugarcane defense systems (Pieretti et al., 2012). The reductive genome evolution may have favored an adaptation of *X. albilineans* to sugarcane xylem vessels by allowing the loss of genes that encode pathogen-associated molecular patterns (PAMPs) recognized by sugarcane surveillance systems. Interestingly, *X. albilineans* is also missing the *gum* genes that are involved in biosynthesis of xanthan gum—an important virulence factor in *Xanthomonas* spp. *X. albilineans* produces surface cell polysaccharides, including LPSs, which appear to play a role in pathogenicity. Researchers found that mutants of the pathogen that do not produce these molecules are affected in their capacity to produce leaf symptoms and/or to invade the sugarcane stalk, but their precise function in the virulence of *X. albilineans* remain unknown (Rott et al., 2011).

Although *X. albilineans* has a reduced genome and a limited "arsenal" of virulence factors, it can be a devastating and destructive plant pathogen when compared to other pathogenic *Xanthomonas* spp. Moreover, large genetic diversity and variation in pathogenicity have been reported in this causal agent of leaf scald disease of sugarcane (Champoiseau et al., 2006; Davis et al., 1997). Further comparative and functional genomics studies are needed to unravel the pathogenicity of this unusual *Xanthomonas* spp.

## • *Xanthomonas citri* pv. *citri*

Several species of *Xanthomonas* are pathogenic to citrus. Before reclassification of the genus in 1995 and the extensive research on *Xanthomonas* taxonomy that followed, most strains were classified as *X. campestris* pv. *citri* (ex Hasse) Dye and further grouped according to pathotypes (A, B, C, D, and E) (Rademaker et al., 2000; Rodriguez et al., 2012; Vauterin and Swings, 1997; Vauterin et al., 1995; Young et al., 2008). Three of these pathotypes, as originally defined (A, B, and C), cause canker symptoms on citrus. The pathovar and subspecies classifications coexist for plant-pathogenic bacteria, possibly causing confusion in the nomenclature.

*X. citri* pv. *citri* (Gabriel et al.) Schaad et al. (syns. *X. citri* subsp. *citri* (Gabriel et al.) Schaad et al. and *X. axonopodis* pv. *citri* (Hasse) Vauterin et al.), which corresponds to pathotype A and its variants (see following), is the causal agent of Asiatic citrus canker (Ah-You et al., 2009; Schaad et al., 2006; Vauterin et al., 1995). This disease threatens most commercial citrus cultivars throughout the world and sporadically infects other members of the Rutaceae family (Graham et al., 2004). Strains of this pathovar are classified as genetic cluster 9.5 (Rademaker et al., 2000).

*X. fuscans* subsp. *aurantifolii* Schaad et al. (syns. *X. citri* pv. *aurantifolii* and *X. axonopodis* pv. *aurantifolii* Vauterin et al.), which corresponds to pathotypes B and C, causes South American canker (Ah-You et al., 2009; Schaad et al., 2006; Vauterin et al., 1995). Strains of this pathovar are categorized as genetic cluster 9.6 (Rademaker et al., 2000).

In addition to these canker-causing bacteria, two pathogens that cause spot diseases of citrus (i.e., water-soaked spots that turn into flat, necrotic lesions) have been described as two distinct *Xanthomonas* pathovars. Namely, *X. axonopodis* pv. *citrumelo* Vauterin et al. (syn. *X. alfalfae* subsp. *citrumelonis* Schaad et al.), which corresponds to pathotype E, is the causal agent of citrus bacterial spot (Schaad et al., 2006; Vauterin et al., 1995), and *X. citri* pv. *bilvae* has been reported to cause spots on rutaceous species in India (Patel et al., 1953). These strains are classified as genetic clusters 9.2 and 9.5, respectively (Rademaker et al., 2000).

All of these diseases except Asiatic citrus canker have low economic significance and are geographically restricted. However, the bacteria that cause them are listed as quarantined organisms in many countries (i.e., the European Union, Australia, countries in northern Africa). Like plants in the genus *Citrus,* the bacterium *X. citri* pv. *citri* likely originated in Asia and spread to most of the other continents. Consistent with this hypothesis, the oldest herbarium specimens with canker lesions were reported from India in the 1830s and from Java in the 1840s (Fawcett and Jenkins, 1933). The first report of a canker outbreak came from Japan in 1899 (Civerolo, 1984). As of 2014, Asiatic canker was reported from more than 30 countries worldwide, including major citrus production areas. The disease had re-emerged in Africa (Balestra et al., 2008; Derso et al., 2009; Leduc et al., 2011; Traoré et al., 2008), and its presence in most areas was considered a continuous and major threat to citriculture (Civerolo, 1984).

The remainder of this section will address *X. citri* pv. *citri* specifically—not other xanthomonads pathogenic to citrus. Extensive descriptions of the symptomatology and biology of this pathovar are available in several reviews (Civerolo, 1984; Goto, 1992; Gottwald et al., 2002a; Graham et al., 2004).

All aerial citrus tissues are susceptible to infection by *X. citri* pv. *citri* (Fig. 21.1). On leaves, the earliest visible signs of Asiatic citrus canker disease are small, water-soaked spots, which turn into slightly raised, blister-like lesions. The lesions further evolve into raised, corky, cankerous lesions with a color that ranges from beige to dark brown. Water-soaked margins may be visible, and a chlorotic zone often surrounds several aging leaf lesions. The morphology of symptoms on other organs is similar to the symptoms described for leaves. Cankers consist of hyperplastic and hypertrophic mesophyll tissue characterized by a rupture of the epidermis,


**FIG. 21.1.** Citrus canker lesions on various aerial citrus organs. **A,** Leaf lesions, displaying typical chlorotic halos. **B,** Fruit lesions on grapefruit. **C,** Lesions on a green shoot. **D,** Twig dieback, typically observed on highly susceptible cultivars (here, makrut or kaffir lime (*Citrus hystrix*)). **E,** Canker lesions on the trunk of a young tree. **F,** Leaf lesions associated with galleries of citrus leafminer (*Phyllocnistis citrella*). (Courtesy Dr. O. Pruvost and Dr. C. Verniére, CIRAD)

which is thought to allow the pathogen to be released onto plant surfaces quickly and efficiently (Pruvost et al., 2002; Timmer et al., 1991).

The marketability of fruit is greatly reduced by Asiatic citrus canker disease in a host species- and environment-dependent manner. Key lime (also known as "Mexican lime") (*Citrus* × *aurantifolia* (Christm.) Swingle (pro sp.)) and grapefruit (*C.* × *paradisi* Macfad. (pro sp.)) display the highest susceptibility to *X. citri* pv. *citri,* followed by sweet orange (*Citrus* × *sinensis* (L.) Osbeck) and lemon (*C.* × *limon* (L.) Burm. f. (pro sp.)), whereas tangerine (or mandarin) (*C. reticulata* Blanco) is partially resistant. Early fruit drop contributes to the economic impact of canker disease primarily on susceptible cultivars. Data from Argentina demonstrated that disease incidence can reach 80% of the fruit in grapefruit plots with no chemical control. Similarly, early fruit drop as high as 50% was reported for sweet orange cultivar Hamlin (Stall and Seymour, 1983). Direct damage also involves tree defoliation and/or twig dieback, which are common consequences of severe infections on highly susceptible cultivars. Severe outbreaks are particularly likely in tropical and subtropical environments, where high temperatures and rainfall occur concomitantly.

Because of the quarantine status of the pathogen, an indirect consequence of Asiatic citrus canker disease is the loss of markets for fruit export in countries or areas where the disease cannot be satisfactorily controlled. In the early 2000s, the annual cost of canker disease in Florida (approximately 0.3 million hectare of commercial citrus at the time) was estimated at $342 million per year (U.S. dollars) (Gottwald et al., 2002a). In Australia, economic analysis of the eradication of a 2004 citrus canker outbreak in Queensland estimated a potential net benefit of $70 million (Australian dollars) (Gambley et al., 2009). In the same country, the economic benefit of averting a national outbreak of citrus canker was estimated as high as $410 million, whereas the estimated cost of a 5-year citrus ban in Australia was $2 billion (Australian dollars) (Alam and Rolfe, 2006).

Infection of citrus occurs through natural openings and wounds, and variations in susceptibility are related to tissue age. Green, actively growing fruits and young leaves (i.e., one-half to two-thirds expansion) are most susceptible. In the absence of wounding, few lesions are observed on mature leaves and fruit. Researchers found that a single *X. citri* pv. *citri* cell forced into a stoma had the capability of inducing infection and subsequently causing disease (Gottwald and Graham, 1992). In the absence of force, bacterial suspensions containing at least $10^4$–$10^5$ cells per millilter were required to induce lesions from spray inoculation. Wounds made by wind, thorns, insects (e.g., the citrus leafminer (*Phyllocnistis citrella* Stainton)) (Fig. 21.1, part F), and grove maintenance are highly efficient sites of ingress. The presence of wounds not only favors infection, but it also widens the period during which infection can occur (Vernière et al., 2003). Temperatures at which *X. citri* pv. *citri* can produce lesions on sweet orange range from 12 to 40°C, with an optimum of 25–35°C (Dalla Pria et al., 2006). The temperature at the time of infection modulates the length of the latency period, which can extend over several weeks (Vernière et al., 2003).

*X. citri* pv. *citri* survives in canker lesions primarily on leaves, twigs, and branches. The population of the bacterium may exceed $10^7$ cells per lesion in young leaf lesions and approximately $10^5$ cells per lesion in old lesions (Pruvost et al., 2002). However, the overwintering of *X. citri* pv. *citri* in canker lesions seems to be influenced by environmental conditions, and greater declines in population have been reported in areas with a definite winter season (Stall et al., 1980). Moreover, the pathogen can survive in twig cankers up to several years. According to studies based primarily on the enumeration of culturable populations, survival outside a citrus host varies widely across media. *X. citri* pv. *citri* was reported from asymptomatic host species, although some asymptomatic populations may represent latent infections (Stall et al., 1993; Timmer et al., 1996).

A 2011 study using an unstable green fluorescent protein- (GFP-) tagged strain expressing fluorescence only in metabolically active cells suggested that *X. citri* pv. *citri* aggregates as biofilms and survives on citrus surfaces when protected from desiccation. Biofilms were found to protect bacterial cells from bactericide treatments applied in the field and in the postharvest fruit disinfection process (Cubero et al., 2011). Asymptomatic survival has also been reported from nonhost plant species (weeds and grass) and from straw mulch and soil (Goto, 1970, 1972; Goto et al., 1975; Leite and Mohan, 1987). *X. citri* pv. *citri* has been reported to survive only a few days in soil and a few months in plant debris that has been incorporated into soil. An unconfirmed report has suggested that *X. citri* pv. *citri* may survive for years in infected tissue that has been kept dry and free of soil (Das, 2003). Saprophytic survival has also been reported from nonhost plant species (weeds and grass) and from straw mulch and soil (Goto, 1970, 1972; Goto et al., 1975; Leite and Mohan, 1987).

Researchers have documented the survival of *X. citri* pv. *citri* on metal (e.g., vehicles, blades), plastic (e.g., fruit crates), leather (e.g., gloves and shoes), cotton (e.g., clothes, gloves), processed wood (e.g., crates, ladders),

bird feathers, and animal fur in both shade and sun. Their findings suggest that the bacterium dies within 24–72 hours, depending on environmental conditions. A potential risk for transmission may exist during the timeframe before bacterial death (Graham et al., 2000).

Although population estimates are based on culturable bacteria, a 2009 report suggested that *X. citri* pv. *citri* may enter a viable but nonculturable (VBNC) state in response to copper and remain virulent (Del Campo et al., 2009). The persistence of VBNC populations over time has not been assessed in previous studies, however, and the biological significance of VBNC populations remains unclear. A 2012 study highlighted interest in messenger RNA (mRNA) of selected genes as the target of quantitative real-time reverse transcription polymerase chain reaction (Q-RT-PCR) for quantification of viable populations of *X. citri* pv. *citri* (Golmohammadi et al., 2012).

*X. citri* pv. *citri* employs multiple virulence factors to cause symptoms and to manipulate the host responses for its survival in the intercellular spaces (Cernadas et al., 2008; De Souza et al., 2012; Fu et al., 2012). This discussion only briefly reviews the involvement of the type III secretion system (T3SS), the substrate type III (T3) effectors, EPS, and LPS in *X. citri* pv. *citri* infections.

Among the bacterium's virulence factors, the T3SS is the most important in causing the characteristic citrus canker lesions. Twenty-five T3SS effectors have been identified in *X. citri* pv. *citri* based on bioinformatic analysis (Jalan et al., 2011). Of these effectors, four have been characterized as playing roles in virulence—*pthA* (*pthA4*), *avrXacE1* (*xopE1*), *avrXacE2* (*xopE3*), and *Xac3090* (*xopL*)—and PthA (PthA4) has been characterized as being responsible for the induction of cell enlargement (hypertrophy) and division (hyperplasia) (Duan et al., 1999; Swarup et al., 1991; Yan et al., 2012).

*X. citri* pv. *citri* strain IAPAR 306 contains four *pthA* homologs, but the number of homologs is strain dependent (da Silva et al., 2002; Lee et al., 2008). Only *pthA* (*pthA4*) functions as the pathogenicity determinant, however (Swarup et al., 1991; Yan and Wang 2012). Citrus leaves inoculated with the *pthA* mutant were observed to remain flat, and no visible hypertrophy and hyperplasia symptoms were observed at the inoculated areas. Expression of the *pthA* gene in *X. axonopodis* pv. *citrumelo,* which does not contain *pthA,* confers the pathogen's ability to elicit erumpent canker lesions in citrus plants, rather than the typical spots caused by the wild-type strain (Swarup et al., 1991). Transient expression of *pthA* in host plant cells induces citrus canker symptoms, including hypertrophy, hyperplasia, and cell death (Duan et al., 1999).

PthA is a typical transcriptional activator-like (TAL) effector and contains N-terminal signals for bacterial T3SS, tandem repeats (17.5) that specify the target nucleotide sequence, nuclear localization signals, and a C-terminal transcriptional activation domain. TAL effectors have been reported to be translocated into the plant cell via the T3SS and to be targeted to the nucleus to function as transcriptional activators (Kay and Bonas, 2009).

Researchers noted that the knockout of *hrp* genes (*hpaB, hrcV, hrcN, hrpB, hrpD,* and *hrpF*) resulted in the loss of pathogenicity of *X. citri* pv. *citri,* probably from the failure of translocation of effectors into the host cells (Dunger et al., 2005; Yan and Wang, 2012). HrcV and HrcN are part of the core secretion apparatus of the T3SS, with HrcV inserting into the inner membrane and HrcN being a cytoplasmic protein that presumably associates with the cytoplasmic site of the secretion apparatus (Galan and Collmer, 1999). The *hpaB* gene encodes a T3SS chaperone and is required for the secretion of multiple effectors, including AvrBs3 in *X. euvesicatoria* Jones et al. (syn. *X. campestris* pv. *vesicatoria* (Doidge) Dye) (Büttner et al., 2004). This finding suggests that *hpaB* is required for the secretion of PthA (PthA4)—a homolog of AvrBs3 in *X. citri* pv. *citri.*

Interestingly, whereas the *hpaB, hrcV,* and *hrcN* mutants were not found to cause canker symptoms, the *pthA4* mutant did cause chlorosis symptoms. Additionally, the cell density of the *pthA4* mutant was 20 times lower than that of the wild type but at least five times higher than that of the T3SS mutants (*hpaB, hrcV,* and *hrcN*) (Yan and Wang, 2012). The difference in symptoms and populations between the *pthA4* mutant and the T3SS mutants *hpaB, hrcV,* and *hrcN* mutants suggests that effectors in addition to PthA4 also contribute to *X. citri* pv. *citri* virulence and survival in planta. Yet individual mutants of 19 putative effector genes (12 of which have been included in the standardized nomenclature of *Xanthomonas* T3 effectors) generated using site-directed mutagenesis revealed that none of the mutants had reduced ability to cause canker disease (Figueiredo et al., 2011). The lack of phenotype of the effector mutants probably results from the fact that effectors are redundant (Galán, 2009). Another study focused on three effector genes: *avrXacE1* (*xopE1*), *avrXacE2* (*xopE3*), and *Xac3090* (*xopL*) (Dunger et al., 2012). Specifically, the *avrXacE1*(*xopE1*) and *avrXacE2* (*xopE3*) mutants caused lesions with larger necrotic areas relative to the wild-type strain when infiltrated in citrus leaves. AvrXacE1 (XopE1) and AvrXacE2 (XopE3) may act separately by affecting different signaling pathways that modify plant tissue necrosis.

In addition to the T3SS, EPS (e.g., xanthan gum) contributes to the virulence of *X. citri* pv. *citri*. EPS is an important component of biofilm and contributes to epiphytic fitness (Rigano et al., 2007). It may also promote colonization of plant tissues by protecting pathogens from harsh environmental conditions, and it may contribute to occlusion of vascular tissues in wilts and blights (Denny, 1995; Kiraly et al., 1997). In one study, researchers generated a gum-deficient mutant by disrupting the *gumB* gene (which is involved in the transport of xanthan gum) and showed that xanthan gum is important for biofilm formation, epiphytic fitness, and bacterial growth in lemon and that it contributes to virulence (Rigano et al., 2007). In another study, researchers identified two *gum* genes (*gumF* and *gumK*) as being involved in the virulence of *X. citri* pv. *citri* in grapefruit (Yan and Wang, 2012). Both *gum* mutants showed reduced water soaking in citrus leaves at an early stage after inoculation, and the cell density of the *gum* mutants was significantly reduced in grapefruit leaves compared with the wild-type strain.

LPS is another major polysaccharide on the cell surface of proteobacteria (Vorhölter et al., 2001), and it contributes to the virulence of *X. citri* pv. *citri*. The roles of *wzt, wzm, nlxA,* and *rfbC* in LPS biosynthesis and bacterial virulence of *X. citri* pv. *citri* have been reported in several studies (Li and Wang, 2011; Petrocelli et al., 2012; Yan et al., 2012). Based on these studies, LPS appears to be involved in biofilm formation, stress resistance, motility on semisolid plates, virulence, and in planta growth of *X. citri* pv. *citri*. As a typical PAMP, LPS is also purported to affect the plant basal defense response (see Chapters 3 and 4). Two LPS mutants (*wzt* and *rfb303*) were found to cause lower increases in the expression levels of host defense-related gene compared with those of the wild-type strain (Petrocelli et al., 2012).

Xanthomonads that are pathogenic to a narrow range of citrus hosts have been reported from nations in southwest and central Asia (i.e., Cambodia, India, Iran, Oman, Saudi Arabia, and Thailand ) and from the U.S. state of Florida. Using several genotyping techniques, as well as DNA:DNA hybridization, researchers determined that these xanthomonads were genetically related to *X. citri* pv. *citri* but not to *X. fuscans* subsp. *aurantifolii* (Bui Thi Ngoc et al., 2009a, 2010; Sun et al., 2004; Vernière et al., 1998). Strains that were first designated as pathotype A* originated primarily from key lime and produced canker-like lesions on this host species after inoculation but not on grapefruit—a species highly susceptible to *X. citri* pv. *citri* pathotype A (Vernière et al., 1998). Strains isolated in Florida that were originally classified as pathotype $A^w$ caused disease in the field on alemow (*C. macrophylla* Wester) in addition to key lime (Hartung et al., 1993; Sun et al., 2004). Findings from pathogenicity tests have suggested that both A* and $A^w$ strains are pathogenic to alemow and to Persian lime (also known as "Tahiti lime") (*C.* × *latifolia* (Yu. Tanaka.) Tanaka), although both of these species are less susceptible than key lime (Bui Thi Ngoc et al., 2010). In addition, several techniques have been used to demonstrate that genetic diversity is higher among pathotype A*/$A^w$ strains than among pathotype A strains (Bui Thi Ngoc et al., 2009a). Furthermore, $A^w$ strains have been determined to be genetically closely related to some A* strains from India (Bui Thi Ngoc et al., 2009a, 2010).

When inoculated on different citrus species, pathotype A* strains were found to be responsible for variable phenotypes when compared with the pathogenically homogenous pathotype A strains. Symptoms ranged from no reaction to small, blister-like lesions without epidermis ruptures, and bacteria reached populations significantly lower than those of pathotype A strains. Some strains induced small canker-like lesions (with epidermis ruptures) when inoculated onto grapefruit and sweet orange but not tangor cultivar Ortanique (*C. reticulata* × *C. sinensis*) (Escalon et al., 2013). The *avrGf1* gene (syn. *xopAG* in the standardized nomenclature of *Xanthomonas* T3 effectors) was identified as an important determinant of host range restriction and determined to be responsible for the HR on sweet orange and grapefruit (Escalon et al., 2013; Rybak et al., 2009). It is present in $A^w$ and some A* strains from India and Oman but not in most pathotype A* strains nor in any pathotype A strains (Escalon et al., 2013). Given that a pathotype $A^w$ *avrGf1*$^-$ mutant strain was not fully restored for pathogenicity to grapefruit and that most A* strains do not carry *xopAG,* other genes are clearly responsible for host range limitation.

Considering the multiple pathovars and pathotypes that are similar in symptomatology but markedly different in economic impact, the reliable identification of citrus canker-causing strains is a key task. Examples of quarantine restrictions that have resulted from misdiagnosis of bacterial strains include the misidentification of a fungal disease in Mexico as a strain of *X. fuscans* subsp. *aurantifolii* and the misidentification of strains that cause citrus bacterial spot as a variant of citrus canker. Both examples outline the importance of accurate strain identification (Rodriguez et al., 1985; Schoulties et al., 1987). In addition, a precise characterization of strains with appropriate molecular markers is required for studies of molecular epidemiology. Molecular epidemiology, which integrates population genetics and epidemiology, offers the potential of finely tracking pathogen movements and identifying the contamination sources and the biotic or abiotic factors that

are linked to their spread. Analyzing this information provides insights about evolutionary forces such as gene flow, genetic drift, and selection pressure imposed by both the host and the environment that have shaped the structures of pathogenic populations and ultimately provides guidance for disease management (Milgroom and Peever, 2003).

Because of the lack of polymorphisms of housekeeping genes of *X. citri* pv. *citri* (Almeida et al., 2010; Bui Thi Ngoc et al., 2010; Young et al., 2008), multilocus sequence typing (MLST)—which has become the reference technique for molecular epidemiology of many bacteria—has insufficient resolution for strain typing of the pathovar (Maiden, 2006). Several genotyping techniques have been developed for *X. citri* pv. *citri,* including repetitive sequence-based PCR (rep-PCR), amplified fragment length polymorphism (AFLP), insertion sequence-based typing, and tandem repeat-based typing (Bui Thi Ngoc et al., 2008, 2009a, 2009b; Cubero and Graham, 2002; Li et al., 2007). In the genus *Xanthomonas,* genetic distances derived from AFLP and rep-PCR data were shown to correlate with the global genome divergence, as estimated by DNA:DNA hybridization (Rademaker et al., 2000). Based on AFLP, pathotype A*/A$^{w}$ strains showed a larger diversity than pathotype A strains and formed several clusters that were related to their area of origin (Fig. 21.2). Most pathotype A strains were closely related; the most divergent strains originated in Bangladesh, India, and Pakistan (Fig. 21.2). Some of the latter pathotype A strains appeared genetically related to pathotype A* strains but had different host ranges (Bui Thi Ngoc et al., 2009a).

The significant correlation between genetic distances derived from independent sets of markers indicates a lack of frequent recombination among *X. citri* pv. *citri* strains and suggests clonal evolution (Bui Thi Ngoc et al., 2009a). The lack of frequent recombination also supports the usefulness of genotyping in epidemiological studies at different spatio-temporal scales. Multilocus variable number of tandem repeats analysis (MLVA) is a simple and robust method that provides the necessary level of resolution for pathogens that cannot be differentiated by MLST. Moreover, it allows reliable interlaboratory comparisons, which may be of increasing use in the coming years. The rate of variation at tandem repeat loci can be influenced by several factors, such as the tandem repeat unit size or total size of the array, the degree of identity of the sequence, and the number of tandem repeats. These parameters can be used in selecting sets of markers that are relevant to the addressed biological question—from local outbreak investigation to global surveillance. Two MLVA schemes have been developed for outbreak investigation of *X. citri* pv. *citri* at small spatio-temporal scales (MLVA-14) (Bui Thi Ngoc et al., 2009a, 2009b; Vernière et al., 2014) and global surveillance analyses (MLVA-31) (Pruvost et al., 2014).

Because of the quarantine status of citrus canker-causing strains, the availability of reliable detection techniques is a major concern. Several PCR-based diagnostic tools have been developed with the specific aim of detecting strains of *X. citri* pv. *citri* (e.g., primers KingF/R (Kingsley and Fritz, 2000), J-RXg/c2 (Cubero and Graham, 2002), Xac01/02 (Coletta-Filho et al., 2006), XACF/R (Park et al., 2006)) and other citrus canker-causing strains, such as pathovars *X. citri* pv. *citri* and *X. fuscans* subsp. *aurantifolii* (e.g., primers 2/3 (Hartung et al., 1993), XCF/R (Miyoshi et al., 1998), 4/7 (Hartung et al., 1996), J-pth1/2 (Cubero and Graham, 2002), and VM3/4 (Mavrodieva et al., 2004)). Delcourt et al. (2013) evaluated the primers' inclusivity (i.e., the ability of a primer to detect all strains of the target organism) and/or exclusivity (i.e., the ability of a primer to generate negative responses from an extensive range of related but nontarget strains, including other *Xanthomonas* species and pathovars and supposedly

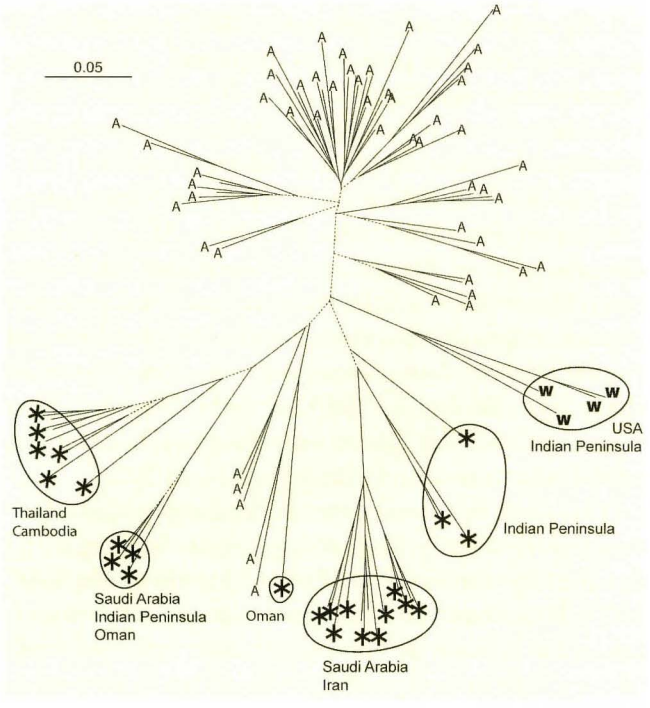


**FIG. 21.2.** Neighbor-joining tree based on the Dice dissimilarity coefficient, showing the relationships between strains of *Xanthomonas citri* pv. *citri* derived from amplified fragment length polymorphism (AFLP) data. Pathotypes are indicated as follows: A = pathotype A; * = pathotype A*; w = pathotype A$^{w}$. Branches with bootstrap values lower than 80% are represented by dotted lines.

saprophytic xanthomonads isolated from asymptomatic citrus). Several real-time PCR assays have been developed to detect strains of *X. citri* pv. *citri*—for instance, using nonspecific DNA binding SYBR Green dye (Mavrodieva et al., 2004) and using a specific fluorescent probe, such as TaqMan (Cubero and Graham, 2005; Golmohammadi et al., 2012). Loop-mediated isothermal amplification (LAMP) has also been applied in diagnosing canker disease (Rigano et al., 2010).

*X. citri* pv. *citri* survives primarily in naturally occurring lesions on leaves, twigs, and branches. Bacterial cells are readily suspended in the water from cankerous lesions and dispersed in droplets following rupture of the epidermis, but bacterial exudation decreases as lesions age (Timmer et al., 1991). Rainwater collected from foliage with lesions was found to contain bacterial populations of $10^5$–$10^8$ colony-forming units per milliliter (CFU $ml^{-1}$) (Goto, 1962; Pruvost et al., 2002; Stall et al., 1980; Timmer et al., 1996). Polluted droplets can also be spread by wind, mostly over short distances—for instance, within a single tree or to neighboring trees, most often causing greater disease severity on the side of the tree exposed to the wind-driven rain (Bock et al., 2010b; Goto, 1962; Pruvost et al., 1999, 2002; Stall et al., 1980). Efficient dispersal from tree to tree occurs when the average wind speed during rains reaches or exceeds 8 meters per second (Kuhara, 1978). The distance of dispersion and the amount of dispersed inoculum are a function of wind speed (Bock et al., 2010b). Similarly, an increase in wind speed fosters greater disease severity by producing more wind-associated leaf injuries (Bock et al., 2010a). Although wind-blown inoculum has reportedly been detected up to 32 meters from infected trees in Argentina, evidence also documents much longer dispersals (up to 17 kilometers (km)) after intense meteorological events in Florida, such as severe tropical storms, hurricanes, and tornadoes (Gottwald et al., 2001; Stall et al., 1980). Dispersion up to 56 km was recorded in the Florida county of Lee/Charlotte as a result of a hurricane in 2004 (Irey et al., 2006).

The citrus leafminer exacerbates the infection of citrus canker via its feeding activity, as seen in Florida and Brazil (Christiano et al., 2007; Gottwald et al., 1997, 2007; Hall et al., 2010). A bacterial concentration as low as $10^1$ cells per millilter can initiate infection when miner galleries are present, whereas a concentration of approximately $10^4$ cells per milliliter is required to infect unwounded leaves through natural openings (Christiano et al., 2007). The citrus leafminer insect can infest young leaves, stems, and fruit and greatly increases the number of individual lesions along the outlines of feeding galleries (Gottwald et al., 1997). Tissues wounded by leafminers remain susceptible for longer periods than wounds caused by wind, thorns, or pruning (Jesus et al., 2006). However, no published data support the role of the leafminer as an important vector of *X. citri* pv. *citri* (Belasque et al., 2005).

In citrus nurseries, disease spread is associated primarily with splash dispersal from rain and overhead irrigation, resulting in aggregations of diseased plants (Gottwald et al., 1989; Pruvost et al., 1999). Numerous secondary foci further develop and coalesce, reducing the strength of aggregation (Gottwald et al., 1992). Disease-induced defoliation of severely infected plants causes fluctuation in the slope of disease gradients.

In contrast with disease spread in citrus nurseries, disease spread in citrus groves is more directional, as dispersal from tree to tree is associated primarily with wind-driven rain (Gottwald et al., 1988, 1992). The proximity patterns are more contiguous in citrus groves because of the greater spacing between trees and the limited effect of splash dispersal to neighboring trees. Spatio-temporal analyses have further indicated that the greater the disease severity, the higher the level of inoculum that is available and as a result, the longer the contiguous area of disease spread (Gottwald et al., 1992). Spatio-temporal analyses of citrus canker on a large scale in an urban Florida environment confirmed the existence of a broad range of distances for bacterial spread following storms (Gottwald et al., 2002b).

Hurricanes can be major means of natural dispersal, leading to the occurrence of widespread secondary disease foci. The geospatially referenced infected citrus trees that were discovered following the devastating 2004–2005 hurricane season were used to develop a predictive model of citrus canker spread associated with extreme weather events (Gottwald and Irey, 2007; Irey et al., 2006). Wind–rain index vectors (WRIVs), based on an index ($I_{wr}$) that accounts for the intensity of wind and rain necessary for long-distance dispersal, were estimated, and the combined thresholds necessary to trigger such events were determined: namely, the wind speed required was 8 meters per second, and the rainfall required was 0.318 centimeters per hour. The application of these vectors in the leeward direction from the initial foci of infection captured 80% or more of the newly infected trees over a 14-month post-hurricane period (Irey et al., 2006). The model that estimates the intensity and the direction of citrus canker spread has proven useful in delimiting putatively infected areas to be surveyed. Applying reverse WRIVs in the windward direction also allowed determining the locations of unknown sources of inoculum.

Management of Asiatic citrus canker relies on prevention and exclusion approaches in areas that the pathogen has not been introduced or is not established

and on integrated control measures in regions that the disease is widely established (Graham et al., 2004). In the latter regions, management of citrus canker should rely on both preventive and curative measures applied to cultural and phytosanitary practices, such as the use of healthy citrus materials, the eradication of inoculum sources, the application of bactericides, and the use of appropriate citrus cultivars and rootstocks. Moreover, the choice of the measures should take into account management costs, effects on environmental and food safety, and the potential for selective pressures to be put upon bacterial populations.

Many citrus-producing countries restrict importation of citrus materials from regions infected by *X. citri* pv. *citri* to prevent introduction of this pathogen. These phytosanitary regulations strongly affect the national and international citrus markets. The epidemiological significance of postpacking survival of the bacterium for dissemination via contaminated fruit is still debated. Postharvest treatments were found to partially reduce the survival of *X. citri* pv. *citri* on fruit after processing via a packing line (Gottwald et al., 2009). No dispersal was observed from untreated fruit or postpacking-line-processed fruit from cull piles placed close to susceptible plants except in one instance when extreme wind (25 meters per second) was applied (Gottwald et al., 2009). In a study involving Satsuma mandarin (*C. unshiu* Marc.), survival of the pathogen on mature fruit was not detected after harvest and no spread from these harvested fruit to susceptible nearby hosts was observed (Shiotani et al., 2009). In another study, however, culturable and pathogenic *X. citri* pv. *citri* was recovered from fresh sweet oranges that had gone through postpacking-line disinfection treatments, storage, and shipment (Golmohammadi et al., 2007).

Eradication programs are nearly always beneficial, provided they are started early, and implementing such programs is certainly preferable to accepting the presence of disease and the concomitant economic effects from treatment costs and production losses (Graham et al., 2004; Schubert et al., 2001). For instance, the net benefit of the eradication of citrus canker to Queensland, Australia, alone was approximately $70 million (Gambley et al., 2009). Decisions should be understood and agreed upon by all parties involved and then quickly executed based on scientific principles. A delay at any stage will be detrimental to the success of the eradication program (Gottwald et al., 2001; Graham et al., 2004; Schubert et al., 2001). In São Paulo state, Brazil, introduction of the citrus leafminer prompted modification of the eradication policy (Gottwald et al., 2007).

In areas where Asiatic citrus canker has been widely established, control measures should focus on reducing the rate of infection and the spread of the disease, so that acceptable disease levels and an economically viable harvest are maintained. In addition, the production of healthy citrus materials through a certified program will help to establish disease-free groves. After the disease has occurred in the plots, drastic measures should be taken to limit disease incidence. Furthermore, as high wind speeds create wounds and disperse bacteria associated with rain, windbreaks should be established around plots to reduce the spread of the pathogen and disease severity (Bock et al., 2010a, 2010b). However, even though spread of the pathogen can be reduced by establishing windbreaks, its efficiency will remain variable (Behlau et al., 2008; Gottwald and Timmer, 1995).

The application of copper-based products is a standard control measure for citrus canker in groves. Copper applications have been shown to significantly decrease citrus canker incidence and severity and to reduce the number of prematurely dropped fruit (Behlau et al., 2008, 2010a, 2010b). Reducing the interval between sprays also increases the treatment efficiency. However, the profitability of the crop must justify spraying more frequently and causing adverse effects on the environment (Behlau et al., 2010b). In addition, frequent exposure to copper bactericides is a factor in selecting resistant strains. Copper-resistant *X. citri* pv. *citri* ($Cu^R$) strains have been reported in Argentina (Behlau et al., 2011; Canteros, 2004), and in Florida, while no $Cu^R$ strains were found after a 21-day-interval copper program, proliferation of epiphytic non-*Xanthomonas* $Cu^R$ bacteria increased the probability of horizontal transfer of copper-resistance genes to *X. citri* pv. *citri* (Behlau et al., 2012). Researchers tested the application of streptomycin treatments to lower the selection pressure for bacterial resistance to copper. Although no streptomycin-resistant ($Sm^R$) *X. citri* pv. *citri* strains were observed after repeated applications, once again, $Sm^R$ bacterial epiphytic populations were recovered from citrus leaves (Behlau et al., 2012). A 2012 report of $Sm^R$ strains of the pathovar in Asia could further impede the control of citrus canker (Hyun et al., 2012).

Pathogens can trigger a wide range of defense mechanisms in plants. The induced systemic acquired resistance (SAR) confers long-lasting protection against a broad spectrum of microorganisms to plants, consequently enhancing their defensive capacity (Durrant and Dong, 2004). SAR is regulated through a signal transduction pathway, in which the signal molecule salicylic acid (SA) plays a central role. Treatment via SA analogs, such as acibenzolar-S-methyl (ASM), can activate this pathway. Foliar applications of ASM were demonstrated to consistently reduce citrus canker severity under greenhouse conditions (Graham and

Leite, 2004). However, foliar applications of ASM alone or with copper formulations were not superior to copper alone in reducing disease incidence in sweet orange groves. Soil application of ASM or another SAR inducer greatly improved treatment efficiency in reducing the disease and in increasing the effectiveness of the treatment over time (Francis et al., 2009).

Most commercial citrus cultivars are highly susceptible to Asiatic citrus canker (Amaral et al., 2010; Gottwald et al., 1993). A few citrus species—mainly within the mandarin group, as well as kumquat (*Fortunella margarita* (Lour.) Swingle) and calamondin (*Citrofortunella microcarpa* (Bunge) Wijnands, an intergenic hybrid)—exhibit high levels of resistance to *X. citri* pv. *citri*. Researchers have characterized the genes involved in defense responses during the interaction between kumquat and *X. citri* pv. *citri* (Fu et al., 2012; Khalaf et al., 2007, 2011). A strong change in the kumquat transcriptional expression has been observed from 6 to 24 hours postinfection (Khalaf et al., 2011). The components involved in the incompatible interaction (such as reactive oxygen species (ROSs) and programmed cell death (PCD)) and in common defense mechanisms were identified. In addition, a number of differentially regulated genes with no homologs in the database were determined to be kumquat specific or associated with new defense mechanisms in citrus (Khalaf et al., 2011).

Conventional citrus breeding is a long, difficult process—partly because of the lack of disease-resistant germplasm and interference of the resistance character with the expression of traits related to fruit quality and production (Viloria et al., 2004). Thus, transgenic approaches have been developed to produce citrus canker-resistant plants based on identification of the genes involved in resistance and tolerance. Transgenic cultivars have been created by integrating the genes that encode antimicrobial proteins (Boscariol et al., 2006; He et al., 2011), the proteins that activate resistance against *Xanthomonas* spp. in other crops (Mendes et al., 2010; Sendin et al., 2012), the SAR regulator (Zhang et al., 2010), and a harpin protein (Barbosa-Mendes et al., 2009. Transgenic lines expressing these various genes showed greater resistance to citrus canker compared with nontransgenic lines.

## • Xanthomonas Leaf Blight of Onion

Xanthomonas leaf blight is a foliar disease that affects onion and other *Allium* species—namely, leek, chives, garlic, shallot, and Welsh onion—across many areas of the world. The natural host range of the pathogen, *X. axonopodis* pv. *allii* Kadota et al., appears limited to *Allium* spp. and certain rutaceous hosts.

On onion (*A. cepa* L.), the disease may appear at any stage of plant development, although symptoms generally develop and are most severe during or after the transition from vegetative to reproductive development. The loss of leaf area results in stunted plants and undersized bulbs, and yield reductions of 20% or more are common when the disease is severe near the time of bulb initiation. Bulb rot is not known to occur.

There is a high degree of genetic and phenotypic diversity among populations of *X. axonopodis* pv. *allii,* and there is evidence that differentiation of populations is associated with geographical isolation. The pathogen survives between susceptible crops in association with contaminated seed and infested crop debris and as an epiphyte or pathogen on volunteer onion, weed, and leguminous plants. The bacterium is readily disseminated within and between fields by surface irrigation water. Seed contamination appears to be an important source of primary inoculum and a key means of long-distance dispersal of the pathogen. Disease management involves an integrated approach that includes selection of cultivars with some level of resistance, sanitation of crop debris, and careful timing and use of irrigation and nitrogen fertility. Copper bactericides, biological control agents, and inducers of SAR also can provide some disease suppression under low to moderate disease pressure.

Xanthomonas leaf blight (also known as "bacterial blight") of onion was first described in 1978 from Hawaii by Alvarez et al. (1978). The taxonomy and nomenclature of the genus *Xanthomonas* were unstable during the time these researchers described Xanthomonas leaf blight, and the original report simply recognized the leaf blight pathogen as a *Xanthomonas* spp. The disease had appeared in a new onion production region in Hawaii that had been cleared of native vegetation, and attempts to isolate the pathogen from nearby plants and crop had failed to yield the bacterium.

The disease description provided by Alvarez et al. (1978) was markedly similar to that of a foliar blight of onion observed by Thomas and Weinhold in southern Colorado in 1953 and attributed to *X. striaformans*. However, the species description provided by Thomas and Weinhold (1953) differed from that provided by Alvarez et al. (1978) by eight physiological characteristics. Unfortunately, making comparative studies of *X. striaformans* and strains of the bacterium collected in Hawaii is not possible, because cultures of *X. striaformans* were not preserved and the pathogen has not been reported since it was originally described .

The Xanthomonas leaf blight pathogen described by Alvarez et al. (1978) was referred to as *X. campestris* (Pammel) Dowson in later publications (Isakeit et al., 2000; Nunez et al., 2002; Schwartz and Otto, 2000), although the basis of the species designation is unclear. In 2000, Kadota et al. (2000) proposed the epithet *X. campestris* pv. *allii* for *Xanthomonas* strains recovered from Welsh onion (*A. fistulosum* L.) in Japan. In independent studies published in 2004, Roumagnac et al. (2004a) and Gent et al. (2004) conducted polyphasic characterizations of a broad collection of *Xanthomonas* strains from *Allium* spp., including the type specimen described by Kadota et al. (2000). Both Roumagnac et al. (2004a) and Gent et al. (2004) found that *X. campestris* pv. *allii* is pathogenic to *A. cepa* and that the strains from *A. fistulosum* were indistinguishable genetically, phenotypically, and pathogenically from other *Xanthomonas* strains recovered from *A. cepa*. Both groups of researchers suggested that the correct species and pathovar designation should be *X. axonopodis* pv. *allii* to represent the correct phylogenetic position of the organism in the *axonopodis* group. This recommendation has been validated and accepted (Bull et al., 2010). The host range of *X. axonopodis* pv. *allii* varies among reports in the literature, but onion appears to be the host most commonly reported. The disease has been reported from numerous *Allium* spp.: namely, leek (*A. ampeloprasum* var. *porrum* (L.) J. Gay), chives (*A. schoenoprasum* L.), garlic (*A. sativum* L.), shallot (*A. cepa* var. *aggregatum* G. Don), and Welsh onion (Kadota et al., 2000; Picard et al., 2008). In Japan, however, *X. axonopodis* pv. *allii* has been reported to be nonpathogenic to chives and Chinese chives (*A. tuberosum* Rottler ex Spreng.) (Kadota et al., 2000). Other studies have found no evidence of host specificity on certain *Allium* spp. among genetically diverse isolates (Picard et al., 2008).

Strains of the pathogen from Barbados have been reported to be pathogenic to leguminous hosts, such as dry edible bean or snap bean (*Phaseolus vulgaris* L.), lima bean (*Phaseolus lunatus* L.), soybean (*Glycine max* (L.) Merr.), winged bean (*Psophocarpus tetragonolobus* (L.) DC.), moth bean (*Vigna aconitifolia* (Jacq.) Marechal), and garden pea or field pea (*Pisum sativum* L.). Attempts to reproduce disease symptoms on leguminous hosts outside Barbados have not been successful, however. Isolates of the pathogen from Hawaii induced a hypersensitive response (HR) in snap bean (Alvarez et al., 1978).

Roumagnac et al. (2004a) noted that some strains of *X. axonopodis* pv. *allii* occasionally caused small, water-soaked lesions on snap bean, but the lesions remained small and become necrotic within 7 days. Gent et al. (2004) found that bacterial blight symptoms were absent on dry bean exposed to *X. axonopodis* pv. *allii*, although the pathogen multiplied and attained high population levels in bean leaves. The bacterium appears able to persist asymptomatically as an epiphyte on some leguminous hosts (Gent et al., 2005c).

The host range of *X. axonopodis* pv. *allii* also includes certain rutaceous hosts. Citrus bacterial spot symptoms were induced following artificial inoculation of *X. axonopodis allii* on key lime and grapefruit cultivar Duncan (Gent et al., 2005a). The multiplication and the persistence of *X. axonopodis* pv. *allii* in Duncan grapefruit leaves were similar to those of an aggressive strain of *X. axonopodis* pv. *citrumelo*. In contrast, populations of *X. axonopodis* pv. *citrumelo* in onion were 1.3 log units lower than populations of *X. axonopodis* pv. *allii* in onion, suggesting that citrus is the primary host of *X. axonopodis* pv. *citrumelo*.

On onion, Xanthomonas leaf blight may appear at any stage of crop development on short-day cultivars, although symptoms generally develop during or after bulb initiation on long-day cultivars. Lesions initially appear as irregularly shaped white flecks, pale spots, or lenticular lesions with water-soaked margins. They quickly enlarge, become tan to brown in color, and are associated with extensive water soaking (Fig. 21.3). Chlorotic streaks develop on some cultivars and may extend the entire lengths of leaves. When weather conditions become hot and dry, infected leaves desiccate

**FIG. 21.3.** Water soaking and lesion development on onion leaves infected by *Xanthomonas axonopodis* pv. *allii*. (Courtesy H. F. Schwartz)

and become brittle but retain their characteristic tan to brown color (Fig. 21.4) (Gent and Schwartz, 2008).

As the disease progresses, the lesions coalesce and cause tip dieback, and extensive blighting of outer, older leaves occurs (Fig. 21.5). The loss of leaf area results in stunted plants and undersized bulbs. Under highly favorable disease conditions, all of the leaves may become completely blighted, resulting in a reduced photosynthetic area, premature plant senescence, and reduction in bulb size. Bulb rot is not known to occur.

Crop damage varies, depending on the timing of disease onset, weather conditions, and cultivar susceptibility. Onion bulb yield is most sensitive to defoliation near the time of bulb initiation, and the plant becomes progressively more tolerant to defoliation as bulbs mature (Bartolo et al., 1994). Because of this timing factor, crop damage from Xanthomonas leaf blight appears most severe when the disease occurs early in the growing season; yield reductions of 20% or more are common when the disease is severe near the time of bulb initiation. Temporal analysis of Xanthomonas leaf blight development in a tropical environment has suggested that bulb initiation is a very susceptible growth stage (Roumagnac et al., 2004b). In tropical and subtropical environments, seedling infection may occur, as well, leading to severe defoliation and complete loss of a marketable crop (Gent and Schwartz, 2008). Symptoms are similar on chive, garlic, leek, shallot, and Welsh onion but tend to be most severe on onion (Gent and Schwartz, 2008).

There is a high degree of genetic diversity among populations of *X. axonopodis* pv. *allii,* although the pathogen clearly belongs in the *X. axonopodis* 9.2 group (Gent et al., 2004, 2005a; Roumagnac et al., 2004a). The *X. axonopodis* group comprises six distinct genetic clusters (referred to as "groups 9.1 through 9.6") that have been characterized using DNA::DNA homology (Rademaker et al., 2000). *X. axonopodis* pv. *allii* rep-PCR "fingerprints" are highly similar genetically, serologically, and phenotypically to those of other *X. axonopodis* pathovars within DNA homology group 9.2 (Alvarez et al., 1991; Gent et al., 2004)—specifically, *X. axonopodis* pv. *alfalfae* (Riker et al.) Vauterin et al. and *X. axonopodis* pv. *citrumelo.* Genomic fingerprinting by rep-PCR identified eight DNA fragments that were common to five genotype groups identified in a collection of 49 isolates from 10 geographic regions. All eight DNA fragments were present in the type strain of *X. axonopodis* pv. *citrumelo,* and seven of the fragments were present in *X. axonopodis* pv. *alfalfae* and *X. axonopodis* pv. *betlicola* (Patel et al.) Vauterin et al. (Gent et al., 2004). Existence of a similar subgroup is evident based on monoclonal antibody reactions (Alvarez et al.,

**FIG. 21.4.** Advanced water soaking and necrosis of an onion leaf infected by *Xanthomonas axonopodis* pv. *allii.* (Courtesy H. F. Schwartz; reproduced, with permission, from H. F. Schwartz and S. K. Mohan. 2008. Compendium of Onion and Garlic Diseases and Pests, 2nd ed. American Phytopathological Society, St. Paul, MN)

**FIG. 21.5.** Advanced symptoms of onion tip death, necrosis, and water soaking, caused by infection with *Xanthomonas axonopodis* pv. *allii.* (Courtesy H. F. Schwartz; reproduced, with permission, from H. F. Schwartz and S. K. Mohan. 2008. Compendium of Onion and Garlic Diseases and Pests, 2nd ed. American Phytopathological Society, St. Paul, MN)

1991). These pathovars cannot be distinguished by genomic fingerprints generated by rep-PCR (Gent et al., 2005a), a method that has somewhat low resolution at the subspecies level, although they differ in virulence to and multiplication in their primary hosts.

Populations of the Xanthomonas leaf blight pathogen are highly diverse phenotypically and genetically, and differentiation of populations appears to be associated with geographical isolation. A diversity of fatty acid and substrate utilization profiles were apparent among 49 isolates collected from 10 geographical locations worldwide, with some evidence of clustering by region. A multivariate analysis of fatty acid composition and substrate utilization yielded a logistic regression model that was able to correctly classify 69% of the strains in the bacterial strains' geographical regions of origin (Gent et al., 2004). Genomic fingerprinting by means of rep-PCR identified five clades that were largely but not entirely explained by the geographical origins of the *X. axonopodis* pv. *allii* strains, indicating largely clonal population structures within individual production areas. Given the worldwide distribution of the populations sampled and the low probability for gene flow, this population differentiation is suggestive of founder effects (perhaps associated with introductions on seed) or bottleneck events that have selected for locally adapted populations of the pathogen (Gent et al., 2004).

In a study of populations of *X. axonopodis* pv. *allii* in the Mascarene Archipelago (east of Madagascar in the Indian Ocean), Picard et al. (2008) found that the strains that caused outbreaks of Xanthomonas leaf blight on Réunion Island were closely related genetically to strains isolated from diseased plants and contaminated seed lots in the neighboring island of Mauritius but distinct from other populations worldwide. Among isolates collected from the Mascarene Archipelago, two genetically related groups within the pathogen (labeled A and B) were distinguished by AFLP fingerprints, differential utilization of three carbon sources, and xanthomonadin pigment production. Group A strains became nearly extinct within 10 years after introduction of the pathogen to Réunion Island, suggesting potential differences in fitness between strains in the two groups. The prevalence and epidemiological significance of the group A and B strains outside the Mascarene Archipelago is unknown (Picard et al., 2008).

Xanthomonas leaf blight is favored by moderate to high temperatures of 25–30°C (Alvarez et al., 1978; Paulraj and O'Garro, 1993) to 28–35°C (Schwartz et al., 2003) and by rainfall at bulb initiation and continuing through bulb development (Schwartz et al., 2003). Frequent rains after bulb initiation, especially when driven by strong winds, favor severe disease epidemics. In Colorado, researchers found that the first appearance of disease was strongly associated with temperature, with some moderating effects due to rain. However, at harvest, the severity of disease was associated primarily with the amount of rain and less influenced by temperature (Schwartz et al., 2003). This phenomenon appears related to the importance of onion bulb initiation for triggering disease susceptibility (Gent and Schwartz, 2005a). Temperature is a primary determinate of host growth and bulb initiation; disease spread and severity are linked to rain once bulb development has been initiated (Gent et al., 2005b; Schwartz et al., 2003). Overhead irrigation and humid, overcast conditions also appear to favor disease.

Secondary infection occurs when aerosols or splashing water deposit *X. axonopodis* pv. *allii* onto leaves. The bacterium multiplies to form large epiphytic populations in the presence of dew or other moisture, infecting hosts through natural openings, such as stomata and wounds. The role of latent infections in the disease cycle has not been investigated extensively. Leaf abrasion by wind and windblown sand favors infection. Sanders et al. (2003) induced disease symptoms within 4–7 days on seedling onion foliage inoculated with $10^7$ CFU $ml^{-1}$ under greenhouse conditions.

*X. axonopodis* pv. *allii* survives between susceptible crops in association with contaminated seed and infested crop debris and as an epiphyte or pathogen on volunteer onion, weed, and leguminous plants (Gent et al., 2005b). Death of the pathogen in crop debris is hastened by burial. In Colorado, Gent et al. (2005b) found that populations of *X. axonopodis* pv. *allii* declined approximately 2–5 log units when infested leaves were overwintered on the soil surface, whereas populations declined 8–9 log units when infested leaves were buried. The pathogen persisted for several months epiphytically on several leguminous hosts commonly grown in rotation with onion and on weeds found in onion fields in the western United States (Gent et al., 2005b, 2005c). However, the bacterium was not recovered from sites where Xanthomonas leaf blight did not occur the previous season, suggesting that these epiphytic sources of the pathogen provide only a temporary refuge.

The pathogen is readily disseminated within and between fields by surface irrigation water. Irrigation tail water flowing through onion fields during outbreaks of Xanthomonas leaf blight was found to harbor up to 45,000 CFU $ml^{-1}$ of *X. axonopodis* pv. *allii* (Gent et al., 2005b). The concentration of the bacterium in irrigation water increased during the season in association

with the increased severity of disease in the field. Presumably, the pathogen also could be transmitted with and among fields by contaminated debris and exudates adhering to workers and equipment (Gent and Schwartz, 2005b, 2008).

Seed contamination appears to be an important source of primary inoculum and a key means of long-distance dispersal of the pathogen. Following the initial outbreak of Xanthomonas leaf blight in Hawaii, Alvarez et al. (1978) attempted unsuccessfully to isolate the pathogen from nearby plants and crops and hypothesized that the pathogen was seedborne. Lack of an efficient method of detecting the seedborne inoculum precluded demonstration of seed contamination. However, later reports confirmed that the pathogen is seedborne (Roumagnac et al., 2000) and that seed may be an epidemiologically important inoculum source when environmental conditions favor disease outbreaks (Gent and Schwartz, 2005a; Roumagnac et al., 2000). The rapid spread of the pathogen internationally is presumed to have occurred via contaminated seed, which is supported by spatial analyses of disease outbreaks (Roumagnac et al., 2004b) and molecular epidemiology studies (Humeau et al., 2006). Distinct disease foci can develop and lead to secondary spread of the pathogen when as few as 0.04% of the seeds are contaminated with the bacterium. Frequent rains or overhead irrigation appear essential for development of an epidemic of Xanthomonas leaf blight from contaminated seed in a semiarid environment (Gent and Schwartz, 2008).

Xanthomonas leaf blight can be managed effectively and economically by planting pathogen-free seed, selecting moderately resistant cultivars, applying strict sanitation and sound cultural practices, and ensuring timely use of chemical controls. The selection of seed and planting materials that are free of the pathogen is also essential for effective disease control. Culture and DNA-based testing procedures for detecting seed contamination are available (Robène-Soustrade et al., 2010; Roumagnac et al., 2000).

Cultivar susceptibility to Xanthomonas leaf blight varies widely. Susceptibility tends to be greatest among Sweet Spanish onion cultivars. In general, cultivars with white and red bulbs tend to be less susceptible than cultivars with yellowish bulbs, although such cultivars are not fully resistant. In Barbados, cultivars H-942 and H-508 were reported to be resistant to the disease (O'Garro and Paulraj, 1997). These cultivars had reduced disease severity compared with other cultivars but did not show complete resistance when inoculated with a Colorado strain of the pathogen, contrary to previous reports (Lang et al., 2004).

A 2-year or longer crop rotation to a nonsusceptible host, such as winter wheat (*Triticum aestivum* L.) or corn (*Zea mays* L.), is advisable. Rotation of onion and garlic with a leguminous crop, such as alfalfa (*Medicago sativa* L.), dry bean, or soybean, may allow epiphytic survival of the pathogen and should be avoided. Prompt and thorough incorporation of crop debris into the soil after harvest reduces pathogen overseasoning and survival between susceptible crops. Overhead irrigation and the reuse of irrigation water should both be avoided. In addition, avoiding excessive nitrogen fertilization can reduce Xanthomonas leaf blight severity (Gent and Schwartz, 2005a, 2008). Excessive nitrogen fertilization was shown to increase disease severity 27–50% compared with moderate fertilizer treatments (Gent and Schwartz, 2005a).

Chemical control measures can provide some disease suppression under low to moderate disease pressure. The critical period for beginning application of chemical control measures is 1–2 weeks before bulb initiation (Gent and Schwartz, 2005b). Copper-based bactericides amended with an ethylene bisdithiocarbamate fungicide, such as maneb or mancozeb, are commonly used. However, researchers have found that maneb amendment does not significantly improve disease control when the bacterial population is sensitive to copper (Gent and Schwartz, 2005b; Schwartz and Otto, 1998). Resistance to commonly applied bactericides was not observed within the collection of strains evaluated in Colorado (Gent et al., 2004) and in Barbados (Paulraj and O'Garro, 1992).

Applications of acibenzolar-S-methyl (Actigard 50WG) were found to reduce in planta and epiphytic populations of *X. axonopodis* pv. *allii* as effectively as applications of copper hydroxide–mancozeb in growth chamber studies. Under field conditions, two to four weekly applications of acibenzolar-*S*-methyl reduced severity of Xanthomonas leaf blight as or more effectively than nine to 12 weekly applications of copper hydroxide or copper hydroxide–mancozeb. However, the use of acibenzolar-S-methyl may cause yield reductions when the number of treatments is excessive or applications are made in the absence of disease (Gent and Schwartz, 2005b).

Some success has been achieved with the biological control of Xanthomonas leaf blight under low to moderate disease severity. A commercial formulation of a mixture of *Pantoea agglomerans* (Ewing & Fife) Gavini et al. strain C9-1 and *Pseudomonas fluorescens* Migula strain A506 provided disease control similar to copper hydroxide in field studies in Colorado (Gent and Schwartz, 2005b). In other studies, biweekly or weekly

applications of bacteriophages reduced disease severity by 26–50%—results superior to those produced by copper hydroxide–mancozeb treatments (Lang et al., 2007). In these studies, acibenzolar-S-methyl also successfully reduced disease severity with or without bacteriophages by up to 50%, but the reductions generally were not associated with improvements in onion bulb size or yield. The narrow spectrum of disease control provided by biological controls and the cost of using them are both impediments to their use in commercial production.

## Xanthomonads Associated with Bacterial Spot Disease of Tomato and Pepper

In general, bacterial spot disease of tomato (*Solanum lycopersicum* L.) and pepper (*Capsicum annuum* L.) is characterized by necrotic lesions on all aboveground plant parts and raised, scabby lesions on fruit (Jones et al., 1991). During rainy weather and when dew is present, spots may appear water soaked. Foliar symptoms typically begin as small (less than 3 millimeters (mm) in diameter), circular, dark-green, water-soaked lesions that turn dark brown to black as they expand and dry. On leaves, halos may be present around spots but not prominent. Leaf spots caused by *X. perforans* Jones et al. often have a shot-hole appearance (Stall et al., 2009). In flower infections, significant blossom drop occurs. Small, water-soaked, slightly raised spots appear on petals, forming greenish-white halos, and they sometimes enlarge to 3–6 mm in diameter (Agrios, 2005). Fruit lesions are typically larger and have distinct raised margins, giving the lesions a scabby appearance. As fruit lesions expand, the centers may become sunken and are often prone to secondary infection by opportunistic pathogens. Dark-brown spots appear as small, wet-to-greasy lesions on both leaf surfaces (Vallad et al., 2004), and lesions often coalesce, giving the plant a blighted appearance. On tomato, infected plants may become huddled in appearance because of increased ethylene production, which results in epinasty. On pepper plants, the increase in ethylene production often leads to leaf abscission. For both plants, the disease may result in significant yield reduction, which may include reduced fruit quality (Pohronezny and Volin, 1983).

Bacterial spot disease on tomato and pepper is caused by four bacterial species: *X. euvesicatoria* Jones et al.; *X. vesicatoria* (ex Doidge) Vauterin et al.; *X. gardneri* (ex Sutic) Jones et al.; and *X. perforans* (only on tomato) (Jones et al., 2004). The bacteria all survive in seed, which is important in disseminating them long distances (Jones et al., 1991). Seed-borne bacteria serve as an important inoculum source for seedling production in greenhouses, where high temperatures, high relative humidity, high plant density, and overhead irrigation create an ideal environment for disease development. The bacteria are also able to survive relatively short periods epiphytically on tomato seedlings. In more temperate climates, the bacteria generally survive less than 2 years in crop residue, but in tropical and subtropical regions, they survive only a few months. The bacteria are also present on volunteers of both tomato and pepper. Disease is favored by various temperatures and by high precipitation, depending on the particular bacterial species (Araújo et al., 2010). The bacteria are disseminated within a field by wind-driven rain droplets, handling of plants (e.g., suckering, clipping, training, harvesting), and aerosols.

Integrated management approaches are available for the control of bacterial spot disease. Among the disease control strategies, the principal strategy has been chemical control, relying on copper compounds, antibiotics, and plant activators. In the 1950s, the use of streptomycin quickly resulted in the selection of streptomycin-resistant strains (Thayer and Stall, 1962). For many decades, copper bactericides have been used to control bacterial diseases, affecting a wide range of annual and perennial crops. However, the continuous application of copper bactericides caused the development of copper-resistant ($Cu^R$) strains of the bacterial spot pathogen and thus reduced these products' effectiveness in disease control (Bouzar et al., 1999; Marco and Stall, 1983; Stall et al., 1986).

Host resistance is a desirable management strategy for control of bacterial spot, but genetic resistance may be ineffective because of race shifts in pathogen populations (Gassmann et al., 2000; Jones et al., 1998). Designing new and possibly durable resistance requires knowledge of the pathogenicity factors that characterize the four species mentioned earlier. Four sources of resistance have been identified in tomato: (1) Hawaii 7998 derived (*Rx1, Rx2* and *Rx3*) (Yang et al., 2005); (2) *Xv3* (Astua-Monge et al., 2000a); (3) *RXopJ4* (Sharlach et al., 2013); and (4) *Bs4* (Bonas et al., 1993). These four sources interact with *avrRxv* (Whalen et al., 1993), *avrXv3, xopJ4,* and *avrBs4,* respectively, to elicit an HR. Six resistance genes have been identified in pepper: (1) *Bs1;* (2) *Bs2;* (3) *Bs3* (Stall et al., 2009); (4) *Bs4C* (Strauss et al., 2012); (5) *Bs7* (Potnis et al., 2012); and (6) *BsT.* They interact with *avrBs1, avrBs2, avrBs3, avrBs4C, avrBs7,* and *avrBst1,* respectively, to provide a hypersensitive type of resistance.

Two recessive resistance genes, *bs5* and *bs6*, provide a nonhypersensitive or multigenic resistance that has been shown to be more durable against strains of *X. euvesicatoria* (Stall et al., 2009). Screening for such novel resistance genes is paramount, since the pathogen continues to evolve as a result of selective pressure and thereby overcomes host resistance in the field (Gassmann et al., 2000; Wichmann et al., 2005). While searching for resistance against bacterial spot pathogens in pepper genotypes, researchers identified a novel resistance in aji pepper (*C. baccatum* var. *pendulum* (Willd.) Eshb.) against races of *X. euvesicatoria* and *X. gardneri*. The resistance is associated with a single dominant resistance gene that confers resistance against two avirulence gene homologs: *avrBs7* and *avrBs1.1* from *X. gardneri* and *X. euvesicatoria*, respectively (Potnis et al., 2012). In later research, a new gene was identified—*Bs4C*, present in rocoto pepper (*C. pubescens* Ruiz & Pav. PI235047)—based on transcriptome profiling with next-generation sequencing (RNA-seq) (Strauss et al., 2012). The method was less labor intensive than positional cloning and resulted in identification of the TAL effector-induced R gene *Bs4C*, which was expressed only in the presence of AvrBs4. Bs4C was determined bioinformatically to encode a structurally unique R protein.

The four bacterial species—*X. euvesicatoria, X. vesicatoria, X. perforans,* and *X. gardneri*—are phenotypically and genotypically distinct and represent four distinct pathogen groups, which have been designated A, B, C, and D, respectively (Jones et al., 2004). Based on 16S ribosomal RNA (rRNA) sequence analysis, *X. euvesicatoria* and *X. perforans* form a monophyletic group, whereas *X. vesicatoria* and *X. gardneri* are in a cluster with *X. campestris* pv. *campestris* (Dowson) Dye et al. Phylogenetic comparisons based on MLST data for A-, B-, C-, and D-group strains and other xanthomonads have shown that *X. euvesicatoria* and *X. perforans* are closely related, whereas *X. gardneri* and *X. vesicatoria* are in separate clades (Almeida et al., 2010).

The sequencing of *X. euvesicatoria* strain 85-10 has provided important insights into pathogenicity mechanisms (Thieme et al., 2005). Representative genomes of the three other bacterial species associated with bacterial spot of tomato and/or pepper were sequenced and compared with the genome of *X. euvesicatoria* to study the evolution of these strains and their convergence on tomato and/or pepper (Potnis et al., 2011). A phylogeny based on a whole-genome comparison correlated well with a phylogeny based on MLST: namely, *X. perforans* and *X. euvesicatoria* belonged to the clade containing *X. citri,* and *X. gardneri* and *X. vesicatoria* belonged to separate clades but were more closely related to *X. campestris* pv. *campestris* than to *X. euvesicatoria* or to *X. perforans*. Different pathogenicity clusters were compared for their sequence identities and overall genetic organization among the four bacterial spot pathogens and other closely related xanthomonads. Two pathogenicity clusters from *X. euvesicatoria* (i.e., the *hrp* cluster and flanking genes and the type II clusters (*xps* and *xcs*)) were similar to the cluster from *X. perforans,* and the clusters from *X. vesicatoria* were similar to those from *X. gardneri*. However, characteristics of some of the pathogenicity clusters suggested acquisition from different closely related pathogens during evolution. For example, *X. perforans* acquired a novel LPS cluster, which is similar to that from *X. citri*. All three pepper pathogens (*X. euvesicatoria, X. vesicatoria,* and *X. gardneri*) possess similar LPS biosynthesis clusters (Potnis et al., 2011).

This difference in the composition of the LPS cluster between the pepper pathogens and *X. perforans* might be a clue to this species' predominance in tomato and its restricted ability to colonize pepper. Using an in planta growth assay, researchers tested the contribution of the LPS O-antigen from the pepper pathogens to pathogenicity on pepper. A mutant of *X. perforans,* Δ*avrXv3* (a functional *avrXv3* elicits an HR in pepper (Astua-Monge, 2000a)), that carries the LPS O-antigen from *X. euvesicatoria* showed increased in planta growth but was not restored to the growth attained by the pepper pathogen, indicating that the LPS O-antigen is not the only determinant of virulence on pepper (Potnis et al., 2011).

T3 effectors are major contributors of pathogenicity in xanthomonads, and many have been implicated in conferring host specificity (see Chapters 7, 8, and 9). A total of 45 effectors have been computationally identified in bacterial spot pathogens, but only a few have been experimentally validated (Potnis et al., 2011). Among the 45 effectors, 11 are core effectors. Effectors XopD, XopL, and XopN have been shown to interfere with PAMP-triggered immune responses, and effectors AvrBs2, XopX, and XopZ have been characterized as serving functions in virulence and fitness of the pathogen (Kearney and Staskawicz, 1990; Kim et al., 2009; Metz et al., 2005). Mutations in core effectors *xopQ* and *xopF1* have been shown not to alter pathogenicity (Roden et al., 2004).

The four bacterial spot pathogens have different repertoires of effectors and include some of the unique variable effectors, which may explain host preferences among strains. Mutation in an individual effector gene does not often result in a change in the virulence phenotype, explaining the redundancy among T3 effectors.

Effectors with little sequence similarity but redundancy in function (known as "redundant effectors") in *Pseudomonas syringae* pv. *tomato* strain DC3000 (Kvitko et al. 2009) are also found in xanthomonads with functional overlap. A classic example is the XopJ effector family, which includes AvrBsT, XopJ, AvrXv4, and AvrRxv. Comparing representative strains from the bacterial spot disease complex, researchers found that *X. euvesicatoria* contains two effectors belonging to the XopJ family (XopJ1 and XopJ3), whereas *X. vesicatoria* and *X. perforans* have only single XopJ-family effectors (AvrBsT and AvrXv4, respectively) and *X. gardneri* has none (Potnis et al., 2011). The AvrXv4 effector from this family has been shown to contribute to suppression of a PAMP-triggered immunity (PTI) response induced during the early stages of infection. The avirulence genes *avrBsT* and *avrBsP* have been determined to be responsible for nonhost resistance on pepper and tomato genotypes, respectively (Minsavage et al., 1990). AvrBsT has also been shown to interfere with effector-triggered immunity (ETI) activated by AvrBs1 and to suppress HR (Szczesny et al., 2010).

The TAL effectors (AvrBs3/pthA family) have been well studied for their contribution to virulence by activating host genes (e.g., hypertrophy) and thereby causing pathogen spread and disease (see Chapter 8). Among the four bacterial spot xanthomonads, *X. euvesicatoria* strain 75-3 contains AvrBs3 and *X. gardneri* strain 444 contains AvrHah1—both major virulence determinants (Schomack et al., 2008). Enhanced watersoaking phenotype by *X. gardneri* was attributed to the presence of AvrHah1 in *X. gardneri*. *X. vesicatoria* and *X. gardneri* have acquired few effectors that are not present in *X. perforans* or *X. euvesicatoria*. Effectors XopAG (AvrGf1) and XopAI—which are found in the citrus pathogens *X. citri* pv. *citri* and *X. fuscans* subsp. *aurantifolii* types B and C —are also present in *X. vesicatoria*.

*X. gardneri* contains few unique effectors—XopAO, XopAQ, XopAS, and an AvrBs1 member, as well as several other virulence factors—compared with *Pseudomonas syringae* van Hall (Potnis et al., 2011). Some of these effectors have been characterized as virulence factors in other plant pathogens and may be candidate determinants for aggressiveness of *X. gardneri* on pepper (Schornack et al., 2008). Similar to *P. syringae*, *X. gardneri* prefers lower temperatures for disease development on tomato. Given this similarity, researchers have hypothesized that *X. gardneri* might have acquired these effectors by horizontal gene transfer (HGT) from *P. syringae* strains on tomato (Potnis et al., 2011.

In a 2012 study, researchers used a transcriptomics approach in *X. euvesicatoria* strain 85-10 to identify the transcriptional start sites and the small RNAs that may be involved in virulence. The study suggested that sx12 small RNAs play a role in virulence of *X. euvesicatoria* on pepper and might interfere with the complex interactions between the pathogen and host (Schmidtke et al., 2012).

## *Xanthomonas axonopodis* pv. *dieffenbachiae*

Bacterial blight of anthurium is caused by *Xanthomonas axonopodis* pv. *dieffenbachiae* (McCulloch & Pirone) Vauterin et al. (previously, *X. campestris* pv. *dieffenbachiae* (McCulloch & Pirone) Dye) (Vauterin et al., 1995). The disease is characterized by chlorotic to necrotic foliar lesions, discoloration of vascular tissues, and systemic wilt that results in death of the plants. The pathogen invades the plant through hydathodes at the leaf margins and moves into the vascular tissues, interrupting water transport and causing general foliar necrosis and wilt. Discolored vascular tissue is observed in cross sections of the stock, which quickly rots, causing plant death. Disease management is dependent on producing pathogen-free propagative materials in tissue culture, practicing sanitation and eradicating diseased plants from fields, and changing cultural practices to reduce disease spread. Foliar sprays with antibiotics, bactericides, and biological control agents reduce disease incidence and severity but generally are not economical means of disease control. Resistant and tolerant cultivars have been developed by both traditional breeding and transgenic methods.

*Anthurium* is a large genus in the family Araceae, which has more than 100 genera, including *Dieffenbachia, Xanthosoma, Spathiphyllum, Epipremnum, Aglaonema,* and *Philodendron,* among others. The genus *Anthurium* comprises more than 1,500 species—more than 600 of which originate from the tropical Americas (Kamemoto and Kuehnle, 1996). The most popular species is *A. andraeanum* Linden, which is native to Colombia and Ecuador.

Bacterial blight, which also affects other aroids, is the most destructive disease of ornamental anthurium plants worldwide and is an ongoing threat to commercial production. The disease was observed in Brazil in 1960 and reported for the first time in the United States in August 1971 on the Hawaiian island of Kauai (*A. andraeanum* cultivar Kansako Red) (Hayward, 1972). Bacterial blight reached epidemic proportions during 1985–1989, destroying the production of

approximately 200 small farms on the island of Hawaii (Alvarez et al., 2006). Following implementation of an integrated disease management program, annual production losses were eventually reduced to 5% or less, but because of the high cost of disease management, a few large farms now dominate the commercial market.

This devastating disease has limited anthurium production throughout the world. By 1992, bacterial blight had been reported in Australia, Guam, Jamaica, Martinique, the Philippines, Puerto Rico, Trinidad, the United States (Florida), and Venezuela (Lipp et al., 1992), and it has subsequently been reported in India (Sathyanarayana et al., 1998) and the Netherlands (OEPP/EPPO, 2004).

Foliar symptoms start as water-soaked spots that are visible near leaf margins, where hydathodes (filled with guttation fluid) serve as the most common port of entry (Fig. 21.6). The pathogen also may invade through wounds or stomates on the same leaf. Tissue surrounding the infected area turns yellow and water-soaked spots coalesce, eventually forming large, necrotic zones at leaf margins. The pathogen moves quickly into the vascular tissues, preventing the translocation of nutrients and water and producing symptoms of water stress that may resemble natural senescence (Fig. 21.7). The main stem of a systemically infected plant turns dark brown and the growing point deteriorates, eventually resulting in plant death. Stomatal invasion often results in limited colonization of the mesophyll tissues but does not necessarily lead to systemic infection. When the spathe is infected, the disease is called "flower blight," even though the spathe is a modified leaf (Fig. 21.8).

Bacterial blight affects most genera and species in the family Araceae (Lipp et al., 1992). A comparison of 323 strains of *X. axonopodis* pv. *dieffenbachiae* resulted in a clear separation of strains isolated from *Anthurium* spp. from those isolated from other aroids (Lipp et al., 1992). Fingerprint analysis using rep-PCR revealed similar groups of anthurium strains and distinguished strains from other aroids (Louws and Alvarez, 2000). Random

**FIG. 21.7.** Anthurium plant showing foliar chlorosis, dried petioles, and disintegrating stem tissues, which are characteristic of systemic infection with *Xanthomonas axonopodis* pv. *dieffenbachiae*. (Courtesy T. Vowell)

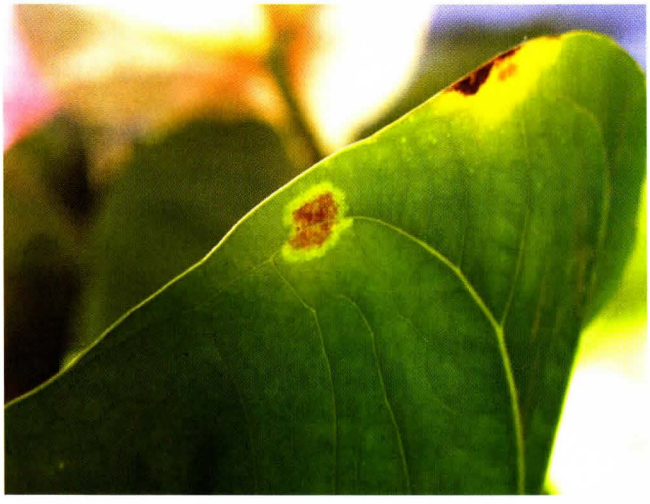

**FIG. 21.6.** Anthurium leaf displaying a darkened area around hydathodes at the leaf margin and a necrotic/chlorotic lesion around a stomate following infection with *Xanthomonas axonopodis* pv. *dieffenbachiae*. (Courtesy T. Vowell)

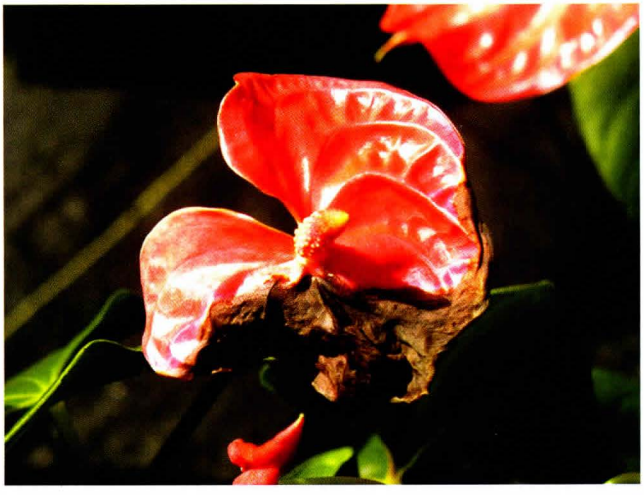

**FIG. 21.8.** *Anthurium andraeanum* with a blighted spathe, caused by infection with *Xanthomonas axonopodis* pv. *dieffenbachiae*. (Courtesy T. Vowell)

amplified polymorphic DNA- (RAPD-) PCR fingerprinting and southern analysis of strains isolated from different aroid hosts and locations confirmed some of the earlier serological groups and formed the basis for a DNA-based diagnostic test (Khoodoo and Jaufeerally-Fakim, 2004; Khoodoo et al., 2005a). An immunocapture PCR was developed using a *Xanthomonas*-specific monoclonal antibody in combination with specific primers in a multiplex PCR (Khoodoo et al., 2005b). A phylogenetic tree based on a DNA marker from a portion of the *dnaA* replication initiation factor (RIF) revealed the heterogeneity of *X. axonopodis* pv. *dieffenbachiae* isolated from various hosts in the Araceae. A tight cluster of 42 strains was associated with anthurium blight, whereas strains from other hosts were dispersed throughout the tree (Schneider et al., 2011). A comprehensive genetic analysis of 175 strains of *X. axonopodis* pv. *dieffenbachiae* from 10 aroid hosts revealed four distinct phylogenetic groups that generally corresponded to the hosts and geographical origins of the strains (Donahoo et al., 2013). Researchers used serotyping to trace the movement of the bacterium in shadehouses through culturing and immunodiagnostic tests and found that the pathogen is spread by contaminated cutting tools, infected plant materials, wind-driven rain and irrigation water, and aerosols (Lipp et al., 1992; Norman and Alvarez, 1989, 1994). The pathogen rarely infects plants through the roots.

The capacity of *X. axonopodis* pv. *dieffenbachiae* to spread through aerosols was demonstrated using Andersen samplers and settling plates (Alvarez et al., 2006). These findings indicate that control procedures based on excluding the pathogen through the use of clean planting stock may be thwarted unless appropriate precautions are taken. The risk of contamination is high if propagation areas for tissue-cultured plantlets are downwind of flower production areas. Tissue-cultured plantlets are often established on misting benches, and young, wet tissues are highly susceptible to infection.

In another study, symptomless cuttings destined for use as planting stock were initially considered pathogen free, so propagative materials grown under cooler conditions at higher elevations were used as planting stock for lower-elevation farms (Norman and Alvarez, 1996). Repeated assays of 1,000 symptomless cuttings revealed that whereas only 0.4% were infected initially, the pathogen spread to neighboring plants when they were propagated on greenhouse benches. Five months later, the disease incidence had increased 10-fold, and within the first year, approximately 67 (6.9%) of 967 symptomless cuttings were found to be infected when transplanted into the production field. Nearly all of the plants in the block developed symptoms before flowering, and the block was destroyed. Spread of the disease from symptomless cuttings convinced growers to develop a pathogen-free tissue culture program, which remains the most important component of a successful disease management program for anthurium blight today.

Other components of an integrated management program for anthurium blight include removal of diseased plants, disinfestation of harvesting implements and containers, spraying with chemicals, modification of cultural practices, production of pathogen-free planting stock in vitro, use of resistant cultivars, and use of biological controls (Alvarez et al., 2006). Disinfestation of cutting tools is essential for preventing the spread of blight, because symptomless plants may have latent infections. Yet while sanitation practices and disinfestation of tools are useful, they are inadequate for stopping disease spread once a crop has been infected. During the blight outbreak in Hawaii, some chemicals and antibiotics were used with limited success during the early stages, but these control methods were later abandoned because of the high cost and the development of streptomycin-resistant strains (Nishijima, 1988). Growing plants under plastic or in glasshouses and irrigating via drip irrigation, rather than an overhead or sprinkler system, was shown to reduce the spread of bacteria through water splashing and aerosol spraying and significantly reduced the incidence of blight in anthurium seedling culture. Growing plants under cool and shaded conditions also slowed disease progress. Inoculated plants exposed to temperatures greater than 31.0°C (87.8°F) were found to be more susceptible to disease than inoculated plants exposed to temperatures of 26.0°C (78.8°F) and lower (Alvarez et al., 1990; Fukui et al., 1999b). This rather small difference in temperature can be regulated in commercial shadehouses by strategically increasing airflow.

Differential susceptibility of anthurium cultivars to foliar and systemic stages of blight was quantified in studies using a bioluminescent strain of *X. axonopodis* pv. *dieffenbachiae* (Fukui et al., 1996, 1998). Crossing *A. antioquiense* Engl. with *A. andraeanum* produced tolerant offspring (Fukui et al., 1998; Kamemoto and Kuehnle, 1996). Additional insights on the genetic basis for resistance have come from employing a transgenic strain of *X. axonopodis* pv. *dieffenbachiae* expressing GFP in infected tissue, enabling detailed and thorough comparisons of tolerant and susceptible cultivars in breeding studies (Elibox and Umaharan, 2007a, 2007b, 2008a, 2008b, 2010).

Breeding plants for tolerance through traditional means is time consuming, so genetic engineering serves as a means of introducing resistance genes from nonplant origins into anthurium plants. *Agrobacterium*-mediated gene transfer was used successfully to transform anthurium plants by using genes that encode the antibacterial peptides attacin and cecropin, isolated from the cecropia moth (*Hyalophora cecropia* L.) (Kuehnle et al., 2004a). Transgenic anthurium plants expressing attacin were less susceptible than nontransgenic plants and were not as extensively colonized by *X. axonopodis* pv. *dieffenbachiae* (Kuehnle et al., 2004b, 2007). Although efforts to produce tolerant transgenic anthurium plants continue, no resistant transgenic cultivars have been released for commercial production.

The use of beneficial organisms, natural or modified, to reduce disease incidence was proposed to anthurium growers during the initial stages of the bacterial blight epidemic in Hawaii (Alvarez et al., 2006). Bacteria were isolated from the guttation fluids of susceptible anthurium cultivars (i.e., Marian Seefurth and UH1060) that resisted infection by *X. axonopodis* pv. *dieffenbachiae* under high inoculum pressure (Fukui et al., 1999c). When applied individually, the beneficial bacteria were not effective in suppressing multiplication of *X. axonopodis* pv. *dieffenbachiae* in guttation fluids, but when applied in combination, they were effective (Fukui et al., 1999a). Foliar applications of the bacterial community reduced infection of anthurium leaves through hydathodes and wounds (Fukui et al., 1999c). Using a combination of bacteriological tests, fatty acid analysis, and 16S ribosomal DNA (rDNA) sequence analysis, the strains were identified as *Sphingomonas chlorophenolica* Nohynek et al.; *Microbacterium testaceum* (Komagata & Iizuka) Takeuchi & Hatano; *Brevundimonas vesicularis* (Büsing et al.) Segers et al.; and *Herbaspirillum rubrisulbalbicans* (Christopher & Edgerton) Baldani et al. All four species survived on the surfaces of microplants up to 2 months following spray inoculation and were effective in protecting microplants against infection (Vowell, 2009).

When applied at weekly intervals, the consortium of beneficial bacteria was shown to enhance the growth of microplants (Toves, 2008). Striking differences were observed when susceptible cultivar Rudolph was inoculated with *X. axonopodis* pv. *dieffenbachiae* and then treated (sprayed weekly) or not treated with the biocontrol agents. Nontreated plants developed typical blight symptoms 12 weeks after inoculation with the pathogen and died. In contrast, 75% of the treated plants survived and produced flowers after 22 weeks. In another study, researchers demonstrated that biological control could be used simultaneously with genetically modified anthurium cultivars. Cultivars Paradise Pink and Mauna Kea, which had been engineered to express the Shiva-1 lytic peptide, did not inhibit the four species of beneficial bacteria (Fujii et al., 2002).

The effectiveness of biological control agents was shown to be enhanced by adding selected amino acids to the formulation (Toves, 2008). Treatments were most effective in glasshouses, where weekly sprays on microplants and 3- to 10-month-old plants could be controlled. Weekly spraying of 1- to 2-year-old plants in shadehouses reduced the disease incidence only by 10–40% in repeated field trials (Toves, 2008). Another endophytic bacterium—*Bacillus amyloliquefaciens* (ex Fukumoto 1943) Priest et al. strain B014—has been evaluated as a potential biocontrol agent for control of bacterial blight (Shu-Bin et al., 2012). This plant-associated bacterium produced anti-*X. axonopodis* pv. *dieffenbachiae* metabolites and decreased both the lesion size and the percentage of leaves with lesions when applied to anthurium plants prior to inoculation with the pathogen. Strain B014 of *B. amyloliquefaciens* also induced an increase of defense-related enzymes (i.e., phenylalanine ammonia lyase, peroxidase, and polyphenol oxidase), making it a very interesting topic for further studies of biological control.

## • *Xanthomonas oryzae* pv. *oryzae*

*X. oryzae* pv. *oryzae* (ex Ishiyama) Swings et al. is the causal agent of bacterial blight on rice (*Oryza* L.) and one of the most prevalent bacterial diseases of a major crop species. The disease, which is endemic in most tropical rice-growing areas, can be controlled through the use of genetic resistance and cultural practices. Outbreaks continue, however, because of the development of adapted strains, the introduction of susceptible cultivars, and the expansion of rice-growing areas (Verdier et al., 2012b).

Strains of *X. oryzae* pv. *oryzae,* both regional and local, are often grouped into races based on now-classic incompatibility assays that identify various plant lines with different combinations of resistance (*R*) genes. From a phylogenetic basis, three main lineages of *X. oryzae* pv. *oryzae* are known to exist, including the strains that have emerged in western Africa and those that have been characterized in the United States, which are of low virulence (Gonzalez et al., 2007; Triplett et al., 2011). The pathogenicity of *X. oryzae* pv. *oryzae* is dependent on many of the same virulence factors that have been identified for other xanthomonads, including

the requirement of a T3SS (Ryan et al., 2011; White et al., 2009). Four T3 effectors from *X. oryzae* pv. *oryzae* have been characterized that contribute to *R* gene specificity and in part to the race classification schemes. These so-called TAL effectors are orthologs of AvrBs3 (White and Yang, 2009) (see Chapter 8). In addition to TAL T3 effectors, strains of *X. oryzae* pv. *oryzae* contain a complement of diverse T3 effectors, and losing at least a few of these effectors has been shown to reduce virulence (Song and Yang, 2010). As occurs in other pathosystems, the effectors appear to target host immunity signaling pathways, suppressing host defense responses to infection. Specific targets are being identified by new investigations into this disease system (Song and Yang, 2010; Yamaguchi et al., 2013a, 2013b).

The characterization of T3 effectors from *X. oryzae* pv. *oryzae* has revealed several interesting features. For instance, *X. oryzae* pv. *oryzae* has a critical requirement for at least one major TAL effector (Antony et al., 2010; Yang et al., 2006; Yu et al., 2011). When this requirement is not met, strains have extremely low virulence and are unable to spread from the site of infection (Yang et al., 2006). The TAL effectors AvrXa7 and PthXo1 of *X. oryzae* pv. *oryzae* serve as excellent examples of the interesting vagaries of the pathogen. Both effectors contribute to virulence. Whereas AvrXa7 triggers incompatibility on plants containing the dominant *R* gene *Xa7,* strains that rely solely on PthXo1 are incompatible on hosts that are homozygous for the recessive *xa13*. Yet both effectors are required for full virulence of strains that rely solely on either effector in susceptible plant lines (Antony et al., 2010; Yang et al., 2006). Extrapolating from the genetic evidence, all strains of *X. oryzae* pv. *oryzae* appear to require at least one major TAL effector for compatibility, of which AvrXa7 and PthXo1 are examples (Yang and White, 2004). The low virulence of the U.S. isolates of *X. oryzae* pv. *oryzae* may be attributable in part to the lack of a major TAL effector gene (Verdier et al., 2012a).

Five major TAL effectors for bacterial blight—PthXo1, PthXo2, PthXo3, AvrXa7, and TalC—have been identified in *X. oryzae* pv. *oryzae,* and some strains have two major TAL effectors (Yang and White, 2004; Yu et al., 2011). PthXo1 binds to a specific effector-binding element (EBE) in the promoter region of the gene *Os8N3* and directs expression of the gene, which is also known as *OsSWEET11* (Anthony et al., 2010). AvrXa7, PthXo3, and TalC target EBEs in a closely related gene named *Os11N3,* or *OsSWEET14* (Antony et al., 2010; Yu et al., 2011).

The product of *OsSWEET11,* along with other orthologs, was recently shown to function as a host sugar transporter, and more specifically, the members of the clade to which the protiens OsSWEET11 and OsSWEET14 belong appear to transport sucrose preferentially (Chen et al., 2010, 2012). Whether sucrose or sucrose transport is required for bacterial blight development or whether a physiological response to sucrose or SWEETs is required remains to be shown. A hypothesis has been proposed that implicates a function in transporting copper and reducing copper toxicity by the SWEET transporters (Yuan et al., 2010). Nevertheless, the use of alleles of *xa13* in traditional breeding programs for resistance to bacterial blight attests to the practicality of targeting SWEET for disease control in the field (Sanchez et al., 2000).

The introduction of promoter mutations in the EBEs of *Os11N3* by TAL/FokI nuclease fusion technology resulted in the isolation of rice plants with recessive resistance to bacterial strains that are dependent on AvrXa7 or PhXo3 for virulence (Antony et al., 2010; Li et al., 2012). Recessive mutations have also been identified in the promoter of another recessive *R* gene (*xa25*) and SWEET homolog in rice (Liu et al., 2011). Bacterial blight, therefore, has both gene-for-gene *R* gene incompatibility attributes and gene-for-gene susceptibility (*S*) gene compatibility attributes of strain behavior.

Interestingly, an α-ketoglutarate transport protein has been shown to be transported by the T3SS of *X. oryzae* pv. *oryzae* and localized to the host cell membrane (Guo et al., 2012). Observing reduced growth of the *kgtP* mutant strain of the pathogen on both synthetic media and rice led researchers to propose that the protein functions both in the secretion of α-ketoglutarate or another organic acid from the host and in its uptake by the pathogen (Guo et al., 2012).

Resistance to bacterial blight can also involve nuclear signaling events. The XA21 receptor kinase, which is a PAMP-type receptor, spans the host cell membrane, and upon binding the cognate elicitor, the intracellular domain is cleaved from the protein, translocating the approximately 70-kilodalton (-kDa) fragment (here, $XA21^{C70}$) to the nucleus (Park and Ronald, 2012). Furthermore, Xa21-mediated resistance has been shown to require nuclear translocation of the C-terminal fragment (Park and Ronald 2012). In the nucleus, $XA21^{C70}$ interacts with the OsWRKY62 transcription factor. OsWRKY62 is a repressor of XA21-mediated resistance, and a model is emerging that describes the nuclear translocation of XA21 and the release of defense gene suppression (Park and Ronald, 2012; Peng et al., 2008).

Additional Xa21-like resistances have been identified in rice, and considerable interest awaits identification of the corresponding elicitors (Cao et al., 2007).

The T3 effector protein Xoo1488 (XopY) has been shown to bind to and inhibit phosphorylation of the cytoplasmic receptor kinase protein OsRLCK 185 in response to chitin (Yamaguchi et al., 2013a, 2013b). OsRLCK 185 mediates both peptidoglycan and chitin-elicited immunity response in rice and is phosphorylated by OsCERK1, an lysM-type PAMP receptor. In time, *X. oryzae* pv. *oryzae* may acquire effectors that target Xa21-like receptors.

## • *Xanthomonas campestris* pv. *campestris*

Black rot of crucifers, caused by *X. campestris* pv. *campestris,* is characterized by chlorotic to necrotic, V-shaped lesions and blackened veins on leaves. The pathogen invades the plant through hydathodes at the leaf margins and moves into the vascular tissues, interrupting water transport and causing general foliar necrosis, wilt, and stunt. Blackened vessels can be seen in cross sections of the stock, which later rots (hence, the name "black rot").

Since first being described by Pammel (1895) and Smith (1897, 1911), the pathogen has undergone four major name changes as it has been more fully characterized. In a comprehensive study based on DNA–DNA hybridization, Vauterin et al. (1995) emended the type species *X. campestris,* restricting it to six pathovars that affect crucifers: namely, *aberrans, armoraciae, barbareae, campestris, incanae,* and *raphani.* A wide variety of cruciferous hosts—including numerous vegetable crops, weeds, and ornamentals in the family Brassicaceae (also called "Cruciferae")—are affected by *X. campestris.* Whereas *X. campestris* pv. *campestris,* pv. *aberrans* Knosel, and pv. *incanae* (Kendrick & Baker) Dye are vascular pathogens, pv. *raphani* (White) Dye et al. and pv. *armoraciae* (McCulloch) Starr & Burkholder are mesophyllic pathogens. Mesophyllic pathogens invade the plant through the stomates and produce limited water-soaked spots and localized necrotic lesions on crucifers, as well as tomato and pepper.

Based on a host range study of representative strains, Fargier and Manceau (2007) concluded that *X. campestris* consists of only three pathovars—*campestris, incanae,* and *raphani*—which cause only three diseases: namely, black rot on cruciferous plants; leaf spot on hosts in the Brassicaceae and some of the Solanaceae; and bacterial blight on ornamental crucifers (i.e., *Matthiola* spp. and *Erysimum cheiri* (L.) Crantz (syn. *Cheiranthus cheiri* L.)). *X. campestris* pv. *barbareae* (Burkholder) Dye was nonpathogenic on the tested hosts, and the status of pv. *armoraciae* was questioned because the original strain from horseradish (*Armoracia rusticana* G. Gaertn., B. Mey., & Scherb.) could not be located (Fargier and Manceau, 2007).

Black rot disease has been reviewed periodically as new information has emerged on host range, epidemiology, disease management, and pathogen characterization (Alvarez, 2000; Schaad and Alvarez, 1993; Walker, 1952; Williams, 1980). In a 2013 review, Vicente and Holub summarized background information and covered research on the pathogen genome, secretion systems, virulence factors, and molecular aspects of host–pathogen interactions.

Black rot has been distributed worldwide through infected seed. Once the disease is established in a field, secondary spread occurs through wind-driven rain, aerosols, contaminated implements, sprinkler irrigation, and groundwater. The bacteria survive epiphytically on cruciferous weeds and alternate crops, such as lettuce (*Lactuca sativa* L.) (Alvarez et al., 1994; Schaad and Dianese, 1981; Williams, 1980). In temperate climates, the black rot pathogen may survive more than 2 years in plant debris and up to 6 weeks in soil. In the tropics, where successive crops of crucifers are grown without adequate rotation with a nonsusceptible crop, soil and plant debris are major inoculum sources. The importance of cabbage (*Brassica oleracea* L.) residues on the perpetuation of disease was confirmed in field studies using phage typing and serotyping to monitor the spread of *X. campestris* pv. *campestris* strains from various inoculum sources (Alvarez and Cho, 1978; Liew, 1979). Cabbage debris has also been confirmed as a source of inoculum in temperate zones, where black rot has caused major losses in cabbage production (Kocks and Zadoks, 1996).

When seed carries a low population of *X. campestris* pv. *campestris,* infected plants may show no symptoms while the pathogen spreads epiphytically throughout the seedbed. Foliar symptoms appear occasionally on infected transplants, but often, no symptoms appear until heads begin to form; at this time, symptoms appear on lower wrapper leaves. Researchers demonstrated the impact of symptomless spread in the seedbed using strain-specific monoclonal antibodies to trace the movements of different strains through the bed to transplant and finally to mature plants (Shigaki, 1996; Yuen et al., 1987). Low levels of seed contamination spread throughout the seedbeds in 6 weeks; no symptoms were observed until transplant, and at harvest, the entire crop was infected.

Smith (1911) observed differences in aggressiveness among *X. campestris* pv. *campestris* strains during

early studies of black rot. In later studies, Alvarez et al. (1994) noted that some aggressive strains produced a blight symptom (i.e., broad patches of necrotic tissue that resemble parchment), indicating that the pathogen had infected mesophyll tissue. Although black veins did not accompany this blight symptom in the early stages of infection, the bacteria entered vascular tissue and the plant developed symptoms more typical of black rot. Chen et al. (1994) changed a mild leaf-spotting pathogen into a more aggressive blight strain by moving a 5.4-kilobase (-kb) DNA fragment from the *X. campestris* pv. *campestris* type strain (ATCC 33913) into a mild mesophyllic strain of *X. campestris* pv. *armoraciae,* conferring the pathogen's ability to cause blight symptoms but not systemic movement. In contrast, Ignatov et al. (2007) found that mildly aggressive strains of *X. campestris* pv. *campestris* produced only flecks or dark-brown leaf spots. Such strains survive epiphytically on wild crucifer hosts and are prevalent along the California coast in the United States, but they do not become widely established in commercial crucifer fields.

The production of crucifer seed in dry, cool areas with a low risk of disease development has been a principal means of disease control (Williams, 1980). Seed testing and certification to ensure quality is the primary means of disease management. Chemical sprays with copper compounds, antibiotics, and other biocides are used when the disease develops despite the use of pathogen-free seed (Williams, 1980). Chemical controls are most effective when applied to the seedbed, where plants are vulnerable to epiphytic spread of the pathogen. For example, a single prophylactic spray and drench on cabbage seedlings with a systemic fungicide fosetyl-aluminum was shown to reduce black rot infection significantly, eliminating the need for foliar sprays after transplant (Mochizuki and Alvarez, 1996). Reducing or otherwise modifying irrigation in the seedbed also can reduce dissemination among transplants and lower disease incidence in the field (Roberts et al., 2007).

Disease management through the deployment of resistant cultivars has been hampered by the limited availability of sources of resistance to black rot. In one study, the cabbage cultivar Early Fuji served as a source of resistance in cabbage-breeding programs (Williams, 1980); however, many of the cabbage cultivars preferred as fresh-market crops are susceptible to black rot. Durable sources of resistance have been sought in cabbage and several other *Brassica* species. Significant advances have been made toward the identification of resistance genes and have been fully reviewed by Vicente and Holub (2013).

The heterogeneity of globally diverse strains of *X. campestris* pv. *campestris* has been shown by phage typing, serotyping, and genetic methods (e.g., restriction fragment length polymorphism (RFLP), AFLP, and rep-PCR (Alvarez et al., 1994; Jensen et al., 2010; Liew and Alvarez, 1981; Singh et al., 2011). Given the worldwide distribution of *X. campestris* pv. *campestris* through infested seed, genetically distinct groups cannot be reliably associated with the geographic origins of strains. Moreover, mixed infections commonly occur in fields planted over several years with seed from different origins.

In an analysis of xanthomonads and other plant pathogens based on a DNA marker designed from the *dnaA* RIF, researchers found that all but two of 152 strains of *X. campestris* tested were clustered in a major clade (E) and clearly distinguished from other *Xanthomonas* spp., indicating that RIF is a useful diagnostic marker for species identification (Schneider et al., 2011). Most of the *X. campestris* pv. *campestris* strains in clade E (92/102) were in a subgroup with the three fully sequenced *X. campestris* pv. *campestris* strains (ATCC33913, 8004, and B100) and with five strains of pv. *aberrans,* three strains of pv. *incanae,* five strains of pv. *raphani,* and 20 strains of pv. *armoraciae*. The RIF framework was similar to the tree produced by multilocus sequence analysis (MLSA) for *Xanthomonas* spp. (Young et al., 2008). Phylogenetic comparisons based on MLSA of 42 strains of *X. campestris* showed that the pathogen was polymorphic at eight loci and had high genetic diversity. Strains that induced black rot (pvs. *aberrans* and *campestris*) were closely related but still separate. Likewise, representative strains of pv. *barbareae* and pv. *incanae* were closely related but separate from strains of pv. *campestris* (Fargier et al., 2011).

Five races were initially described for *X. campestris* pv. *campestris* based on the abilities of specific groups of strains to infect different crucifer species (Kamoun et al., 1992). This classification was refined and additional races were described based on reactions of a different set of brassicas (Ignatov et al., 1998; Vicente et al., 2001, 2002), and new races (7, 8, and 9) were later described (Fargier and Manceau, 2007). In a broad geographical survey of crucifer production fields along the California coast, researchers showed how specific races corresponded to characteristic AFLP patterns and how different *X. campestris* pv. *campestris* populations were distributed among commercial farms (Ignatov et al., 2007).

LPSs are surface-associated virulence factors in *X. campestris* pv. *campestris* that are associated with the bacterial outer membrane and involved in PTI (Erbs

and Newman, 2012; Erbs et al., 2008). LPSs consist of membrane-anchored lipid A, a core oligosaccharide, and polysaccharide side chains (O-antigens) (Wicken, 1985) and induce host defense responses, such as pathogenesis-related gene expression, oxidative burst, and thickening of the plant cell wall (Dow et al., 2000; Newman et al., 2002). Variations in LPS composition allow the bacteria to evade host resistance mechanisms (Ojanen et al., 1993).

An LPS-specific antigen has been associated with blight-inducing strains of *X. campestris* pv. *campestris,* but the blight-associated epitopes and the DNA fragments from the strains are not required for blight to occur (Chen et al., 1994; Shigaki, 1996; Shigaki et al., 2001). Mutations in LPS gene clusters have been associated with reduced virulence and reduced capacity to withstand adverse environmental conditions (Dow et al., 1995; Kingsley et al., 1993; Newman et al., 2002). LPS synthesis in *X. campestris* pv. *campestris* is dependent on 15 genes of the *wxc* gene cluster (Vicente and Holub, 2013; Vorhölter et al., 2001). Differences in the compositions of LPS clusters between various pathogens might explain why some strains of *X. campestris* pv. *campestris* are predominant on certain hosts but have restricted ability to colonize other hosts.

Unique host–pathogen signaling mechanisms in *X. campestris* pv. *campestris* are encoded by *rpf* genes (Crossman and Dow, 2004). T3 effectors and ETI responses are also common to the xanthomonads (Büttner and Bonas, 2010; Kay and Bonas, 2009), and the role of T3 effectors in the virulence and fitness of *X. campestris* pv. *campestris* and related pathogens has been described. Mutations in core effectors alter the pathogenicity of xanthomonads, and the different repertoires of effectors confer host specificity among strains. Sequenced genomes are available of representative strains of *X. campestris* pv. *campestris* (i.e., ATCC 33913 (da Silva et al., 2002), 8004 (Qian et al., 2005), and B100 (Vorhölter et al., 2008)) and of strain 756C of *X. campestris* pv. *raphani* (Bogdanove et al., 2011), enabling the comparison of genomes (Vicente and Holub, 2013). Fundamental differences between *X. campestris* pathovars that colonize vascular tissues and those that are restricted to the mesophyll can be explained using these data.

As noted earlier, molecular aspects of host–pathogen interactions have been thoroughly covered in a review by Vicente and Holub (2013). Transcriptome profiling and further studies of functional and comparative genomics are providing new insights into the adaptation and evolution of pathogens, as well as factors that contribute to virulence in the xanthomonads (Liu et al., 2013; Ryan et al., 2011).

## CONCLUDING REMARKS

The genus *Xanthomonas* represents a diverse and expanding range of bacterial species, and new (or at least previously considered) minor pathogens have emerged as current threats. In two host species (i.e., citrus and tomato), at least three phylogenetically distinct groups target the same host and, as is true for most xanthomonads, require a functional Hrp-T3SS and accompanying effectors. *X. albilineans* does not contain an Hrp-T3SS and relies on other strategies, including toxin production for disease. Genus-specific genes reflect the constant adaptive forces at work that enhance and limit the spread of strains to new niches.

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