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REVIEW

Coffee bacterial diseases: a plethora of scientific opportunities

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Coffee is a very important crop for several tropical countries across different continents. The diseases bacterial halo blight (BHB), bacterial leaf spot (BLS), bacterial leaf blight (BLB) and coffee leaf scorch (CLS), caused by the bacterial pathogens *Pseudomonas syringae* pv. *garcae* (Psgc), *P. syringae* pv. *tabaci* (Psta), *Pseudomonas cichorii* (Pch) and *Xylella fastidiosa* subsp. *pauca* (Xfp), respectively, cause significant reductions in coffee production, although other minor bacterial diseases have also been reported in some countries. Little research progress has been made on aspects that are relevant for control and management of these diseases. In all cases, there is an urgent need to develop rapid and more reliable methods for early detection of the pathogens in order to minimize their negative impact on coffee production. Because of the high rate of intra- and intersubspecific recombination occurring in *X. fastidiosa*, a permanent revision of the detection methods is necessary. Greater efforts should be made to understand the genetic and virulence diversity of Psgc, Psta and Pch populations. Early studies reported the identification of potential sources of resistance against Psgc and Psta, but, to date, no resistance gene has been isolated. Little effort has been made to understand the biology and molecular mechanisms underlying the interaction between *Coffea* spp. and these pathogenic bacteria. This review discusses the recent progress on the molecular mechanisms used by these bacteria to cause diseases on other plant species, in order to provide a guideline for the establishment of future research programmes.

Keywords: bacterial blight, *Coffea*, leaf scorch, pathogen detection, *Pseudomonas syringae*, *Xylella fastidiosa*

Introduction

Coffee (*Coffea* spp.) is a very important commodity for a number of tropical countries in South America, Central America, Asia and Africa. It plays an important socioeconomic role contributing to employment and settlement of families in production areas. Coffee production in several countries is threatened by abiotic and biotic stresses, including diseases caused by phytopathogenic bacteria. The four bacterial species *Robbsia andropogonis* (synonym *Burkholderia andropogonis*) (Rodrigues Neto *et al.*, 1981), *Pseudomonas syringae* (Franco Do Amaral *et al.*, 1958; Ramos & Shavdia, 1976; Rodrigues *et al.*, 2006, 2017c; Deséfano *et al.*, 2010), *Pseudomonas cichorii* (Robbs *et al.*, 1974; Almeida *et al.*, 2012b) and *Xylella fastidiosa* (Paradela Filho *et al.*, 1997; Lima *et al.*, 1998) have been reported to cause disease on coffee plants under field conditions. Within *P. syringae*, pathovars *garcae* (Amaral *et al.*, 1956; Ramos & Shavdia, 1976; Korobko & Wondinagegne, 1997; Chen, 2002; Bai *et al.*, 2013), *tabaci* (Rodrigues *et al.*, 2006, 2017c; Destéfano *et al.*, 2010) and *syringae* (Franco Do Amaral *et al.*, 1958; Baker, 1972; Ramos & Shavdia, 1976; Okioga, 1977; Cortés Monllor, 1988) have been associated with similar disease symptoms.

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Despite the considerable damage these bacteria cause on coffee, a crop of high socioeconomic importance, neither efficient methods for their reliable detection nor their efficient control are currently available. Also, little is known about the biology of the interaction of these bacterial species with coffee plants. This review discusses the relevant aspects of coffee diseases caused by these bacterial pathogens and brings together information on their pathogenicity mechanisms that could be used as guidelines for establishing research programmes aimed to better understand the biology of the interactions and to develop effective alternatives for disease management and control. Herein, the current methods used to control these diseases will not be discussed, because they mostly rely on the implementation of preventive measures such as planting certified seedlings, avoiding pathogen entry in the area by planting natural windbreaks and avoiding injuries on the leaves and branches by mechanical

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implements. Chemical treatment in areas with history of the disease usually involves preventive application of copper-based compounds alone or in mixtures with mancozeb (and insecticides in the case of *X. fastidiosa*) sometimes in alternation with kasugamycin (Zambolim *et al.*, 2005; Patrício *et al.*, 2010; Patrício & Oliveira, 2014). In the case of *X. fastidiosa*, severe pruning has also been proposed as an efficient alternative to control the disease (Queiroz-Voltan *et al.*, 2006; Rocha *et al.*, 2010b).

Bacterial halo blight

Disease symptoms, aetiology and dissemination

Pseudomonas syringae pv. *garcae* (Psgc), the causal agent of bacterial halo blight (BHB) of coffee, was first described in 1955 by Amaral *et al.* (1956) in the municipality of Garça in the Brazilian state of São Paulo. It was also reported in the states of Paraná (Mohan *et al.*, 1978) and Minas Gerais (Kimura *et al.*, 1973; Zoccoli *et al.*, 2011) in Brazil. Bacterial halo blight has been reported in Kenya (Ramos & Shavdia, 1976), Ethiopia (Korobko & Wondinagegne, 1997), Uganda (Chen, 2002) and China (Chen, 2002; Bai *et al.*, 2013). Although the disease was initially considered of minor importance, recurrent outbreaks have occurred in coffee plantations in several Brazilian states, mainly at higher altitudes (Kimura *et al.*, 1973; Mohan, 1976; Cafepoint, 2011; Zoccoli *et al.*, 2011; Rodrigues *et al.*, 2013).

Characteristic symptoms of the disease include the formation of necrotic spots surrounded or not by chlorotic haloes (Fig. 1a). The lesions are more evident on the leaf borders, but flowers, fruits and branches can also be affected. Under severe infection, the disease can cause blackening of twigs and blighting of branch tips, resulting in dieback, defoliation, and apical meristem death, which in turn induces over-branching (Fig. 1b,c) (Zambolim *et al.*, 1999, 2005; Rodrigues *et al.*, 2013). Disease development is favoured by mild temperatures, high wind intensity and rain, especially hail stone rain, which produces injuries that facilitate bacterial penetration (Rodrigues *et al.*, 2013). High increases in disease incidence have occurred after frost, for example after the 1973–1975 period in the state of Paraná (Kimura *et al.*, 1973; Mohan, 1976), which could be facilitated by the ice-nucleation activity exhibited by Psgc (Gonçalves & Massambani, 2011; Araújo *et al.*, 2018).

It has been estimated that BHB can cause losses of up to 70% in nurseries and in the field, predominantly in regions above 1000 m a.s.l. in the presence of severe wind (Zoccoli *et al.*, 2011). The bacterium survives mainly as an epiphyte and associated with plant debris. It penetrates the host tissue through natural openings (stomata) or wounds and is mainly disseminated by water and wind-driven aerosol particles (Zoccoli *et al.*, 2011). A recent study suggests that coffee seed may also be a source of inoculum (Belan *et al.*, 2016).

Plant genetic resistance

Early studies on the resistance to Psgc indicated that Ethiopian *C. arabica* cultivars Hara, Dilla and Alghe, S12 and Geisha were resistant to the bacterium, which was attributed to the *SH1* gene that also confers resistance to some races of the rust fungus *Hemileia vastatrix* (Moraes *et al.*, 1974; Petek *et al.*, 2006). Additional potential sources of resistance to BHB were identified in some varieties of *C. arabica*, some genotypes derived from Caturra × Timor hybrids, and the species *Coffea eugenioides* and *Coffea stenophylla* (Mohan *et al.*, 1978). The existence of genes that confer resistance to BHB, in addition to *SH1*, is supported by results obtained with progenies derived from crosses of Caturra with the Timor hybrid (Mohan *et al.*, 1978; Cardoso & Sera, 1983), IAPAR-59 with descendants of *C. arabica SH1* × Catuaí Vermelho IAC 81 (Petek *et al.*, 2001, 2006), and Catuaí Vermelho IAC 24 with IAC 1137-5 of Geisha (Rodrigues *et al.*, 2017b).

In Brazil, in studies conducted in the state of Paraná, *C. arabica* IPR 102 was found to be completely resistant under natural field infections whereas other cultivars, such as IPR 103, IPR 104, IPR 108 and IPR 59 exhibited incomplete resistance (Ito *et al.*, 2005, 2008). Petek *et al.* (2001, 2006) reported that IAPAR-59 was a source of potentially important resistance in the state of Paraná. In contrast, Zoccoli *et al.* (2011) did not observe high levels of resistance when IAPAR-59 was inoculated with a strain from Carmo do Paranaíba, Minas Gerais state. It is probable that virulence variations between bacterial strains may be responsible for the observed differences. Such a possibility was suggested by Petek *et al.* (2006) when progenies of Catuaí × Icatú PRFB 2-27-1 did not exhibit the same level of resistance that was observed in previous studies (Kairu, 1997). In Ethiopia in field surveys conducted by Hinkosa *et al.* (2016, 2017), differences in disease severity and incidence were observed between the Sidama and southwestern zones. In most studies reported to date, the majority of coffee genotypes exhibited disease symptoms although at different levels, meaning that a classical gene-for-gene interaction was not clearly detected. However, in some studies, such as those conducted by Mohan *et al.* (1978) and Petek *et al.* (2006), *Coffea* genotypes that exhibited immunity to the disease were found, suggesting potential sources of major gene resistances. However, no reports on the identification of the resistance genes have been published so far.

One of the major difficulties when trying to establish comparisons among different studies is that several inoculation methods have been used to evaluate plant resistance. In some studies, leaves were previously wounded using different techniques (Ithiru *et al.*, 2013; Rodrigues *et al.*, 2017a; Maciel *et al.*, 2018); in others they were not wounded (Rodrigues *et al.*, 2017a). Some of those studies have shown that the reactions of the coffee genotypes were highly dependent on the inoculation method and that some methods seem to be better in discriminating differences in resistance to the pathogen (Ithiru *et al.*,

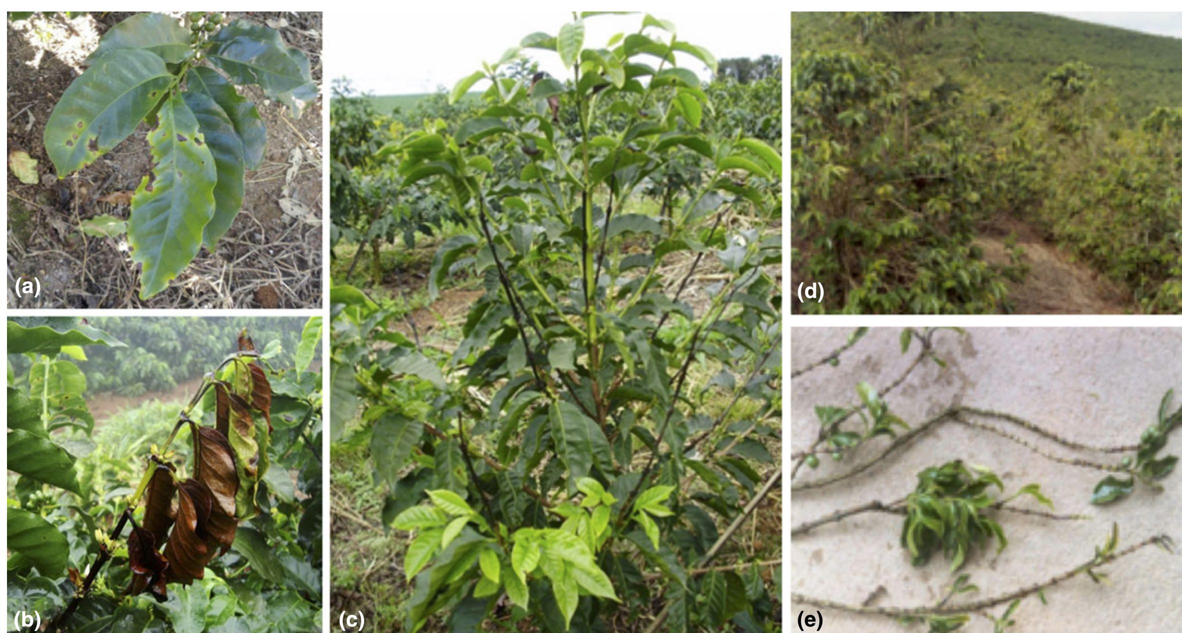

Figure 1 Symptoms of coffee bacterial diseases. (a) Foliar necrotic spots surrounded by chlorotic haloes, (b) death of branches and apical meristem, and (c) stem necrosis and plant defoliation caused by *Pseudomonas syringae* pv. *garcae*. (d) Leaf chlorosis and plant defoliation and (e) internode shortening and defoliation caused by *Xylella fastidiosa* subsp. *pauca*. Photos courtesy Rui P. Leite Jr, Instituto Agronômico do Paraná (a, b), Flávia R. A. Patrício, Instituto Agronômico de Campinas (c), and Laércio Zambolim, Universidade Federal de Viçosa (d, e). [Colour figure can be viewed at wileyonlinelibrary.com].

2013; Rodrigues *et al.*, 2017a). Therefore, there is an urgent need to establish standardized methods for plant resistance evaluation. More importantly, the aggressiveness (and possible virulence) variability of the pathogen population should be taken into consideration when selecting resistant genotypes.

Taxonomy, host range and genetic diversity

Pseudomonas syringae pv. *garcae* is a rod-shaped, strictly aerobic, fluorescent, Gram-negative bacterium that belongs to the family *Pseudomonadaceae*, order *Pseudomonadales* of the *Gammaproteobacteria*. It clusters in genomospecies 4 based on DNA-DNA hydridization (Gardan *et al.*, 1999) and *rpoD*-based phylogenetic analysis (Parkinson *et al.*, 2011; Raimundi, 2017), along with informal nomospecies *Pseudomonas coronafaciens*. Hence, Psgc has lately been referred to as *P. coronafaciens* pv. *garcae*.

Although it is well known that the species *P. syringae* is highly variable, the genetic variability of Psgc has been poorly investigated. Early studies demonstrated biochemical and genetic variability at the intrapathovar level. For instance, Kairu (1997) reported that Brazilian and Kenyan strains differed with regard to production of bacteriocins and fluorescent pigments. Mwangi *et al.* (2018) reported variability in the 16S rDNA sequence and found that the most aggressive strains clustered together in the phylogenetic tree, but no correlation with geographic origin was observed. In contrast, Maciel *et al.* (2018) when using rep-PCR to evaluate the genetic diversity of Brazilian and Kenyan strains, found clustering according to geographic origin, although no clear correlation with aggressiveness was detected. A major caveat of those studies is that a small number of strains was evaluated and it is difficult to draw clear conclusions. Studies using larger populations, from different geographic areas, are needed in order to gain a better understanding of the genetic diversity and population structure of the phytopathogen.

Aggressiveness variation among Psgc strains is supported by studies conducted in Kenya and Brazil (Ithiru *et al.*, 2013; Rodrigues *et al.*, 2017a; Maciel *et al.*, 2018; Mwangi *et al.*, 2018). Interestingly, Kairu (1997) found that cultivar SL 28 was resistant to Brazilian strains but susceptible to Kenyan strains, suggesting a possible virulence variability among bacterial populations. Also, a possible geographic adaptation of Psgc to infect additional plant species is suggested by a study indicating that strains from Kenya can also cause disease on oat plants (at least under experimental conditions) whereas those from Brazil cannot. It is not clear whether this specialization is at the host species or cultivar level, because only one oat cultivar was used in the experiments (Barta & Willis, 2005). These results suggest the possibility that some bacterial genes could restrict the host range of some Psgc populations to additional hosts. Furthermore, although that study was conducted with only a few strains from each country, its results suggest that genes that confer resistance to coffee bacterial blight could be procured in distantly related plant species, including monocots.

Pathogen detection

Molecular and serological methods for specific detection of Psgc in infected tissue have not been reported. Current methods rely on isolation and identification of the bacterium using morphological, physiological and biochemical tests (Zoccoli *et al.*, 2011; Belan *et al.*, 2016; Hinkosa *et al.*, 2016; Rodrigues *et al.*, 2017c). Distinction between Psgc and other *P. syringae* pathovars is commonly achieved by LOPAT testing (Rodrigues *et al.*, 2017c). *Pseudomonas syringae* pv. *garcae* can be differentiated from *P. syringae* pv. *tabaci* and *P. syringae* pv. *syringae* by assaying for production of pectate lyase and utilization of L-trigonelline, L-(+)-tartrate and lactate (Young & Triggs, 1994). At the present time, specific identification using molecular methods has been attempted only using rep-PCR (Kairu, 1997; Belan *et al.*, 2016). The availability of the genome sequence of Psgc strain ICMP 4323 (Thakur *et al.*, 2016) could help identify pathovar-specific DNA regions useful for development of PCR-based and serological detection methods.

Mechanisms of pathogenicity

Studies on the mechanisms by which Psgc causes disease on coffee plants have not been reported. Like many other phytopathogenic pseudomonads, Psgc possesses genes coding for a type III secretion system (T3SS), considered a major pathogenicity determinant of this taxonomic group. The T3SS is coded by *hrp*/*hrc* genes that are widely conserved in species of the genera *Xanthomonas*, *Pseudomonas*, *Ralstonia*, *Erwinia* and *Pantoea* (Collmer *et al.*, 2000). The presence of such a secretion system as well as of its cargo proteins was verified in the genome sequence of Psgc ICMP 4323 (Thakur *et al.*, 2016). Furthermore, it was also demonstrated that Psgc 1202 carries a functional T3SS able to translocate chimeric fusions of adenylate cyclase (CyaA) or *H. vastatrix* effectors with N-terminal secretion signals of bacterial T3SS effectors into the cytoplasm of *C. arabica* cells (Maia *et al.*, 2016). Nonetheless, the importance of the T3SS for the pathogenicity of Psgc remains to be investigated.

Pseudomonas syringae pathovars also deliver phytotoxins that contribute to symptom development during the interaction with their hosts. It has long been demonstrated that Psgc produces the monocyclic β-lactam tabtoxin that has been associated with the formation of chlorotic haloes (Bender *et al.*, 1999). Consistently, some Psgc strains that do not cause chlorotic haloes have been shown not to produce tabtoxin (Barta & Willis, 2005). Tabtoxin is cleaved by a plant amino peptidase releasing tabtoxinine-β-lactam, which causes inhibition of glutamine synthase (Bender *et al.*, 1999).

Bacterial leaf spot

Disease symptoms, aetiology and dissemination

Bacterial leaf spot (BLS) was first reported by Rodrigues *et al.* (2006) in coffee nurseries in São Paulo state. This disease has been reported only in Brazil (Rodrigues *et al.*, 2006, 2017c; Destéfano *et al.*, 2010; Raimundi, 2017). In the state of São Paulo, BLS was associated with leaf spots on Catuaí plants in a commercial nursery located in Arandu at a disease incidence of 1–2%. Symptoms appeared initially in the leaves as small brown lesions that turned into black and angular spots (Destéfano *et al.*, 2010). More recently, the aetiologic agent was also detected in plant samples collected in several localities in the states of Paraná and Minas Gerais (Raimundi, 2017). *Pseudomonas syringae* pv. *tabaci* (Psta) was found to be sympatric and causing mixed infections with Psgc (Rodrigues *et al.*, 2017c). Because symptoms of BLS highly resemble those of BHB, and differentiation between the two *P. syringae* pathovars causing them using biochemical methods is sometimes difficult, it is probable that the importance of the disease in Brazil has been underestimated. As a consequence, studies on the epidemiology, survival and dissemination of the pathogen are scarce. The sympatric and mixed infection occurring between Psta and Psgc could provide an explanation for why some coffee genotypes that exhibited high levels of resistance to Psgc (Petek *et al.*, 2001, 2006) showed more severe disease symptoms when tested in subsequent studies (Zoccoli *et al.*, 2011).

Plant genetic resistance

Studies on the identification of resistance against Psta in coffee germplasm have not been reported. The types of resistance (quantitative or qualitative) that act in coffee against the bacterial pathogen remain unknown. Sources of resistance against Psta have been identified in several *Nicotiana* species (Burk & Heggestad, 1966; Stavely, 1979; Goy *et al.*, 1992). Some of the resistance genes have been transferred to cultivated *Nicotiana tabacum* via interspecific hybridization (Clayton, 1947; Smeeton & Ternouth, 1992; Woodend & Mudzengerere, 1992). Soybean cultivars resistant to Psta have also been obtained (Rose, 1989; Carnielli *et al.*, 1992). These results suggest the possibility of developing coffee cultivars resistant to Psta using genes identified in other host plant species. Nonetheless, there are no recent reports on the identification of new sources of resistance and development of resistant cultivars. Consequently, the effectiveness of genes identified in early studies should be verified, because strains that overcome plant resistance have been reported (Valleau *et al.*, 1962).

Alternatives to endogenous resistance for the control of Psta have also been explored. Enhanced resistance has been obtained by cisgenic overexpression and by expression of genes from other plant species, insects, bacteria and even coding for synthetic peptides in the model plant *N. tabacum* (Table 1). Whether these genes enhance resistance to Psta in coffee plants remains to be investigated and they are candidates for development of coffee resistant varieties. Importantly, because Psta strains that produce tabtoxin also possess a cognate tabtoxin resistance gene (*ttr*) for detoxification, the *ttr* genes can

Table 1 Genes that have been shown to confer enhanced resistance against *Pseudomonas syringae* pv. *tabaci*.

Gene	Function	Source	Reference
PAO	Polyamine oxidase	Tobacco (*Nicotiana tabacum*)	Moschou *et al.* (2009)
OPBP1	Osmotin promoter-binding protein	Tobacco (*N. tabacum*)	Guo *et al.* (2004)
OsDRF1	F-box protein	Rice (*Oryza sativa*)	Cao *et al.* (2008)
OsBIRF1	RING finger protein	Rice (*O. sativa*)	Liu *et al.* (2008)
OsLOL2	*Arabidopsis* LSD1 (zinc finger protein) orthologue	Rice (*O. sativa*)	Bhatti *et al.* (2008)
α-thionin	Cystein-rich peptide	Barley (*Hordeum vulgare*)	Carmona *et al.* (1993)
VvPR1b	Pathogenesis-related protein 1	Interspecific grapevine (*Vitis*) hybrid BN5-4	Li *et al.* (2011)
pflp	Ferredoxin-like protein	Sweet pepper (*Capsicum annuum*)	Huang *et al.* (2004)
LTP	Lipid transfer protein	Pepper (*C. annuum*) cv. Bugang	Sarowar *et al.* (2009)
HRAP	Hypersensitive response-assisting protein	Sweet pepper (*C. annuum*)	Ger *et al.* (2003)
GhCyp1	Cyclophilin	Cotton (*Gossypium hirsutum*) cv. Zhongmian 35	Zhu *et al.* (2011)
Sarcotoxin IA	Bactericidal peptide	Flesh fly (*Sarcophaga peregrina*)	Ohshima *et al.* (1999), Mitsuhara *et al.* (2000)
ipt	Isopentenyl transferase	*Agrobacterium tumefaciens*	Großkinsky *et al.* (2011)
ttr	Tabtoxin resistance	*Pseudomonas syringae* pv. *tabaci*	Anzai *et al.* (1989), Batchvarova *et al.* (1998)
MB39	Cecropin-like lytic peptide	Synthetic lytic peptide	Huang *et al.* (1997)
D2A21	Antimicrobial peptide (AMP)	Synthetic AMP	Hao *et al.* (2017)
MSI-99	Analogue of magainin 2 (African clawed frog, *Xenopus laevis*)	Synthetic AMP	DeGray *et al.* (2001)

potentially be used to create plants resistant to the toxin. Using such a rationale, Anzai *et al.* (1989) and Batchvarova *et al.* (1998) demonstrated that constitutive expression of the *ttr* gene from Psta in several cultivars of *N. tabacum* conferred enhanced resistance to the pathogen. Because many strains of both Psta and Psgc produce tabtoxin, this approach could be effective to control both pathogens. Reports of studies using *ttr* under the control of tissue-specific promoters in coffee plants have not been reported.

Another alternative to control Psta is the induction of plant resistance. Exogenous application of theobroxide, a natural epoxy cyclohexene produced by strain OCS71 of *Lasiodiplodia theobromae*, caused restriction of Psta growth and reduction in symptom development in *Nicotiana benthamiana* (Ahn *et al.*, 2013). Application of acibenzolar-*S*-methyl, an inducer of systemic acquired resistance (Cole, 1999), and of pyraclostrobilurin, a strobilurin fungicide (Herms *et al.*, 2002) to *N. tabacum* have been shown to induce resistance against Psta.

Pathogen detection

As for other *Pseudomonas* that infect coffee, detection of Psta is currently based on morphological, physiological and biochemical tests. Even though Psgc, Psta and *Pseudomonas cichorii* (Pch) can be clearly distinguished using *rpoD*-based phylogenetic analysis after a somewhat long and cumbersome process that requires well-trained laboratory personnel (Parkinson *et al.*, 2011; Raimundi, 2017), practical methods for detection must be developed. Attempts to develop simple detection methods based on the polymorphisms present in the *rpoD* locus have not been reported. Detection methods could also be based on initial comparisons of the genomes of strains that belong to the three coffee pathogens that are available in the databases to identify DNA regions or biochemical functions specific to each one of them. The establishment of such methods could help determine the real impact of the disease in coffee plantations.

Taxonomy, host range and genetic diversity

Pseudomonas syringae pv. *tabaci* is morphologically very similar to Psgc and was traditionally classified in the same suprapathovar taxonomic groups. However, Psta clusters in genomospecies 2 ('amygdali' group) based on DNA-DNA hybridization (Gardan *et al.*, 1999) and *rpoD*-based phylogenetic analysis (Parkinson *et al.*, 2011; Raimundi, 2017). Because the nomospecies *Pseudomonas amydali* was already validly published, Psta has lately been referred to as *Pseudomonas amygdali* pv. *tabaci*.

Studies on the genetic diversity of Psta associated with coffee have not been reported in peer-reviewed articles and little is known about the genetic structure of populations associated with other host plants. Results from a single study suggest that the population of Psta has a high genetic diversity (Peng *et al.*, 2003). Nonetheless, understanding of the pathogen population associated with coffee should be furthered. This *P. syringae* pathovar is widely distributed in all continents and has a wide host range, comprising species of several different botanical families (Bradbury, 1986; Tanaka *et al.*, 1993;

Beriam *et al.*, 2006; Malavolta Júnior *et al.*, 2008; CABI, 2018). The specificity of strains toward a particular host is largely unknown. In a recent study, Raimundi (2017) found that strains from *N. tabacum*, *Phaseolus vulgaris*, *Cucumis sativus*, *Carica papaya*, *Desmodium canum*, *Celosia plumosa* and *Aster* sp. were able to infect *C. arabica* 'Catuaí Vermelho IAC 99', causing symptoms in the greenhouse similar to those observed under natural conditions. These results suggest that other plant species (associated with coffee plantations) could serve as sources of inoculum in the field and that cross-infection could occur. However, this hypothesis must be proved by conducting field experiments.

With regard to the pathotypic composition of the bacterial population, early studies considered the existence of two races, 0 and 1, the latter not being capable of causing disease on various *Nicotiana* species (*N. nudicaulis*, *N. repanda*, *N. rustica* and *N. undulata*; Stavely, 1979). However, the differential interaction of bacterial isolates with coffee cultivars and species remains largely unknown.

Mechanisms of pathogenicity

As for other phytopathogenic pseudomonads, the T3SS is a major pathogenicity determinant in Psta. In a genome comparison study, it was shown that Psta strain 11528 carries a *hrp*/*hrc* region, containing a conserved effector locus (CEL) and an exchangeable effector locus (EEL) (Studholme *et al.*, 2009), similar to the tripartite island previously identified in *P. syringae* pv. *tomato* (Pst) DC3000 (Alfano *et al.*, 2000). In Psta 11528, the CEL is well conserved whereas the EEL is reduced compared to the Pst DC3000 EEL and contains only one effector (*avrPphE*) (Alfano *et al.*, 2000; Deng *et al.*, 2003). Fifteen of the Psta 11528 effectors are conserved in Pst DC3000, whereas only one effector (*hopAE1*) is conserved in *P. syringae* pv. *phaseolicola* 1448a and *P. syringae* pv. *syringae* B728a, two strains that cause disease on common bean (*P. vulgaris*) (Studholme *et al.*, 2009). Interestingly, Psta 11528 carries a truncated version of *hopM1*, which has been shown to play a key role in the pathogenicity of Pst DC3000 (Badel *et al.*, 2003, 2006). However, Psta 11528 carries *AvrE1*, which is also a key determinant in the pathogenicity of Pst DC3000 (Badel *et al.*, 2006). As expected, Psta 11528 does not carry a copy of *hopQ1*, a gene that confers avirulence toward *N. benthamiana* (Wei *et al.*, 2007). It is worth mentioning that the T3SS effector complement varies according to the strain. Some effectors are present in some strains but not in others that belong to the same pathovar, as demonstrated for Pst strains DC3000 and T1 (Almeida *et al.*, 2009). Effectors have been shown to suppress the plant defence response to contribute to virulence. For example, HopX1 from Psta 11528 is a cysteine protease that causes degradation of JAZ proteins (a family of proteins that repress jasmonic acid (JA)-responsive genes) to promote susceptibility in plants (Gimenez-Ibanez *et al.*, 2014).

Psta produces tabtoxin, which has been associated with the formation of the chlorotic haloes that surround the necrotic lesions (Clayton, 1934; Anzai *et al.*, 1990). The involvement of additional nonproteinaceous effectors was suggested recently by Lee *et al.* (2013). The authors reported that an extracellular, thermostable secondary metabolite produced by Psta suppresses several *N. benthamiana* defence responses against nonadapted bacterial pathogens, including stomatal closure and the hypersensitive response (HR). However, the nature of the metabolite and its involvement in suppression of plant defence responses during compatible interactions remain to be determined (Lee *et al.*, 2013).

The ability of Psta to cause disease on its host plants is the result of the concerted action of several factors. In addition to tabtoxin and T3SS effectors, the assembly of type IV pili (T4P) (Taguchi & Ichinose, 2011; Nguyen *et al.*, 2012) and flagella (Ichinose *et al.*, 2003; Taguchi *et al.*, 2009) and the production of the siderophore pyoverdine (Taguchi *et al.*, 2010) have also been shown to play important roles in the pathogenicity of Psta. Type IV pili are assembled through the general secretion pathway and a protein machinery that resembles the type II secretion system (T2SS) (Tanassi *et al.*, 2012). Several of those factors, such as tabtoxin and siderophore production, T3SS assembly and T3SS effector gene expression are coordinately regulated by complex regulatory networks that include the GacS/GacA two-component *quorum-sensing* system (Marutani *et al.*, 2008), AefR (a member of the TetR transcription factors; Kawakita *et al.*, 2012; Yun *et al.*, 2015), Fur (involved in iron acquisition; Cha *et al.*, 2008, 2012) and the virulence factor regulator Vfr (a member of the 3′,5′-adenosine monophosphate (cAMP) receptor proteins; Taguchi & Ichinose, 2013).

Bacterial leaf blight

Disease symptoms, aetiology and dissemination

Pseudomonas cichorii (Pch) has been reported in several Brazilian states causing bacterial leaf blight (BLB) on coffee plants (Robbs *et al.*, 1974; Almeida *et al.*, 2012b). The bacterium seems to establish epiphytic populations on the leaf surface and its penetration to the plant has been associated with injuries caused by fungi (*Phoma* and *Cercospora*) and by the coffee leaf miner *Leucoptera coffeela* (Robbs *et al.*, 1974; Kimura *et al.*, 1976). Although BLB mainly occurs in seedlings and reports in adult plants have treated the pathogen as an epiphyte, a recent study demonstrated that it can cause disease symptoms under field conditions in the state of Minas Gerais, Brazil (Raimundi, 2017). Pathogenic Pch isolates were also recovered from *C. arabica* nurseries in eight municipalities of Puerto Rico (Sánchez *et al.*, 2003). It has been reported that BLB can cause the loss of between 64% and 85% of coffee plants in the nurseries (Sánchez *et al.*, 2003). It is likely that the importance of BLB has been underestimated because it is difficult to

phenotypically differentiate it from BHB, as the two diseases can be found in the same geographic area (Raimundi, 2017).

Plant genetic resistance

To the best of the authors' knowledge only one study has reported on variations of susceptibility to BLB among coffee genotypes. Sánchez *et al.* (2003) tested the susceptibility of 16 hybrids, three *C. arabica* varieties and two species (*Coffea canephora* and *Coffea liberica*) to a Pch isolate. The varieties Bourbón, Pacas and Caturra were susceptible to that isolate whereas *C. liberica* 'Excelsa' and *C. canephora* 'Robusta' were resistant.

Taxonomy, host range and genetic diversity

Pseudomonas cichorii is a rod-shaped, strictly aerobic, fluorescent, Gram-negative bacterium that belongs to the *Pseudomonadaceae* family. Besides coffee, it is also a pathogen of many other plant species of economic importance, including lettuce (Cottyn *et al.*, 2009) and eggplant (Hojo *et al.*, 2008). Phylogenetic studies on Pch are scarce and the very few reported did not include strains obtained from coffee plants with symptoms.

Pathogen detection

Pseudomonas cichorii identification in diseased samples have been accomplished mostly using morphological, physiological and biochemical methods (Sánchez *et al.*, 2003; Cottyn *et al.*, 2009; Yu & Lee, 2012; Imriz & Çinar, 2015). Recently, an ELISA-based method for strains that cause pith necrosis on tomato plants with potential use in the detection of coffee-associated strains was reported (Imriz & Çinar, 2015). Also, a real-time PCR method based on amplification of the *hrpRST* region (Cottyn *et al.*, 2011) for specific detection of Pch associated with lettuce was developed, which was successfully used to detect Pch recovered from coffee samples with leaf blight symptoms (Raimundi, 2017). Several genome sequences of coffee-associated Pch strains that could aid in designing methods for host-specific detection of the pathogen are publicly available.

Mechanisms of pathogenicity

Little effort has been made to understand the molecular mechanisms underlying the ability of Pch to cause disease on coffee plants. Some knowledge about its pathogenicity has been gained studying strains that cause disease on some other host plants. Interestingly, like many other phytopathogenic bacteria, Pch carries a T3SS (Hojo *et al.*, 2008; Ramkumar *et al.*, 2015), but it seems not to be essential for pathogenesis on some host plants. Hojo *et al.* (2008) demonstrated that the functions of the *hrcG*, *hrcT*, *hrpL* and *hrpS* genes in Pch SPC9018 are required to cause disease on eggplant (*Solanum melongena* 'Senryo No. 2'), celery (*Apium graveolens* 'Topseller'), sweet pepper (*Capsicum annuum* 'Shosuke') and okra (*Abelmoschus esculentus* 'Gulliver') but not on lettuce (*Lactuca sativa* 'Success') plants. Similar Pch dependence on *hrp* genes to cause disease on some hosts but not on others was observed when *hrpW* (Kajihara *et al.*, 2012) and *hopA1* (Hung *et al.*, 2014) were deleted in strain JBC1. In Pch, the *aldH* and *pat* genes, coding for an aldehyde dehydrogenase and a phosphinothricin *N*-acetyltransferase, respectively, are linked to the *hrp* gene cluster, and their deletions also result in the inability of the bacterium to cause disease on eggplant but not on lettuce (Tanaka *et al.*, 2012). Overall, these observations indicate that Pch uses different mechanisms to cause disease on different hosts. Whether the functions of the *hrp*, *aldH* or *pat* genes are required for pathogenicity on coffee plants remains to be determined.

Other genes have also been implicated in the virulence of Pch, including those coding for the formation of the flagellum (*fliI* and *fliJ*) (Hung *et al.*, 2016), for the production of the corpeptin-related cyclic lipopeptide cichopeptin A (*cipA*), and linear lipopeptides of the syringafactin family named cichofactins A and B (*cifA* and *cifB*) (Pauwelyn *et al.*, 2013; Huang *et al.*, 2015). Lipopeptides are molecules composed of a fatty acid tail linked to an oligopeptide that, due to their amphipathic nature, can insert into plasma membranes forming pores and causing cell death (Raaijmakers *et al.*, 2006).

Coffee leaf scorch

Disease symptoms, aetiology and dissemination

Coffee leaf scorch (CLS), also referred to as atrophy of branches, was first recorded in São Paulo state (Paradela Filho *et al.*, 1997), and demonstrated to be caused by *X. fastidiosa* in 1998 (Lima *et al.*, 1998). The bacterium is transmitted from plant to plant by insects of the Cicadellidae family that acquire it when feeding on infected branches (Silva *et al.*, 2007; Marucci *et al.*, 2008). Although vector transmission is an important factor contributing to short-range dissemination, introduction of the bacterium into new geographic areas is more likely through infected but symptomless plant material (Almeida & Nunney, 2015). The bacterium colonizes the xylem vessels of host plants, as well as the foregut of its insect vectors (Newman *et al.*, 2004; Chatterjee *et al.*, 2008; Almeida *et al.*, 2012a).

Disease symptoms resemble those caused by nutritional deficiency and herbicide toxicity and are frequently observed in the upper part of the plant. They include plant decay concomitant with chlorosis and defoliation symptoms, leaf malformation, premature leaf senescence at the base of branches, internode shortening (zinc deficiency) (Fig. 1d,e), and reductions in fruit size and quality (Paradela Filho *et al.*, 1997; Lima *et al.*, 1998). As a consequence, plants affected by CLS commonly exhibit dwarfism, marginal necrosis, reduced yield and are more susceptible to abiotic stresses (Rocha *et al.*, 2010a,b; Coletta-Filho *et al.*, 2016). Disease incidence of

c. 100% and high severity have been reported in some Brazilian localities (Rocha *et al.*, 2010a).

Plant genetic resistance

The coffee genetic resistance to *X. fastidiosa* has been poorly investigated. A few reports conducted under field conditions in the Brazilian state of São Paulo indicate variation in the level of infection among *C. arabica* cultivars, *C. liberica* varieties and their interspecific hybrids. For instance, it was observed that Acaiá IAC 474-19 was less severely affected by the disease than Catuaí Vermelho IAC 81 in Campinas (Queiroz-Voltan *et al.*, 2006); cultivars Catuaí and Mundo Novo showed more disease severity than other commercial cultivars in Mococa, particularly during rainy periods (Queiroz-Voltan *et al.*, 2004). Accessions of *C. liberica* var. *liberica*, *C. liberica* var. *dewevrei* and *C. arabica* × *C. liberica* var. *dewevrei* exhibited less infection incidence and supported lower bacterial numbers in Londrina, Paraná state (Yorinori *et al.*, 2003). So far, complete resistance to *X. fastidiosa* in *Coffea* spp. has not been reported.

Taxonomy, host range and genetic diversity

Xylella fastidiosa is a Gram-negative bacterium that belongs to the family *Xanthomonadaceae*, order *Xanthomonadales* of the *Gammaproteobacteria*. Although the bacterium does not grow on culture media routinely used in the laboratory, several special (well-defined) culture media can be used instead, on which it grows very slowly at an optimal temperature of 26–28 °C. The first disease caused by *X. fastidiosa* was reported in the late 19th century on grapevine in California, USA, and the bacterium properly identified later by Wells *et al.* (1987). Based on phylogenetic studies, the species has been proposed to consist of several distinct subspecies, namely, *fastidiosa* (causing Pierce's disease of grapevines and leaf scorch of almond), *multiplex* (leaf scorch of peach, oak and almond), *pauca* (Xfp; citrus variegated chlorosis and leaf scorch of coffee and olive), *sandyi* (leaf scorch of oleander), *tashke* (isolated from *Chitalpa tashkenensis*) and *morus* (leaf scorch of mulberry) (Mendonça *et al.*, 2017; Rapicavoli *et al.*, 2017).

Diseases on different host species caused by strains of subspecies *pauca* have been reported to be sympatric. For instance, citrus variegated chlorosis (CVC), CLS and olive leaf scorch are all present in some Brazilian states (Coletta-Filho *et al.*, 2016; Francisco *et al.*, 2017). Although there appears to be some degree of host specialization within the subspecies *pauca* (i.e. strain specificity for either citrus or coffee; Almeida *et al.*, 2008; Prado *et al.*, 2008), cross-infection has been reported (Li *et al.*, 2001; Almeida *et al.*, 2008; Francisco *et al.*, 2017). In addition, some strains that cause CVC and CLS can also cause disease symptoms in several commercial grapevine cultivars (Li *et al.*, 2002). Subspecies *pauca* was also associated with disease symptoms in *Prunus persica* and *Quercus ilex* in France (Denancé *et al.*, 2017) and in olive trees in Italy (Giampetruzzi *et al.*, 2017).

Currently, multilocus sequence typing (MLST) is the most widely accepted genotyping technique for assessing the genetic diversity of *X. fastidiosa* (Nunney *et al.*, 2010, 2014; Yuan *et al.*, 2010). However, it has been shown that even MLST is not enough to distinguish between closely related strains, sometimes requiring the use of additional gene sequences (Bleve *et al.*, 2016). In any case, an MLST system based on seven housekeeping genes (*leuA*, *petC*, *malF*, *cysG*, *holC*, *nuoL* and *glrT*) to rapidly classify *X. fastidiosa* strains into sequence types (STs) has been proposed (Yuan *et al.*, 2010; Nunney *et al.*, 2014). This system provides good insights into the phylogenetic position of the strains but is not clearly associated with host specificity.

Considerable effort has been made to study the genetic variability of *X. fastidiosa* populations that cause disease on coffee, citrus and olive (Almeida *et al.*, 2008; Nunney *et al.*, 2012; Saponari *et al.*, 2013; Elbeaino *et al.*, 2014; Haelterman *et al.*, 2015; Coletta-Filho *et al.*, 2016). Phylogenetic analysis using different molecular markers has consistently grouped strains from coffee and citrus in a distinct monophyletic group separated from strains from North America. Overall, those studies also indicate that citrus strains are genetically different from coffee strains, but that some recombination between the two populations can occur (Almeida *et al.*, 2008; Nunney *et al.*, 2012). MLST analysis showed that different *X. fastidiosa* STs can cause disease on the same host plant (Coletta-Filho *et al.*, 2016; Bergsma-Vlami *et al.*, 2017), in particular on *C. arabica* (Bergsma-Vlami *et al.*, 2017). Also, the same ST can cause disease on different host plants (Nunney *et al.*, 2014) and the same ST that affects coffee plants has been recorded in very distant geographic areas (Loconsole *et al.*, 2014; Nunney *et al.*, 2014; Almeida & Nunney, 2015; Coletta-Filho *et al.*, 2016). Importantly, sequence types ST72 and ST76, corresponding to subspecies *sandyi*, were identified in coffee samples intercepted in France (Denancé *et al.*, 2017) and Italy (Loconsole *et al.*, 2014; Marcelletti & Scortichini, 2016).

Results of several studies have demonstrated a high rate of genetic recombination among *X. fastidiosa* strains (Almeida *et al.*, 2008; Jacques *et al.*, 2016; Coletta-Filho *et al.*, 2017; Denancé *et al.*, 2017). Importantly, genetic recombination between citrus and coffee strains collected from citrus and coffee plants with symptoms was evidenced by the sequences of the gene markers *leuA*, *rfbD*, *petC*, *malF*, *cysG* and *holC* (Almeida *et al.*, 2008; Nunney *et al.*, 2012). Based on their observations, Nunney *et al.* (2012) suggested that Xfp became pathogenic to citrus and coffee recently via intersubspecific recombination, possibly via introgression from subspecies *multiplex*, and concluded that recombination contributes more than point mutations to the genetic variation of *X. fastidiosa*. Such a high rate of recombination makes it difficult to make phylogenetic inferences and develop reliable methods for detection.

Besides the high rate of genetic recombination revealed by MLST analysis, clear understanding of the biology of

X. fastidiosa is further complicated by the large proportion of flexible gene pools present in its genomes. In a genome comparison study, differences in the flexible genome among *X. fastidiosa* strains causing disease in diverse hosts were found (Nunes *et al.*, 2003). Several genomic islands, some of them putatively associated with pathogenicity, were identified. Notably, citrus and coffee strains share genomic islands (such as GI_1) not present in strains that infect other hosts. It was estimated that the flexible pool of *X. fastidiosa* constitutes at least 18% of the total genome (Nunes *et al.*, 2003).

Pathogen detection

Several immunological and molecular methods for specific detection of *X. fastidiosa* have been developed by diverse research groups (comprehensively reviewed by Baldi & La Porta (2017)). Some of the PCR-based methods were designed to detect the pathogen at the species level using primers targeted to genomic regions conserved among all subspecies (Francis *et al.*, 2006), others to differentiate among a subset of subspecies (Hernandez-Martinez *et al.*, 2006), and yet others to detect a specific subspecies (Pooler & Hartung, 1995; Brady *et al.*, 2012). Although it is believed that the method that best discriminates among *X. fastidiosa* strains is MLST (Almeida *et al.*, 2008; Yuan *et al.*, 2010; Nunney *et al.*, 2012), lack of correlation has been observed between detection using subspecies-specific primers and MLST (Denancé *et al.*, 2017). Recently, a protocol referred to as single nucleotide primer extension (SNuPE) to differentiate between citrus and coffee strains was proposed (Montes-Borrego *et al.*, 2015). The authors of this protocol noticed that SNPs present in the *gyrB* locus were able to better discriminate between these two populations than the loci commonly used in MLST.

Mechanisms of pathogenicity

Studies on the molecular mechanisms underlying the pathogenicity of *X. fastidiosa* toward coffee plants have not been published. The vast majority of knowledge on the pathogenicity of *X. fastidiosa* has been obtained from strains that cause Pierce's disease and CVC (Mendonça *et al.*, 2017; Rapicavoli *et al.*, 2017). It is presumed that some of those mechanisms are shared among strains that cause disease on different host species. As in other hosts (De Souza *et al.*, 2004; Cardinale *et al.*, 2018), *X. fastidiosa* forms extensive biofilms in the xylem vessels of coffee plants causing their occlusion (Alves *et al.*, 2004), which is thought to be responsible for nutrient deficiency and poor development of aerial parts.

Several bacterial factors contribute to biofilm formation. The importance of the Rpf (for regulation of pathogenicity factors) *quorum-sensing* regulatory system and the diffusible signal factor (DSF) for biofilm formation and disease development in host plants as well as in insect vectors for bacterium transmission has been demonstrated (Newman *et al.*, 2004; Almeida *et al.*, 2012a; Ionescu *et al.*, 2014). Similarly, the involvement of the extracellular polysaccharide (EPS; Killiny *et al.*, 2013), T4P (Smolka *et al.*, 2003; Meng *et al.*, 2005; De La Fuente *et al.*, 2007, 2008; Li *et al.*, 2007) and genes coding for adhesins (Van Sluys *et al.*, 2002; Guilhabert & Kirkpatrick, 2005; Voegel *et al.*, 2010) in *X. fastidiosa* biofilm formation and virulence has also been well documented. Adhesins are high molecular weight proteins that are predicted to be secreted by type V secretion systems (T5SS) (De Souza *et al.*, 2004).

Xylella fastidiosa possesses the main bacterial secretion systems, except for the T3SS, which is usually required for pathogenicity (Mendonça *et al.*, 2017; Rapicavoli *et al.*, 2017). The absence of a T3SS could explain why gene-for-gene resistance has not been observed in its host species. It is believed that bacterial colonization, mobilization of plant nutrients and subversion of plant defence responses are facilitated by the function of the type II secretion system (T2SS) (Roper *et al.*, 2007; Pérez-Donoso *et al.*, 2010). Reports on the contribution of the type I and type IV secretion systems to the pathogenicity of *X. fastidiosa* have not been published. Importantly, several studies conducted with mutations in genes coding for proteins secreted by T2SS (Gouran *et al.*, 2016; Nascimento *et al.*, 2016), T5SS (Guilhabert & Kirkpatrick, 2005; Voegel *et al.*, 2010) as well as with *quorum-sensing rpf* mutants (Ionescu *et al.*, 2014) revealed that the ability of *X. fastidiosa* to form biofilm and to cause disease symptoms can be uncoupled. Consistently, lack of correlation between severity of symptoms and bacterial biofilm formation in the xylem vessels was observed in other studies (Newman *et al.*, 2003; Alves *et al.*, 2004). Those results challenge the notion that the ability of *X. fastidiosa* to cause disease depends exclusively on occlusion of the xylem vessels.

Minor bacterial diseases

Bacterial leaf spot symptoms caused by *P. syringae* pv. *syringae* (Pss) on coffee trees have been reported in Puerto Rico (Cortés Monllor, 1988) and Africa (Baker, 1972; Ramos & Shavdia, 1976; Okioga, 1977). The disease is particularly important in nurseries, in which it can cause plant losses of up to 85% (Ramos & Shavdia, 1976; Okioga, 1977). In Brazil, similar symptoms have been reported for Psgc (Franco Do Amaral *et al.*, 1958; Ramos & Shavdia, 1976), Pch (Robbs *et al.*, 1974; Almeida *et al.*, 2012b; Raimundi, 2017), Psta (Rodrigues *et al.*, 2006, 2017c; Destéfano *et al.*, 2010) and *Robbsia andropogonis* (Ran) (Lopes-Santos *et al.*, 2017; Rodrigues *et al.*, 2017c), making disease diagnosis difficult in the absence of isolation and characterization of the causal agent or of appropriate detection methods. It is not clear if the importance of the disease caused by Pss has been underestimated because it is confused with BHB caused by Psgc or if some misidentification of the aetiologic agent has occurred. A guidance to biochemical identification of bacteria causing necrotic spot symptoms in coffee trees has been provided by Rodrigues *et al.*

(2013). However, simpler and more reliable molecular or serological methods are required.

Robbsia andropogonis was recorded causing the disease bacterial brown spot on *C. arabica* in nursery plants in Brazil (Rodrigues Neto *et al.*, 1981). This species has a broad host range affecting plants belonging to different families (Lopes-Santos *et al.*, 2017). Nevertheless, it has not been found constantly associated with disease in commercial coffee plantations resulting in severe losses.

Concluding remarks

Recurrent outbreaks of bacterial diseases cause significant damage to coffee crops especially in nurseries. One of the major limitations to efficiently control these diseases is the lack of appropriate methods for early detection of the aetiologic agents. In the case of pseudomonads, comparative genomics studies could help identify regions that are specific to each bacterial pathogen and could be useful for the development of sensitive, rapid and simple serological or DNA-amplification-based methods. In that regard, efforts should be made to develop not only methods that require considerable laboratory infrastructure but also methods that could be applied directly in the orchard without the need for highly qualified professionals, such as loop-mediated isothermal amplification (LAMP) or recombinase polymerase amplification (Donoso & Valenzuela, 2018). More efforts should also be made to try to determine the genetic variability and virulence/aggressiveness of the pseudomonad populations causing blight symptoms on coffee plants, which is important for plant resistance selection and deployment. In the case of *X. fastidiosa*, considering its high rates of recombination and introgression, permanent revision of the virulence and genetic variability is necessary, which has direct implication on the specificity of the detection methods. In the absence of evidence for the existence of complete resistance against these coffee bacterial pathogens, it is important to undertake studies aimed to understand the molecular mechanisms underlying horizontal resistance and to identify genes associated with it. Comparative transcriptomic, proteomic and genome-wide association studies (GWAS) could provide important insights into the mechanisms underlying this type of resistance. Such information can be useful for plant breeding or biotechnology programmes in order to obtain genotypes with enhanced (and probable durable) field resistance.

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