



Review

# Citrus Canker—Distribution, Taxonomy, Epidemiology, Disease Cycle, Pathogen Biology, Detection, and Management: A Critical Review and Future Research Agenda

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**Abstract:** *Xanthomonas citri* subsp. *citri*, a causative agent of the citrus canker (CC) disease, belongs to one of the essential groups of the bacterial phytopathogen family, *Xanthomonadaceae*. It has been a potential threat to the globally significant citrus fruit crop, which has remained under investigation for disease management and epidemiology since the 1980s. In Pakistan, the average yield of citrus is 11 t/ha, which is lower than other countries, including China, Brazil, and India, having average productions of 27, 26, and 22 tons/hectare, respectively. Citrus canker is one of the most devastating diseases, posing a significant threat to crop yield and fruit quality. To date, five distinct types (or forms) of the citrus canker have been recognized; the Asiatic (Canker A) form is most destructive and affects most citrus cultivars. Severe infection outcomes include dieback, defoliation, severely blemished fruit, premature fruit drop, and reduced fruit quality. The infection increases under humid, warm, cloudy climate, wind, and heavy rainfall. The analysis of plasmid and chromosomal DNA of *X. citri* subsp. *citri* depicted an evolutionary relationship among pathovars of *Xanthomonas*. The extensive study on the genome of *X. citri* subsp. *citri* has contributed to the current knowledge of plant host recognition of pathogens, host specificities, dissemination, and propagation. Regulatory programs, i.e., quarantine or exclusion, continued to be practiced, prohibiting infected citrus plant material into the existing stock. Other measures include removal of inoculum sources, resistant hosts, protective copper-containing sprays, and windbreak systems. In this review, we explored the latest trends in the areas of epidemiology, pathogenome, detection, host–pathogen interaction, biofilm formation, and management of *X. citri* subsp. *citri*.

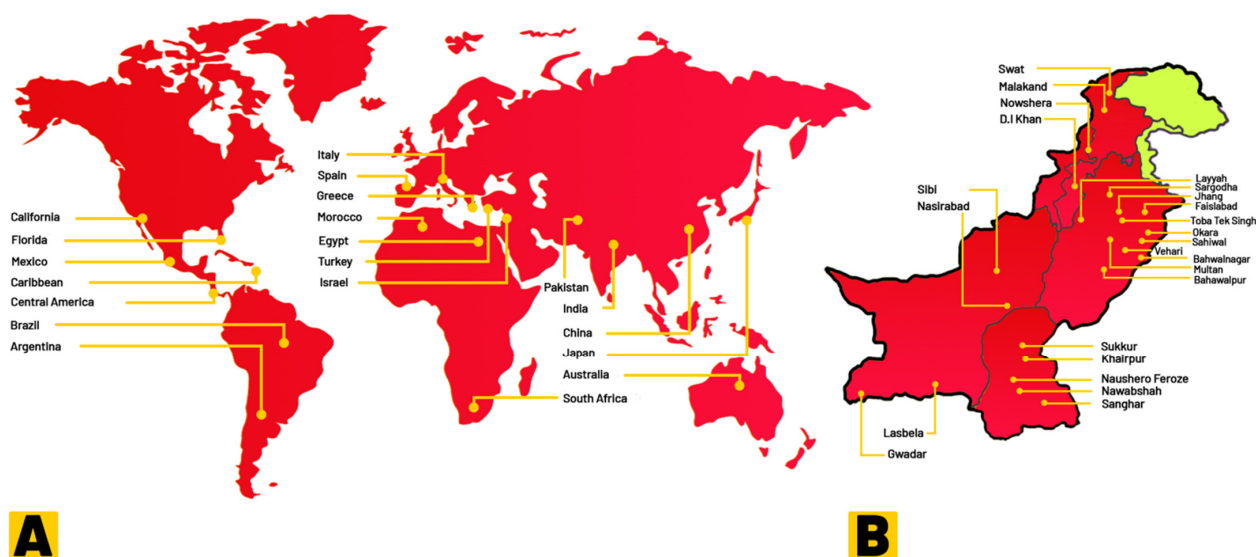
**Keywords:** *Xanthomonas citri* subsp. *citri*; bacterium; plant pathogenic; local infection; industry; economic importance; management

## 1. Introduction

Citrus is one of the world's major fruit crops (second to bananas), with more than 200,400 hectares cultivated and an annual production of 158 million tons. Globally, China produces the most citrus fruits [1]. As of 2020, citrus fruit production in China amounted to 44.6 million tons, accounting for 28.21% of the world's citrus fruit production, with Brazil, India, Mexico, and the USA rounding up the top 5 countries (accounting for 59.45% of citrus fruit production) [2]. Pakistan is one of the largest citrus-producing countries, ranking 13th in citrus fruit production; Pakistan's total citrus fruit production (primarily Kinnow) is approximately 2.0 million metric tons annually. Although there is no remarkable increase in citrus production which has increased 30.8% since 1991–1992 [1]. In 1991–1992, Pakistan produced 1.62 million tons of citrus, which increased to 2.1 million tons in 2008–2009 and 2.4 million tons in 2014–2015. In 2020, citrus fruit yield for Pakistan was 115,554 hg per ha; though Pakistan citrus fruit yield fluctuated substantially in recent years, it increased from 1971 to 2020, with 115,554 hg per ha in 2020 [1]. Pakistan's citrus fruit production increased from 1993 to 1994; however, it started to decline in 1999 [3]. Citrus fruit crop requires a critically low temperature for its ripening, which, if not achieved, may lead to a decline in the production of fruit [3]. Therefore, one of the reasons for varied citrus fruit production might be the temperature variations in the citrus-growing areas of Pakistan [3]. Such an excellent temperature variation was recorded in 2006–2007 in citrus-producing regions, due to which, citrus production dropped from 2.4 to 1.4 million tons; however, the area under citrus fruit orchards remained the same [3]. In Pakistan, the average yield of citrus is 11 t/ha, which is lower than other countries, including China, Brazil, India, Mexico, and the USA, having average productions of 27, 26, 22, 21, and 20 tons/hectare, respectively [3]. Pakistan exports about 533,000 tons of citrus annually to Saudi Arabia, Kuwait, Dubai, Bahrain, Qatar, Netherlands, Oman, UK, Indonesia, Malaysia, Singapore, and Russia [3]. New citrus hybrids are being developed to produce delectable, juicy, and seedless fruits. Citrus fruits and juices contain carbohydrates, fiber, vitamin C, low fats, potassium, calcium, folate, thiamine, vitamin B6, niacin, vitamin A, magnesium, phosphorus, copper, flavonoids, riboflavin, limonoids, lignin, polysaccharides, fiber, carotenoids, and phenolic compounds [4]. These substances contribute to different pharmacological effects, e.g., anti-microbial, anti-oxidant, anti-cancer, cardiovascular, central nervous system, anti-inflammatory, anti-diabetic, reproductive, gastrointestinal, immune-logical, respiratory, obesity, and many others [4]. Pakistan has a very short product lifespan for citrus plants, 20–30 years compared to 50 years in other countries [3]. Lack of information regarding management practices, such as low doses of fertilizers and inter-cropping with wheat, maize, fodders, and other crops in orchards are responsible for low production and short lifespans [3]. In Pakistan, citrus fruit is predominantly cultivated in four provinces, namely: Punjab, Khyber Pakhtunkhwa (KPK), Sindh, and Baluchistan, where the Punjab province, according to the Pakistan Horticulture Development and Export Company [5], produces more than 90% of the total Kinnow production; KPK mainly produces oranges among all citrus fruits in the country [6]. Sargodha, Toba Tek Singh, and Mandi Bahauddin are three districts known for their citrus production in the Punjab province. Mandarins (Feutrell's Early and Kinnow) and sweet orange (Mausami or Musambi and Red Blood) are important among all the citrus varieties cultivated in Pakistan [3,6] (Figure 1).

Citrus production is constrained due to numerous diseases, insect pests, nutritional imbalances, improper cultural practices, and sudden climatic changes [7]. Numerous diseases, e.g., gummosis, citrus canker, citrus tristeza virus, citrus greening, and citrus tree decline, attack citrus plants, hampering their production, causing heavy economic losses, and socially impacting growers, consumers, and the industry [3,7]. Citrus canker is a bacterial disease that affects all commercial citrus cultivars; there is no cure to minimize pathogen spread in the field. Control strategies are limited to the application of copper-based compounds and the removal of diseased trees [8]. Several plants from the cultivated family, Rutaceae [9], particularly the *Citrus* spp., i.e., *Fortunella* and *Poncirus* spp., are infected by *Xanthomonas citri* subsp. *citri* [10]. The aerobic bacterium requires maximum

growth temperatures of 35–39 °C and optimal temperatures of 28–30 °C [11,12]. A wide range of virulence factors is included in CC development, such as structures for surface attachment, enzymes for degradation of the cell wall, a few secretion systems and their effectors, and the diffusible signal factor (DSF), which mediates the quorum sensing (QS) system [13]. *Xanthomonas citri* subsp. *citri*, a member of *Xanthomonadaceae* family, is one of the largest and most important groups of bacterial phytopathogens; it has been used as a model organism for pathogenesis and the phylogeny study, and is the causative agent of citrus canker (CC) disease, which has been the subject of extensive research in terms of epidemiology and management [3,7]. There is controversy over the geographical origin of citrus canker, and it is assumed *Fortunella hindsii* may have been a wild host plant in southern China [14]. Yet, some scientists reported that citrus canker originated in India; citrus canker was found in the oldest citrus herbarium of Herbaria of the Royal Botanical Gardens, England [15]. It is assumed that the disease originated first in tropical regions, such as South China, Indonesia, and India. In 1910, in Florida, the disease was first identified and transported through infected nursery stock imported from Japan in the nineteenth century, and spread throughout the southeastern US [16,17]. The disease also occurred in South America [18], South Africa [19], and Australia [20] earlier this century. In southern Iran, an atypical strain, XAC, was discovered, which showed extreme virulence on Mexican limes, grape fruits, and sweet oranges [21]. In Taiwan, citrus bacterial canker was first reported in 1932 [22]. Citrus canker is prevalent in over thirty citrus-growing countries in Asia, the Pacific, Indian Oceans, South America, and in the southeastern US [23]. The transportation of fruit from an infested zone to a production area free of disease imposes trade restrictions under regulations [24]. The causal agent is considered a quarantine organism in citrus-producing areas of Europe, where canker has not been reported so far. Exclusion or quarantine practices for *X. citri* subsp. *citri* are still being refined wherever citrus is grown worldwide, while new methods and tools for managing and eradicating CC are being developed [25]. The current review presents recent developments in the research of *X. citri* subsp. *citri* and CC, including taxonomy, distribution, epidemiology, disease cycle, pathogen biology, detection, and management.



**Figure 1.** (A) World citrus production areas and (B) Pakistan's various districts participating in world citrus production. In Pakistan, citrus fruit is predominantly cultivated in four provinces.

## 2. Taxonomy of Citrus Canker Bacterium

Citrus canker, also known as Asiatic CC, was initially reported on in the United States of America in the early 1900s following an outbreak in numerous southeastern states [26]. In 1914, Hasse received samples from Florida, Texas, and Mississippi, and was able to isolate

the bacterium [27]. After completing characterization and pathogenicity tests, Hasse called the bacterium *Pseudomonas citri* [27]. Since then, the bacterium has been classified into several genera, including *Bacterium*, *Phytomonas*, and, finally, *Xanthomonas citri* in 1939 [8,28,29]. *Xanthomonas* genus consists of 27 phytopathogens that cause critical diseases in ornamental plants and other crops [8]. The genus has a broad range of 68 host families, over 240 genera and 140 different pathovars [30]. The genus *Xanthomonas* can infect more than 350 species, including 268 dicots and 124 monocots, including grains, fruits, nuts, and plants belonging to *Brassicaceae* and *Solanaceae* families [8]. The strains of *Xanthomonas citri* have been assigned to the A strain within this species to show that they are linked to Asiatic CC [8]. Two more CC-producing *Xanthomonads* were discovered in the 1970s and were first classified as group C strains, which induce canker lesions solely in key lime (*Citrus aurantifolia*), and group B strains, which have a broader host range [31,32]. CC bacterium continued as *X. citri* until 1978; in the same year, 1978, Dye placed *X. citri* in *X. campestris* pv. *citri* to uphold *citri* at the infra subspecific level [33]. CC bacterium was again reassigned the title of *X. citri* by [34], while the B and C strains were placed in *X. campestris* pv. *aurantifolii*. Reference [35] disagreed with previous research and argued that more work was needed to place CC strains in *X. citri* and suggested A, B, and C strains continued as *X. campestris* pv. *citri*; then [36] performed research using DNA–DNA hybridization (DDH) based on renaturation rates with a diverse array of *Xanthomonas* strains, recommending strain A to *X. axonopodis* pv. *citri* and B and C to *X. axonopodis* pv. *aurantifolii*, respectively [7,8]. The research on CC bacterium taxonomy continued and, Ref. [37] again, using the S1 nuclease DNA–DNA hybridization technique, the researchers recommended *X. axonopodis* pv. *citri* strains in *X. smithii* subsp. *smithii* and the *X. axonopodis* pv. *aurantifolii* strains in *X. fuscans* subsp. *aurantifolii*, although the placement of strains in *X. smithii* after due deliberations was later considered illegitimate and was agreed upon by the previous legitimate proposal by [34]. Hence, the authors of reference [38] published an erratum “Emended classification of *Xanthomonad* pathogens on citrus” in systematic and microbiology and recommended the placement of strains in *X. citri*. Finally, the authors of reference [39] formally validated *X. citri* as *X. citri* subsp. *citri*. Reference [40] proposed important modifications to the taxonomy of *Xanthomonads*, including within *X. citri*, recommending the addition of several pathovars within *X. axonopodis*, as well as the placement of members of *X. fuscans*, into *X. citri*, using a polyphasic approach that included a multilocus sequence analysis (MLSA), a DDH calculation of whole-genome average nucleotide identity values, and phenotypic analyses. As a result, it has been suggested that *X. fuscans* subsp. *aurantifolii* be transferred to *X. citri* as *X. citri* pv. *aurantifolii* [40]. The authors submitted their recommendations for these adjustments to the *International Journal of Systematic and Evolutionary Microbiology*, which were accepted; from now on, the prokaryotic names *X. citri* subsp. *citri* (XCC) and *X. fuscans* subsp. *aurantifolii* (XFA) will be used in nomenclature for bacteria that cause CC [40]. The bacterium was gram-negative, rod-shaped, and polar flagella. In contrast, colonies of bacterium showed yellow colors on petri plates due to the presence of a carotenoid pigment called Xanthomonadin. Because of exopolysaccharide (EPS), it is known as xanthan, showing a glossy appearance, invitro [40]. The classification of bacterium consists of kingdom: Prokaryote, phylum: proteo-bacteria, class: Gamma-proteobacteria, order: *Xanthomonadales*, family: *Xanthomonadaceae*, genus: *Xanthomonas*, specie: *citri*, and subsp.: *citri* [7] (Table 1).

**Table 1.** Citrus canker bacterium A strain classification details, from the start of the studies.

Sr. No.	Genus	Specie	*f.sp./*pv/subsp.	Year	Reference
1.	<i>Pseudomonas</i>	<i>citri</i>	not reported	1915	[27]
2.	<i>Xanthomonas</i>	<i>citri</i>	not reported	1915	[27]
3.	<i>Bacterium</i>	<i>citri</i>	not reported	1916	[28]
4.	<i>Bacillus</i>	<i>citri</i>	not reported	1920	[41]
5.	<i>Phytomonas</i>	<i>citri</i>	not reported	1923	[42]
6.	<i>Xanthomonas</i>	<i>citri</i>	not reported	1939	[29]
7.	<i>Xanthomonas</i>	<i>citri</i>	<i>aurantifolia</i>	1972	[43]



Table 1. Cont.

Sr. No.	Genus	Specie	*f.sp./*pv/subsp.	Year	Reference
8.	<i>Xanthomonas</i>	<i>campestris</i>	<i>aurantifolia</i>	1978	[33]
9.	<i>Xanthomonas</i>	<i>campestris</i>	<i>citri</i>	1980	[44]
10.	<i>Xanthomonas</i>	<i>citri</i>	<i>aurantifolia</i>	1989	[34]
11.	<i>Xanthomonas</i>	<i>axonopodis</i>	<i>citri</i>	1995	[36]
12.	<i>Xanthomonas</i>	<i>smithii</i>	<i>citri</i>	2005	[37]
13.	<i>Xanthomonas</i>	<i>citri</i>	<i>citri</i>	2006	[38]
14.	<i>Xanthomonas</i>	<i>citri</i>	subsp. <i>citri</i>	2007	[39]
15.	<i>Xanthomonas</i>	<i>citri</i>	subsp. <i>citri</i>	2016	[40]

\*f.sp. stands for forma special and \*pv for pathovar (classification of a pathogen beyond sub specie levels). Subsp.: Sub specie.

### 3. Phylogenetically Distinct Groups of CC

*X. citri* subsp. *citri* and *X. citri* subsp. *aurantifolia* have been further divided into subgroups based on their significant differences in the host range, which is also a reason for true pathogenic variants [34,45]. It was reported that the division of these groups based on citrus host type and symptoms was made on bacterial isolation on various nutrient media [31].

#### 3.1. Asiatic Citrus Canker Strains

The most important and widespread pathovar is the Asiatic-canker (also named cancrasis-A or true-canker), *X. citri* subsp. *citri* A strain is the most virulent and has a wide host range, including cultivars of citrus [46]. South-West Asian strains *X. citri* subsp. *citri* A are relatively less widespread [7]. Most recently, in Florida, at one location, a third pathogenic strain was found, which was designated Aw, apparently of Asiatic origin [47].

#### 3.2. South-American Canker Strains

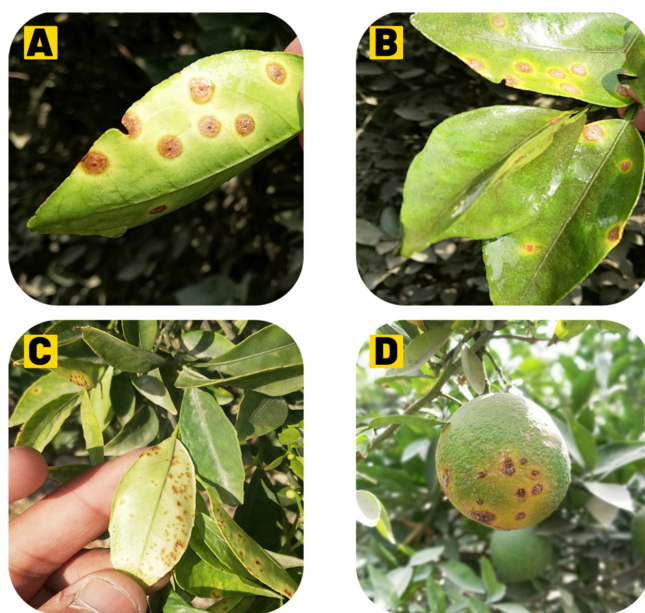
There are two types of South American canker strains which causes the same symptoms on the susceptible host as those produced by *X. citri* subsp. *citri* A strains, but all strains of South America have narrow host ranges [47]. *X. citri* subsp. *aurantifolia* B strain, also referred to as false canker or cancrasis B, has a more restricted host range and is found to primarily infect lemons and limes [32]. The B strain (XAUB as an acronym, XAC pathotype B; XAC-B) first appeared in Argentina in 1923 and it eventually extended to nearby Uruguay and Paraguay [32,33]. The B strain generally causes severe infections on lemon fruits (*Citrus limon*), limes (*C. aurantiifolia*), sour oranges (*C. aurantium*), but seldom on sweet oranges (*C. sinensis*) and pummelo fruit (*C. maxima*). Moreover, this strain does not infect grapefruit (*C. paradisi*) [30,32]. Hence, the B strain is not present in nature longer. Mexican lime cancrasis, CBC-C (XAC type C; XAC-C) or the C strain (XAUC as an acronym) was discovered in 1963 and present only in São Paulo, Brazil, where it just infects the Mexican lime [48]. The B and C strains are currently classified as *X. axonopodis* subsp. *aurantifolia* and produce many similar symptoms on the host as produced by the canker A strain [49,50]. The strains XAC, XAUB, and XAUC were compared and analyzed phenotypically and phylogenetically; all three strains were shown to have polar flagella with noticeable motility when cultured in semi-solid media [51]. In the presence of maltose and aspartic acid, only XAC can grow and hydrolyze pectate and gel [52]. Polyclonal antisera were prepared against XAC, but XAUB and XAUC showed little or no affinity to antisera. In contrast, XAC is susceptible to CP1 and CP2 bacteriophages, and XAUB and XAUC are not affected by these bacteriophages [53]. It was observed in culture media that XAUB has fastidious growth; XAC and XAUC both grow well in nutrient-agar (NA) and tryptophan–sucrose agar media. Moreover, these three strains show good growth in glutamic-acid rich media. A molecular analysis confirmed that XAUB and XAUC are more interlinked with one another than XAC [54–56]. In contrast, data obtained from physiological tests, i.e., phage-typing [57] total protein profiles after SDS-PAGE, DNA–DNA solution hybridizations [56,58], plasmid–DNA fingerprints [59], plasmid-based hybridization probes [59], PCR assay [60], DNA fragment sequence of gene *hrp*, restriction enzymes to analyze DNA fragments [61] confirm

these conclusions. Furthermore, a gene required to cause CBC symptoms is the *pthA* gene, which was obtained from the XAC-A strain [62,63]. Total DNA hybridization with a *pthA* fragment revealed several profiles among XAC-A, XAUB, and XAUC; no hybridization with strains of *X. axonopodis* pv. *citrumelo* was observed [10].

Provisionally, two more CC strains were classified, named D and E strains, but now they do not exist or are categorized differently [64]. The D strain, which is also referred to as bacteriosis, induces disease in limes in Colima (Mexico), but its etiology is not confirmed yet [65,66]. This disease causes typical leaves and twig lesions, but no symptoms are observed on key Mexican lime fruit [67]. In this area, the suspected citrus pathogen no longer exists. It is now believed that the disease was caused by *Alternaria limicola* [68,69]. The second pathogen was the E strain, previously identified as a citrus canker in a Florida nursery. The disease is 'called' a bacterial spot of citrus produced by *X. axonopodis* subsp. *citrumelo* [34,50,70]. It can be distinguished based on these studies that three groups of *X. axonopodis* strains, i.e., A strain, B strain (including C and D strains), cause citrus cankers [50] and, notably, these strains have controversial taxonomy [71]. This interpretation was confirmed when the *Xanthomonas* genus was reclassified based on DNA–DNA hybridization and metabolic activity studies [7,8,10]. Moreover, *Xanthomonas*, causing diseases on citrus, was transferred from *X. campestris* to *X. axonopodis* species. Perhaps now, pathotype A, pathotypes B and C, and the CBS strains of *X. axonopodis*, are named *X. axonopodis* subsp. *citri*, *X. axonopodis* subsp. *aurantifolii*, and *X. axonopodis* subsp. *citrumelo*, respectively, but the subcommittee on the taxonomy of plant pathogenic bacteria did not support this proposal [7,8,71].

#### 4. Symptomatology

Canker symptoms are observed in all aerial parts of the plant [72]. They are characterized mainly by the formation of erumpent, corky, and raised pustules on the surface of leaves, fruits, and twigs, which serve as sources of bacterial inoculums [72]. Defoliation and fruit drop are also observed as plant responses to the infection [73,74]. Notably, *Xanthomonas citri* can survive in such plant debris for two months [75]. Severe symptoms are produced on trifoliate oranges, grapefruit, Mexican lime, and some sweet oranges; however, the actual host range depends primarily on a strain of citrus canker [74]. Generally, the susceptibility of young tissues to the citrus canker is more than mature tissues as there is a period of vulnerability in each flush around three times a year [76] (Figure 2).



**Figure 2.** (A) Raised, corky, and sunken lesions on the upper side of the leaf. (B) Lesions on the lower side of the leaf. (C) Initial lesions on the lower surface of the leaf. (D) Canker symptoms on the fruit.

#### 4.1. Leaf Lesions

CC bacterium naturally penetrates the host tissues through stomata [77], hydathodes, lenticels, or wounds [78]. Citrus canker disease symptoms first appear as tan, brown, or grey-oily circular lesions, 2 to 10 mm in size, depending on the susceptibility of the host, the number of cycles of the infection, and optimal environmental conditions, i.e., the presence of water film and 20 to 30 °C temperature; canker protrudes from both surfaces of leaf tissue around 4–7 days after inoculation [78]. Symptoms might appear after more than 60 days under optimum conditions [79,80]. As the disease advances, host cell expansion (hypertrophy) and cell division (hyperplasia) occur, due to which the lesions become visible from small water-soaked spots and are surrounded by a yellow halo, which turns into slightly raised blister-like lesions and can be viewed with transmitted light [74,80]. The hyperplastic mesophyll tissue is an essential diagnostic symptom of the disease characterized by the formation of the canker due to rupturing the epidermis [78] and it releases abundant *X. citri* subsp. *citri* on the leaves. These lesions are elevated, are ‘corky’ in leaves, stems, and fruits, and then become dark and thick into the distinctive citrus canker under dry conditions [73]. A wound on the leaves or fruits or an injury by the Asiatic citrus leaf miner (*Phyllocnistis citrella*) significantly increases symptom severity [81–83].

#### 4.2. Fruit Lesions

Fruits are susceptible for 90–120 days when they grow between 2.0 and 6.0 mm in diameter, depending on citrus species [77]. The lesions in the early stages look similar to large oily glands on the peel and become progressively dark and corky in texture, usually circular, and may occur individually or in groups, leading to premature fruit fall [84].

#### 4.3. Twig Lesions

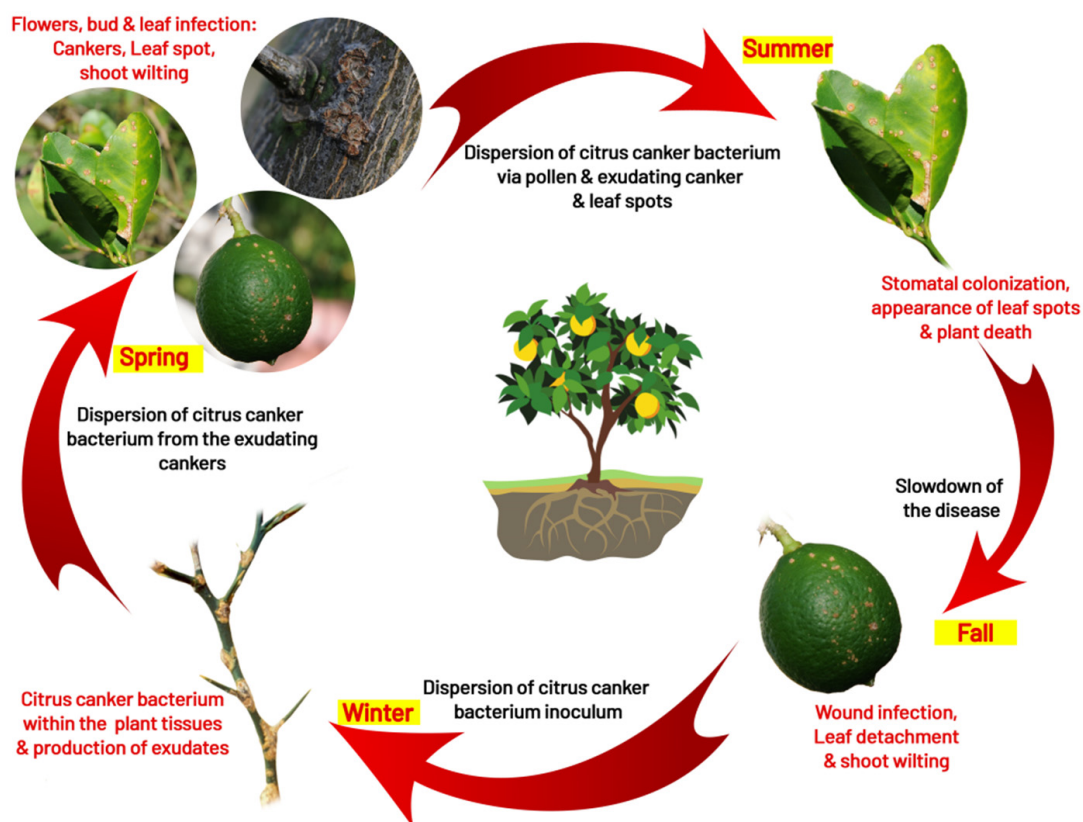
Twig lesions generally occur when leaves and fruits pass through one or more cycles of infection. Similar symptoms are produced on both twigs and fruits; twig lesions are not surrounded by chlorosis (but fruit lesions do) [24]. Citrus canker is endemic, the inoculum spreads by twig lesions on young shoots, and *X. axonopodis* subsp. *citri* survival is prolonged in these areas; lesions with raised corky patches may persist for many years until girdling infections do not kill the twigs [75]. The highest susceptibility of citrus to *X. axonopodis* subsp. *citri* infection is during the last half of the growth development phase in all of the above ground citrus tissues [85]. Lesion incidence is seasonal, but sometimes severe precipitation and high temperatures coincide with periods of flush growth [84]. As leaves, stems, and fruits are fully grown, they become resistant to infection; once leaves are expanded between 50 and 80%, they become most susceptible [86]. New flushes, tender leaves, and stems are more likely to be vulnerable to citrus cankers than fully grown citrus [85]. When a pathogen severely attacks the host, it leads to defoliation, dieback, early fruit drop, and tree decline; hence, infected fruit is less valuable or unmarketable [87,88].

### 5. Disease Cycle and Epidemiology

#### 5.1. Infection

The bacteria penetrate the host by disrupting the leaf epidermis, inducing cell hyperplasia, and colonizing the apoplast [89]. Under optimum conditions, the pathogen multiplies 3 to 4 log units per lesion; for further disease development, bacterial cells may emerge from stomata openings to provide inoculum within five days [75]. For successful infection and lesion formation, free moisture for 20 min is required for the bacterial cells to ooze out from the lesion. As a result of water congestion, one to two bacterial cells are released from stomatal openings during inoculation [77,90]. After the initiation of growth of the host, almost all infections take place on stems and leaves within the first six weeks, while the first 90 days after petals falling is the most crucial time period for fruit infection [90]. Small and unnoticeable pustules are formed due to infections after this time period [10]. It has been reported that fruits are more susceptible to disease than leaves;

hence, observations have been made that lesions of different sizes can be found on the rind of the same fruit during the infection of the bacterium [91] (Figure 3).

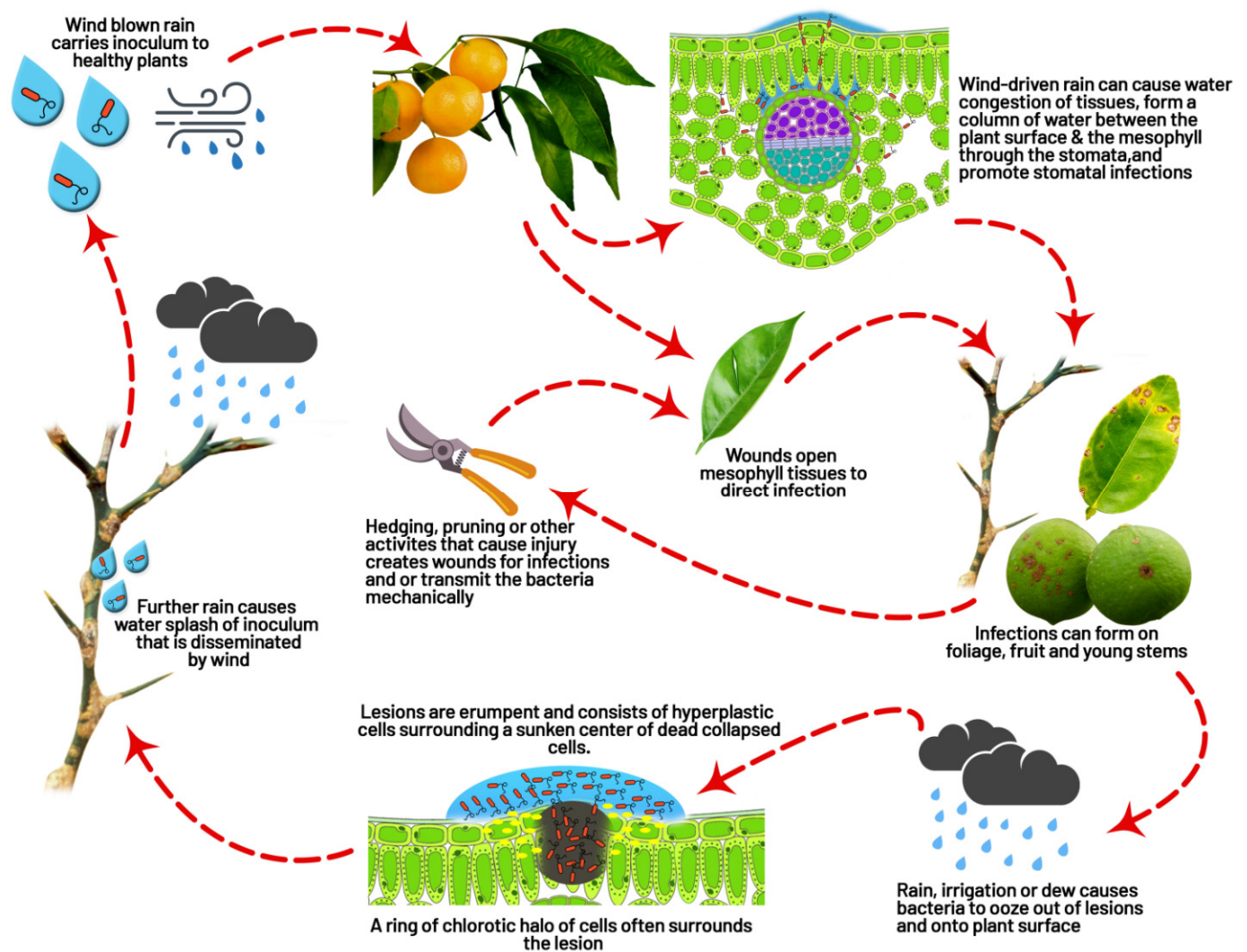


**Figure 3.** Dispersion of citrus canker bacterium in orchards.

### 5.2. Survival

The main inoculum sources are branches, leaves, and twigs infected with cankers [84]. The disease is primarily carried in cankers on twigs and branches from one season to another, serving as a primary inoculum [10]. In leaf and fruit lesions, the bacteria remain alive until they fall; because the affected leaves fall off early, they may not act as the primary inoculum source [75]. Still, it was reported that, in infected leaves, the bacterium survived up to six months [92]. Reference [93] found that the bacterium survived for over 6 months in the infected leaves, for 52 days in sterilized soil, and only 9 days in unsterilized soils, respectively. It was also observed that the organism could survive for 11–12 days under desiccation at 30 °C [93]. On citrus hosts, the bacterium survives epiphytically with a lower population without developing the symptoms, combined with non-citrus weeds, grass host, and soil [94–96]. However, in the absence of plant tissue or debris, the saprophytic existence of soil pathogens has not been observed [93,95]. The survival capability of the pathogen in subtropical soil is very limited [97], and bacterial inoculum dies within 24 to 72 h on different inert surfaces, such as cloths, metals, plastics, and processed wood in both sunlight and shade [91]. Due to antagonisms and competition with saprophytic microorganisms, the bacterial population decreases to an undetectable level 1–2 months after leaves or fruits fall to the ground [91]. In Japan and Brazil, it has been reported that *X. axonopodis* subsp. *citri* may survive on non-host plant material and in the root zone of certain weeds under eradicated diseased trees for a few weeks [98] (Figure 4).

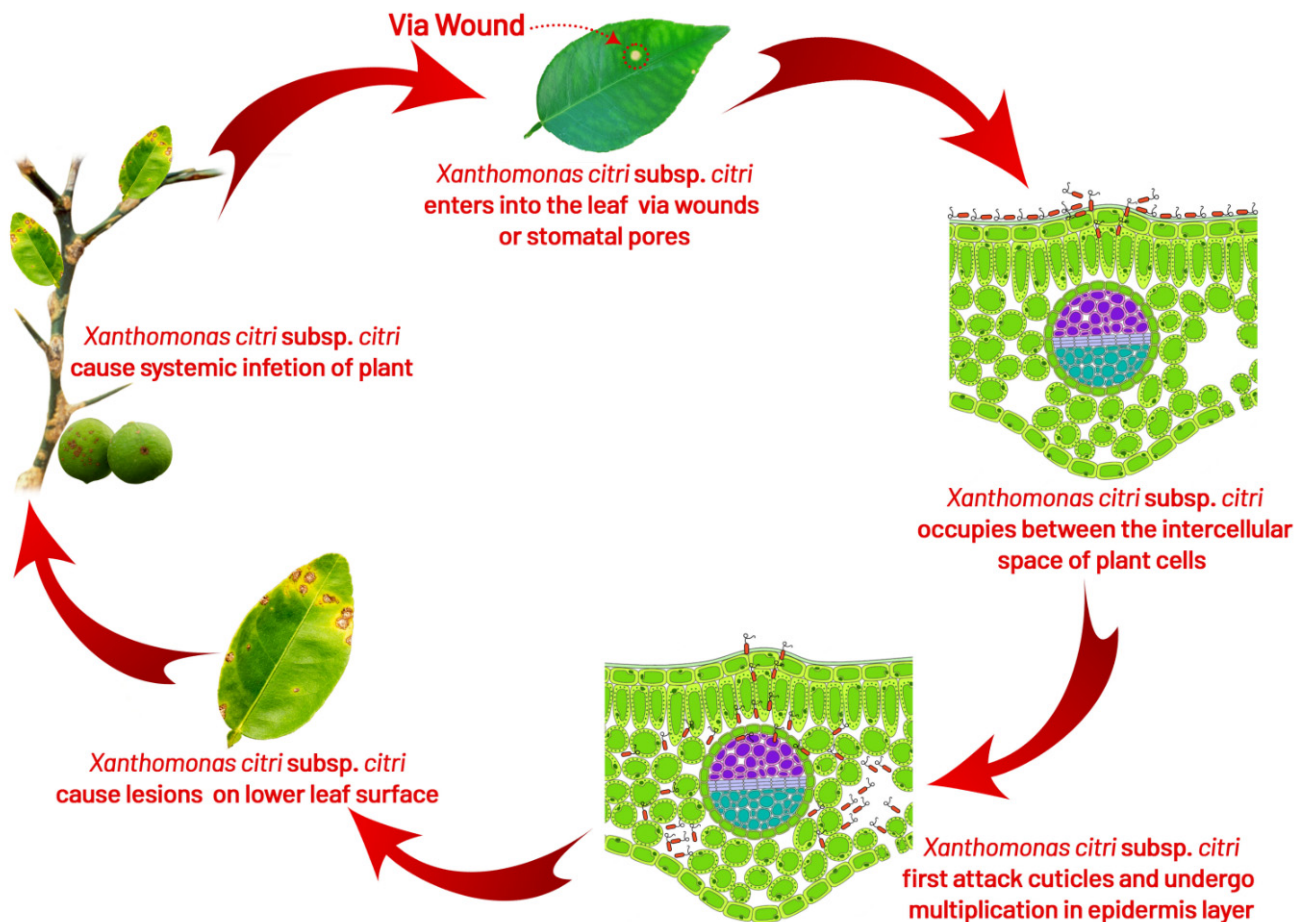




**Figure 4.** How canker bacterium initiates local infection into leaves, twigs, and fruits.

### 5.3. Dispersal

Under natural conditions, rainfall splashes and rainfall associated with wind are reasons for the short-distance spread of disease. Still, dissemination to long-distances between geographical regions is mainly expected to occur through infected plant material [24]. During severe storms, such as tornadoes, disease spread takes up to 10 to 15 km [90]. Dispersal of diseases have been further explored with model-based data on wind direction and threshold parameters, which showed wind speed eight m/s and rainfall of nearly 0.32 cm/h helped the insects, such as *P. citrella*, and blowing sand penetrates the bacteria through stomatal pores or injuries caused by thorns [99]. The major reason for the dispersal of bacteria to average distance is the wind-driven rain. In Argentina, the wind-blown rain dispersed the bacteria from infected trees up to a distance of 32 m [100]. A drop of rainwater may contain up to 105 to 108 cfu/mL of bacteria [80,100]. Under globalization, the transmission of bacteria from one region to another through frequent communication and transportation increases the risk of infecting citrus farming in free areas of disease [101] (Figure 5).



### Pathogenicity of *Xanthomonas citri* subsp. *citri*

**Figure 5.** Cellular interaction of *Xanthomonas citri* subsp. *citri* with the host and how it expresses symptoms on host plant parts.

#### 5.4. Role of Insect (Leaf Miner Interaction)

The leaf miner plays an important role in the spread of citrus canker, but it has not yet been reported as a disease vector [81]. In earlier 1994, the distribution of citrus leaf miners was restricted to southeast and southwest Asia, while it spread after the mid-1990s to most of the world's major citrus areas [81]. It was first reported in Florida and Brazil in 1993 and 1996 [102]. The feeding activities of the citrus leaf miner provide bacterial infections to the host in three ways: (1) wind-blown rain disseminates the bacteria, contacts the surface of the leaf; the leaf miner tears the cuticle and opens the mesophyll of the leaf, providing direct bacterial infection; (2) the leaf miner injuries are cured more slowly than mechanical injuries, which allow for longer exposure to bacterial infections; (3) leaf miner larvae may become contaminated with bacteria and carry it to the feeding galleries, where feeding activities lead to an increase in mesophyll cells infection [99,103]. Trees with leaf miner injuries remain susceptible for 7–14 days, compared with only 24 h for wind, thorns, or pruning injuries [104]. The prevalence of citrus canker increases in Brazil and Florida due to leaf miner injuries [74,90,104]. Still, it is believed that the host can tolerate some loss of leaf area without yield being affected because of leaf miner damage (up to 10%) [105]; there are reports that loss of 16–23% of leaf areas can lead to significant yield loss [106].

## 6. Detection and Identification of Citrus Bacterial Canker

The diagnosis of CC can be made using various methods and in most circumstances; however, when no official confirmation is required, the disease diagnosis can be made by recognizing common symptoms [8,107]. It is also possible to confirm the causal agent by isolating XCC from lesions on a solid medium and looking for xanthomonad-like colonies, which are yellow, convex, circular, semi-translucent, and have regular edges [52]. Infiltration of a bacterial suspension adjusted to 10<sup>8</sup> colony-forming units (CFU)/mL into the leaf mesophyll, followed by observation of water soaking and raised margins in the infiltrated portion of the leaf 2–4 days after inoculation, can be used to test pathogenicity in susceptible citrus species [76,107]. DNA-based assays and serological testing are routinely utilized methods for CC diagnosis when symptoms are atypical or an official confirmation is required for quarantine purposes [107]. Although molecular approaches can identify the presence of XCC in infected plant tissue before canker lesions occur, serology-based assays are usually sufficient for detecting XCC in symptomatic tissue [8]. Several primers based on rDNA sequences, plasmid-borne genes, and pathogenicity regulatory factors have been devised for polymerase chain reaction (PCR) detection of XCC [54,108–111]. In recent years, the introduction of real-time PCR and loop-mediated isothermal amplification procedures has improved the accuracy of diagnostic testing for XCC [11,112–115]. All existing conventional PCR methods need gel-visualization or primers, but all strains are not detected [54,90,116]. PCR primers are very effective for *X. citri* subsp. *citri* ‘A type’ detection, but these primers do not show consistency in *X. citri* subsp. *Aurantifolii* ‘B’ or ‘C’ strain detections [60,117]. New PCR primers based on the gene-sequence of *pthA* did not even detect one canker strain [54]. This technique, based on rep-PCR with BOX and ERIC primers, was developed to separate and distinguish the CBC pathotypes worldwide and the subgroups of pathotypes of citrus associated within specific geographical areas around the world [54,118]. Rapid, sensitive, and reliable real-time PCR assays were developed along with designed primers to detect all citrus-canker strains, which are important for both specificity and sensitivity [75,110]. Real-time PCR is easier to perform, less labor-intensive (no need for agarose gels), and much faster than conventional PCR [110]. If the sampling method is performed accurately, exact results can be obtained within 1 h. For plant pathogen detection, real-time PCR is becoming increasingly useful, i.e., for fungi [111,119–220], bacteria [122–124], and viruses [125,126]. A reliable, sensitive, SYBR Green real-time PCR assay in which primers are used to amplify conserved regions of a desired gene of pathogenicity to detect all known strains of XAC is based on sampling techniques conducted in the field samples [127]. The detection of the bacterium through PCR is based on an internal standard to make sure the quality of the DNA template for the reaction and ratio of PCR products is used to evaluate the early concentration of bacteria in citrus leaf tissues with lesions using internal standards and target pathogens [118]. Detection and comparison of *X. axonopodis* subsp. *citri* (XAC) from imported citrus fruits was based on an integrated approach involving isolation of bacteria from three conventional protocols viz., PCR, real-time PCR with SYBR-green, or a TaqMan-probe in canker lesions and LAMP [8,116,117]. The real-time PCR for fresh fruit samples with a TaqMan probe is the fastest screening method for the detection of bacteria [111]. Enzyme-linked immunosorbent assay (ELISA), or serological tests, have also proved to be useful for rapid detection of XCC, which are based on the ability of an antibody to recognize and bind to a specific antigen [111]. These tests are usually performed in the laboratory. Still, they are also available as strip-based kits that are easy to use in the field where the disease is suspected; these kits do not require special equipment or training, and the results are obtained within a few minutes [111,128]. Other older techniques for the detection of XCC have been developed, including physiological characterization, fatty acid profile analyses, protein profiling, hybridization, restriction fragment length polymorphism analysis, and comparison of plasmid DNA patterns [8,10,111,128] (Table 2).

**Table 2.** List of various primers used for the detection of *Xanthomonas citri* pv. *citri*.

Sr. No.	Primer	Target Region	Sequence	Reference
1.	P16SF1/P16SR2	16S rDNA	5-AGAGTTTGATCCTGGCTCAG-3 5-ACGGCTACCTTGTACGACTT-3	[129]
2.	FD1/RP2	16S rDNA	5-AGAGTTTGATCCTGGCTCAG-3 5-ACGGCTACCTTGTACGACTT-3	[130]
3.	X-ITS, F3/X-ITS R2	Internal transcribed spacer	5-GGCGGGGACTTCGAGTCCCTAA-3 5-CTGCAGGATACTGCCGAAGCA-3	[131]
4.	X-fyuaF/X-fyuaR	FyuA	5-GCCGGTGGACTACGATTGGAATTA-3 5-GTCGCGGCGCCACTTCA-3	[131]
5.	J- <i>pth</i> 1/J- <i>pth</i> 2	Pathogenicity	5-CTTCAACTCAAACGCCGGAC-3 5-CATCGCGCGCTGTTCCGGAG-3	[54]
6.	DLH 1/DLH 2	Pathogenicity	5-TTGGTGTCTCGCTTGTAT-3 5-CACGGGTGCAAAAAATCT-3	[60]

## 7. Genome

The sequence of the genome of *X. axonopodis* subsp. *citri* (306-strain) has been completed [132]. The bacteria have 5,175,554 bp and two plasmids that are pXAC33 (33,699 bp) and pXAC64 (64,920 bp) with a rounded chromosome [132]. The comparison shows a high degree of resemblance (about 80%) between *X. campestris* pv. *campestris* (pathogens of crucifers) genome and *X. axonopodis* subsp. *citri* genome, where both contain many genes specific to their sequenced strains [132]. The specificity of the host and variation in pathogenesis processes can be explained through these genes [7,132]. Genomics studies of CC bacterium have significantly opened new avenues for better understanding of XAC pathogenicity and virulence [7]. Several molecular methods for analyzing the population structure of a few plant pathogens have been utilized, e.g., RAPD, Rep, Eric-PCR, RFLP, plasmid profile analysis, PCR amplification, SDS-PAGE, 16S rDNA sequence [16,133,134,136–140]. There are 4314 projected open reading frames (ORFs) on the single circular chromosome, which has a G1C content of 64.7% [141]. The G1C content of the two plasmids, pXAC64 (64,920 bp) and pXAC33 (33,699 bp), is 61.4 and 61.9%, respectively [132,141]. Pathogenicity, virulence, and ecological adaptation are involved in about 7% of XCC genes [141]. The XCC-A genome contains a large number of cell wall-degrading enzymes (CWDEs), proteases, iron receptors, genes related to energy metabolism pathways, the type 2 secretion system (T2SS), type 3 secretion system (T3SS) genes for flagella structural units, chemotactic protein genes, the xanthomonadin, and xanthan gum synthesis gene cluster (*gumB* to *gumM*), which are important in the epiphytic phase of the life cycle [142]. The 23-kb *hrp* (for hypersensitive reaction and pathogenicity) region has six operons, designated as *hrpA* to *hrpF* [143]. The *hrp* cluster is part of a pathogenicity island in the XCC genome and encodes the T3SS. The genes *hrpG* and *hrpX* are involved in the regulation of amino acid biosynthesis, oxidative phosphorylation, pentose–phosphate pathway, phenolic catabolism, and transport of sugar, iron, and potassium in response to exposure to the host environment [144], while an additional 124 and 90 unknown genes are regulated by *hrpG* and *hrpX*, respectively [144]. *HrpG* induces the expression of 11 proteins secreted by the T2SS and is a regulator of the T3SS [145,146]. In 2010, several XCC-A, XCC-A\* (produces canker lesions in Mexican lime but not in grapefruit; Ref [147], XCC-Aw and *X. citri* pv. *Bilvae*, which causes CC-like symptoms in key lime) strains were characterized by amplified fragment length polymorphism (AFLP) and MLSA based on four partial housekeeping gene sequences (*atpD*, *dnaK*, *efp*, and *gyrB*) [147]. The study was performed on 157 XCC strains from Brazil, which were compared for their T3SS effector profiles using a qualitative PCR Southern blot technique [148]. Low genetic variability was observed for strains isolated in the northern part of the country, but more diversity was present in the strains isolated in the southern part [148]. The host plant genes, where products recognize pathogen effectors, are known



as *R* genes, and the pathogen pathogenicity (*pth*) genes, which encode these recognizable effectors, are also referred to as avirulence (*avr*) genes [148]. The products of *R* genes, either directly or indirectly, interact with the products of *avr* genes; interaction between products of *R* and *avr* genes is termed gene-for-gene resistance [149] whereas if no *R* genes correspond to XCC, then it leads imparting resistance to XCC host plant, which has been identified in citrus and citrus-related species [7,8,45].

## 8. Virulence

### 8.1. Type III Secretion System (T3SS)

Hypersensitive response and pathogenicity (*hrp*) of CC have a cluster of 24 genes on locus arranged in six operons from *hrpA* to *hrpF*, which are regulated by *hrpG*/*hrpX* genes and codes T3SS in *X. citri* subsp. *citri* [144,150]. It was observed that XAC remained unsuccessful at inducing disease and HR in the cotton plant by the deletion of *hrpB*, *hrpD*, and *hrpF* operons [151]. This system was presumed to secrete the effector proteins [152,153], and *pthA*, the member of the *avrBs3* gene family, targets the host susceptible gene lateral boundaries of organ 1 [154]. XAC uses T3SS against the host by injecting virulence proteins into host plant cells required for canker development in susceptible citrus plants and resistant plants, developing a hypersensitive response (HR) [151,155]. Effectors (virulence protein) are transported through specialized T3SS and use a hollow exterior (*Hrp*-pilus) that crosses the plant cell wall, delivering effectors across the plant plasma membrane through a translocon [156]. *Xanthomonas* strains usually harbor about 30 various effectors but their molecular activity is still unknown in most cases [156]. Transcription-activator-like (TAL) effectors form a large and important family of effectors found almost exclusively in *Xanthomonas* [157]. They act as transcription factors for plant genes and few induce expression of sugar exporters [13].

### 8.2. Citrus Specific *pthA* and Its Requirement for Canker Development

The first member of the gene family *avrBs3/pthA* is the *pthA* gene necessary for pathogenicity and was cloned through screening for pathogenicity [62,63,98]. Genes of *avrBs3* are broadly spread in the genus *Xanthomonas*, but are not present in all *Xanthomonas* spp. [63]; there are a minimum of 27 cloned members of the *avrBs3* family [158]. Without evidence of the *pth* function, mostly *avrBs3* members are isolated as *avr* genes for the first time while all genes *pthA*, *pthB*, *pthC*, and *pthW* of this family induce the citrus canker, and two genes, *pthN* and *pthN2*, are involved in the induction of cotton blight [159]. Intensive studies have been reported on the molecular virulence mechanism of the pathogen for the citrus canker [45,160]. Gene *pthA*, an effector of the type III secretion system (T3SS), is a determinant of canker pathogenicity and is commonly found in *Xanthomonas* spp., which causes citrus canker [54,63,98]. The *pthA* gene's exogenous insertion into *X. axonopodis* subsp. *citrumelo* causes bacterial citrus spot disease without causing erumpent lesions [62]. The transient expression of *pthA* produces citrus canker symptoms, causing cell hyperplasia, hypertrophy, and eventually, the plant dies [89]. In addition, *pthB* and *pthC* are functionally homologous genes that were cloned from *X. citri* subsp. *aurantifolii* B and C, and are important in inducing citrus canker [161]. Therefore, all three genes are functionally exchangeable and can be transmitted horizontally between strains of *X. citri* and *X. campestris* subsp. *aurantifolii* on plasmids [162]. Members of the *pthA* gene family are more than 3.8 kb in length and possess a high level of identity with DNA sequences (more than 90%) over their total length [138]. It seems that functionally homologous genes based on DNA hybridizations were found in all canker-causing strains and have not been found in isolated citrus strains that do not induce cankers, e.g., *X. campestris* subsp. *citrumelo* [55,161]; therefore, the single common gene was needed to induce citrus canker by *Xanthomonas* [45], while the XAC genome possessed several genes coding putative effectors [62,162]. The *pthA* is the most important effector that induces canker-like symptoms even in the absence of a pathogen when expressed transiently in plants [89,98]. Transient expression of *pthA* in host plant cells was sufficient at inducing symptoms of citrus canker in 10–14 days and *pthA*'s

deletion eliminates the pathogen to cause citrus canker [154]. In all citrus species, *pthA* induces canker symptoms, but in other plant species, it changes the plant to immune; hence, this characteristic makes the XAC specific toward the citrus species [53,62,63]. The *pthA* mutation prevents XAC from inducing hyperplasia, hypertrophy, water soaking symptoms, and pathogen losses of the ability to grow within plants [62,63]. All strains of *X. citri* subsp. *citri* group A were examined, which has three *pthA* alleles in addition to *pthA*; two of them are slightly functional to produce cankers in citrus, and one seems non-functional [63,163]. In the case of host recognition or avoidance, multiple homologs seem to provide rapid development of new genes of pathogenicity through recombination [164]. *X. axonopodis* subsp. *citri* have three homologs of *avrBs3/pthA* while only *ap11* homologue was reported to participate in virulence to induce canker formation, while the functions of other homologous were insignificant or not assessable [163]. The *pthA* is transported through T3SS, which consists of *hrp* gene cluster products, and the transcription of *hrp* genes is induced and regulated by the *hrpG* and *hrpX* regulators [144,155,165,166]. The *hrpG* or *hrpX* gene mutations in *X. citri* subsp. *citri* led to a loss of pathogenicity in citrus [144,167]. Other virulence-related genes are required for *X. citri* subsp. *citri*, in addition to the *pthA* and *hrp* genes, to cause disease [45], e.g., it was reported that *opsX* gene plays an important role in the production of lipopolysaccharides (LPS) and extracellular polysaccharides (EPS), growth, and virulence in planta [168].

### 8.3. Adhesion and Extracellular Polysaccharides (EPSs)

Extracellular polysaccharides (EPSs) and lipopolysaccharides (LPSs) defend the bacterium from unfavorable environmental conditions [169]. *Xanthomonas* spp. Produce characteristic EPS and xanthan, which results in bacterial colonies being mucoid; it is known as xanthan gum [170]. Xanthan is a polymer of repetitive units of pentasaccharide, having a backbone of side chains of cellulose and trisaccharide, used in the nutritional and pharmaceutical industries commercially as a thickening agent [171,172]. The xanthan production is managed by various genetic loci, including the 12 gum gene clusters from *Gum B* to *Gum M* that are highly maintained among *Xanthomonas* spp. [172,173]. Xanthan production in *Xanthomonas* is regulated hierarchically by the gene cluster regulation of pathogenicity factors (*rpf*) [174]. Host plants wilted due to infections caused by vascular pathogens that obstructed the flow of water in xylem vessels due to xanthan production [175,176]. A plethora of research depicted various gum genes of *Xanthomonas* spp. e.g., *X. axonopodis* subsp. *citri*, *X. axonopodis* subsp. *manihotis*, *X. campestris* subsp. *campestris*, and *X. oryzae* pv. *oryzae* has been involved in epiphytic survival and the development of bacteria in plants to induce disease symptoms [172,177–181]. It is interesting that gum genes from *X. axonopodis* subsp. *citri* are not essential in *Citrus sinensis* for disease development and growth of bacteria. Still, in *Citrus limon*, these gum genes play an important role in bacterial virulence, showing that xanthan virulence depends on the host plant and environmental conditions [177,178]. The *rpf* protein has been involved in DSF synthesis to regulate the genes and determine the synthesis of extracellular polysaccharides [183]. In many xanthomonads, the gum gene cluster is involved in EPS biosynthesis. The *Gum B* mutant showed defective EPS production and biofilms formation, hence, decreased infection in lemon [13,184].

XAC produces abundant extracellular polysaccharides (EPS) [184]. Bacterial cells are incorporated in a dense matrix of EPS of canker lesions and are disseminated with EPS through rain [13]. The EPS molecules effectively protect the bacteria in water from the ‘dilution effect’ and desiccation in air, hence playing an important role in bacterial ecology [185]. Bacteria enter the cell through stomata or wounds and remain stick to the host’s cell wall through an interaction between EPS and citrus agglutinins [186]. Experimental evidence suggests that basal plant defense responses are always suppressed by xanthan, e.g., deposition of callose in the plant cell wall. The chelating divalent calcium ions in the apoplast of the plant are required to activate plant defense responses [187,188]. Further, xanthan plays an important role in biofilms formation in *X. axonopodis* subsp. *citri*,

and *X. campestris* subsp. *campestris* [177,189]. Microbes create a biofilm matrix, made up of proteins, extracellular DNA, and polysaccharides, essential for the establishment of bacterial colonies while polysaccharide overproduction has been shown to alter colony shape and help to identify certain species [190]. The matrix's polysaccharide component can give a variety of benefits to the biofilm's cells, including adhesion, protection, and structure, and aggregative polysaccharides operate as molecular glue, allowing bacteria to stick to both biotic and abiotic surfaces by allowing them to withstand physical stresses, such as fluid movement that might detach them from a nutrition source [191–193].

#### 8.4. Lipopolysaccharides (LPSs)

In plant pathogenic bacteria, the major virulence factor responsible for host infectivity is lipopolysaccharides and it is increasingly recognized as a major plant pathogen-associated molecular pattern (PAMP) [98,194]. LPSs are major components and characteristic structures of the outer membrane of Gram-negative bacteria [194]. LPS molecules are usually composed of hydrophilic heteropolysaccharides formed by three major substructures, the O-specific polysaccharide (O-antigen), a repetitive sugar sub-unit; the core oligosaccharide region, which is covalently connected to the lipid A of the glycolipid moiety, and the lipid 'A' attached to the external plasma membrane [195,196].

LPSs have been recognized as a virulence factor during plant pathogenic interactions and involved in bacterial pathogenicity [197]. LPSs are present in bacteria's outer membrane, which protects the pathogen from hostile medium found within plant tissues, reduces the membrane permeability, and allows the bacteria to grow under unfavorable environmental conditions [197]. LPSs can prevent hypersensitive response (HR) in plants by avirulent bacteria that have been widely studied in plant cells [198]. In various *Xanthomonas* spp., insilico analyses have been carried out to identify and characterize the genes involved in LPS biosynthesis, showing *wxacO* and *rfbC* genes involved in LPS biosynthesis that reduces the pathogen motility, lack of resistance to stress, and virulence in grapefruit [199,200]. Further, the two-component regulatory system (TCRS) *ColR/ColS* has been reported to play multiple roles in LPSs and catalase production, biofilm formation, resistance to stress, transcription of *hrpD6* and *hpaF* genes, and knockout of either *colR* and *ColS*, causing loss of pathogenicity in grapefruit [13].

#### 8.5. Quorum Sensing

Virulence factors involve pathogen's ability to express their genetic, biochemical, and structural features to the host [13]. Quorum sensing (QS) is a mechanism that facilitates communication among bacterial cells through production and detection of signal molecules [13,201,202]. *Xanthomonas* has QS regulatory systems facilitated by molecules of the diffusible signal factor (DSF) family, which regulates the QS pathway of *Xanthomonas* spp. comprising three major QS regulons: *RpfF*, *RpfC*, and *RpfG* [203–205]. Furthermore, transcriptome analysis characterized *RpfF*, *RpfC*, and *RpfG* regulons, which showed *RpfF* controls the unique genes responsible for the QS-pathway complexity and other sensory mechanisms involved in canker development [204]. *RpfC* and *RpfG* control individual genes that play a wider role in gene regulation and their involvement in chemotaxis, motility and flagellate biosynthesis, extracellular enzymes production, adhesion, stress tolerance and regulations, transport, and transport detoxification [206]. The QS process depends on the production, release, and detection of small signaling molecules known as autoinducers (AIs) [206]. The MJ Daniels Research Group, for the first time, reported the detection of activity of DSF molecules as autoinducers [174]. The synthesis of DSF in *X. axonopodis* subsp. *citri* is based on genes *rpfF* and *rpfB*, and it is an autoinducer in bacteria and regulates quorum sensing [203]. The *Rpf/DSF* system involves the initial attachment of XAC to the host and controls a range of virulence-related characteristics, such as the synthesis of extracellular enzymes (proteases, endoglucanases), extracellular polysaccharides, EPSs biosynthesis, and biofilm formation in several strains of pathogens [13].

## 9. Nutrition

Bacteria can take nutrients from their hosts through enzyme secretions that degrade the host's cell wall and bacteria utilize the cell wall's breakdown products as nutrition sources [62]. In XAC genome pectin esterases are not present, but bacteria possess three pectate lyases, six cellulases, five xylanases, and an endoglucanase, while in cellulose, it contains endoglucanaseBcsZ (gi | 22001634), which hydrolyzes 1,4- $\beta$ -D-glucosidic linkages [62]; moreover, permease, through which degraded pectin products are imported into the bacterial cells in a hydrogen-transport coupled fashion [62]. The *pthA* is also necessary for optimal growth of the bacteria in the host, which is probably directly injected into the host cell [62]. *X. citri* subsp. *citri* produces less enzymes as compared to *X. campestris* subsp. *campestris*, which degrades the cell wall; because of this, the two pathogens may cause their hosts to suffer from different symptoms [132].

## 10. Integrated Management Programs

IDM has been introduced to control the occurrence of citrus bacterial canker disease (CBCD) in new seedlings [9]. This program recommends that only citrus bacterial canker disease-resistant cultivars may be planted [107]. For commercial cultivation, it is recommended to plant sweet oranges, such as Tahiti lime, Pera pre-immunized, FolhaMurcha, Valencia, and Navelina, mandarins, such as Ponkan, Dancy, Loose Jacket, Satsuma, Batangas, and Willowleaf accessions [9]. Many studies have been carried out to control CC disease through cultural, chemical, or biological management strategies but have shown limited effects [207,208]. When leaf miners attack citrus varieties, or under changing weather conditions, the development of disease becomes far more complex and harder to control the eradication of diseased trees is the only way to control the CC disease [7,9,11]. Resistance genotypes in citrus and relative genera have long been researched around the world, and several types of citrus germplasms with certain resistance levels have been reported, e.g., calamondin (*C. mitis*) and kumquats (*Fortunella* spp.) are highly resistant, and mandarins (*C. reticulata*) were also reported to be resistant [78,162,209]. Suppose plants are inoculated artificially with the bacterium or planted in combination with sweet oranges. In that case, all plants show characteristic symptoms of citrus cankers without any complete or active resistant citrus genotypes [7,9]. As no resistant varieties were identified, breeding efforts have made little progress in the production of resistant cultivars, and few experiments in molecular breeding have shown transformants of some resistance through the transfer of antibacterial genes to citrus fruits. Still, only a lower disease incidence has been achieved without complete resistance [8,9]. The molecular mechanism of pathogenesis remains unclear, and there are no resistance genes isolated; it is very tough to obtain resistant genotypes through breeding programs [210,211]. The resistant mandarin varieties are grown in Southeast Asia, where the climate is most favorable for epidemics; the citrus canker was not a major issue until more vulnerable sweet oranges were brought into the disease regions of China and Japan [78]. Since the 1950s, eradication/control programs have been established in São Paulo, Brazil, to prevent the pathogen spread in the production area of sweet oranges [9]. Contrarily, the nearby zones of Paraná State, Brazil, Misiones, Corrientes, and Argentina have adopted integrated program strategies to efficiently prevent and control citrus cankers in sweet oranges [212–214]. The program is mainly concerned with shifting of citrus plantations to the disease-free area, having resistant citrus varieties, and in these regions, regulations are there, not only to deal with more resistant varieties, but also to produce disease-free nursery trees, as well as other means to exclude the XAC from citrus plantations [212]. The regulations for the management of CC disease include (i) nurseries must be situated in disease-free areas; (ii) the design of citrus production areas must be managed in order to decrease the danger of an epidemic of CC by constructing windbreaks, applying preventive copper sprays, building fences to avoid the entrance of bacteria to the citrus plantation; (iii) the planting and harvesting tools should be disinfected; workers should also disinfect their clothes, shoes, and gloves; (iv) fresh fruits should be strictly inspected for domestic and export markets to prevent the fruits in citrus groves from



citrus cankers; workers should also disinfect the storage and packaging houses; (v) infected summer and autumn shoots should be pruned; (vi) disease management forecasts should be considered; (vii) control of citrus leaf miners; (viii) use of chemical inducers, which induce systemic acquired resistance (SAR) in plants [215,216].

#### 10.1. Quarantines

Federal quarantine barriers are regulatory responses to diseases, which could be found in almost any country. Still, the exact locations of such barriers are difficult issues, for biological and political reasons [9,24]. These barriers are usually placed two or more two miles away from any known infestation [24]. The distribution of host plant materials is limited within quarantine areas, affecting both the citrus agriculture sector and homeowners with citrus trees [9]. In commercial production, it is recommended to disinfect the fruits in packing houses and disinfect the harvesting and transport equipment [24]. Fresh fruits are often restricted in market distributions from regulated areas [7]. Commercial citrus planting needs sanitization stations at plantation doors, a caution that has become a national demand, even outside quarantine areas [7]. Citrus replanting in commercial or residential areas that have undergone eradication efforts is against the law before the disease is declared eliminated [7,9]. People are informed in residential areas—that it is illegal to transport fruits to neighbors and family; decontamination of all equipment that is moved between properties during lawn and garden services is required [7,9]. These measures are publicized through intensive media reporting and expertise in community relations [24].

#### 10.2. Cultural Control

Eradication of any disease is the method used to manage that specific problem if it has not been endemic in a region [7]. Quarantine and eradication are key measures to control the entry and dissemination of pathogens in many countries [9]. Eradication measures involve destruction by cutting and burning citrus species [24]. Sometimes, herbicides are used instead of cutting and burning to kill citrus plants [18]. The infested property is quarantined, followed by the eradication procedure for at least a year without planting or propagation of citrus fruits, with inspection at least twice a year [24].

Data from Argentina showed that the pathogen could disseminate in rainfall with wind up to 32 m (105 ft), which provided the scientific basis for eradicating this disease [50]. This has been translated in the U.S. and many other countries into regulating policies, allowing survey teams to locate diseased citrus trees, to remove and kill the trees, as well as exposed trees within a radius of 38.1 m (125 ft) of a diseased tree [76,217]. Now, Brazil uses a distance of 30 m (98 ft) to remove exposed infected trees. If infections of the Brazilian plants are 0.50% or lower, all trees will be removed within a radius of 30 m of the infected plantation. When the infection exceeds 0.5%, the whole block will be removed [217]. New canker infections occur in known source trees, at about 1900 ft (579 m) [17]. In January 2000, a new regulation, “the 1900 ft rule”, was set up. In March 2000, it was implemented, which involved the eradication of infected citrus trees along with healthy trees within 1900-ft of an infected tree [17,218]. Each 1900 ft radius circle has a surface area of 1.06 km<sup>2</sup> (0.41 miles), leading to the removal of dooryard citrus in infected areas by implementing the 1900 ft rule [9]. Pruning of infected twigs, along with application of a 1% Bordeaux mixture at regular intervals, before the onset of monsoon, also proved to be very effective in the management of disease [219–223].

#### 10.3. Chemical Control

For the management of CC, it has been reported that every year, from November to December, pruning of infected twigs with three to four sprays of a 1% Bordeaux mixture could be used to reduce disease [224]. Control of disease by applying four sprays of 5000 ppm copper oxychloride or a 1% Bordeaux mixture and two prunings gave excellent results [225,226]. Chemicals such as Perenox, Ultrasulphur, and a mixture of Blitox + nickel chloride, sodium arsenate + copper sulphate, were used against citrus cankers [227–230].

The application of 1% glycerin spray and 500–1000 ppm of streptomycin–sulphate was found useful in controlling disease on acid lime [231]. Acid lime canker was reduced by six sprays of 1000 ppm streptomycin with two prunings [232]. Streptocycline, in combination with Bordeaux mixture, and Agrimycin, are effective antibiotics against CC [233]. For field tests with different chemicals, Paushamycin + Blitox and Bordeaux mixture showed the best control of CC [234]. In nurseries, treatments of young plants have been reported by applying neem cake solution on the leaves [235,236]. The application of streptocycline + copper oxychloride (0.1%), preferably at intervals of seven days or fifteen days, has been found very effective against CC [236]. Integrated application of copper oxychloride (0.3%), streptocycline (100 ppm), and suspension of neem cake on pruned infected twigs has shown to be very useful to control the disease [237].

In field experiments in Argentina, copper ammonium carbonate with 8% metallic copper was consistently found better than other products to control CC; regarding field trials on trees of ripened grapefruits, three applications of copper ammonium carbonate (CAC) or copper hydroxide + maneb per season were examined and reduced the number of lesions found on fruits but not on leaves [238]. The recommendation to add mancozeb to copper spray was effective for copper resistance [239]. Sanitary procedures have been explained for persons or tools that encounter citrus in quarantine regions, e.g., by applying sprayable ammonium detergent disinfectants [23].

#### 10.4. Biological Control

There has been a surge in the hunt for more environmentally-friendly plant disease treatment methods [7]. Researchers are looking for more ecological approaches to manage phytopathogens in the field due to chemical residues in soils and water bodies and increased consumer concerns [7]. Some recent research used the antagonistic activity of microorganisms and plant-derived compounds to suppress citrus cankers [7]. Studies on biological control are still in the initial phases, to control CC [239,240]. Some bacterial strains, including *Pseudomonas syringae*, *Erwinia herbicola*, *Bacillus subtilis*, and *Pseudomonas fluorescence* isolated from citrus phylloplane have been found to be antagonistic to the citrus canker pathogen, *X. citri*, in vitro [133,241–244]. However, it appears hard to find antagonistic bacteria that stabilize on mature citrus tree leaves [245]. For example, *P. aeruginosa* LV produces a bioactive combination of secondary metabolites, the most important of which is an organocopper antibiotic that reduces the formation of citrus canker lesions in Valencia oranges by 90% [245]. The authors discovered a bacteriolytic impact on *X. citri* that was not accompanied by any signs of phytotoxicity [246]. At low micromolar concentrations, several secondary metabolites from *P. aeruginosa* also reduced canker formation [247,248]. However, because *P. aeruginosa* is an opportunistic human infection, its use as a biocontrol agent is fraught with dangers and must be strictly managed [9]. *Bacillus* spp. isolated from citrus rhizosphere and leaves have also been proposed as a citrus canker biocontrol agent because they inhibited *X. citri* growth in vitro and in field circumstances [76,240,249,250]. *X. citri* and several species of *Pseudomonas* and *Bacillus* suppressed growth and canker formation, and *Citrobacter* isolated from sweet orange phylloplane, by degrading the *X. citri* quorum sensing molecule DSF [251]. Other bacterial genera, such as *Cronobacter* and *Enterobacter*, similarly inhibited *X. citri* growth in vitro by producing bacteriocins; however, the protective effect of these bacterial species on *X. citri*-infected citrus trees was not assessed [252]. The sensitivity of bacteriophages (phages) can be utilized to identify intra-species sub-groups in bacteria [252]. Cp1 and Cp2 phages have long been employed to identify *X. citri* strains [253]. The employment of phages in biological control, on the other hand, poses significant difficulties [254]. Phages have a very short active life on the leaf surface and must be administered at high concentrations to be effective [255]. The authors of reference [256] found that the jumbo phage *XacN1*, originally isolated from an orange orchard soil in Japan, can infect a wide range of *X. citri* isolates, making it a promising choice for future field experiments. In greenhouse and field testing, a mixture of phages obtained from orange orchards combined with ASM successfully decreased canker

symptoms [257]. Compared to copper–mancozeb alone, the combination of phages and copper–mancozeb did not improve citrus canker control [258]. Interestingly, filamentous integrative phages, such as XACF1, reduced the virulence of *X. citri*, suggesting that they could be used as citrus canker biocontrol agents [259].

#### 10.5. Field Screening

Globally, field screening was carried out to assess the response of citrus varieties in local environmental conditions to CC [9,24]. Suppose very intensive control programs have been carried out. In that case, highly susceptible varieties, i.e., several early–mid-season sweet oranges, grapefruits, and Mexican limes (e.g., Navel, Hamlin), are not recommended for planting [212]. Mid and late season oranges, tangerines (tangerine, tangelo, and tangerine), and Tahiti limes have been identified with acceptable resistance to citrus cankers in screening programs [9]. These cultivars may be susceptible in the young stages and need to be sprayed to control the leaf miner to avoid damage to emerging flushes that predispose them to infection [9].

#### 10.6. Induced Systemic Resistance

Induced systemic resistance (ISR) is an active resistance mechanism in plants activated through biotic or abiotic infection. The mechanism increases physical or chemical barriers of host plants against infection [260]. Different inducer compounds, e.g., salicylic acid, harpin protein, and benzothiadiazoles are used effectively to induce resistance in plants against diseases [261,262]. ISR mechanisms can simultaneously control the disease and decrease the risk of developing pathogen resistance [263]. ISR activity may be used early in the season to complement the protectant activity of Cu, which slows the growth of bacteria in rapidly growing leaves [263]. Chemicals sold for the treatment of citrus canker include ‘Actigard’, approved for acibenzolar-S-methyl, a benzothiadiazole in the USA, and ‘Bion’ in Europe and South-America (Syngenta Crop Protection); ‘Messenger’ (Eden Bioscience) is approved for the harpin protein (a *hrp*-gene product) [63]. In Florida, several ISR inducers (e.g., Messenger, Nutri-phite, Oxycom, and FNX-100) are evaluated for control of *X. citri* subsp. *citri* [9].

#### 10.7. Leaf Miner Control

Leaf miners do not propagate cankers, but extensive bacterial invasion through leaf miner galleries increases inoculum levels significantly, making it difficult to control the disease [24]. Leaf miner control on the first summer flush can significantly reduce the pressure of the disease, but there is no effective control of leaf miners on late summer flushes. On spring flush, it causes no damage; therefore, control is required [63]. Applications of Agri-mek, petroleum oil, Assail, Micromite, or Spintor on time minimize leaf miner damage [24,63].

#### 10.8. Control through Plant Extracts

To avoid or reduce the deleterious effects of synthetic pesticides on the ecosystem, it is necessary to find alternative approaches to manage plant pathogenic microorganisms [264,265]. As an alternative to synthetic pesticides, green plants have proved to be effective chemo-therapeutics and can be used as valuable sources of natural pesticides [266]. The use of several plant byproducts that possess antimicrobial properties on several pathogenic bacteria and fungi has been reported by many researchers [267–272]. In Pakistan, where the farms are small, and the economic conditions of farmers are also not good, standard antibiotics, on account of their high costs, are often beyond the reach of an average farmer [272]. Under such conditions, the use of plant extracts/diffusates to manage bacterial plant diseases appeared to have good potential [273]. Different plant extracts, e.g., *Allium sativum* L., *Allium cepa* L., *Azadirachta indica*, *Calotropis gigantea*, *Dalbergia sissoo*, *Eucalyptus camelduensis*, *Gardenia florida*, and *Melia azedarach* have been used by farming communities to mitigate the multiplication of XCC [273]. Essential oil is a broad word that

refers to any volatile aromatic molecule generated from plants [274]. Essential oils have long been known for their antibacterial properties against pathogenic and phytopathogenic microorganisms [275]. Many essential oils from *Citrus aurantium*, *Citrus aurantifolia*, and *Fortunella* sp. have been shown to kill *X. citri* [276]. Citral from *C. aurantifolia* inhibited *X. citri* growth, the most in disc diffusion experiments, while limonene, geranyl acetate, and trans-caryophyllene from *Fortunella* sp. had little effect [276]. Citral has a MIC of 0.5 mg/mL, indicating that substantial doses would be needed to control *X. citri* in the field [276]. Other plant-derived chemicals may be useful in the fight against citrus canker, e.g., water and acetone extracts from the leaf gallnuts of the Chinese “sumac” (*Rhus chinensis*) suppressed the development of *X. citri* at a dose of 1 mg/mL [277]. After further separation of the gallnut leaf extracts, the bioactive chemicals were methyl gallates and gallic acids [277]. Gallic acids (MIC 4 mg/mL) were active at significantly lower quantities (MIC 0.1 mg/mL) than methyl gallates (MIC 0.1 mg/mL) [277]. At low micromolar concentrations, synthetic gallates also reduced *X. citri* growth in vitro, but when administered to fully formed cankers, these chemicals prevented *X. citri* host colonization after artificial infiltration and reduced the bacterial population [278]. Alkyl gallate amphiphile structures exhibit enhanced chemical entry in target cells, similar to pyridinium-tailored molecules [279]; membrane permeabilization and the divisional septum have been identified as primary targets of these molecules in *X. citri* [280]. These compounds were found to be low in toxicity in human cells, and further molecule development led to more lipophilic and lethal monoacetylated alkyl gallates [280]. Reference [281] studied that *C. coriaria* is a potential candidate plant for managing phytopathogenic *Xanthomonas*. In forest trees, it has been reported that diffusates of *Phyllanthus emblica*, *Acacia nilotica*, *Sapindus mukorossi*, and *Terminalia chebula* (large, black type) were found to be most effective against XCC [282]. Methanolic leaf extracts of *Psidium guajava* L. could be developed as antibacterial agents to control plant pathogenic bacteria as they were able to inhibit the growth of *Xanthomonas* spp. with all concentrations [283].

#### 10.9. Factors Affecting Successful Eradication of Citrus Canker

*X. citri* subsp. *Citri* possess some unique characteristics that make them desirable for eradication: (i) for long periods; the bacterium cannot live outside the host lesion; (ii) bacteria lack an effective vector; (iii) the typical elevated lesions are easily recognized and can be diagnosed quickly and accurately; (iv) the bacterial host range is limited to a highly valuable perennial fruit plant; (v) many commercial citrus species are moderately to highly susceptible. For that reason, disease control measures were moderately efficient and comparatively expensive; during previous campaigns in Florida, Australia, and South-Africa, it has been eliminated successfully [9].

### 11. Conclusions and Future Prospects

Citrus canker, as the most feared citrus disease worldwide, continues to be a potential threat to citriculture. Broad-spectrum pathogenicity and cultivation of susceptible varieties of citrus canker and the emergence of new strains are major threats to the world's production of citrus. The new citrus canker strains are also evolving because of mutations in the genome. Various physiological, biochemical, serological, molecular, and pathogenic variations are found among these strains. Therefore, detailed biological and molecular characterizations of the pathogens and their genomes are crucial for their proper identification [84]. Recent conclusions have shown that the vascular systems of plants generally contain internal microbes (endophytes). There is a considerable chance that an antagonistic microbe found among these endophytes will help control citrus canker, biologically [84]. The risk of citrus canker must be prevented or reduced by establishing and using wind-breaks, constructing barriers to prevent bacterial access to orchards, applying antibiotics, utilizing preventive copper-based sprays, biological control approaches, and developing genetic engineering-based canker resistant varieties.



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## References

- Ministry of National Food Security and Research. *Agricultural-Statistics-of-Pakistan-2019–2020*; Ministry of National Food Security and Research: Islamabad, Pakistan, 2020.
- FAO STAT, Statistics Division, Food and Agriculture Organization of the United Nations. *Agriculture Statistics 2019–2020*; Food and Agriculture Organization of the United Nations: Rome, Italy, 2020.
- Siddique, M.I.; Garnevska, E. Citrus Value Chain(s): A Survey of Pakistan Citrus Industry. In *Agricultural Value Chain*; IntechOpen: London, UK, 2017. [\[CrossRef\]](#)
- Al-Snafi, A.E. Nutritional value and pharmacological importance of citrus species grown in Iraq. *IOSR J. Pharm.* **2016**, *6*, 76–108. [\[CrossRef\]](#)
- Pakistan Horticulture Development Export Company (PHDECo). *Citrus Marketing Strategy*; Pakistan Horticulture Development Export Company: Lahore, Pakistan, 2018.
- Agriculture Marketing Information Service of Pakistan. *District Wise Data of Citrus*; Agriculture Marketing Information Service of Pakistan: Lahore, Pakistan, 2018.
- Martins, P.M.M.; de Oliveira Andrade, M.; Benedetti, C.E.; de Souza, A.A. Xanthomonas citri subsp. citri: Host interaction and control strategies. *Trop. Plant Pathol.* **2020**, *45*, 213–236. [\[CrossRef\]](#)
- Ference, C.M.; Gochez, A.M.; Behlau, F.; Wang, N.; Graham, J.H.; Jones, J.B. Recent advances in the understanding of Xanthomonas citri ssp. citri pathogenesis and citrus canker disease management. *Mol. Plant Pathol.* **2018**, *19*, 1302–1318. [\[CrossRef\]](#) [\[PubMed\]](#)
- Randsborg, K. Introduction. *EPPO Bull.* **1979**, *9*, 341–342. [\[CrossRef\]](#)
- da Gama, M.A.S.; de Lima Ramos Mariano, R.; da Silva J'uniior, W.J.; de Farias, A.R.G.; Barbosa, M.A.G.; da Silva Velloso Ferreira, M.A.; J'uniior, C.R.L.C.; Santos, L.A.; de Souza, E.B. Taxonomic Repositioning of Xanthomonas campestris pv. viticola (Nayudu 1972) Dye 1978 as Xanthomonas citri pv. viticola (Nayudu 1972) Dye 1978 comb. nov. and Emendation of the Description of Xanthomonas citri pv. anacardii to Include Pigmented Isolates Pathogenic to Cashew Plant. *Phytopathology* **2018**, *108*, 1143–1153. [\[PubMed\]](#)
- Mehrotra, R. Bacteria and Bacterial Diseases. In *Plant Pathology*; Tata McGraw-Hill Pub. Co. Ltd.: New Delhi, India, 1980; pp. 636–638.
- Whiteside, J.O.; Garnsey, S.M. *Compendium of Citrus Diseases*; APS Press: College Park, MD, USA, 1988.
- Li, L.; Li, J.; Zhang, Y.; Wang, N. Diffusible signal factor (DSF)-mediated quorum sensing modulates expression of diverse traits in Xanthomonas citri and responses of citrus plants to promote disease. *BMC Genom.* **2019**, *20*, 55. [\[CrossRef\]](#)
- Lee, H.A. Further data on the susceptibility of rutaceous plants to citrus-canker. *J. Agric. Res.* **1918**, *15*, 661–665.
- Fawcett, H.S.; Jenkins, A.E. Records of Citrus Canker from Herbarium Specimens of the Genus Citrus in England and the United States. *Phytopathology* **1933**, *23*, 820–824.
- Graham, J.; Hartung, J.; Stall, R.; Chase, A. Pathological, restriction-fragment length polymorphism, and fatty acid profile relationships between Xanthomonas campestris from citrus and noncitrus hosts. *Phytopathology* **1990**, *80*, 829–836. [\[CrossRef\]](#)
- Gottwald, T.R.; Hughes, G.; Graham, J.H.; Sun, X.; Riley, T. The citrus canker epidemic in Florida: The scientific basis of regulatory eradication policy for an invasive species. *Phytopathology* **2001**, *91*, 30–34. [\[CrossRef\]](#)
- Rossetti, V. Citrus canker in Latin America: A review. In Proceedings of the International Society of Citriculture, Orlando, FL, USA, 1–8 May 1977; pp. 918–924.
- Doidge, E.M. Citrus canker in South Africa. *S. Afr. Fruit Grow.* **1916**, *3*, 265–268.
- Garnsey, S.; Ducharme, E.; Lightfield, J.; Seymour, C.; Griffiths, J. Citrus canker. *Citrus Ind.* **1979**, *60*, 5.
- Bártová, V.; Bárta, J.; Jarošová, M. Antifungal and antimicrobial proteins and peptides of potato (*Solanum tuberosum* L.) tubers and their applications. *Appl. Microbiol. Biotechnol.* **2019**, *103*, 5533–5547. [\[CrossRef\]](#) [\[PubMed\]](#)
- Okabe, N. Bacterial Diseases of Plants Occurring in Formosa I. *J. Soc. Trop. Agric.* **1932**, *4*, 470–483.
- Del Campo, R.; Russi, P.; Mara, P.; Mara, H.; Peyrou, M.; De León, I.P.; Gaggero, C. Xanthomonas axonopodis pv. citri enters the VBNC state after copper treatment and retains its virulence. *FEMS Microbiol. Lett.* **2009**, *298*, 143–148. [\[CrossRef\]](#)
- Schubert, T.S.; Rizvi, S.A.; Sun, X.; Gottwald, T.R.; Graham, J.H.; Dixon, W.N. Meeting the challenge of eradicating citrus canker in Florida—Again. *Plant Dis.* **2001**, *85*, 340–356. [\[CrossRef\]](#)

25. Naseem, S.; Shah, H.A.; Ali, Z. First report on characterization of citrus disease causing bacteria and related phages isolated in Pakistan. *Int. J. Agric. Biol.* **2017**, *19*, 857–864. [\[CrossRef\]](#)
26. Stevens, H.E. Citrus canker. A preliminary bulletin. *Fla. Agric. Expt. Sta. Bull.* **1914**, *122*, 113–118.
27. Hasse, C.H. *Pseudomonas citri*, the cause of citrus canker—A preliminary report. *J. Agric. Res.* **1915**, *4*, 97–100.
28. Doidge, E.M. The Origin and Cause of Citrus Canker in South Africa. *Union So. Afr. Dept. Agric. Sci. Bul.* **1916**, *8*, 20.
29. Dowson, W.J. On the systematic position and generic names of the gram negative bacterial plant pathogens. *Zentr. Bakteriell. Parasitenk. Abt. II* **1939**, *100*, 177–193.
30. Sena-Velez, M.; Redondo, C.; Graham, J.H.; Cubero, J. Presence of extracellular DNA during biofilm formation by *Xanthomonas citri* subsp. *citri* strains with different host range. *PLoS ONE* **2016**, *11*, e0156695. [\[CrossRef\]](#) [\[PubMed\]](#)
31. Bansal, K.; Kumar, S.; Patil, P.B. Phylogenomic insights into diversity and evolution of nonpathogenic *Xanthomonas* strains associated with citrus. *mSphere* **2020**, *5*, e00087-20. [\[CrossRef\]](#) [\[PubMed\]](#)
32. Patané, J.S.L.; Martins, J.; Rangel, L.T.; Belasque, J.; Digiampietri, L.A.; Facincani, A.P.; Ferreira, R.M.; Jaciani, F.J.; Zhang, Y.; Varani, A.M.; et al. Origin and diversification of *Xanthomonas citri* subsp. *citri* pathotypes revealed by inclusive phylogenomic, dating, and biogeographic analyses. *BMC Genom.* **2019**, *20*, 700. [\[CrossRef\]](#) [\[PubMed\]](#)
33. Young, J.M.; Dye, D.W.; Bradbury, J.F.; Panagopoulos, C.G.; Robbs, C.F. Proposed nomenclature and classification for plant pathogenic bacteria. *N. Z. J. Agric. Res.* **1978**, *21*, 153–177. [\[CrossRef\]](#)
34. Gabriel, D.W.; Kingsley, M.T.; Hunter, J.E.; Gottwald, T. Reinstatement of *Xanthomonas citri* (ex Hasse) and *Xanthomonas phaseoli* (ex Smith) to species and reclassification of all *Xanthomonas campestris* pv. *citri* strains. *Int. J. Syst. Bacteriol.* **1989**, *39*, 14–22. [\[CrossRef\]](#)
35. Young, J.M.; Bradbury, J.F.; Gardan, L.; Gvozdyak, R.I.; Stead, D.E.; Takikawa, Y.; Vidaver, A.K. Comment on the reinstatement of *Xanthomonas citri* (Ex Hasse 1915) Gabriel et al. 1989 and *X. phaseoli* (Ex Smith 1897) Gabriel et al. 1989—Indication of the need for minimal standards for the genus *Xanthomonas*. *Int. J. Syst. Bacteriol.* **1991**, *41*, 172–177. [\[CrossRef\]](#)
36. Vauterin, L.; Hoste, B.; Kersters, K.; Swings, J. Reclassification of *Xanthomonas*. *Int. J. Syst. Bacteriol.* **1995**, *45*, 472–489. [\[CrossRef\]](#)
37. Schaad, N.W.; Postnikova, E.; Lacy, G.; Sechler, A.; Agarkova, I.; Stromberg, P.E.; Stromberg, V.K.; Vidaver, A.K. Reclassification of *Xanthomonas campestris* pv. *citri* (ex Hasse 1915) Dye 1978 forms A, B/C/D, and E as *X. smithii* subsp. *citri* (ex Hasse) sp. nov. nom. rev. comb. nov., *X. fuscans* subsp. *aurantifolii* (ex Gabriel 1989) sp. nov. nom. rev. comb. nov., and *X. alfalfae* subsp. *citrumelo* (ex Riker and Jones) Gabriel et al., 1989 sp. nov. nom. rev. comb. nov.; *X. campestris* pv. *malvacearum* (ex Smith 1901) Dye 1978 as *X. smithii* subsp. *smithii* nov. comb. nov. nom. nov.; *X. campestris* pv. *alfalfae* (ex Riker and Jones, 1935) Dye 1978 as *X. alfalfae* subsp. *alfalfae* (ex Riker et al., 1935) sp. nov. nom. rev.; and “var. *fuscans*” of *X. campestris* pv. *phaseoli* (ex Smith, 1987) Dye 1978 as *X. fuscans* subsp. *fuscans* sp. nov. *Syst. Appl. Microbiol.* **2005**, *28*, 494–518.
38. Schaad, N.W.; Postnikova, E.; Lacy, G.; Sechler, A.; Agarkova, I.; Stromberg, P.E.; Stromberg, V.K.; Vidaver, A.K. Emended classification of *Xanthomonad* pathogens on citrus. *Syst. Appl. Microbiol.* **2006**, *29*, 690–695. [\[CrossRef\]](#)
39. Euzéby, J. List of new names and new combinations previously effectively, but no validly, published, list. *Int. J. Syst. Evol. Microbiol.* **2007**, *57*, 893–897.
40. Constantin, E.C.; Cleenwerck, I.; Maes, M.; Baeyen, S.; Van Malderghem, C.; De Vos, P.; Cottyn, B. Genetic characterization of strains named as *Xanthomonas axonopodis* pv. *dieffenbachiae* leads to a taxonomic revision of the *X. axonopodis* species complex. *Plant Pathol.* **2016**, *65*, 792–806. [\[CrossRef\]](#)
41. Holland, D.F.V. The families and genera of the bacteria. V. Generic index of the commoner forms of bacteria. *J. Bacteriol.* **1920**, *5*, 191–229.
42. Bergey, D.H.; Harrison, F.C.; Breed, R.S.; Hammer, B.W.; Huntoon, F.M. *Bergey's Manual of Determinative Bacteriology*, 1st ed.; Williams Wilkins: Baltimore, MD, USA, 1923.
43. Namekata, T.; Oliveira, A.D. Comparative serological studies between *Xanthomonas citri* and a bacterium causing canker on Mexican lime. In Proceedings of the Third International Conference on Plant Pathogenic Bacteria; Maas Geesteranus, H.P., Ed.; Centre of the Agricultural Publication and Documentation: Wageningen, The Netherlands, 1972; pp. 151–152.
44. Dye, D.W.; Bradbury, J.F.; Goto, M.; Hayward, A.C.; Lelliott, R.A.; Schroth, M.N. International standards for naming pathovars of phytopathogenic bacteria and a list of pathovar names and pathotypes. *Rev. Plant Pathol.* **1980**, *59*, 153–168.
45. Brunings, A.M.; Gabriel, D.W. *Xanthomonas citri*: Breaking the surface. *Mol. Plant Pathol.* **2003**, *4*, 141–157. [\[CrossRef\]](#)
46. Maloy, O.; Baudoin, A. Disease control principles. In *Encyclopedia of Plant Pathology*; Maloy, O.C., Murray, T.D., Eds.; Wiley: New York, NY, USA, 2001; pp. 330–332.
47. Izadiyan, M.; Taghavi, S.M.; Farahbakhsh, F. Characterization of *Xanthomonas citri* subsp. CITRI isolated from grapefruit in Iran. *J. Plant Pathol.* **2018**, *100*, 257–267. [\[CrossRef\]](#)
48. Civerolo, E. Bacterial canker disease of citrus [*Xanthomonas campestris*]. *J. Rio Gd. Val. Hortic. Soc.* **1984**, *35*, 811–818.
49. Civerolo, E. Citrus bacterial canker disease in tropical regions. *Colloques-Inra* **1994**, *66*, 45.
50. Stall, R.E.; Civerolo, E.L. Research relating to the recent outbreak of citrus canker in Florida. *Annu. Rev. Phytopathol.* **1991**, *29*, 399–420. [\[CrossRef\]](#)
51. Humphries, J. *Bacteriology*; John Murray Albermack Street: London, UK, 1974; p. 452.
52. Schaad, N.W.; Jones, J.B.; Chun, W. *Laboratory Guide for the Identification of Plant Pathogenic Bacteria*; American Phytopathological Society (APS Press): St. Paul, MN, USA, 2001.

53. Vernière, C.; Hartung, J.S.; Pruvost, O.P.; Civerolo, E.L.; Alvarez, A.M.; Maestri, P.; Luisetti, J. Characterization of phenotypically distinct strains of *Xanthomonas axonopodispv. citri* from Southwest Asia. *Eur. J. Plant Pathol.* **1998**, *104*, 477–487. [\[CrossRef\]](#)
54. Cubero, J.; Graham, J. Genetic relationship among worldwide strains of *Xanthomonas* causing canker in citrus species and design of new primers for their identification by PCR. *Appl. Environ. Microbiol.* **2002**, *68*, 1257–1264. [\[CrossRef\]](#) [\[PubMed\]](#)
55. Leite, R.; Minsavage, G.V.; Bonas, U.; Stall, R.E. Detection and identification of phytopathogenic *Xanthomonas* strains by amplification of DNA sequences related to the *hrp* genes of *Xanthomonas campestris* pv. *vesicatoria*. *Appl. Environ. Microbiol.* **1994**, *60*, 1068–1077. [\[CrossRef\]](#) [\[PubMed\]](#)
56. Vauterin, L.; Yang, P.; Hoste, B.; Vancanneyt, M.; Civerolo, E.; Swings, J.; Kersters, K. Differentiation of *Xanthomonas campestris* pv. *citri* strains by sodium dodecyl sulfate-polyacrylamide gel electrophoresis of proteins, fatty acid analysis, and DNA-DNA hybridization. *Int. J. Syst. Evol. Microbiol.* **1991**, *41*, 535–542. [\[CrossRef\]](#)
57. Wu, M.K.; Gee, A.D.; Wesselink, P.; Moor, W. Fluid transport and bacterial penetration along root canal fillings. *Int. Endod. J.* **1993**, *26*, 203–208. [\[CrossRef\]](#)
58. Egel, D.; Graham, J.; Stall, R. Genomic relatedness of *Xanthomonas campestris* strains causing diseases of citrus. *Appl. Environ. Microbiol.* **1991**, *57*, 2724–2730. [\[CrossRef\]](#)
59. Pruvost, O.; Hartung, J.; Civerolo, E.; Dubois, C.; Perrier, X. Plasmid DNA fingerprints distinguish pathotypes of *Xanthomonas campestris* pv. *citri*, the causal agent of citrus bacterial canker disease. *Phytopathology* **1992**, *82*, 485–490. [\[CrossRef\]](#)
60. Hartung, J.; Daniel, J.-F.; Pruvost, O. Detection of *Xanthomonas campestris* pv. *citri* by the polymerase chain reaction method. *Appl. Environ. Microbiol.* **1993**, *59*, 1143–1148. [\[CrossRef\]](#)
61. Zhang, M.; Meng, Q. Automatic citrus canker detection from leaf images captured in field. *Pattern Recognit. Lett.* **2011**, *32*, 2036–2046. [\[CrossRef\]](#)
62. Swarup, S.; De Feyter, R.; Brlansky, R.H.; Gabriel, D.W. A pathogenicity locus from *Xanthomonas citri* enables strains from several pathovars of *X. campestris* to elicit cankerlike lesions on citrus. *Phytopathology* **1991**, *81*, 802–809. [\[CrossRef\]](#)
63. Swarup, S.; Yang, Y.; Kingsley, M.T.; Gabriel, D.W. An *Xanthomonas citri* pathogenicity gene, *pthA*, pleiotropically encodes gratuitous avirulence on nonhosts. *Mol. Plant Microbe Interact.* **1992**, *5*, 204–213. [\[CrossRef\]](#)
64. Medina-Urrutia, V.M.; Stapleton, J.J. Control of Mexican lime bacteriosis with copper-based products. *Proc. Fla. State Hortic. Soc.* **1987**, *99*, 22–25.
65. Stapleton, J.; Garza-Lopez, J. Epidemiology of a citrus leaf-spot disease in Colima, Mexico. *Phytopathology* **1988**, *78*, 440–443. [\[CrossRef\]](#)
66. Urrutia, M. Isolation, pathogenicity, and partial host range of *Alternaria limicola*, causal agent of mancha foliar de los citricos in Mexico. *Plant Dis.* **1994**, *78*, 879.
67. Graham, J.H.; Gottwald, T. Research perspectives on eradication of citrus bacterial diseases in Florida. *Plant Dis.* **1991**, *75*, 1193–1200. [\[CrossRef\]](#)
68. *International Standards for Phytosanitary Measures (ISPM) ISPM 27 Diagnostic Protocols, DP 6: Xanthomonas citri subsp. citri*; IPPC, FAO: Rome, Italy, 2014.
69. Timmer, L. Anthracnose diseases. In *Compendium of Citrus Diseases*, 2nd ed.; Timmer, L.W., Garnsey, S.M., Graham, J.H., Eds.; APS Press: St. Paul, MN, USA, 2000; pp. 21–22.
70. Cernadas, R.A.; Benedetti, C.E. Role of auxin and gibberellin in citrus canker development and in the transcriptional control of cell-wall remodeling genes modulated by *Xanthomonas axonopodis* pv. *citri*. *Plant Sci.* **2009**, *177*, 190–195. [\[CrossRef\]](#)
71. Swings, J.; Van den Mooter, M.; Vauterin, L.; Hoste, B.; Gillis, M.; Mew, T.; Kersters, K. Reclassification of the Causal Agents of Bacterial Blight (*Xanthomonas campestris* pv. *oryzae*) and Bacterial Leaf Streak (*Xanthomonas campestris* pv. *oryzicola*) of Rice as Pathovars of *Xanthomonas oryzae* (ex Ishiyama 1922) sp. nov., nom. rev. *Int. J. Syst. Evol. Microbiol.* **1990**, *40*, 309–311. [\[CrossRef\]](#)
72. Mahaffee, W.F.; Kloepper, J.W. Temporal changes in the bacterial communities of soil, rhizosphere and endorhiza associated with field-grown cucumber (*Cucumis sativus* L.). *Microb. Ecol.* **1997**, *34*, 210–223. [\[CrossRef\]](#)
73. Holt, J.G.; Krieg, N.R.; Sneath, P.H.A.; Staley, J.T.; Williams, S.T. *Bergey's Manual of Determinative Bacteriology*, 9th ed.; Williams and Wilkins: Baltimore, MD, USA, 1994.
74. Gottwald, T.R.; Sun, X.; Riley, T.; Graham, J.H.; Ferrandino, F.; Taylor, E.L. Geo-referenced spatiotemporal analysis of the urban citrus canker epidemic in Florida. *Phytopathology* **2002**, *92*, 361–377. [\[CrossRef\]](#)
75. Graham, J.H.; Gottwald, T.R.; Cubero, J.; Achor, D.S. *Xanthomonas axonopodispv. citri*: Factors affecting successful eradication of citrus canker. *Mol. Plant Pathol.* **2004**, *5*, 1–15. [\[CrossRef\]](#)
76. Daungfu, O.; Youpensuk, S.; Lumyong, S. Endophytic Bacteria Isolated from Citrus Plants for Biological Control of Citrus Canker in Lime Plants. *Trop. Life Sci. Res.* **2019**, *30*, 73–88. [\[CrossRef\]](#)
77. Graham, J.; Gottwald, T.; Riley, T.; Achor, D. Penetration through leaf stomata and growth of strains of *Xanthomonas campestris* in citrus cultivars varying in susceptibility to bacterial diseases. *Phytopathology* **1992**, *82*, 1319–1325. [\[CrossRef\]](#)
78. Koizumi, M. *Citrus Canker: The World Situation*. *Citrus Canker: An International Perspective*; Timmer, L.W., Ed.; University of Florida: Lake Alfred, FL, USA, 1985; pp. 2–7.
79. Loucks, K.W. *Citrus Canker and Its Eradication in Florida*; Department of Agriculture, Division of Plant Industry: St. Gainesville, FL, USA, 1934.
80. Goto, M. Citrus canker. *Plant Dis. Int. Importance* **1992**, *3*, 170–208.

81. Chagas, M.; Parra, J.R.; Namekata, T.; Hartung, J.S.; Yamamoto, P.T. *Phyllocnistiscitrella* Stainton (Lepidoptera: Gracillariidae) and its relationship with the citrus canker bacterium *Xanthomonas axonopodispv citri* in Brazil. *Neotrop. Entomol.* **2001**, *30*, 55–59. [[CrossRef](#)]
82. Christiano, R.; Dalla Pria, M.; Jesus Junior, W.C.; Parra, J.R.P.; Amorim, L.; Bergamin Filho, A. Effect of citrus leaf-miner damage, mechanical damage and inoculum concentration on severity of symptoms of Asiatic citrus canker in Tahiti lime. *Crop Prot.* **2007**, *26*, 59–65. [[CrossRef](#)]
83. Hall, D.G.; Gottwald, T.R.; Bock, C.H. Exacerbation of citrus canker by citrus leafminer *Phyllocnistiscitrella* in Florida. *Fla. Entomol.* **2010**, *93*, 558–566. [[CrossRef](#)]
84. Das, A. Citrus canker—A review. *J. Appl. Hortic.* **2003**, *5*, 52–60. [[CrossRef](#)]
85. Stall, R. *Xanthomonas campestris* pv. *citri* detection and identification by enzyme-linked immunosorbent assay. *Plant Dis.* **1982**, *231*, 231–236.
86. Bergamin Filho, A.; Hughes, G. Citrus Canker Epidemiology-Methodologies and Approaches: A Moderated Discussion Session. In Proceedings of the International Citrus Canker Research Workshop, Ft. Pierce, FL, USA, 20–22 June 2022; pp. 24–25.
87. Francis, M.; Redondo, A.; Burns, J.; Graham, J. Soil application of imidacloprid and related SAR-inducing compounds produces effective and persistent control of citrus canker. *Eur. J. Plant Pathol.* **2009**, *124*, 283–292. [[CrossRef](#)]
88. Dewdney, M.; Graham, J. *Florida Citrus Pest Management Guide: Citrus Canker*; Institute of Food and Agricultural Sciences, University of Florida: Gainesville, FL, USA, 2012; p. 4.
89. Duan, Y.P.; Castaneda, A.; Zhao, G.; Erdos, G.; Gabriel, D. Expression of a single, host-specific, bacterial pathogenicity gene in plant cells elicits division, enlargement, and cell death. *Mol. Plant-Microbe Interact.* **1999**, *12*, 556–560. [[CrossRef](#)]
90. Gottwald, T.; Graham, J.; Schubert, T. An epidemiological analysis of the spread of citrus canker in urban Miami, Florida, and synergistic interaction with the Asian citrus leafminer. *Fruits* **1997**, *6*, 383–390.
91. Graham, J.; Gottwald, T.; Riley, T.; Cubero, J.; Drouillard, D. Survival of *Xanthomonas campestris* pv. *citri* (Xcc) on various surfaces and chemical control of Asiatic Citrus Canker (ACC). In Proceedings of the International Citrus Canker Research Workshop, Ft. Pierce, FL, USA, 20–22 June 2000; p. 7.
92. Rao, Y.; Hingorani, M. Survival of *Xanthomonas citri* (Hasse) Dowson in leaves and soil. *Indian Phytopath* **1963**, *16*, 362–364.
93. Verniere, C.; Gottwald, T.; Pruvost, O. Disease development and symptom expression of *Xanthomonas axonopodispv. citri* in various citrus plant tissues. *Phytopathology* **2003**, *93*, 832–843. [[CrossRef](#)] [[PubMed](#)]
94. Goto, M.; Serizawa, S.; Morita, M. *Studies on Citrus Canker Disease. III. Survival of Xanthomonas Citri (Hasse) Dowson in Soils and on the Surface of Weeds*; Bulletin of the Faculty of Agriculture, Shizuoka University: Shizuoka, Japan, 1970; Volume 20, pp. 21–29.
95. Goto, M. Survival of *Xanthomonas citri* in the bark tissues of citrus trees. *Can. J. Bot.* **1972**, *50*, 2629–2635. [[CrossRef](#)]
96. Leite, R., Jr.; Mohan, S. Evaluation of citrus cultivars for resistance to canker caused by *Xanthomonas campestris* pv. *citri* (Hasse) Dye in the State of Paraná, Brazil. *Proc. Int. Soc. Citric.* **1984**, *1*, 385–389.
97. Graham, J.; Gottwald, T.; Civerolo, E.; McGuire, R. Population dynamics and survival of *Xanthomonas campestris* in soil in citrus nurseries in Maryland and Argentina. *Plant Dis.* **1989**, *43*, 423–427. [[CrossRef](#)]
98. Teper, D.; Pandey, S.S.; Wang, N. The HrpG/HrpX Regulon of *Xanthomonads*—An Insight to the Complexity of Regulation of Virulence Traits in Phytopathogenic Bacteria. *Microorganisms* **2021**, *9*, 187. [[CrossRef](#)]
99. Luthra, J.C.; Sattar, A. Citrus canker and its control in Punjab. *Punjab Fruit J.* **1942**, *6*, 179–182.
100. Stall, R.E.; Miller, J.; Marco, G.M.; de Echenique, B.C. Population dynamics of *Xanthomonas citri* causing canker of citrus in Argentina. In Proceedings of the Florida State Horticultural Society; Florida State Horticultural Society: Alexandria, VA, USA, 1980; pp. 10–14.
101. Traoré, Y.N.; Ngoc, L.B.T.; Vernière, C.; Pruvost, O. First report of *Xanthomonas citri* pv. *citri* causing citrus canker in Mali. *Plant Dis.* **2008**, *92*, 977. [[CrossRef](#)]
102. Heppner, J.B. Citrus leafminer, *Phyllocnistiscitrella*, in Florida (Lepidoptera: Gracillariidae: Phyllocnistinae). *Trop. Lepid. Res.* **1993**, *1*, 49–64.
103. Parsai, P.S. Citrus canker. In Proceedings of the Seminar on Diseases of Horticultural Plants, Simla, India, 10–15 June 1959; pp. 91–95.
104. Bacon, C.W.; Hinton, D.M. Endophytic and biological control potential of *Bacillus mojavenis* and related species. *Biol. Control* **2002**, *23*, 274–284. [[CrossRef](#)]
105. Knapp, J. *Citrus Leafminer, Phyllocnistiscitrella Stainton: Current Status in Florida-1995*; University of Florida: Gainesville, FL, USA, 1995.
106. Peña, J.E.; Hunsberger, A.; Schaffer, B. Citrus leafminer (Lepidoptera: Gracillariidae) density: Effect on yield of ‘Tahiti’ lime. *J. Econ. Entomol.* **2000**, *93*, 374–379. [[CrossRef](#)] [[PubMed](#)]
107. Behlau, F.; Belasque, J. *Cancro Citrico—A Doença e Seu Controle*; Fundecitrus: Araraquara, Brazil, 2014.
108. Coletta-Filho, H.D.; Takita, M.A.; Souza, A.A.; Neto, J.R.; Destefano, S.A.L.; Hartung, J.S.; Machado, M.A. Primers based on the rpf gene region provide improved detection of *Xanthomonas axonopodis* pv. *citri* in naturally and artificially infected citrus plants. *J. Appl. Microbiol.* **2006**, *100*, 279–285. [[CrossRef](#)] [[PubMed](#)]
109. Hartung, J.S. Plasmid-based hybridization probes for detection and identification of *Xanthomonas campestris* pv. *citri*. *Plant Dis.* **1992**, *76*, 889–893. [[CrossRef](#)]



110. Mavrodieva, V.; Levy, L.; Gabriel, D.W. Improved sampling methods for real-time polymerase chain reaction diagnosis of citrus canker from field samples. *Phytopathology* **2004**, *94*, 61–68. [[CrossRef](#)] [[PubMed](#)]
111. Sun, X.; Stall, R.E.; Jones, J.B.; Cubero, J.; Gottwald, T.R.; Graham, J.H.; Dixon, W.N.; Schubert, T.S.; Chaloux, P.H.; Stromberg, V.K.; et al. Detection and characterization of a new strain of citrus canker bacteria from key Mexican lime and alemow in South Florida. *Plant Dis.* **2004**, *88*, 1179–1188. [[CrossRef](#)] [[PubMed](#)]
112. Cubero, J.; Graham, J.H. Quantitative real-time polymerase chain reaction for bacterial enumeration and allelic discrimination to differentiate *Xanthomonas* strains on citrus. *Phytopathology* **2005**, *95*, 1333–1340. [[CrossRef](#)] [[PubMed](#)]
113. Golmohammadi, M.; Cubero, J.; Penalver, J.; Quesada, J.M.; Lopez, M.M.; Llop, P. Diagnosis of *Xanthomonas axonopodis* pv. citri, causal agent of citrus canker, in commercial fruits by isolation and PCR-based methods. *J. Appl. Microbiol.* **2007**, *103*, 2309–2315. [[CrossRef](#)]
114. Park, D.S.; Wook Hyun, J.; Jin Park, Y.; Sun Kim, J.; Wan Kang, H.; Ho Hahn, J.; Joo Go, S. Sensitive and specific detection of *Xanthomonas axonopodis* pv. citri by PCR using pathovar specific primers based on hrpW gene sequences. *Microbiol. Res.* **2006**, *161*, 145–149. [[CrossRef](#)]
115. Rigano, L.A.; Siciliano, F.; Enrique, R.; Sendin, L.; Filippone, P.; Torres, P.S.; Qüesta, J.; Dow, J.M.; Castagnaro, A.P.; Vojnov, A.A. Biofilm formation, epiphytic fitness, and canker development in *Xanthomonas axonopodis* pv. citri. *Mol. Plant-Microbe Interact.* **2007**, *20*, 1222–1230. [[CrossRef](#)]
116. Hartung, J.S.; Pruvost, O.P.; Villemot, I.; Alvarez, A. Rapid and sensitive colorimetric detection of *Xanthomonas axonopodis* pv. citri by immunocapture and a nested-polymerase chain reaction assay. *Pathology* **1996**, *8695*, 101.
117. Gabriel, D.; Gottwald, T.R.; Lopes, S.A.; Wulff, N.A. Bacterial pathogens of citrus: Citrus canker, citrus variegated chlorosis and Huanglongbing. In *The Genus Citrus*; Woodhead Publishing: Sawston, UK, 2020; pp. 371–389.
118. Cubero, J.; Graham, J.; Gottwald, T. Quantitative PCR method for diagnosis of citrus bacterial canker. *Appl. Environ. Microbiol.* **2001**, *67*, 2849–2852. [[CrossRef](#)] [[PubMed](#)]
119. Qi, M.; Yang, Y. Quantification of *Magnaporthe grisea* during infection of rice plants using real-time polymerase chain reaction and northern blot/phosphoimaging analyses. *Phytopathology* **2002**, *92*, 870–876. [[CrossRef](#)] [[PubMed](#)]
120. Winton, L.; Stone, J.; Watrud, L.; Hansen, E. Simultaneous one-tube quantification of host and pathogen DNA with real-time polymerase chain reaction. *Phytopathology* **2002**, *92*, 112–116. [[CrossRef](#)] [[PubMed](#)]
121. Vandemark, G.; Barker, B.; Gritsenko, M. Quantifying *Aphanomyces euteiches* in alfalfa with a fluorescent polymerase chain reaction assay. *Phytopathology* **2002**, *92*, 265–272. [[CrossRef](#)]
122. Schaad, N.; Berthier-Schaad, Y.; Sechler, A.; Knorr, D. Detection of *Clavibacter michiganensis* subsp. *sepedonicus* in potato tubers by BIO-PCR and an automated real-time fluorescence detection system. *Plant Dis.* **1999**, *83*, 1095–1100. [[CrossRef](#)]
123. Schaad, N.W.; Frederick, R.D. Real-time PCR and its application for rapid plant disease diagnostics. *Can. J. Plant Pathol.* **2002**, *24*, 250–258. [[CrossRef](#)]
124. Weller, S.; Elphinstone, J.; Smith, N.; Stead, D. Detection of *Ralstonia solanacearum* from potato tissue by post-enrichment TaqMan PCR. *EPPO Bull.* **2000**, *30*, 381–383. [[CrossRef](#)]
125. Roberts, C.A.; Dietzgen, R.G.; Heelan, L.A.; Maclean, D.J. Real-time RT-PCR fluorescent detection of tomato spotted wilt virus. *J. Virol. Methods* **2000**, *88*, 1–8. [[CrossRef](#)]
126. Mumford, R.; Walsh, K.; Boonham, N. A comparison of molecular methods for the routine detection of viroids. *EPPO Bull.* **2000**, *30*, 431–435. [[CrossRef](#)]
127. Mackay, I.M.; Arden, K.E.; Nitsche, A. Real-time PCR in virology. *Nucleic Acids Res.* **2002**, *30*, 1292–1305. [[CrossRef](#)]
128. Al-Saleh, M.A.; Widiyawan, A.; Saleh, A.A.; Ibrahim, Y.E. Distribution and pathotype identification of *Xanthomonas citri* subsp. citri recovered from southwestern region of Saudi Arabia. *Afr. J. Microbiol. Res.* **2014**, *8*, 673–679.
129. Adriko, J.; Aritua, V.; Mortensen, C.N.; Tushemereirwe, W.K.; Kubiriba, J.; Lund, O.S. Multiplex PCR for specific and robust detection of *Xanthomonas campestris* pv. *musacearum* in pure culture and infected plant material. *Plant Pathol.* **2012**, *61*, 489–497. [[CrossRef](#)]
130. Weisburg, W.G.; Barns, S.M.; Pelletier, D.A.; Lane, D.J. 16S ribosomal DNA amplification for phylogenetic study. *J. Bacteriol.* **1991**, *173*, 697–703. [[CrossRef](#)] [[PubMed](#)]
131. Adriko, J.; Mbega, E.R.; Mortensen, C.N.; Wulff, E.G.; Tushemereirwe, W.K.; Kubiriba, J.; Lund, O.S. Improved PCR for identification of members of the genus *Xanthomonas*. *Eur. J. Plant Pathol.* **2014**, *138*, 293–306. [[CrossRef](#)]
132. da Silva, A.R.; Ferro, J.A.; Reinach, F.d.C.; Farah, C.S.; Furlan, L.R.; Quaggio, R.B.; Monteiro-Vitorello, C.B.; Van Sluys, M.-A.; Almeida, N.A.; Alves, L. Comparison of the genomes of two *Xanthomonas* pathogens with differing host specificities. *Nature* **2002**, *417*, 459–463. [[CrossRef](#)] [[PubMed](#)]
133. Kalita, P.; Bora, L.C.; Bhagabati, K.N. Phylloplane microflora of citrus and their role in management of citrus canker. *Indian Phytopathol.* **1996**, *49*, 234–237.
134. Jalan, N.; Kumar, D.; Andrade, M.O.; Yu, F.; Jones, F.B.; Graham, J.H.; White, F.F.; Setubal, J.C.; Wang, N. Comparative genomic and transcriptome analyses of pathotypes of *Xanthomonas citri* subsp. citri provide insights into mechanisms of bacterial virulence and host range. *BMC Genom.* **2013**, *14*, 551. [[CrossRef](#)]
135. Hartung, J.S.; Civerolo, E. Genomic Fingerprints of *Xanthomonas campestris* pv. citri Strains. *Phytopathology* **1987**, *77*, 282–285. [[CrossRef](#)]

136. Lazo, G.R.; Roffey, R.; Gabriel, D.W. Pathovars of *Xanthomonas campestris* are distinguishable by restriction fragment-length polymorphism. *Int. J. Syst. Evol. Microbiol.* **1987**, *37*, 214–221. [\[CrossRef\]](#)
137. Cooksey, D.A.; Graham, J.H. Genomic fingerprinting of two pathovars of phytopathogenic bacteria by rare-cutting restriction enzymes and field inversion gel electrophoresis. *Phytopathology* **1989**, *79*, 745–750. [\[CrossRef\]](#)
138. Leach, J.; White, F.; Rhoads, M.; Leung, H. A repetitive DNA sequence differentiates *Xanthomonas campestris* pv. *oryzae* from other pathovars of *X. campestris*. *Mol. Plant-Microbe Interact.* **1990**, *3*, 238–246. [\[CrossRef\]](#)
139. Pooler, M.R.; Ritchie, D.F.; Hartung, J.S. Genetic relationships among strains of *Xanthomonas fragariae* based on random amplified polymorphic DNA PCR, repetitive extragenic palindromic PCR, and enterobacterial repetitive intergenic consensus PCR data and generation of multiplexed PCR primers useful for the identification of this phytopathogen. *Appl. Environ. Microbiol.* **1996**, *62*, 3121–3127. [\[PubMed\]](#)
140. Hauben, L.; Vauterin, L.; Swings, J.; Moore, E. Comparison of 16S ribosomal DNA sequences of all *Xanthomonas* species. *Int. J. Syst. Evol. Microbiol.* **1997**, *47*, 328–335. [\[CrossRef\]](#) [\[PubMed\]](#)
141. Van Sluys, M.A.; Monteiro-Vitorello, C.B.; Camargo, L.E.A.; Menck, C.F.M.; Da Silva, A.C.R.; Ferro, J.A.; Simpson, A.J. Comparative genomic analysis of plant-associated bacteria. *Annu. Rev. Phytopathol.* **2002**, *40*, 169–189. [\[CrossRef\]](#) [\[PubMed\]](#)
142. Dunger, G.; Relling, V.M.; Tondo, M.L.; Barreras, M.; Ielpi, L.; Orellano, E.G.; Ottado, J. Xanthan is not essential for pathogenicity in citrus canker but contributes to *Xanthomonas* epiphytic survival. *Arch. Microbiol.* **2007**, *188*, 127–135. [\[CrossRef\]](#)
143. Rossier, O.; Van den Ackerveken, G.; Bonas, U. HrpB2 and HrpF from *Xanthomonas* are type III-secreted proteins and essential for pathogenicity and recognition by the host plant. *Mol. Microbiol.* **2000**, *38*, 828–838. [\[CrossRef\]](#)
144. Guo, Y.; Figueiredo, F.; Jones, J.; Wang, N. HrpG and HrpX play global roles in coordinating different virulence traits of *Xanthomonas axonopodis* pv. *citri*. *Mol. Plant-Microbe Interact.* **2011**, *24*, 649–661. [\[CrossRef\]](#)
145. Yamazaki, A.; Hirata, H.; Tsuyumu, S. HrpG regulates type II secretory proteins in *Xanthomonas axonopodis* pv. *citri*. *J. Gen. Plant Pathol.* **2008**, *74*, 138–150. [\[CrossRef\]](#)
146. Yamazaki, A.; Hirata, H.; Tsuyumu, S. Type III regulators hrpG and hrpXct control synthesis of alpha-amylase, which is involved in in planta multiplication of *Xanthomonas axonopodis* pv. *citri*. *J. Gen. Plant Pathol.* **2008**, *74*, 254–257. [\[CrossRef\]](#)
147. Sturz, A.V.; Christie, B.R.; Nowak, J. Bacterial endophytes: Potential role in developing sustainable systems of crop production. *Crit. Rev. Plant Sci.* **2000**, *19*, 1–30. [\[CrossRef\]](#)
148. Jaciani, F.J.; Ferro, J.A.; Ferro, M.I.T.; Vernière, C.; Pruvost, O.; Belasque, J., Jr. Genetic diversity of a Brazilian strain collection of *Xanthomonas citri* subsp. *citri* based on the type III effector protein genes. *Plant Dis.* **2012**, *96*, 193–203. [\[CrossRef\]](#) [\[PubMed\]](#)
149. Mysore, K.S.; Ryu, C.M. Nonhost resistance: How much do we know? *Trends Plant Sci.* **2004**, *9*, 97–104. [\[CrossRef\]](#) [\[PubMed\]](#)
150. Weber, E.; Berger, C.; Bonas, U.; Koebnik, R. Refinement of the *Xanthomonas campestris* pv. *vesicatoria* hrpD and hrpE operon structure. *Mol. Plant-Microbe Interact.* **2007**, *20*, 559–567. [\[CrossRef\]](#) [\[PubMed\]](#)
151. Dunger, G.; Arabolaza, A.; Gottig, N.; Orellano, E.; Ottado, J. Participation of *Xanthomonas axonopodis* pv. *citri* hrp cluster in citrus canker and nonhost plant responses. *Plant Pathol.* **2005**, *54*, 781–788. [\[CrossRef\]](#)
152. Dunger, G.; Garofalo, C.G.; Gottig, N.; Garavaglia, B.S.; Rosa, M.C.P.; Farah, C.S.; Orellano, E.G.; Ottado, J. Analysis of three *Xanthomonas axonopodis* pv. *citri* effector proteins in pathogenicity and their interactions with host plant proteins. *Mol. Plant Pathol.* **2012**, *13*, 865–876. [\[CrossRef\]](#)
153. Sgro, G.G.; Ficarra, F.A.; Dunger, G.; Scarpeci, T.E.; Valle, E.M.; Cortadi, A.; Orellano, E.G.; Gottig, N.; Ottado, J. Contribution of a harpin protein from *Xanthomonas axonopodis* pv. *citri* to pathogen virulence. *Mol. Plant Pathol.* **2012**, *13*, 1047–1059. [\[CrossRef\]](#)
154. Al-Saadi, A.; Reddy, J.D.; Duan, Y.P.; Brunings, A.M.; Yuan, Q.; Gabriel, D.W. All five host-range variants of *Xanthomonas citri* carry one pthA homolog with 17.5 repeats that determines pathogenicity on citrus, but none determine host-range variation. *Mol. Plant-Microbe Interact.* **2007**, *20*, 934–943. [\[CrossRef\]](#)
155. Alegria, M.C.; Docena, C.; Khater, L.; Ramos, C.H.; Da Silva, A.C.; Farah, C.S. New protein-protein interactions identified for the regulatory and structural components and substrates of the type III Secretion system of the phytopathogen *Xanthomonas axonopodis* Pathovar *citri*. *J. Bacteriol.* **2004**, *186*, 6186–6197. [\[CrossRef\]](#)
156. Büttner, D.; Bonas, U. Regulation and secretion of *Xanthomonas* virulence factors. *FEMS Microbiol. Rev.* **2010**, *34*, 107–133. [\[CrossRef\]](#)
157. Boch, J.; Bonas, U. *Xanthomonas* AvrBs3 family-type III effectors: Discovery and function. *Annu. Rev. Phytopathol.* **2010**, *48*, 419–436. [\[CrossRef\]](#)
158. Leach, J.E.; White, F.F. Bacterial avirulence genes. *Annu. Rev. Phytopathol.* **1996**, *34*, 153–179. [\[CrossRef\]](#) [\[PubMed\]](#)
159. Chakrabarty, P.; Duan, Y.; Gabriel, D. Cloning and characterization of a member of the *Xanthomonas* avr/pth gene family that evades all commercially utilized cotton R genes in the United States. *Phytopathology* **1997**, *87*, 1160–1167. [\[CrossRef\]](#) [\[PubMed\]](#)
160. Moreira, L.M.; Almeida, N.F.; Potnis, N.; Digiampietri, L.A.; Adi, S.S.; Bortolossi, J.C.; da Silva, A.C.; da Silva, A.M.; de Moraes, F.E.; de Oliveira, J.C. Novel insights into the genomic basis of citrus canker based on the genome sequences of two strains of *Xanthomonas fuscans* subsp. *aurantifolii*. *BMC Genom.* **2010**, *11*, 238. [\[CrossRef\]](#) [\[PubMed\]](#)
161. Gabriel, D. Why do pathogens carry avirulence genes? *Physiol. Mol. Plant Pathol.* **1999**, *55*, 205–214. [\[CrossRef\]](#)
162. Koizumi, M.; Kochinotsu, B. Relation of temperature to the development of citrus canker lesions in the spring. *Proc. Int. Soc. Citric.* **1977**, *3*, 924–928.
163. Kanamori, H.; Tsuyumu, S. Comparison of nucleotide sequences of canker-forming and non-canker-forming pthA homologues in *Xanthomonas campestris* pv. *citri*. *Jpn. J. Phytopathol.* **1998**, *64*, 462–470. [\[CrossRef\]](#)

164. Yang, B.; Zhu, W.; Johnson, L.B.; White, F.F. The virulence factor AvrXa7 of *Xanthomonas oryzae* pv. *oryzae* is a type III secretion pathway-dependent nuclear-localized double-stranded DNA-binding protein. *Proc. Natl. Acad. Sci. USA* **2000**, *97*, 9807–9812. [\[CrossRef\]](#)
165. Wengelnik, K.; Bonas, U. HrpXv, an AraC-type regulator, activates expression of five of the six loci in the hrp cluster of *Xanthomonas campestris* pv. *vesicatoria*. *J. Bacteriol.* **1996**, *178*, 3462–3469. [\[CrossRef\]](#)
166. Wengelnik, K.; Van den Ackerveken, G.; Bonas, U. HrpG, a key hrp regulatory protein of *Xanthomonas campestris* pv. *vesicatoria* is homologous to two-component response regulators. *Mol. Plant-Microbe Interact.* **1996**, *9*, 704–712. [\[CrossRef\]](#)
167. Laia, M.L.; Moreira, L.M.; Dezajacomo, J.; Brigati, J.B.; Ferreira, C.B.; Ferro, M.I.; Silva, A.C.; Ferro, J.A.; Oliveira, J.C. New genes of *Xanthomonas citri* subsp. *citri* involved in pathogenesis and adaptation revealed by a transposon-based mutant library. *BMC Microbiol.* **2009**, *9*, 12. [\[CrossRef\]](#)
168. Kingsley, M.T.; Gabriel, D.W.; Marlow, G.C.; Roberts, P.D. The opsX locus of *Xanthomonas campestris* affects host range and biosynthesis of lipopolysaccharide and extracellular polysaccharide. *J. Bacteriol.* **1993**, *175*, 5839–5850. [\[CrossRef\]](#) [\[PubMed\]](#)
169. Casabuono, A.; Petrocelli, S.; Ottado, J.; Orellano, E.G.; Couto, A.S. Structural analysis and involvement in plant innate immunity of *Xanthomonas axonopodis* pv. *citri* lipopolysaccharide. *J. Biol. Chem.* **2011**, *286*, 25628–25643. [\[CrossRef\]](#) [\[PubMed\]](#)
170. Zimaro, T.; Thomas, L.; Marondedze, C.; Sgro, G.G.; Garofalo, C.G.; Ficarra, F.A.; Gottig, N. The type III protein secretion system contributes to *Xanthomonas citri* subsp. *citri* biofilm formation. *BMC Microbiol.* **2014**, *14*, 1–15. [\[CrossRef\]](#)
171. Jansson, P.-E.; Kenne, L.; Lindberg, B. Structure of the extracellular polysaccharide from *Xanthomonas campestris*. *Carbohydr. Res.* **1975**, *45*, 275–282. [\[CrossRef\]](#)
172. Becker, A.; Katzen, F.; Pühler, A.; Ielpi, L. Xanthan gum biosynthesis and application: A biochemical/genetic perspective. *Appl. Microbiol. Biotechnol.* **1998**, *50*, 145–152. [\[CrossRef\]](#) [\[PubMed\]](#)
173. Vojnov, A.A.; Zorreguieta, A.; Dow, J.M.; Daniels, M.J.; Dankert, M.A. Evidence for a role for the gumB and gumC gene products in the formation of xanthan from its pentasaccharide repeating unit by *Xanthomonas campestris*. *Microbiology* **1998**, *144*, 1487–1493. [\[CrossRef\]](#) [\[PubMed\]](#)
174. Tang, J.-L.; Liu, Y.-N.; Barber, C.; Dow, J.; Wootton, J.; Daniels, M. Genetic and molecular analysis of a cluster of rpf genes involved in positive regulation of synthesis of extracellular enzymes and polysaccharide in *Xanthomonas campestris* pathovar *campestris*. *Mol. Gen. Genet. MGG* **1991**, *226*, 409–417. [\[CrossRef\]](#)
175. Denny, T. Involvement of bacterial polysaccharides in plant pathogenesis. *Annu. Rev. Phytopathol.* **1995**, *33*, 173–197. [\[CrossRef\]](#)
176. Chan, J.W.; Goodwin, P.H. The molecular genetics of virulence of *Xanthomonas campestris*. *Biotechnol. Adv.* **1999**, *17*, 489–508. [\[CrossRef\]](#)
177. Hao, G.; Stover, E.; Gupta, G. Overexpression of a modified plant thionin enhances disease resistance to citrus canker and huanglongbing (HLB). *Front. Plant Sci.* **2016**, *7*, 1078. [\[CrossRef\]](#)
178. Duan, S.; Jia, H.; Pang, Z.; Teper, D.; White, F.; Jones, J.; Zhou, C.; Wang, N. Functional characterization of the citrus canker susceptibility gene CsLOB1. *Mol. Plant Pathol.* **2018**, *19*, 1908–1916. [\[CrossRef\]](#) [\[PubMed\]](#)
179. Chou, F.-L.; Chou, H.-C.; Lin, Y.-S.; Yang, B.-Y.; Lin, N.-T.; Weng, S.-F.; Tseng, Y.-H. The *Xanthomonas campestris* gumD Gene Required for Synthesis of Xanthan Gum Is Involved in Normal Pigmentation and Virulence in Causing Black Rot. *Biochem. Biophys. Res. Commun.* **1997**, *233*, 265–269. [\[CrossRef\]](#) [\[PubMed\]](#)
180. Dharmapuri, S.; Sonti, R.V. A transposon insertion in the gumG homologue of *Xanthomonas oryzae* pv. *oryzae* causes loss of extracellular polysaccharide production and virulence. *FEMS Microbiol. Lett.* **1999**, *179*, 53–59. [\[CrossRef\]](#) [\[PubMed\]](#)
181. Kemp, B.P.; Horne, J.; Bryant, A.; Cooper, R.M. *Xanthomonas axonopodis* pv. *manihotis* gumD gene is essential for EPS production and pathogenicity and enhances epiphytic survival on cassava (*Manihot esculenta*). *Physiol. Mol. Plant Pathol.* **2004**, *64*, 209–218. [\[CrossRef\]](#)
182. Kim, J.-G.; Li, X.; Roden, J.A.; Taylor, K.W.; Aakre, C.D.; Su, B.; Lalonde, S.; Kirik, A.; Chen, Y.; Baranage, G. *Xanthomonas* T3S effector XopN suppresses PAMP-triggered immunity and interacts with a tomato atypical receptor-like kinase and TGT1. *Plant Cell* **2009**, *21*, 1305–1323. [\[CrossRef\]](#)
183. Siciliano, F.; Torres, P.; Sendín, L.; Bermejo, C.; Filippone, P.; Vellice, G.; Ramallo, J.; Castagnaro, A.; Vojnov, A.; Marano, M.R. Analysis of the molecular basis of *Xanthomonas axonopodis* pv. *citri* pathogenesis in Citrus limon. *Electron. J. Biotechnol.* **2006**, *9*, 3–13. [\[CrossRef\]](#)
184. Huang, Q.; Wu, J.; Li, X.; Liu, M.; Kong, Y. Toxicity and biochemical action of amicarbazol on citrus canker pathogen, *Xanthomonas citri* ex Hasse. *Pesticide Biochem. Physiol.* **2006**, *84*, 188–195. [\[CrossRef\]](#)
185. Goto, M.; Hyodo, H. Role of extracellular polysaccharides of *Xanthomonas campestris* pv. *citri* in the early stage of infection. *Jpn. J. Phytopathol.* **1985**, *51*, 22–31. [\[CrossRef\]](#)
186. Takahashi, T.; Doke, N. A role of extracellular polysaccharides of *Xanthomonas campestris* pv. *citri* in bacterial adhesion to citrus leaf tissues in preinfectious stage. *Jpn. J. Phytopathol.* **1984**, *50*, 565–573. [\[CrossRef\]](#)
187. Yun, M.H.; Torres, P.S.; El Oirdi, M.; Rigano, L.A.; Gonzalez-Lamothe, R.; Marano, M.R.; Castagnaro, A.P.; Dankert, M.A.; Bouarab, K.; Vojnov, A.A. Xanthan induces plant susceptibility by suppressing callose deposition. *Plant Physiol.* **2006**, *141*, 178–187. [\[CrossRef\]](#)
188. Aslam, S.N.; Newman, M.-A.; Erbs, G.; Morrissey, K.L.; Chinchilla, D.; Boller, T.; Jensen, T.T.; De Castro, C.; Ierano, T.; Molinaro, A. Bacterial polysaccharides suppress induced innate immunity by calcium chelation. *Curr. Biol.* **2008**, *18*, 1078–1083. [\[CrossRef\]](#) [\[PubMed\]](#)



189. Dow, J.M.; Crossman, L.; Findlay, K.; He, Y.-Q.; Feng, J.-X.; Tang, J.-L. Biofilm dispersal in *Xanthomonas campestris* is controlled by cell–cell signaling and is required for full virulence to plants. *Proc. Natl. Acad. Sci. USA* **2003**, *100*, 10995–11000. [[CrossRef](#)] [[PubMed](#)]
190. Limoli, D.H.; Jones, C.J.; Wozniak, D.J. Bacterial Extracellular Polysaccharides in Biofilm Formation and Function. *Microbiol. Spectr.* **2015**, *26*, 3. [[CrossRef](#)]
191. Branda, S.S.; Vik, Å.; Friedman, L.; Kolter, R. Biofilms: The matrix revisited. *Trends Microbiol.* **2005**, *13*, 20–26. [[CrossRef](#)]
192. Sutherland, I.W. Microbial polysaccharides from Gram-negative bacteria. *Int. Dairy J.* **2001**, *11*, 663–674. [[CrossRef](#)]
193. Stoodley, P.; Sauer, K.; Davies, D.G.; Costerton, J.W. Biofilms as complex differentiated communities. *Annu. Rev. Microbiol.* **2002**, *56*, 187–209. [[CrossRef](#)] [[PubMed](#)]
194. Hilario, E.; De Keyser, S.; Fan, L. Structural and biochemical characterization of a glutathione transferase from the citrus canker pathogen *Xanthomonas*. *Acta Crystallogr. Sect. D Struct. Biol.* **2020**, *76*, 778–789. [[CrossRef](#)]
195. Omar, A.A.; Murata, M.M.; El-Shamy, H.A.; Graham, J.H.; Grosser, J.W. Enhanced resistance to citrus canker in transgenic mandarin expressing Xa21 from rice. *Transgenic Res.* **2018**, *27*, 179–191. [[CrossRef](#)]
196. Vorhölter, F.-J.; Niehaus, K.; Pühler, A. Lipopolysaccharide biosynthesis in *Xanthomonas campestris* pv. *campestris*: A cluster of 15 genes is involved in the biosynthesis of the LPS O-antigen and the LPS core. *Mol. Genet. Genom.* **2001**, *266*, 79–95. [[CrossRef](#)]
197. Dow, J.M.; Osbourn, A.E.; Wilson, T.G.; Daniels, M.J. A locus determining pathogenicity of *Xanthomonas campestris* is involved in lipopolysaccharide biosynthesis. *MPMI-Mol. Plant Microbe Interact.* **1995**, *8*, 768–777. [[CrossRef](#)]
198. Petrocelli, S.; Tondo, M.L.; Daurelio, L.D.; Orellano, E.G. Modifications of *Xanthomonas axonopodispv. citri* lipopolysaccharide affect the basal response and the virulence process during citrus canker. *PLoS ONE* **2012**, *7*, e40051. [[CrossRef](#)] [[PubMed](#)]
199. Patil, P.B.; Bogdanove, A.J.; Sonti, R.V. The role of horizontal transfer in the evolution of a highly variable lipopolysaccharide biosynthesis locus in xanthomonads that infect rice, citrus and crucifers. *BMC Evol. Biol.* **2007**, *7*, 243. [[CrossRef](#)] [[PubMed](#)]
200. Li, J.; Wang, N. Genome-wide mutagenesis of *Xanthomonas axonopodispv. citri* reveals novel genetic determinants and regulation mechanisms of biofilm formation. *PLoS ONE* **2011**, *6*, e21804.
201. Waters, C.M.; Bassler, B.L. Quorum sensing: Cell-to-cell communication in bacteria. *Annu. Rev. Cell Dev. Biol.* **2005**, *21*, 319–346. [[CrossRef](#)]
202. Papenfort, K.; Bassler, B.L. Quorum sensing signal–response systems in Gram-negative bacteria. *Nat. Rev. Microbiol.* **2016**, *14*, 576. [[CrossRef](#)]
203. Ryan, R.P.; Vorhölter, F.-J.; Potnis, N.; Jones, J.B.; Van Sluys, M.-A.; Bogdanove, A.J.; Dow, J.M. Pathogenomics of *Xanthomonas*: Understanding bacterium–plant interactions. *Nat. Rev. Microbiol.* **2011**, *9*, 344–355. [[CrossRef](#)]
204. Ryan, R.P.; An, S.-Q.; Allan, J.H.; McCarthy, Y.; Dow, J.M. The DSF family of cell–cell signals: An expanding class of bacterial virulence regulators. *PLoS Pathog.* **2015**, *11*, e1004986. [[CrossRef](#)]
205. Dow, J.M. Diffusible signal factor-dependent quorum sensing in pathogenic bacteria and its exploitation for disease control. *J. Appl. Microbiol.* **2017**, *122*, 2–11. [[CrossRef](#)]
206. Guo, Y.; Zhang, Y.; Li, J.-L.; Wang, N. Diffusible signal factor-mediated quorum sensing plays a central role in coordinating gene expression of *Xanthomonas citri* subsp. *citri*. *Mol. Plant-Microbe Interact.* **2012**, *25*, 165–179. [[CrossRef](#)]
207. Tan, X.; Huang, S.; Ren, J.; Yan, W.; Cen, Z. Study on a bacterial strain Bt8 for biocontrol against citrus bacterial canker. *Wei Sheng Wuxue Bao Acta Microbiol.* **2006**, *46*, 292–296.
208. Balogh, B.; Canteros, B.I.; Stall, R.E.; Jones, J.B. Control of citrus canker and citrus bacterial spot with bacteriophages. *Plant Dis.* **2008**, *92*, 1048–1052. [[CrossRef](#)] [[PubMed](#)]
209. McCollum, G.; Bowman, K.; Gottwald, T. (262) Screening Citrus Germplasm for Resistance to *Xanthomonas axonopodispv. Citri*. *HortScience* **2006**, *41*, 1048E–1049E. [[CrossRef](#)]
210. Lahaye, T.; Bonas, U. Molecular secrets of bacterial type III effector proteins. *Trends Plant Sci.* **2001**, *6*, 479–485. [[CrossRef](#)]
211. Cernadas, R.A.; Camillo, L.R.; Benedetti, C.E. Transcriptional analysis of the sweet orange interaction with the citrus canker pathogens *Xanthomonas axonopodispv. citri* and *Xanthomonas axonopodispv. aurantifolii*. *Mol. Plant Pathol.* **2008**, *9*, 609–631. [[CrossRef](#)] [[PubMed](#)]
212. Leite, R., Jr.; Mohan, S. Integrated management of the citrus bacterial canker disease caused by *Xanthomonas campestris* pv. *citri* in the State of Paraná, Brazil. *Crop Prot.* **1990**, *9*, 3–7. [[CrossRef](#)]
213. Nazaré, A.C.; Polaquini, C.R.; Cavalca, L.B.; Anselmo, D.B.; Saiki MD, F.C.; Monteiro, D.A.; Zielinska, A.; Rahal, P.; Gomes, E.; Scheffers, D.; et al. Design of antibacterial agents: Alkyl dihydroxybenzoates against *Xanthomonas citri* subsp. *citri*. *Int. J. Mol. Sci.* **2018**, *19*, 3050. [[CrossRef](#)]
214. Stall, R.E.; Seymour, C.P. Canker, a threat to citrus in the Gulf-Coast states. *Plant Dis.* **1983**, *67*, 581–585. [[CrossRef](#)]
215. Behlau, F.; Barelli, N.; Belasque, J., Jr. Lessons from a case of successful eradication of citrus canker in a citrus-producing farm in São Paulo State, Brazil. *J. Plant Pathol.* **2014**, *96*, 561–568.
216. Stein, B.; Ramallo, J.; Foguet, L.; Graham, J.H. Citrus leafminer control and copper sprays for management of citrus canker on lemon in Tucuman, Argentina. *Florida State Hortic. Soc.* **2007**, *120*, 127–131.
217. McGuire, R.G. Evaluation of bactericidal chemicals for control of *Xanthomonas* on citrus. *Plant Dis.* **1988**, *72*, 1016–1020. [[CrossRef](#)]



218. Bock, C.H.; Graham, J.H.; Gottwald, T.R.; Cook, A.Z.; Parker, P.E. Wind speed effects on the quantity of *Xanthomonas citri* subsp. *citri* dispersed downwind from canopies of grapefruit trees infected with citrus canker. *Plant Dis.* **2010**, *94*, 725–736. [[CrossRef](#)] [[PubMed](#)]
219. Talibi, I.; Boubaker, H.; Boudyach, E.H.; Ait Ben Aoumar, A. Alternative methods for the control of postharvest citrus diseases. *J. Appl. Microbiol.* **2014**, *117*, 1–17. [[CrossRef](#)] [[PubMed](#)]
220. Cacciola, S.O.; Lio, G.M.D.S. Management of citrus diseases caused by *Phytophthora* spp. In *Integrated Management of Diseases Caused by Fungi, Phytoplasma and Bacteria*; Springer: Dordrecht, The Netherlands, 2008; pp. 61–84.
221. Luiz, M.H.R.; Takahashi, L.T.; Bassanezi, R.C. Optimal control in citrus diseases. *Comput. Appl. Math.* **2021**, *40*, 1–13. [[CrossRef](#)]
222. Govinda Rao, P. Citrus diseases and their control in Andhra State. *Andhra Agric. J.* **1954**, *1*, 187–192.
223. Paracer, C. Some important diseases of fruit trees. *Punjab Hort. J.* **1961**, *1*, 45–47.
224. Graham, J.H.; Johnson, E.G.; Myers, M.E.; Young, M.; Rajasekaran, P.; Das, S.; Santra, S. Potential of nano-formulated zinc oxide for control of citrus canker on grapefruit trees. *Plant Dis.* **2016**, *100*, 2442–2447. [[CrossRef](#)]
225. Patel, R.; Desai, M. Control of Citrus Canker1. *Indian J. Hortic.* **1970**, *27*, 93–98.
226. Kishun, R.; Chand, R. Studies on germplasm resistance and chemical control of citrus canker. *Indian J. Hortic.* **1987**, *44*, 126–132.
227. Chowdhury, S. Citrus canker in Assam. *Pl. Prot. Bull* **1951**, *3*, 78–79.
228. Nirvan, R. Citrus canker and its control. *Hort. Adv.* **1961**, *5*, 171–175.
229. Patel, M.; Padhya, A. Sodium arsenite-Copper sulphate spray for the control of citrus canker. *Curr. Sci.* **1964**, *33*, 87–88.
230. Ram, G.; Nirvan, R.; Saxena, M. Control of citrus canker. *Prog. Hort.* **1972**, *12*, 240–243.
231. Rangaswami, G.; Rao, R.R.; Lakshaman, A. Studies on the control of citrus canker with Streptomycin. *Phytopathology* **1959**, *49*, 224–226.
232. Balaraman, K.; Purushotman, R. Control of citrus canker on acid lime. *South Indian Hortic.* **1981**, *29*, 175–177.
233. El-Goorani, M.A. The occurrence of citrus canker disease in United Arab Emirates (UAE). *J. Phytopathol.* **1989**, *125*, 257–264. [[CrossRef](#)]
234. Kale, K.; Raut, J.; Ohekar, G. Efficacy of fungicides and antibiotics against acidlime (*Citrus aurantifolia* (Christm.) swingle) canker. *Pesticides* **1988**, *22*, 26–27.
235. Reddy, G.; Rao, A. Control of canker in citrus nurseries. *Agric. J.* **1960**, *7*, 1–13.
236. Dakshinamurthi, V.; Rao, D. Preliminary studies on the control of citrus Canker on acid lime. *Andhra Agric. J.* **1959**, *6*, 145–148.
237. Kale, K.; Kolte, S.; Peshney, N. Economics of chemical control of citrus canker caused by *Xanthomonas campestris* pv. *citri* under field conditions. *Indian Phytopathol.* **1994**, *47*, 253–255.
238. Kumar, A.; Sharma, N.; Ahmad, M.; Siddiqui, M.W. Climate change, food security, and livelihood opportunities in mountain agriculture. *Clim. Dyn. Hortic. Sci.* **2015**, *28*, 349–360.
239. Timmer, L. Evaluation of bactericides for control of citrus canker in Argentina. In Proceedings of the Florida State Horticultural Society; Florida State Horticultural Society: Alexandria, VA, USA, 1988; pp. 6–9.
240. Canteros, B.I.; Gochez, A.M.; Moschini, R.C. Management of citrus canker in Argentina, a success story. *Plant Pathol. J.* **2017**, *33*, 441. [[CrossRef](#)] [[PubMed](#)]
241. Das, K.; Datta, K.; Karmakar, S.; Datta, S.K. Antimicrobial Peptides—Small but Mighty Weapons for Plants to Fight Phytopathogens. *Protein Pept. Lett.* **2019**, *26*, 720–742. [[CrossRef](#)] [[PubMed](#)]
242. Das, R.M.B.; Mondal, P.; Khatua, D.; Mukherjee, N. Biological management of citrus canker on acid lime through *Bacillus subtilis* (S-12) in West Bengal, India. *J. Biopestic.* **2013**, *7*, 38–41.
243. Ota, T. Interactions in vitro and in vivo between *Xanthomonas campestris* pv. *citri* and *Antagonistic pseudomonas* sp. *Jpn. J. Phytopathol.* **1983**, *49*, 308–315. [[CrossRef](#)]
244. Goto, M.; Yaguchi, Y. Relationship between defoliation and disease severity in citrus canker. *Jpn. J. Phytopathol.* **1979**, *45*, 689–694. [[CrossRef](#)]
245. Unnamalai, N.; Gnanamanickam, S. *Pseudomonas fluorescens* is an antagonist to *Xanthomonas citri* (Hasse) Dye, the incitant of citrus canker. *Curr. Sci.* **1984**, *53*, 703–704.
246. Marco, G.M.; Stall, R.E. Control of bacterial spot of pepper initiated by strains of *Xanthomonas campestris* pv. *vesicatoria* that differ in sensitivity to copper. *Plant Dis.* **1983**, *67*, 779–781. [[CrossRef](#)]
247. de Oliveira, A.G.; Spago, F.R.; Simionato, A.S.; Navarro, M.O.; da Silva, C.S.; Barazetti, A.R.; Andrade, G. Bioactive organocopper compound from *Pseudomonas aeruginosa* inhibits the growth of *Xanthomonas citri* subsp. *citri*. *Front. Microbiol.* **2016**, *7*, 113. [[CrossRef](#)]
248. Murate, L.S.; de Oliveira, A.G.; Higashi, A.Y.; Barazetti, A.R.; Simionato, A.S.; da Silva, C.S.; Simões, G.C.; Santos, I.M.O.D.; Ferreira, M.R.; Cely, M.V.T.; et al. Activity of secondary bacterial metabolites in the control of citrus canker. In Embrapa Soja-Artigo em anais de congresso (ALICE). *Agric. Sci.* **2015**, *6*, 295–303.
249. Spago, F.R.; Mauro, C.I.; Oliveira, A.G.; Beranger JP, O.; Cely MV, T.; Stanganelli, M.M.; Andrade, G. *Pseudomonas aeruginosa* produces secondary metabolites that have biological activity against plant pathogenic *Xanthomonas* species. *Crop Prot.* **2014**, *62*, 46–54. [[CrossRef](#)]
250. Huang, T.P.; Tzeng, D.D.S.; Wong, A.C.L.; Chen, C.H.; Lu, K.M.; Lee, Y.H.; Huang, W.D.; Hwang, B.F.; Tzeng, K.C. DNA polymorphisms and biocontrol of *Bacillus* antagonistic to citrus bacterial canker with indication of the interference of phyllosphere biofilms. *PLoS ONE* **2012**, *7*, e42124. [[CrossRef](#)] [[PubMed](#)]

251. Rabbee, M.F.; Ali, M.S.; Baek, K.-H. Endophyte *Bacillus velezensis* isolated from Citrus spp. Controls streptomycin-resistant *Xanthomonas citri* subsp. *citri* that causes citrus bacterial canker. *Agriculture* **2019**, *9*, 470. [\[CrossRef\]](#)
252. Choi, J.; Moon, E. Identification of novel bioactive hexapeptides against phytopathogenic bacteria through rapid screening of a synthetic combinatorial library. *J. Microbiol. Biotechnol.* **2009**, *19*, 792–802. [\[CrossRef\]](#) [\[PubMed\]](#)
253. Gholami, D.; Goodarzi, T.; Aminzadeh, S.; Alavi, S.M.; Kazemipour, N.; Farrokhi, N. Bacterial secretome analysis in hunt for novel bacteriocins with ability to control *Xanthomonas citri* subsp. *citri*. *Iran J. Biotechnol.* **2015**, *13*, 10–19. [\[CrossRef\]](#)
254. Ahmad, A.A.; Askora, A.; Kawasaki, T.; Fujie, M.; Yamada, T. The filamentous phage XacF1 causes loss of virulence in *Xanthomonas axonopodis* pv. *citri*, the causative agent of citrus canker disease. *Front. Microbiol.* **2014**, *5*, 321. [\[CrossRef\]](#)
255. Nilsson, A.S. Phage therapy—constraints and possibilities. *Uppsala J. Med. Sci.* **2014**, *119*, 192–198. [\[CrossRef\]](#)
256. Kering, K.K.; Kibii, B.J.; Wei, H. Biocontrol of phyto-bacteria with bacteriophage cocktails. *Pest. Manag. Sci.* **2019**, *75*, 1775–1781. [\[CrossRef\]](#)
257. Yoshikawa, G.; Askora, A.; Blanc-Mathieu, R.; Kawasaki, T.; Li, Y.; Nakano, M.; Ogata, H.; Yamada, T. *Xanthomonas citri* jumbo phage XacN1 exhibits a wide host range and high complement of tRNA genes. *Sci. Rep.* **2018**, *8*, 4486. [\[CrossRef\]](#)
258. Ibrahim, Y.E.; Saleh, A.A.; Al-Saleh, M.A. Management of Asiatic citrus canker under field conditions in Saudi Arabia using bacteriophages and acibenzolar-s-methyl. *Plant Dis.* **2017**, *101*, 761–765. [\[CrossRef\]](#)
259. Buttner, C.; McAuliffe, O.; Ross, R.P.; Hill, C.; O'Mahony, J.; Coffey, A. Bacteriophages and bacterial plant diseases. *Front. Microbiol.* **2017**, *8*, 34–41. [\[CrossRef\]](#)
260. Lin, Y.; He, Z.; Roskopf, E.N.; Conn, K.L.; Powell, C.A.; Lazarovits, G. A nylon membrane bag assay for determination of the effect of chemicals on soilborne plant pathogens in soil. *Plant Dis.* **2010**, *94*, 201–206. [\[CrossRef\]](#) [\[PubMed\]](#)
261. Kessmann, H.; Staub, T.; Hofmann, C.; Maetzke, T.; Herzog, J.; Ward, E.; Uknes, S.; Ryals, J. Induction of systemic acquired disease resistance in plants by chemicals. *Annu. Rev. Phytopathol.* **1994**, *32*, 439–459. [\[CrossRef\]](#)
262. Romero, A.; Kousik, C.; Ritchie, D. Resistance to bacterial spot in bell pepper induced by acibenzolar-S-methyl. *Plant Dis.* **2001**, *85*, 189–194. [\[CrossRef\]](#) [\[PubMed\]](#)
263. Wei, Z.-M.; Laby, R.J.; Zumoff, C.H.; Bauer, D.W.; He, S.Y.; Collmer, A.; Beer, S.V. Harpin, elicitor of the hypersensitive response produced by the plant pathogen *Erwinia amylovora*. *Science* **1992**, *257*, 85–88. [\[CrossRef\]](#) [\[PubMed\]](#)
264. Oostendorp, M.; Kunz, W.; Dietrich, B.; Staub, T. Induced disease resistance in plants by chemicals. *Eur. J. Plant Pathol.* **2001**, *107*, 19–28. [\[CrossRef\]](#)
265. Hostettmann, K.; Wolfender, J.L. The search for biologically active secondary metabolites. *Pestic. Sci.* **1997**, *51*, 471–482. [\[CrossRef\]](#)
266. Mahajan, A.; Das, S. Plants and microbes—Potential source of pesticide for future use. *Pestic. Inf.* **2003**, *28*, 33–38.
267. Balandrin, M.F.; Klocke, J.A.; Wurtele, E.S.; Bollinger, W.H. Natural plant chemicals: Sources of industrial and medicinal materials. *Science* **1985**, *228*, 1154–1160. [\[CrossRef\]](#)
268. Dorman, H.; Deans, S.G. Antimicrobial agents from plants: Antibacterial activity of plant volatile oils. *J. Appl. Microbiol.* **2000**, *88*, 308–316. [\[CrossRef\]](#)
269. Parameswari, C.; Tulasi Latha, A. Antibacterial activity of *Ricinus communis* leaf extract. *Indian Drugs* **2001**, *38*, 587–588.
270. Rath, C.; Dash, S.; Mishra, R. In vitro susceptibility of Japanese mint (*Mentha arvensis* L.) essential oil against five human pathogens. *Indian Perfum.* **2001**, *45*, 57–62.
271. Britto, S.J.; Senthilkumar, S. Antibacterial activity of *Solanum incanum* L. leaf extracts. *Asian J. Microbiol. Biotechnol. Environ. Sci.* **2001**, *3*, 65–66.
272. Bylka, W.; Szafer-Hajdrych, M.; Matławska, I.; Goślińska, O. Antimicrobial activity of isocytiside and extracts of *Aquilegia vulgaris* L. *Lett. Appl. Microbiol.* **2004**, *39*, 93–97. [\[CrossRef\]](#) [\[PubMed\]](#)
273. Shimpi, S.; Bendre, R. Stability and antibacterial activity of aqueous extracts of *Ocimumcanum* leaves. *Indian Perfum.* **2005**, *49*, 225.
274. Tahir, H.A.; Sahi, S.T.; Habib, A.; Haq, I.U.; Ahmad, A.; Ashraf, W. Evaluation of plant extracts as biocontrol agents against *Xanthomonas axonopodis* pv. *citri* the cause of citrus canker. *Pak. J. Phytopathol.* **2016**, *28*, 35–43.
275. Rios, J.L. Essential oils: What they are and how the terms are used and defined. In *Essential Oils in Food Preservation, Flavor and Safety*; Academic Press: London, UK, 2016; pp. 3–10.
276. Pandey, A.K.; Kumar, P.; Singh, P.; Tripathi, N.N.; Bajpai, V.K. Essential oils: Sources of antimicrobials and food preservatives. *Front. Microbiol.* **2017**, *7*, 2161. [\[CrossRef\]](#)
277. Mirzaei-Najafgholi, H.; Tarighi, S.; Golmohammadi, M.; Taheri, P. The effect of citrus essential oils and their constituents on growth of *Xanthomonas citri* subsp. *citri*. *Molecules* **2017**, *22*, 591. [\[CrossRef\]](#)
278. Feng, C.T.; Su, H.J.; Chen, C.T.; Ho, W.C.; Tsou, Y.R.; Chern, L.L. Inhibitory effects of Chinese medicinal herbs on plant-pathogenic bacteria and identification of the active components from gallnuts of Chinese sumac. *Plant Dis.* **2012**, *96*, 1193–1197. [\[CrossRef\]](#)
279. Silva, I.C.; Regasini, L.O.; Petrário, M.S.; Silva, D.H.S.; Bolzani, B.S.; Belasque, J., Jr.; Sacramento, L.V.S.; Ferreira, H. Antibacterial activity of alkyl gallates against *Xanthomonas citri* subsp. *citri*. *J. Bacteriol.* **2013**, *195*, 85–94. [\[CrossRef\]](#)
280. Wang, P.Y.; Fang, H.S.; Shao, W.B.; Zhou, J.; Chen, Z.; Song, B.A.; Yang, S. Synthesis and biological evaluation of pyridinium-functionalized carbazole derivatives as promising antibacterial agents. *Bioorg Med. Chem. Lett.* **2017**, *27*, 4294–4297. [\[CrossRef\]](#)
281. Król, E.; de Sousa Borges, A.; da Silva, I.L.; Polaquini, C.R.; Regasini, L.O.; Ferreira, H.; Scheffers, D.J. Antibacterial activity of alkyl gallates is a combination of direct targeting of FtsZ and permeabilization of bacterial membranes. *Front. Microbiol.* **2015**, *6*, 390. [\[CrossRef\]](#) [\[PubMed\]](#)

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282. Mohana, D.; Raveesha, K. Anti-bacterial activity of *Caesalpinia coriaria* (Jacq.) Willd. against plant pathogenic *Xanthomonas* pathovars: An eco-friendly approach. *J. Agric. Technol.* **2006**, *2*, 317–327.
283. Akhtar, M.A.; Rahber-Bhatti, M.; Aslam, M. Antibacterial activity of plant diffusate against *Xanthomonas campestris* pv. *citri*. *Int. J. Pest Manag.* **1997**, *43*, 149–153. [[CrossRef](#)]