Title

Top 10 plant pathogenic bacteria in molecular plant pathology.

Journal

Molecular plant pathology, 13(6)

ISSN

1364-3703

Authors

Mansfield, John
Genin, Stephane
Magori, Shimpei
et al.

Publication Date

2012-08-05

Peer reviewed

Molecular Plant Pathology

MOLECULAR PLANT PATHOLOGY (2012) **13**(6), 614–629 DOI: 10.1111/J.1364-3703.2012.00804.X

Review

Top 10 plant pathogenic bacteria in molecular plant pathology

JOHN MANSFIELD[1], STEPHANE GENIN[2], SHIMPEI MAGORI[3], VITALY CITOVSKY[3], MALINEE SRIARIYANUM[4,†], PAMELA RONALD[4], MAX DOW[5], VALÉRIE VERDIER[6], STEVEN V. BEER[7], MARCOS A. MACHADO[8], IAN TOTH[9], GEORGE SALMOND[10] AND GARY D. FOSTER[11,*]

[1]*Division of Biology, Imperial College, London SW7 2AZ, UK*
[2]*INRA, CNRS, Laboratoire des Interactions Plantes-Microorganismes (LIPM), UMR 441-2594, F-31326 Castanet Tolosan, France*
[3]*Department of Biochemistry and Cell Biology, State University of New York, Stony Brook, NY 11794-5215, USA*
[4]*Department of Plant Pathology, University of California, Davis, CA 95616, USA*
[5]*Department of Microbiology, BioSciences Institute, University College Cork, Cork, Ireland*
[6]*Institut de Recherche pour le Développement, UMR Résistance des Plantes aux Bioagresseurs, IRD-CIRAD-UM2, 911 Avenue Agropolis BP 64501, 34394 Montpellier Cedex 5, France*
[7]*Department of Plant Pathology and Plant-Microbe Biology, 306 Plant Science Building, Cornell University, Ithaca, NY 14853, USA*
[8]*Centro de Citricultura Sylvio Moreira, Instituto Agronômico, Cordeirópolis, Caixa Postal 04, São Paulo 13490-970, Brazil*
[9]*James Hutton Institute, Invergowrie, Dundee DD2 5DA, UK*
[10]*Department of Biochemistry, University of Cambridge, Tennis Court Road, Cambridge CB2 1QW, UK*
[11]*School of Biological Sciences, University of Bristol, Bristol BS8 1UG, UK*

SUMMARY

Many plant bacteriologists, if not all, feel that their particular microbe should appear in any list of the most important bacterial plant pathogens. However, to our knowledge, no such list exists. The aim of this review was to survey all bacterial pathologists with an association with the journal *Molecular Plant Pathology* and ask them to nominate the bacterial pathogens they would place in a 'Top 10' based on scientific/economic importance. The survey generated 458 votes from the international community, and allowed the construction of a Top 10 bacterial plant pathogen list. The list includes, in rank order: (1) *Pseudomonas syringae* pathovars; (2) *Ralstonia solanacearum*; (3) *Agrobacterium tumefaciens*; (4) *Xanthomonas oryzae* pv. *oryzae*; (5) *Xanthomonas campestris* pathovars; (6) *Xanthomonas axonopodis* pathovars; (7) *Erwinia amylovora*; (8) *Xylella fastidiosa*; (9) *Dickeya (dadantii* and *solani)*; (10) *Pectobacterium carotovorum* (and *Pectobacterium atrosepticum*). Bacteria garnering honourable mentions for just missing out on the Top 10 include *Clavibacter michiganensis* (*michiganensis* and *sepedonicus*), *Pseudomonas savastanoi* and *Candidatus Liberibacter asiaticus*. This review article presents a short section on each bacterium in the Top 10 list and its importance, with the intention of initiating discussion and debate amongst the plant bacteriology community, as well as laying down a benchmark. It will be interesting to see, in future years, how perceptions change and which bacterial pathogens enter and leave the Top 10.

*Correspondence: Email: gary.foster@bristol.ac.uk
†Present address: Department of Chemical and Process Engineering, Thai-German Graduate School of Engineering, King Mongkut's University of Technology, North Bangkok, Bangkok 10800, Thailand.

INTRODUCTION

Recently, the journal *Molecular Plant Pathology* considered which viruses would appear in a Top 10 of plant viruses based on their perceived importance, scientifically or economically, in terms of the views of the contributors to the journal (Scholthof *et al*., 2011). This was followed by a similar review on fungi (Dean *et al*., 2012). These surveys were carried out as many papers, reviews and grant applications claim that a particular plant virus or fungal pathogen is of huge importance, and this is probably rightly so.

As a result of the interest generated by the plant virus and fungal pathogen surveys, a similar survey was carried out for plant pathogenic bacteria and, as before, bacterial pathologists with an association with the journal *Molecular Plant Pathology* were contacted and asked to nominate three plant pathogenic bacteria that they would expect to see in a list of the most scientifically/economically important bacterial pathogens. The review, by its very nature, is similar in format and layout to the Top 10 Virus and Top 10 Fungal Reviews (Dean *et al*., 2012; Scholthof *et al*., 2011).

The survey generated 458 votes from the international community, and allowed the construction of a Top 10 bacterial plant pathogen list for the journal *Molecular Plant Pathology* (see Table 1).

The bacterium, or group of pathovars, making the strongest appearance on scientific and economic grounds is *Pseudomonas syringae*, with many voters grouping the various pathovars

Table 1 Top 10 bacterial plant pathogens. The table represents the ranked list of bacteria as voted for by plant bacteriologists associated with the journal *Molecular Plant Pathology*.

Rank	Bacterial pathogen	Author of bacterial description
1	*Pseudomonas syringae* pathovars	John Mansfield
2	*Ralstonia solanacearum*	Stéphane Genin
3	*Agrobacterium tumefaciens*	Shimpei Magori, Vitaly Citovsky
4	*Xanthomonas oryzae* pv. *oryzae*	Malinee Sriariyanum, Pamela Ronald
5	*Xanthomonas campestris* pathovars	Max Dow
6	*Xanthomonas axonopodis* pv. *manihotis*	Valérie Verdier
7	*Erwinia amylovora*	Steven V. Beer
8	*Xylella fastidiosa*	Marcos A. Machado
9	*Dickeya* (*dadantii* and *solani*)	Ian Toth
10	*Pectobacterium carotovorum* (and *P. atrosepticum*)	George Salmond

together, and others voting for individual pathovars. It is clear that *P. syringae* has had a huge impact on our scientific understanding of microbial pathogenicity, and continues to cause economically important plant diseases.

In second place is *Ralstonia solanacearum*, which rates very highly on economic importance worldwide, especially as it has a very broad host range, with affected crops ranging from potato to banana.

In third position is *Agrobacterium tumefaciens*, making a very strong appearance based primarily on its scientific importance. Although this bacterium can cause significant damage in particular crops, its role in scientific breakthroughs and applications clearly attracted votes.

In fourth, fifth and sixth positions are *Xanthomonas* species, all clearly distinctive in their pathology and host targets, with each attracting significant votes as individuals. In fourth and sixth positions are xanthomonads with relatively specific crop targets, namely *Xanthomonas oryzae* pv. *oryzae*, one of the most serious pathogens of rice, and *Xanthomonas axonopodis* pv. *manihotis*, the causal agent of cassava bacterial blight (CBB). *Xanthomonas campestris* pathovars, which cause diseases in a range of crops worldwide, reached fifth position.

In seventh position comes *Erwinia amylovora*, which causes the well-known fire blight disease of ornamentals, fruit trees and bushes. This disease has significant scientific history and is of continuing economic importance.

Xylella fastidiosa rightly has a place in the Top 10 in eighth position, as it is associated with several important diseases of crops and trees. It also has the important scientific claim of being the first phytopathogen (outside of plant viruses) to have had its genome sequenced.

For the entry in ninth position, it was decided to group two species of *Dickeya* together, namely *Dickeya dadantii* and *solani*, as *Dickeya* attracted significant votes, many of which were simply referred to as *Dickeya* spp. This is perhaps understandable as the taxonomy of these bacteria may be described as being in a state of flux. Indeed, the name *Dickeya solani* has not been officially accepted, but it is clear that *Dickeya* spp. cause economically important diseases, particularly in potato.

The final entry in tenth place is *Pectobacterium carotovorum* (also covering *P. atrosepticum*), meriting a place in the Top 10 because of the economic losses linked with the soft rot diseases, but also being responsible for several scientific milestones. This is in addition to some long-standing translational breakthroughs, such as involvement in the treatment of some leukaemias.

Although the aim of this review article was to identify the views of contributors to *Molecular Plant Pathology* with regard to the Top 10 most important plant pathogenic bacteria, the authors are very much aware that importance and priorities can vary locally across continents and disciplines. We are also aware that not all bacteria can make it into any Top 10, for obvious numerical limits, although such bacteria can still be regarded as hugely important. We therefore felt it appropriate to make honourable mentions to bacteria just missing out on the Top 10 list, including *Clavibacter michiganensis* (*michiganensis* and *sepedonicus*) (Eichenlaub and Gartemann, 2011), *Pseudomonas savastanoi* (Rodríguez-Palenzuela *et al*., 2010) and *Candidatus Liberibacter* (pv. *asiaticus*) (Duan *et al*., 2009), all clearly important.

This review contains single-page descriptions of the Top 10, including illustrative figures and key references for further reading. We hope that the review triggers discussion and debate amongst the plant bacteriology community, as well as laying down a benchmark. It will be interesting to see how perceptions change in future years and which bacteria enter and leave the Top 10 list.

1. *PSEUDOMONAS SYRINGAE* PATHOVARS

It seems a little unfair that a team of pathovars has been voted for an award, a bit like a relay team winning the 400-m individual Olympic gold medal. It may of course be argued that the pathovar designation is really unjustified and that we are dealing with one remarkably versatile single species, *Pseudomonas syringae*. This debate is now being resurrected by the emerging detail from genomic sequencing. The criteria for this award were importance to basic science and impact on food production and/or the environment—*P. syringae* scores heavily on all counts.

The economic impact of *P. syringae* is increasing, with a resurgence of old diseases, including bacterial speck of tomato (pv. *tomato*; Shenge *et al*., 2007), and the emergence of new infections of importance worldwide, such as bleeding canker of horse-chestnut (pv. *aesculi*; Green *et al*., 2010). The *European Handbook of Plant Diseases* (Smith *et al*., 1988) describes 28 pathovars, each attacking a different host species. We can now add pv. *aesculi* to this list. Several pathovars cause long-term problems in trees, often through the production of distortions and cankers (e.g. pathovars *savastanoi* and *morsprunorum*). Infections of annual crops are more sporadic, and outbreaks are often caused by sowing contaminated seed. Many reports highlight the seed-borne nature of *P. syringae*, but it is a remarkably adaptive pathogen, emerging in some apparently bizarre sites, such as snow melt waters (Morris *et al*., 2007). Once new infections have established, given favourable conditions of rainfall and temperatures, disease outbreaks are often devastating, as observed with bean halo blight caused by pv. *phaseolicola* (Murillo *et al*., 2010).

Research into the molecular biology of virulence and plant defence against *P. syringae* has opened up new insights into microbial pathogenicity, not only with regard to plants but also with more general significance to human diseases. Pathovars *phaseolicola* and *tomato* have emerged as excellent models for fundamental studies on bacterial attack and plant defence (Arnold *et al*., 2011; Preston, 2000). Notable examples are discoveries concerning the hypersensitive response and pathogenicity (*hrp*) gene cluster encoding the type III secretion system (see Fig. 1), effector trafficking and host targets for defence suppression (Huynh *et al*., 1989; Jovanovic *et al*., 2011; Kvitko *et al*., 2009; Li *et al*., 2002; Zhang *et al*., 2010).

Pseudomonas syringae leads the field in the impact of high-throughput sequencing technologies on our understanding of pathogenicity. Remarkably, the prediction by O'Brien *et al*. (2011) that, '. . . at least two dozen new *P. syringae* genomes will be released this year', has been proven to be correct with the publication of the landmark study by Baltrus *et al*. (2011). So far, a perhaps unexpected feature is that pathovars colonizing strongly unrelated plants are being closely grouped together, for example pv. *savastanoi* (olive) and pv. *phaseolicola* (bean) both lie within the same clade. Genomic analysis, initiated by Joardar *et al*. (2005) and Lindeberg *et al*. (2008), has perhaps the most potential for unravelling the determinants of host specificity. As more genomic sequences are completed, further insight should be gained into the still puzzling role of effector proteins and toxins in defining host range within the species.

Pseudomonas syringae pathovars represent not only the premier plant pathogenic bacterial grouping, but would also probably top the all time pathogen charts including fungi and oomycetes. Research on the effector biology of the filamentous pathogens is very much following in the wake of advances made with *P. syringae* (Cunnac *et al*., 2009; Hann *et al*., 2010; Oliva *et al*., 2010).

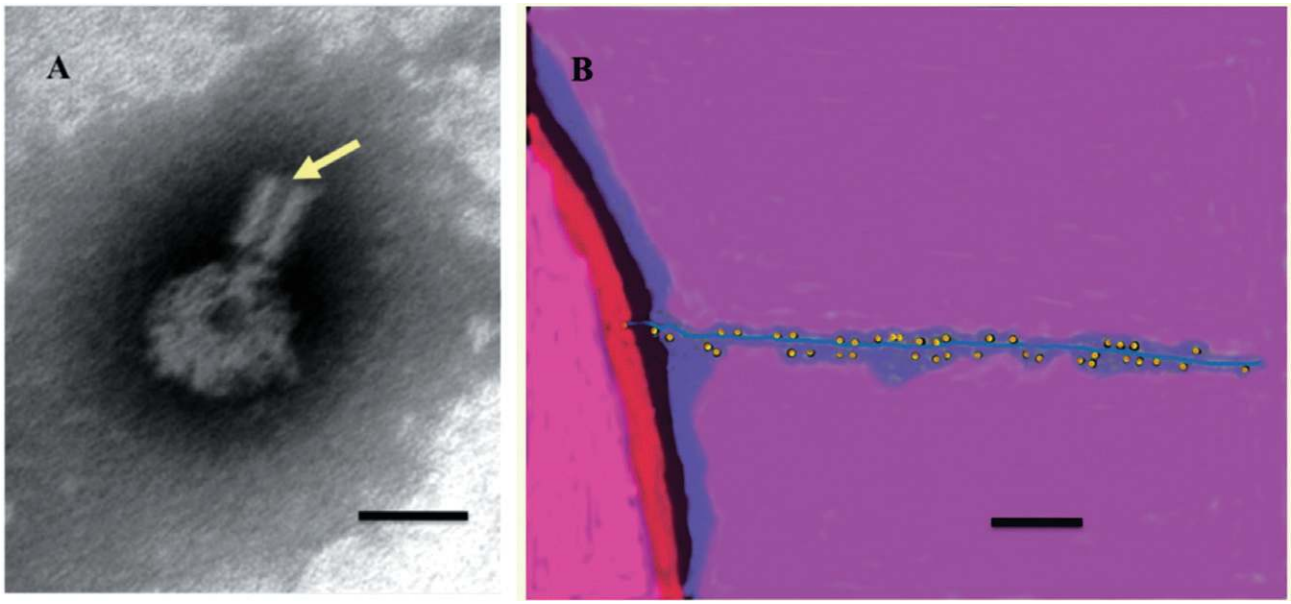

Fig. 1 The type III secretion system (T3SS) of *Pseudomonas syringae* pv. *tomato*. (A) Putative basal body of the T3SS released from membrane preparations after growth in *hrp* inducing medium. The arrow marks the attachment point of the Hrp pilus. Bar, 25 nm. (B) False colour image of the Hrp pilus gold labelled with antibodies to the subunit protein HrpA, emerging from the bacterial surface. Bar, 50 nm. Both images kindly provided by Ian Brown (University of Kent).

2. *RALSTONIA SOLANACEARUM*

Ralstonia solanacearum is probably the most destructive plant pathogenic bacterium worldwide. One of the reasons for this is that the *R. solanacearum* species is composed of a very large group of strains varying in their geographical origin, host range and pathogenic behaviour (Denny, 2006; Genin, 2010). This heterogeneous group is nowadays recognized as a 'species complex' which has been divided into four main phylotypes (phylogenetic grouping of strains). The species as a whole has a very broad host range, infecting 200 plant species in over 50 families, and is the causal agent of potato brown rot, bacterial wilt of tomato, tobacco, eggplant and some ornamentals, as well as Moko disease of banana.

Ralstonia solanacearum is a soil-borne pathogen that infects plants via wounds, root tips or cracks at the sites of lateral root emergence. The bacterium subsequently colonizes the root cortex, invades the xylem vessels and reaches the stem and aerial parts of the plant through the vascular system (Fig. 2). *Ralstonia solanacearum* can rapidly multiply in the xylem up to very high cell densities, leading to wilting symptoms and plant death.

The direct economic impact of *R. solanacearum* is difficult to quantify, but the pathogen is extremely damaging because of its wide geographical distribution and host range; on potato alone, it is responsible for an estimated US$1 billion in losses each year worldwide (Elphinstone, 2005). The incidence of the disease is particularly dramatic for agriculture in many developing countries in inter-tropical regions in which *R. solanacearum* is endemic. In areas in which the organism has quarantine status, it is also responsible for important losses because of regulatory eradication measures and restrictions on further production on contaminated land. Disease management remains limited and is hampered by the faculty of the pathogen to survive for years in wet soil, water ponds, on plant debris or in asymptomatic weed hosts, which act as inoculum reservoirs. Breeding for resistance, although effective in a few cases, is hampered by the broad diversity of the pathogenic strains.

As a root and vascular pathogen, *R. solanacearum* is a model system for the study of bacterial pathogenicity. The bacterium was one of the first plant pathogen genomes to be entirely sequenced (Salanoubat *et al*., 2002), and the development of pathosystems with model plants, such as *Arabidopsis*, or the legume *Medicago truncatula* has facilitated genetic and molecular studies on both the plant and bacterial partners. The pathogenicity of *R. solanacearum* relies on a type III secretion system, and many studies have been conducted on this topic since the first description of a *hrp* mutant phenotype by Boucher *et al*. (1985). Many other pathogenicity factors have been identified and characterized, whose expression is orchestrated by an atypical quorum-sensing molecule structurally related to the diffusible signal factor (DSF) family (Flavier *et al*., 1997).

Future research in this field will include a better understanding of the molecular bases underlying the adaptation of this versatile group of strains to such a diverse range of hosts. Another major task to address is how our increasing knowledge of the sophisticated mechanisms developed by *R. solanacearum* to promote plant susceptibility could be used to engineer novel and durable protection strategies to fight this devastating disease.

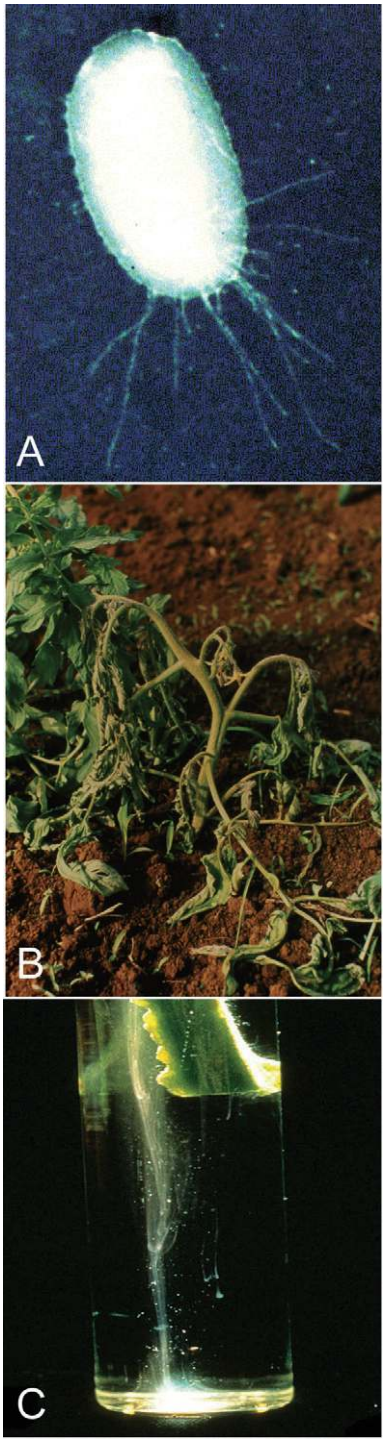

Fig. 2 *Ralstonia solanacearum* (A, photograph J. Vasse) and disease wilting symptoms on tomato (B) with bacteria oozing from the vascular system after stem section (C).

3. *AGROBACTERIUM TUMEFACIENS*

More than a century ago, Smith and Townsend (1907) identified *Agrobacterium tumefaciens* as the causative agent of crown gall tumour, one of the most serious plant diseases affecting various crop species worldwide. In nature, this soil-borne bacterium induces neoplastic growths (Fig. 3) at wound sites on host plants and severely limits crop yield and growth vigour. This deleterious effect of *A. tumefaciens* has unquestionably contributed to a driving force behind long-lasting *Agrobacterium* research. However, *A. tumefaciens* is not just another phytopathogen, but possesses a very rare feature: the ability for genetic transformation.

The 'Eureka' moment came in the late 1970s when Mary-Dell Chilton and Eugene Nester with their colleagues demonstrated that the specific DNA segment (now known as the T-DNA) of the bacterial tumour-inducing (Ti) plasmid was present in the genome of infected plant cells (Chilton *et al*., 1977). This landmark discovery cast the spotlight on *Agrobacterium* as the first organism capable of trans-kingdom gene transfer. Since then, a great deal has been learned about the molecular mechanisms underlying *A. tumefaciens*-mediated genetic transformation, which has emerged as a highly complex process regulated by numerous bacterial and host factors (reviewed by Gelvin, 2010; Pitzschke and Hirt, 2010; Tzfira and Citovsky, 2002; Zupan *et al*., 2000). Briefly, *A. tumefaciens* perceives phenolic compounds exuded from plant wound tissues and activates the expression of several effectors, termed virulence (Vir) proteins. Some of these factors mediate the generation of a single-stranded copy of T-DNA (T-strand) and its transport into the host cell through a type IV secretion system. In addition to the T-strand, several Vir proteins are also translocated into plant cells. These exported effectors, together with multiple host factors, facilitate the nuclear import of the T-strand and its subsequent integration into the host genome. Finally, genes involved in auxin and cytokinin biosynthesis are expressed from the integrated T-DNA, leading to abnormal cell proliferation in the infected tissues and the formation of tumours, i.e. crown galls (Fig. 3).

Although the details on its molecular basis are still emerging, the discovery of the *Agrobacterium*-mediated genetic transformation of plants ushered in a new era of plant molecular biology. In 1983, Chilton and colleagues reported that an engineered T-DNA carrying a foreign gene could be transferred to tobacco plants and maintained through regeneration (Barton *et al*., 1983). Since this first demonstration of transgenic plants, substantial conceptual and technical advances have been achieved to make the *Agrobacterium*-mediated genetic engineering of plants more feasible in the daily practice of basic research as well as biotechnology (Fig. 4). For example, the advent of binary vectors, a system of two separate replicons that house the T-DNA and virulence genes and function in both *Escherichia coli* and *A. tumefaciens*, has made it much easier to manipulate the T-DNA. Owing to its incredibly wide host range, which, under laboratory conditions, includes most eukaryotic organisms (Lacroix *et al*., 2006), high efficiency and sophisticated modern transformation technology, *A. tumefaciens* is now a transformation vehicle of choice for the genetic manipulation of most plant species, including the model plant *Arabidopsis thaliana*, as well as numerous fungal species.

Agrobacterium tumefaciens never ceases to amaze plant biologists and pathologists. Even after 100 years of research, we are still discovering novel mechanisms that underlie the interactions of *A. tumefaciens* with its hosts, and are only beginning to understand how truly clever this pathogen is. For instance, recent studies have revealed that *A. tumefaciens* can subvert the host defence machinery for the active promotion of infection (Djamei *et al*., 2007; Zaltsman *et al*., 2010). In the foreseeable future, therefore, *A. tumefaciens* will continue to serve not only as a powerful tool for plant genetic engineering, but also as an excellent model organism to decipher host–pathogen interactions.

Fig. 3 A crown gall on cherry trunk caused by *Agrobacterium tumefaciens*.

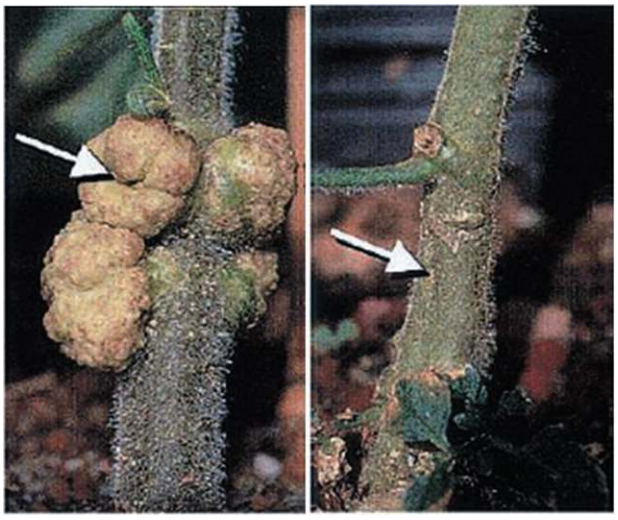

Fig. 4 Wild-type tomato plant developing crown gall tumours (left) and *Agrobacterium tumefaciens*-resistant transgenic tomato plant generated by *A. tumefaciens*-mediated genetic transformation (right) illustrate two important aspects of *A. tumefaciens*: one as a pathogen and another as a tool for genetic engineering (reproduced with permission from Escobar *et al*., 2001).

4. *XANTHOMONAS ORYZAE* (ORYZAE)

Bacterial leaf blight (BLB), caused by *Xanthomonas oryzae* pv. *oryzae* (*Xoo*), is found in both tropical and temperate regions. BLB also occurs in Australia, Africa, Latin America, the Caribbean and the USA (Mew *et al*., 1993; Mizukami and Wakimoto, 1969). Yield losses of 10%–50% from BLB have been reported (Ou, 1972). Outbreaks of BLB are most common during the monsoon season in South-East Asia and India (Mew *et al*., 1993). Rice was introduced for cultivation into the USA (North Carolina) more than 200 years ago and has been cultivated in other parts of the USA for over 100 years. Although many rice diseases have either been introduced or developed on rice during the history of its cultivation in the USA, *Xoo* has not established in the USA. The climates of rice-producing areas in the USA and USA rice cultivation practices are not conducive to the long-term survival or spread of *Xoo*. For these reasons, *Xoo* is of low risk to US agriculture.

BLB is efficiently controlled by the use of resistant rice cultivars. However, because *Xoo* has the capacity to express effectors that suppress some host defence responses, often this resistance is eventually overcome (Verdier *et al*., 2011). Resistance genes of the non-RD pattern recognition receptor class typically confer long-lasting resistance because they recognize conserved microbial signatures, which, when mutated, cripple the virulence of the pathogen (Han *et al*., 2011; Ronald and Beutler, 2010; Schwessinger and Ronald, 2012). Control of the disease with copper compounds, antibiotics and other chemicals has not proven to be effective (Mew, 1989; Singh *et al*., 1980).

Xanthomonas oryzae pv. *oryzae* is a rod-shaped, Gram-negative bacterium. It produces a yellow soluble pigment, called xanthomonadin (Fig. 5), and extracellular polysaccharide (EPS). EPS is important in the protection of bacteria from desiccation and for the attenuation of wind- and rain-borne dispersal (Ou, 1972; Swings *et al*., 1990). *Xoo* is disseminated by irrigation water systems, splashing or wind-blown rain, as well as by contaminated rice stubble from the previous crop season, which is the most important source of primary inoculum (Mizukami and Wakimoto, 1969; Murthy and Devadath, 1984). *Xoo* infects the rice leaf typically through hydathodes at the leaf tip, broken trichomes, leaf margins and wounds in the leaves or roots, multiplies in the intercellular spaces and enters into xylem vessels (Fig. 5) (Noda and Kaku, 1999; Ou, 1985; Park *et al*., 2010). Within a few days of infection, bacterial cells and EPS fill the xylem vessels and ooze out from the hydathodes, and form beads of exudate on the leaf surface, a characteristic sign of the disease and a source of secondary inoculum (Mew *et al*., 1993).

Similar to *Xanthomonas campestris* pv. *campestris* (*Xcc*), *Xoo* also produces a range of virulence factors, including EPS, extracellular enzyme and type III effectors, which are essential for virulence (Mole *et al*., 2007). *Xoo* employs two different types of quorum-sensing factors, DSF and Ax21 (activator of Ax21-mediated immunity), a small, N-terminally processed, type I secreted protein (Han *et al*., 2011; He *et al*., 2010). A dual role for Ax21 in quorum sensing and in the activation of the host innate immune response has recently been demonstrated (Han *et al*., 2011). Ax21 mediates biofilm formation, motility and virulence. Whereas the *rpf* (regulation of pathogenicity factors) gene cluster is required for DSF-mediated quorum sensing (Jeong *et al*., 2008), *rax* genes are required for Ax21-mediated quorum sensing (Lee *et al*., 2006). Ax21 is broadly conserved in all *Xanthomonas* species and in related genera, and some of these orthologues can also activate XA21-mediated immunity (Lee *et al*., 2009).

The genome sequences of three *Xoo* strains (MAFF311018, KACC10331, PXO99A) have been completed (Lee *et al*., 2005; Ochiai *et al*., 2005; Salzberg *et al*., 2008) and the genome sequencing of eight additional *Xoo* strains is underway (Verdier *et al*., 2011). Comparative genomic analysis of different *Xoo* strains has revealed a large number of genomic rearrangements and transcriptional activator-like (TAL) effector gene recombinations, as well as a large number of insertion sequence (IS) elements (Ochiai *et al*., 2005; Ryan *et al*., 2011; Salzberg *et al*., 2008). Several genetic studies have suggested that the activity of IS elements and recombination among TAL effector genes have contributed to the diverse race structure within *Xoo* (Ochiai *et al*., 2005; Ponciano *et al*., 2004; Rajeshwari and Sonti, 2000). The comparative analysis of the genomic sequence has facilitated an understanding of the diversity and evolution of *Xoo* (Salzberg *et al*., 2008). Complete genome sequences have also facilitated the development of markers that are useful for epidemiological studies.

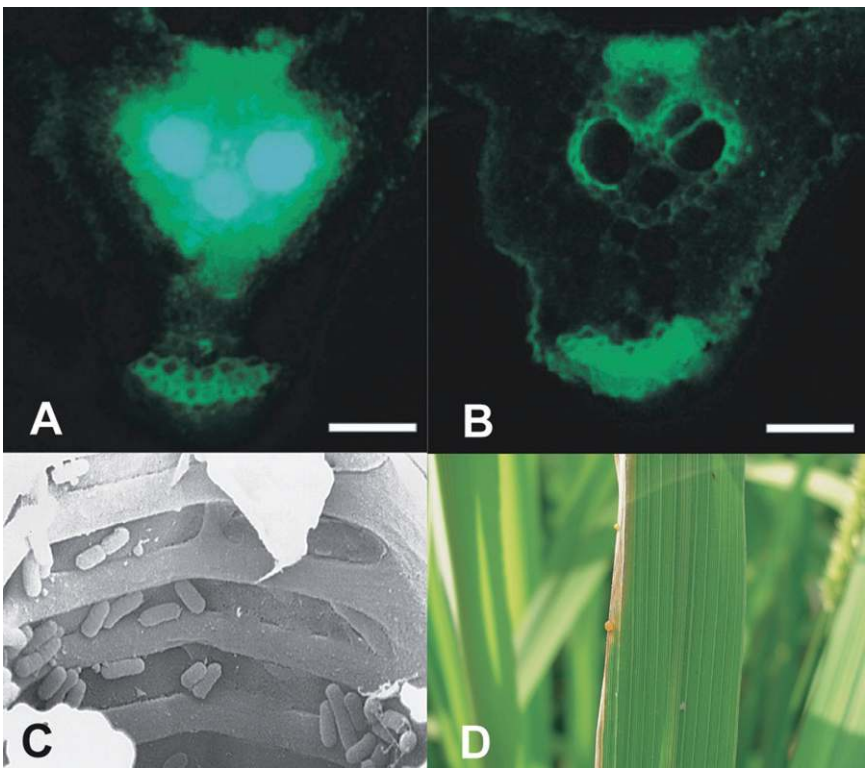

Fig. 5 Visualization of *Xanthomonas oryzae* pv. *oryzae* (*Xoo*) in rice plants. (A, B) Transverse leaf sections of rice infected with *Xoo* strain PXO99 expressing the green fluorescence of rice cultivar TP309 (susceptible) (A) and TP309-XA21 (resistant) (B). Images were observed with excitation from 450 to 490 nm and emitted light collected at 520 nm at 40× magnification using a Zeiss Axiophot fluorescence microscope, 12 days after inoculation. The bars in (A) and (B) represent 50 μm. (C) Scanning electron micrograph of *Xoo* cells in the xylem vessel of a rice leaf. (D) Close-up of *Xoo*-infected rice leaf. Bacterial cells fill the xylem vessels and ooze out at hydathodes, forming beads or strands of exudate on the leaf surface, a characteristic sign of the disease. Photographs in (A) and (B) courtesy of S. W. Han (reprinted from *BMC Microbiol*. 2008; 8: 164). Photograph in (C) courtesy of J. Leach (reprinted from *Mol*. *Plant Pathol*. 2006; 7(5): 303–324). Photograph in (D) courtesy of the Bureau of Rice Research and Development, Thailand (http://www.brrd.in.th).

5. *XANTHOMONAS CAMPESTRIS* PATHOVARS

Pathovars of *Xanthomonas campestris* cause diseases of agronomic importance throughout the world. Among the most notable of these pathogens are *Xanthomonas campestris* pv. *campestris* (*Xcc*), the causal agent of black rot of crucifers that affects all cultivated brassicas, *X. campestris* pv. *vesicatoria* (*Xcv*), now reclassified as *X. euvesicatoria*, the causal agent of bacterial spot of pepper and tomato, and *X. campestris* pv. *malvacearum* (*Xcm*, now *X. axonopodis* pv. *malvacearum*), which causes angular leaf spot of cotton. The diseases caused by these bacteria are particularly severe in regions with a warm and humid climate, although black rot is also economically important in temperate regions, e.g. in Cornwall and other western areas of the UK. *Xcc* is also important as a producer of the EPS xanthan, which is used as a food additive and in the pharmaceutical and oil-drilling industries.

Studies of these bacteria have had considerable scientific impact, which has not been restricted to the discipline of molecular plant pathology. Work on *Xcm* provided the first demonstration for the hypothesis that a gene-for-gene pattern governs interactions between bacterial pathogens and plants (Gabriel *et al*., 1986). Work on *Xcv* established the genetic basis of the triggering of disease resistance in pepper, leading to the isolation of genes specifying avirulence on pepper cultivars containing the *Bs1*, *Bs2* or *Bs3* (for bacterial spot) resistance genes (Boch and Bonas, 2010; Minsavage *et al*., 1990). AvrBs3 is the paradigm member of the large family of TAL type III effector proteins in *Xanthomonas* spp. It was subsequently established that this effector is translocated to the nucleus of the plant cell, where it influences gene expression by binding to plant promoters (Boch and Bonas, 2010). The 'code' governing promoter recognition by the majority of effectors of this family has been determined (Fig. 6). The knowledge of this code affords great potential for biotechnology, e.g. by engineering promoters with boxes for TAL effectors to drive the expression of resistance genes or by allowing the generation of custom-designed DNA-binding specificities.

Work on *Xcc* led to the identification of the genes involved in xanthan biosynthesis (Capage *et al*., 1987; Vorhölter *et al*., 2008) and the *rpf* gene cluster, which acts to control the synthesis of extracellular enzymes and xanthan, and contributes to virulence. Studies of the function of the Rpf gene products led to the discovery of the cell–cell signalling system mediated by DSF, which was subsequently identified as a *cis*-unsaturated fatty acid (Ryan and Dow, 2011). The *rpf* genes involved in DSF synthesis and perception are conserved in all xanthomonads, including *Xylella fastidiosa* and *Stenotrophomonas* spp., some strains of which are nosocomial human pathogens. Furthermore, DSF signalling controls virulence in some, but not all, of these bacteria, although the precise role differs between organisms (Ryan and Dow, 2011). RpfG, the regulatory protein involved in DSF signal transduction, contains a histidine-aspartic acid-glycine-tyrosine-proline (HD-GYP) domain. Studies in *Xcc* were the first to establish the regulatory function of an HD-GYP domain regulator and its enzymatic activity as a phosphodiesterase degrading the second messenger cyclic di-guanosine monophosphate (di-GMP) (Ryan *et al*., 2006). These observations have contributed to an understanding of cyclic di-GMP signalling in many organisms, as the HD-GYP domain is widely conserved in bacteria, including plant, animal and human pathogens.

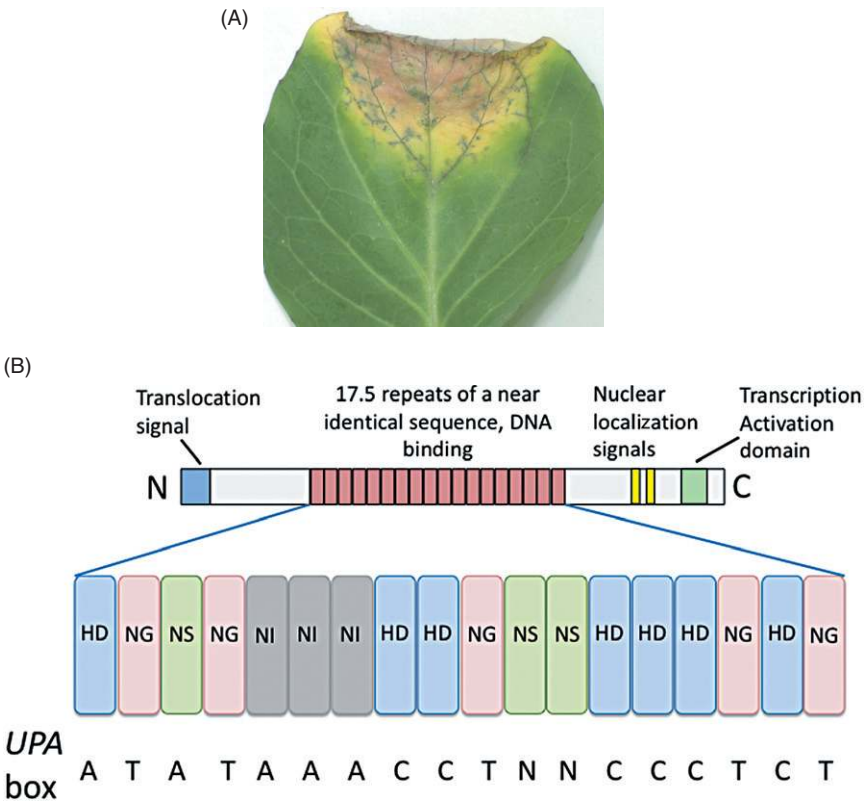

Fig. 6 (A) Black rot disease symptoms on cabbage caused by *Xanthomonas campestris* pv. *campestris*, showing the characteristic blackening of the leaf veins (image kindly provided by Sarah Schatschneider and Karsten Niehaus, University of Bielefeld). (B) Domain architecture of the AvrBs3 effector showing the variations at positions 12 and 13 in the repeats and the nucleotides recognized in the consensus *UPA* (upregulated by AvrBs3) box (see Boch and Bonas, 2010).

6. *XANTHOMONAS AXONOPODIS*

Xanthomonas axonopodis pv. *manihotis*

The genus *Xanthomonas* currently consists of 20 species including *X. axonopodis* (Vauterin *et al*., 2000). Six distinct genomic groups have been defined within *X. axonopodis*, with many pathovars causing economically important diseases on different host plants of agronomic significance (Rademaker *et al*., 2005; Young *et al*., 2008).

Cassava (*Manihot esculenta*) is the staple food of nearly 600 million people in the world's tropical regions. *Xanthomonas axonopodis* pv. *manihotis* (*Xam*) is the causal agent of CBB, a major disease, endemic in tropical and subtropical areas. This foliar and vascular disease severely affects cassava production worldwide. Losses of between 12% and 100% affect both yield and planting material (Lozano, 1986; Verdier *et al*., 2004). Over recent years, a significant recurrence of the disease has been reported in different regions in Africa and Asia. *Xam* induces a wide combination of symptoms, including angular leaf lesions, blight, wilt, stem exudates and stem canker (Figs. 7 and 8). Host resistance is still the most effective way to control this disease. However, no breeding strategy is being developed for the control of CBB disease. Only two cassava CBB resistance genes have been identified so far (C. Lopez, personal communication, Universidad Nacional, Bogota, Colombia). Plant defence responses to *Xam* have been well characterized (Fig. 9) (Boher and Verdier, 1995; Boher *et al*., 1997; Kpémoua *et al*., 1996). Genomic tools for cassava, such as a large expressed sequence tag (EST) database and a cassava microarray, have been developed and used for *Xam*–plant expression studies (Lopez *et al*., 2004, 2005).

The pathogenicity of *Xam* relies, in part, on a type III secretion system which translocates effectors into plant cells. A strong effect in *Xam* pathogenicity has been demonstrated for a small number of effectors, including transcriptional activator-like effector (A. Bernal, personal communication, Universidad de Los Andes, Bogota, Colombia). Different pathotypes of *Xam* have been reported in different countries in Africa and South America (Restrepo *et al*., 2000a; Wydra *et al*., 2004), and studies using DNA fingerprinting methods have shown that *Xam* pathogen populations are variable both within and across Africa, South America and Asia (Restrepo and Verdier, 1997; Restrepo *et al*., 2000b; Verdier *et al*., 1993). In Colombia, the existence of a geographical differentiation of *Xam* strains in different ecozones has been shown (Restrepo and Verdier, 1997). The exchange of contaminated cassava materials has contributed to the migration of strains and, consequently, has influenced the genetic structure of *Xam* populations. Climate changes may also influence the genetic diversity and population structure of *Xam* (Restrepo *et al*., 2000b).

Xam is considered as a quarantine organism in all countries that grow cassava. A simple and fast procedure has been employed to rapidly identify *Xam* strains (Ojeda and Verdier, 2000; Verdier *et al*., 1998), and can easily be implemented to certify plant materials.

Recently, the sequencing of a *Xam* genome (Colombian strain CIO151) was completed at the Universidad de los Andes (Bogota, Colombia) and the annotation is in progress through the French *Xanthomonas* consortium (http://www.reseau-xantho.org, http://www.xanthomonas.org). Access to this and subsequent *Xam* genomes should open up new applications for the comparative and functional genomics of *Xam*, and will accelerate the development of new molecular typing techniques useful for epidemiological and phylogenetic studies of *Xam*, as well as diagnostic primers. Much remains to be carried out to improve our ability to combat this economically important plant disease.

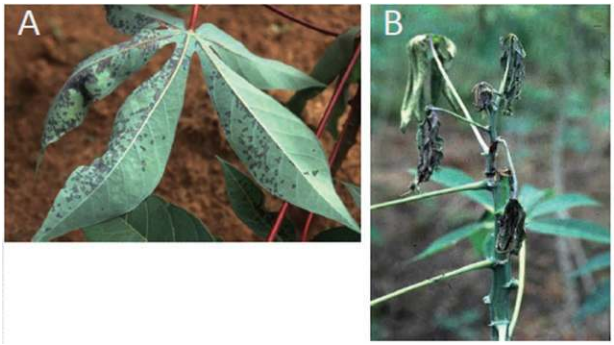

Fig. 7 Bacterial blight symptoms caused by *Xanthomonas axonopodis* pv. *manihotis*: (A) angular leaf spots (Courtesy of V. Verdier, IRD Montpellier, France); (B) leaf wilting (courtesy of B. Boher, IRD Montpellier, France).

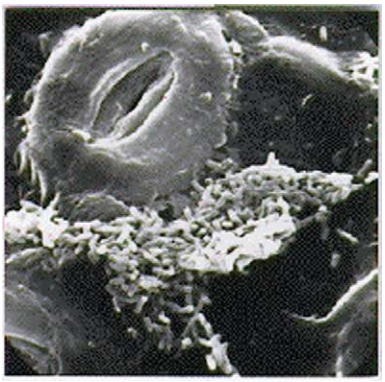

Fig. 8 Scanning electron microscopy showing a large amount of bacteria near the stomata (Courtesy of V. Verdier, IRD Montpellier, France).

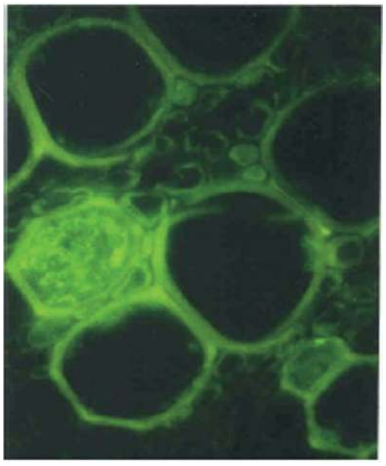

Fig. 9 *Xanthomonas axonopodis* pv. *manihotis* in xylem vessels (courtesy of B. Boher, IRD Montpellier, France).

7. *ERWINIA AMYLOVORA*

Erwinia amylovora causes fire blight disease of apple, pear, quince, blackberry, raspberry and many wild and cultivated rosaceous ornamentals (Vanneste, 2000). The disease develops sporadically, but, occasionally, it is highly destructive, especially to young fruit trees that may be killed outright by infections that girdle the trunk or the rootstock. The pathogen is distributed widely in temperate regions in which rosaceous plants thrive. It was described initially as *Micrococcus amylovorus*, and then *Bacillus amylovorus* (Burrill), under the erroneous assumption that it destroys starch. It is Gram negative, rod shaped and motile with peritrichous flagella. It was renamed *Erwinia amylovora* (Burrill) Winslow *et al*. in the early 1900s and remains the type species of the genus (Lelliott and Dickey, 1984). Closely related bacteria that elicit symptoms reminiscent of fire blight, particularly, but not exclusively, in pear, have been described as new species, e.g. *E. pyrifoliae* (Kim *et al*., 1999) and *E. piriflorinigrans* (Lopez *et al*., 2011).

Erwinia amylovora is of great historical importance to phytobacteriologists in that it was the first bacterium clearly demonstrated to cause disease in plants shortly after the pioneering work of Pasteur and Koch on bacterial pathogens of humans and animals in the late 1800s (see Griffith *et al*., 2003 for the pioneering papers of Burrill, Arthur and Waite). Thus, *E. amylovora* is justifiably referred to as the 'premier phytopathogenic bacterium'.

Symptoms of fire blight were first reported from orchards close to New York City. From there, the pathogen spread westward and across continents, particularly during the 20th century. Although *E. amylovora* is now widespread, stringent quarantine regulations against the movement of rosaceous plant materials continue, in effect, to prevent the introduction of *E. amylovora* into areas free, or potentially free, of the pathogen.

The management of fire blight is based on sanitation, cultural practices and the use of a limited number of bactericides and biological control products (Johnson and Stockwell, 1998), mainly to combat blossom blight. An analysis of materials tested for control in recent years in the eastern USA concluded that, in spite of more than two centuries of knowledge and 'tremendous research efforts, effective control remains an elusive goal' (Ngugi *et al*., 2011). Furthermore, streptomycin, which was introduced more than 50 years ago, remains the most effective control material in areas in which sensitive strains of *E. amylovora* are present. However, in many areas, resistant strains are prevalent or regulations against the use of antibiotics in plant agriculture preclude the use of streptomycin. The development of genetic resistance, particularly in apple rootstocks and scions, holds promise for the future (Norelli *et al*., 2003).

Interestingly, the genome of *E. amylovora* is amongst the smallest of the plant pathogenic bacteria sequenced so far, at only 3.89 Mb (Sebaihia *et al*., 2010). Its small size is consistent with its lack of plant cell-degrading tools, which are common to most other phytopathogenic bacteria, e.g. cell wall-degrading enzymes and low-molecular-weight toxins. Its most important pathological tools appear to be components of the *hrp* pathogenicity island and the exopolysaccharides amylovoran and levan (Oh and Beer, 2005). The type III secreted proteins DspA/E and HrpN are essential to pathogenicity (Bocsanczy *et al*., 2008), whereas approximately 20 additional proteins that secrete or regulate the expression of Hrp proteins also play a role. Amylovoran and levan are involved in biofilm formation and pathogenicity (Koczan *et al*., 2009). Genomes of several strains and species closely related to *E. amylovora* have become available recently. Bioinformatic comparisons undoubtedly will reveal additional genetic bases for the virulence capability of the fire blight pathogen.

The developing fruits in Fig. 10 exhibit grey–green watersoaking typical of fire blight infection, which precedes necrosis, which is apparent on the dead blossoms at the bottom left and top right of the figure. Several drops of ooze exuding from infected blossoms and fruits, which contain billions of cells in a matrix of polysaccharides and plant sap, should be noted. Blossom cluster infection often leads to devastating losses to pome-fruit growers.

In Fig. 11, the two outer circles depict the genes of *E. amylovora* on the forward (outermost) and complementary strands of chromosomal DNA, respectively. The genes in blue have predicted orthologues in *E. coli* K12, whereas the genes in red do not. Loci coloured orange, yellow and purple are RNA genes. The inner circles depict the predicted orthologous genes of related organisms. Purple and red indicate genes of enterobacterial plant pathogens, orange *Yersinia*, black *E. coli*, yellow *Shigella*, green *Salmonella*, dark blue enterobacterial endosymbionts (e.g. *Sodalis glossinidius*) and light blue *Pseudomonas syringae*. The absence of a particular colour indicates the absence of an orthologue. The innermost circle represents genome coordinates. The two plasmids inside the chromosomal diagram follow the same colour scheme as the two outer circles of the chromosome genome.

Fig. 10 Apple blossom cluster infected by *Erwinia amylovora*.

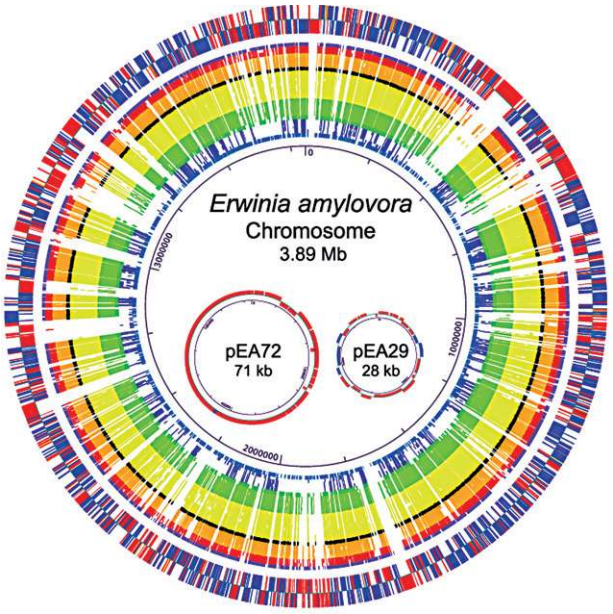

Fig. 11 Circular representation of the genome of *Erwinia amylovora* strain ATCC 49946 (Ea273) and comparison with related genomes. The figure and legend were provided courtesy of Bryan S. Biehl and Nicole T. Perna (University of Wisconsin, MI, USA), and Ana Maria Bocsanczy and Steven V. Beer (Cornell University, Ithaca, NY, USA).

8. *XYLELLA FASTIDIOSA*

Xylella fastidiosa (*Xanthomonadales*, *Xanthomonadaceae*) is a Gram-negative, nonflagellate, xylem-limited and nutritional pathogenic bacterium associated with several important plant diseases, including Pierce's disease of grapevine (PD), citrus variegated chlorosis (CVC) and almond leaf scorch disease (ALSD). Elm, oak, oleander, maple, sycamore, coffee, peach, mulberry, plum, periwinkle, pear and pecan are also other host species of the bacterium. There is only a single species in the genus, but different strains have been well characterized as pathotypes, with cross-infections among different hosts and strains having been reported, but without the development of disease symptoms.

Xylella fastidiosa was the first phytopathogen to have its genome completely sequenced (Simpson *et al*., 2000). The genome size changes from 2475 to 2731 kb between strains, and consists of a circular chromosome and plasmids. In addition to the pathotype 9a5C (CVC), Temecula-1 (PD) and others (including Dixon, Ann1, M12, M23 and GB514) have now been sequenced completely. Genome-wide analyses among strains have revealed genes unique to each strain (60 of 9a5c, 54 of Dixon, 83 of Ann1 and nine of Temecula-1). Indels and strain-specific genes are the main source of variation among strains. The Pierce's disease strain Temecula-1 genome represents the ancestral genome of *X. fastidiosa* (Doddapaneni *et al*., 2006). Over the past 10 years, the increasing number of publications related to genomic information has considerably expanded our knowledge on the bacterium and its pathosystems (Chatterjee *et al*., 2008).

Xylella fastidiosa does not carry a type III secretion system, and it is therefore assumed that this pathogen does not translocate effectors into plant cells for the elicitation of a host response. This hypothesis is supported by the fact that, in the xylem vessels, there is only fibre and dead cells, and the pathogen is introduced into this tissue by its vector, the sharpshooter leafhopper (Homoptera, Cicadellidae). However, *X. fastidiosa* has active type I and type II secretion systems, which could be associated with the efflux pump and the secretion of hydrolytic enzymes, respectively, allowing lateral movement of the bacterium through pit membranes and the digestion of plant cell walls.

The development of symptoms in diseases caused by *X. fastidiosa* is strictly associated with the ability of the bacterium to spread, colonize and block xylem vessels. The colonies grow in biofilms, which can occlude xylem vessels, and reduce water and nutrient transport. The different virulences exhibited by strains of *X. fastidiosa* are often associated with differences in their abilities to spread, colonize and block xylem vessels. Type I and type IV pili are involved in twitching motility and migration, and attachment and biofilm formation, respectively. Biofilms are important for this pathogen to survive in environments with high turbulence, differential pressure and poor nutrient availability, such as xylem vessels and insect foreguts.

Fig. 12 Symptoms of citrus variegated chlorosis in leaves and plant of sweet orange (photograph Marcos A. Machado).

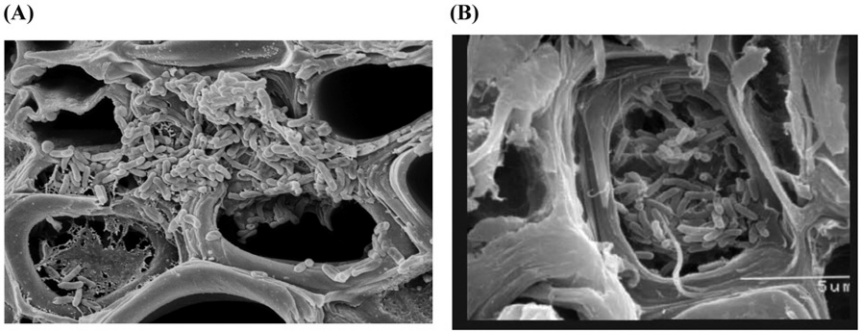

Fig. 13 (A, B) Biofilm of *Xylella fastidiosa* blocking the xylem vessels of sweet orange tree. Photographs in (A) by E.W. Kitajima (Escola Superior de Agricultura Luis de Queiróz, USP, Piracicaba, SP, Brazil) and in (B) by J.O. Lima (Citrulima Viveiros, São João da Boa Vista, SP, Brazil) and Marcos A. Machado.

9. *DICKEYA* (*DADANTII* AND *SOLANI*)

In 1995, *Erwinia chrysanthemi* was transferred to the new genus *Dickeya* and divided into six species: *D. dianthicola*, *D. dadantii*, *D. zeae*, *D. chrysanthemi*, *D. paradisiaca* and *D. dieffenbachiae* (Samson *et al*., 2005). Since then, it has become clear that some strains do not fall into any of these species and may constitute new species, e.g. '*D. solani*' (Parkinson *et al*., 2009; Sławiak *et al*., 2009). All *Dickeya* spp. cause economically important diseases on different plant hosts worldwide, including 10 monocot and 16 dicot families (Ma *et al*., 2007; Samson *et al*., 2005). However, *D. dadantii* and '*D. solani*' have been selected here for two very different reasons.

Dickeya dadantii causes disease mainly in tropical and subtropical environments and has a wide host range, including *Saintpaulia* and potato (Samson *et al*., 2005) (Fig. 14). The reason for its inclusion is that *D. dadantii* strain 3937 (*Dda*3937) has been the *Dickeya* strain of choice for molecular studies for over 25 years (Diolez and Coleno, 1985). These studies have been instrumental in our understanding of bacterial plant pathogenesis, including the roles of exoenzymes and sugar catabolism, iron transport, secretion and regulation, complementing related studies in other 'soft rot erwiniae' (including *Pectobacterium carotovorum* and *P. atrosepticum*—see next section) (Hommais *et al*., 2008; Kazemi-Pour *et al*., 2004; Lemanceau *et al*., 2009; Rodionov *et al*., 2004; Toth *et al*., 2003; Venkatesh *et al*., 2006; Yang *et al*., 2002). Other recent areas of study include plant defence and pathogen response to defence (Antunez-Lamas *et al*., 2009; Fagard *et al*., 2007; Li *et al*., 2009; Segond *et al*., 2009; Yang *et al*., 2010), pathogenesis in the pea aphid (Costechareyre *et al*., 2010) and the interaction between phytopathogens and human pathogens on plants (Yamazaki *et al*., 2011). The availability of a genome sequence for *Dda*3937, annotated through an international consortium, combined with functional genomics and systems biology approaches, is furthering our knowledge of this and related pathogens (Babujee *et al*., 2007; Glasner *et al*., 2011; Kepseu *et al*., 2010; Yang *et al*., 2010) (Fig. 14).

The name '*D. solani*' has not yet been officially accepted. However, the sudden rise to prominence of this 'species' in European potato production has made it worthy of inclusion (Fig. 15). The 'species' was first recognized on potato around 2005, possibly transferring host from an ornamental plant, and has since spread to many potato-growing regions in Europe and beyond (Sławiak *et al*., 2009; Toth *et al*., 2011; Tsror (Lahkim) *et al*., 2009). Moreover, in some regions, it appears to have displaced existing 'soft rot' pathogens, possibly as a result of its increased aggressiveness and/or mode of infection (Czajkowski *et al*., 2010; Toth *et al*., 2011) (Fig. 16). In 2010, Scotland became the first country to introduce legislation in an attempt to keep its seed industry free from this pathogen; a strategy that has so far succeeded. '*D. solani*' causes disease at a range of temperatures, conducive to the current European climate, but also shows increased aggressiveness in warmer conditions, raising concerns that climate change could lead to increased disease problems in the future (Sławiak *et al*., 2009; Tsror (Lahkim) *et al*., 2009). Little is known about the biology of '*D. solani*', but scientists (including those studying *Dda*3937) are working together to better understand the biology of this pathogen and its control.

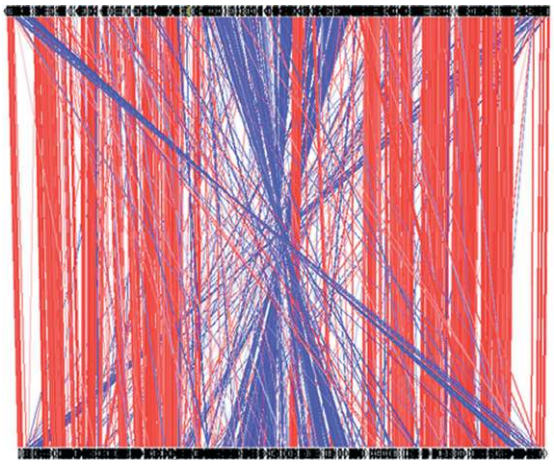

Fig. 14 Artemis screenshot showing reciprocal best hit analysis of coding sequences (CDS) between *Pectobacterium atrosepticum* (top) and *Dickeya dadantii* 3937 (bottom). Coloured lines represent orthologues; red, same orientation; blue, opposite orientation.

Fig. 15 Potato tuber rot caused by '*Dickeya solani*'. Fera crown copyright.

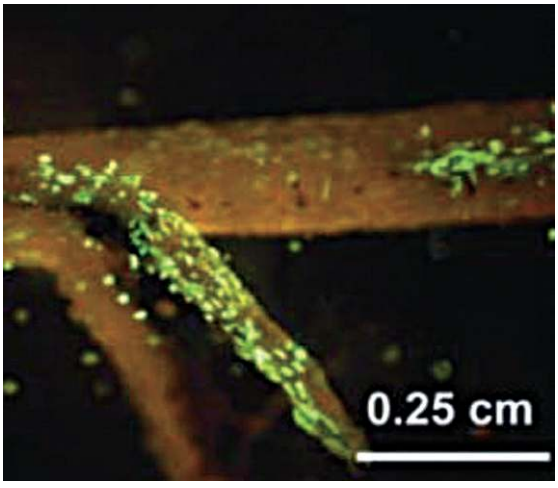

Fig. 16 '*Dickeya solani*' expressing green fluorescent protein (GFP) on potato roots (courtesy of J. van der Wolf, Plant Research International, Wageningen, the Netherlands).

10. *PECTOBACTERIUM CAROTOVORUM* (AND *P. ATROSEPTICUM*)

Pectobacterium carotovorum (*Pcc*) and *Pectobacterium atrosepticum* (*Pca*) were originally classified as *Erwinia carotovora* subspecies *carotovora* and subspecies *atroseptica*, respectively. These species (or subspecies) were members of the soft rot group of erwinias and are taxonomically closely related to *Erwinia chrysanthemi* (recently reclassified as multiple *Dickeya* species; see previous section).

Pectobacterium carotovorum is geographically widely distributed, whereas *Pca* is largely confined to cooler climates (Pérombelon, 2002; Pérombelon and Kelman, 1980; Pérombelon and Salmond, 1995; Salmond, 1992; Toth *et al*., 2003). *Pcc* is the aetiological agent of soft rot diseases of several crop plants, and *Pca* is of particular importance in the commercially important blackleg disease of potato in temperate regions (Fig. 17) (Pérombelon, 2002; Pérombelon and Kelman, 1980). These soft rot pectobacteria were important 'model' pathogens in the early days of the genetic analysis of phytopathogenesis. Their taxonomic relatedness to *E. coli* (family Enterobacteriaceae) allowed the facile transfer, or development, of many genetic tools from *E. coli* to enable the molecular analysis of virulence (Fig. 18) (see, for example, Diolez and Coleno, 1985; Hinton *et al*., 1989; Kotoujansky, 1987; Mulholland and Salmond, 1995; Toth *et al*., 1993, 1997). This genetic tractability underpinned the first studies on the structure and virulence roles of plant cell wall-degrading enzymes (PCWDEs); particularly assorted pectinases, cellulases and proteases (Hinton *et al*., 1990; Kotoujansky, 1987; Liu *et al*., 1994). The central catabolic pathway for plant pectin degradation and assimilation by the pathogen was extensively investigated. Moreover, the analysis of the roles of PCWDEs in virulence led to the discovery of the enzyme secretion systems (type I and type II secretory pathways) and the fundamental appreciation that protein secretion systems operate by common mechanisms in molecular pathogenesis across plant and animal pathogens (Evans *et al*., 2009; Salmond, 1994; Wharam *et al*., 1995). This acknowledgement of common themes in plant and animal pathogens is now widespread.

In addition to the role of PCWDE synthesis and secretion in virulence, the analysis of PCWDE regulation mechanisms in *Pcc* uncovered the phenomenon of 'quorum sensing' through which the pathogen controls the elaboration of the virulence determinants in concert with bacterial cell population density (Barnard *et al*., 2007; Coulthurst *et al*., 2007; Jones *et al*., 1993; Liu *et al*., 2008; Pirhonen *et al*., 1993; Whitehead *et al*., 2001). The crucial importance of quorum sensing pectobacterial pathogenesis was confirmed by studies on genetically engineered plants (Dong *et al*., 2001; Toth *et al*., 2004). Density-dependent control of virulence factors, modulated by freely diffusible *N*-acyl homoserine lactone intercellular signalling molecules, is now a well-established trait of various plant and animal pathogens (Waters and Bassler, 2005). Furthermore, *Pcc* was one of the first bacteria shown to produce 1-carbapen-2-em-3-carboxylic acid, a member of the carbapenem class of β-lactam antibiotics, and the production of this antibiotic is co-regulated with the PCWDE virulence factors via quorum sensing (Barnard *et al*., 2007; Coulthurst *et al*., 2005). It has been shown by *in planta* transcriptomic studies that quorum sensing plays an essential role during plant infection in the control of several hundred genes encoding diverse products impacting on the physiology of plant pathogenesis (Liu *et al*., 2008). These genes encode traits such as multiple protein secretion pathways (including type II, III, IV and VI machines), secondary metabolite production and an interesting selection of proteins of unknown function. Studies on PCWDE regulation have also demonstrated a key role for post-transcriptional control of gene expression via the RsmAB system (Liu *et al*., 1998; Mukherjee *et al*., 2000), another regulatory system that has been shown to occur in other plant and animal pathogens.

Pectobacterium atrosepticum was the first enterobacterial phytopathogen to be genomically sequenced and, at the time, this uncovered various unexpected predicted traits in the pathogen, including the possession of type IV and type VI secretion machines, the production of new secondary metabolite toxins and nitrogen fixation capability (Bell *et al*., 2004; Liu *et al*., 2008; Mattinen *et al*., 2008). Furthermore, the genome sequence highlighted fascinating evolutionary relationships between this enterobacterial plant pathogen and taxonomically related animal pathogens. In particular, *Pca* has been shown to carry a series of genomic islands, some of which are obvious loci for virulence, and ecological adaptation genes acquired by horizontal transfer. Genomic information is now available for *Pcc* strains and other 'former *Erwinia*' species now reclassified in the genus *Dickeya* (see previous section; Glasner *et al*., 2008; Ma *et al*., 2007).

Ecological studies of *Pcc* (and *Pca*) have been classically phenomenological (Pérombelon, 2002; Pérombelon and Kelman, 1980). However, recent studies have shown important roles for specific proteins in the possible ecological dissemination of *Pcc* by insect vectors, such as *Drosophila*. Interestingly, the fly also benefits from this interaction with the phytopathogen through a stimulation of the insect innate immune system (Basset *et al*., 2003; Muniz *et al*., 2007).

Finally, in addition to their agricultural impacts, we should not ignore the long-standing translational significance of *Pectobacterium* spp. For example, a periplasmic L-asparaginase from soft rotting *Pcc* is used clinically in the treatment of acute lymphocytic leukaemias and, historically, some related recombinant *Erwinia* spp have been considered as possible tools for the biotechnological manufacture of vitamin C (Robert-Baudouy, 1991).

Fig. 17 Blackleg disease of potato caused by *Pectobacterium atrosepticum*. Apparently healthy mother tubers can be seen, but stem rotting is also clear.

Fig. 18 Identification of *Pectobacterium* mutants affected in potato plant virulence (stem inoculation assays). Left, wild-type; others, reduced virulence.

ACKNOWLEDGEMENTS

The authors would like to thank Dr Diane Hird for the distribution of voting information and the collation of voting results.

REFERENCES

Antunez-Lamas, M., Cabrera, E., Lopez-Solanilla, E., Solano, R., Gonzalez-Melendi, P., Chico, J.M., Toth, I.K., Birch, P.R.J., Pritchard, L., Liu, H. and Rodriguez-Palenzuela, P. (2009) Bacterial chemoattraction towards jasmonate plays a role in the entry of *Dickeya dadantii* through wounded tissues. *Mol. Microbiol.* **74**, 662–671.

Arnold, D., Lovell, H., Jackson, R.W. and Mansfield, J.W. (2011) *Pseudomonas syringae* pv. *phaseolicola*: from 'has bean' to supermodel. *Mol. Plant Pathol.* **12**, 617–627.

Babujee, L., Venkatesh, B., Yamazaki, A. and Tsuyumu, S. (2007) Proteomic analysis of the carbonate insoluble outer membrane fraction of the soft-rot pathogen *Dickeya dadantii* (syn. *Erwinia chrysanthemi*) strain 3937. *J. Proteome Res.* **6**, 62–69.

Baltrus, D.A., Nishimura, M.T., Romanchuk, A., Chang, J.H., Mukhtar, M.S., Cherkis, K., Roach, J., Grant, S.R., Jones, C.D. and Dangl, J.L. (2011) Dynamic evolution of pathogenicity revealed by sequencing and comparative genomics of 19 *Pseudomonas syringae* isolates. *PLoS Pathog.* **7**, e1002132. Epub: 1 July 2011.

Barnard, A., Bowden, S., Burr, T., Corbett, M., Coulthurst, S., Monson, R. and Salmond, G.P.C. (2007) Quorum sensing, virulence and secondary metabolite production in plant soft rotting bacteria. *Proc. R. Soc. London, Ser. B: Biol. Sci.* **362**, 1165–1183.

Barton, K.A., Binns, A.N., Matzke, A.J. and Chilton, M.D. (1983) Regeneration of intact tobacco plants containing full length copies of genetically engineered T-DNA, and transmission of T-DNA to R1 progeny. *Cell*, **32**, 1033–1043.

Basset, A., Tzou, P., Lemaitre, B. and Boccard, F. (2003) A single gene that promotes interactions between a phytopathogenic bacterium with its insect vector, *Drosophila melanogaster*. *EMBO Rep.* **4**, 205–210.

Bell, K., Sebaihia, M., Pritchard, L., Holden, M., Holeva, M.C., Thomson, N.R., Bentley, S.D., Churcher, L.J.C., Mungall, K., Atkin, R., Bason, N., Brooks, K., Chillingworth, T., Clark, K., Frazer, A., Hance, Z., Hauser, H., Jagels, K., Moule, S., Norbertczak, H., Ormons, D., Price, C., Quali, M.A., Sanders, M., Walker, D., Whitehead, S., Salmond, G.P.C., Birch, P.R.J., Parkhill, J. and Toth, I.K. (2004) Genome sequence of the enterobacterial phytopathogen, *Erwinia carotovora* subsp. *atroseptica* and characterisation of novel virulence factors. *Proc. Natl. Acad. Sci. USA*, **101**, 11 105–11 110.

Boch, J. and Bonas, U. (2010) *Xanthomonas* AvrBs3 family-type III effectors: discovery and function. *Annu. Rev. Phytopathol.* **48**, 419–436.

Bocsanczy, A.M., Nissinen, R.M., Oh, C.-S. and Beer, S.V. (2008) HrpN of *Erwinia amylovor*a functions in the translocation of DspA/E into plant cells. *Mol. Plant Pathol.* **9**, 425–434.

Boher, B. and Verdier, V. (1995) Cassava bacterial blight in Africa: the state of knowledge and implications for designing control strategies. *Afr. Crop Sci. J.* **2**, 1–5.

Boher, B., Nicole, M., Potin, M. and Geiger, J.P. (1997) Extracellular polysaccharides from *Xanthomonas axonopodis* pv. *manihotis* interact with cassava cell walls during pathogenesis. *Mol. Plant–Microbe Interact.* **10**, 803–811.

Boucher, C.A., Barberis, P., Trigalet, A.P. and Demery, D.A. (1985) Transposon mutagenesis of *Pseudomonas solanacearum*: isolation of Tn*5*-induced avirulent mutants. *J. Gen. Microbiol.* **131**, 2449–2457.

Capage, M.A., Doherty, D.H., Betlach, M.R. and Vanderslice, R.W. (1987) Recombinant-DNA mediated production of xanthan gum. International Patent WO87/05938.

Chatterjee, S., Almeida, R.P.P. and Lindow, S. (2008) Living in two worlds: the plant and insect lifestyles of *Xylella fastidiosa*. *Annu. Rev. Phytopathol.* **46**, 243–271.

Chilton, M.D., Drummond, M.H., Merio, D.J., Sciaky, D., Montoya, A.L., Gordon, M.P. and Nester, E.W. (1977) Stable incorporation of plasmid DNA into higher plant cells: the molecular basis of crown gall tumorigenesis. *Cell*, **11**, 263–271.

Costechareyre, D., Dridi, B., Rahbe, Y. and Condemine, G. (2010) Cyt toxin expression reveals an inverse regulation of insect and plant virulence factors of *Dickeya dadantii*. *Environ. Microbiol.* **12**, 3290–3301.

Coulthurst, S., Barnard, A. and Salmond, G.P.C. (2005) Regulation and biosynthesis of carbapenem antibiotics in bacteria. *Nat. Rev. Microbiol.* **3**, 295–306.

Coulthurst, S., Monson, R. and Salmond, G.P.C. (2007) Quorum sensing in the soft rot erwinias. In: *Bacterial Cell–Cell Communication* (Winans, S. and Bassler, B., eds), pp. 185–199. Washington, DC: ASM Press.

Cunnac, S., Lindeberg, M. and Collmer, A. (2009) *Pseudomonas syringae* type III effectors: repertoires in search of functions. *Curr. Opin. Microbiol.* **12**, 53–60.

Czajkowski, R., de Boer, W.J., Velvis, H. and van der Wolf, J.M. (2010) Systemic colonization of potato plants by a soilborne, green fluorescent protein-tagged strain of *Dickeya* sp. Biovar 3. *Phytopathology*, **100**, 134–142.

Dean, R., van Kan, J.A.L., Pretorius, Z.A., Hammond-Kosack, K.E., Di Pietro, A., Spanu, P.D., Rudd, J.J., Dickman, M., Kahmann, R., Ellis, J. and Foster, G.D. (2012) The top 10 fungal pathogens in molecular plant pathology. *Mol. Plant Pathol.* **13**, 414–430.

Denny, T.P. (2006) Plant pathogenic *Ralstonia* species. In: *Plant-Associated Bacteria* (Gnanamanickam, S.S., ed.), pp. 573–644. Dordrecht: Springer Publishing.

Diolez, A. and Coleno, A. (1985) Mu-*lac* insertion-directed mutagenesis in a pectate lyase gene of *Erwinia chrysanthemi*. *J. Bacteriol.* **163**, 913–917.

Djamei, A., Pitzschke, A., Nakagami, H., Rajh, I. and Hirt, H. (2007) Trojan horse strategy in *Agrobacterium* transformation: abusing MAPK defense signaling. *Science*, **318**, 453–456.

Doddapaneni, H., Yao, J., Lin, H., Walker, M.A. and Civerolo, E.L. (2006) Analysis of the genome-wide variations among multiple strains of the plant pathogenic bacterium *Xylella fastidiosa*. *BMC Genomics*, **7**, 225.

Dong, Y.H., Wang, L.H., Xu, J.L., Zhang, H.B., Zhang, X.F. and Zhang, L.H. (2001) Quenching quorum sensing-dependent bacterial infection by an *N*-acyl homoserine lactonase. *Nature*, **411**, 813–817.

Duan, Y., Zhou, L., Hall, D.G., Li, W., Doddapaneni, H., Lin, H., Liu, L., Vahling, C.M., Gabriel, D.W., Williams, K.P., Dickerman, A., Sun, Y. and Gottwald, T. (2009) Complete genome sequence of citrus huanglongbing bacterium, '*Candidatus* Liberibacter asiaticus' obtained through metagenomics. *Mol. Plant–Microbe Interact.* **22**, 1011–1020.

Eichenlaub, R. and Gartemann, K.-H. (2011) The *Clavibacter michiganensis* subspecies: molecular investigation of Gram-positive bacterial plant pathogens. *Annu. Rev. Phytopathol.* **49**, 445–464.

Elphinstone, J.G. (2005) The current bacterial wilt situation: a global overview. In: *Bacterial Wilt Disease and the* Ralstonia solanacearum *Species Complex* (Allen, C., Prior, P. and Hayward, A.C., eds), pp. 9–28. St Paul, MN: APS Press.

Escobar, M.A., Civerolo, E.L., Summerfelt, K.R. and Dandekar, A.M. (2001) RNAi-mediated oncogene silencing confers resistance to crown gall tumorigenesis. *Proc. Natl. Acad. Sci. USA*, **98**, 13 437–13 442.

Evans, T.J., Perez-Mendoza, D., Monson, R., Stickland, H.G. and Salmond, G.P.C. (2009) Secretion systems of the enterobacterial phytopathogen, Erwinia. In: *Bacterial Secreted Proteins*. (Wooldridge, K. ed), pp. 479–503. Norfolk: Caister Academic Press.

Fagard, M., Dellagi, A., Roux, C., Perino, C., Rigault, M., Boucher, V., Shevchik, V.E. and Expert, D. (2007) *Arabidopsis thaliana* expresses multiple lines of defense to counterattack *Erwinia chrysanthemi*. *Mol. Plant–Microbe Interact.* **20**, 794–805.

Flavier, A.B., Clough, S.J., Schell, M.A. and Denny, T.P. (1997) Identification of 3-hydroxypalmitic acid methyl ester as a novel autoregulator controlling virulence in *Ralstonia solanacearum*. *Mol. Microbiol.* **26**, 251–259.

Gabriel, D.W., Burges, A. and Lazo, G.R. (1986) Gene-for-gene interactions of five cloned avirulence genes from *Xanthomonas campestris* pv. *malvacearum* with specific resistance genes in cotton. *Proc. Natl. Acad. Sci. USA*, **83**, 6415–6419.

Gelvin, S.B. (2010) Plant proteins involved in *Agrobacterium*-mediated genetic transformation. *Annu. Rev. Phytopathol.* **48**, 45–68.

Genin, S. (2010) Molecular traits controlling host range and adaptation to plants in *Ralstonia solanacearum*. *New Phytol.* **187**, 920–928.

Glasner, J.D., Marquez-Villavicencio, M., Kim, H.S., Jahn, C.E., Ma, B., Biehl, B.S., Rissman, A.I., Mole, B., Yi, X., Yang, C.H., Dangl, J.L., Grant, S.R., Perna, N.T. and Charkowski, A.O. (2008) Niche-specificity and the variable of the *Pectobacterium* pan-genome. *Mol. Plant–Microbe Interact.* **21**, 1549–1560.

Glasner, J.D., Yang, C.-H., Reverchon, S., Hugouvieux-Cotte-Pattat, N., Condemine, G., Bohin, J.-P., Van Gijsegem, F., Yang, S., Franza, T., Expert, D., Plunkett, G., Francisco, M.J.S., Charkowski, A.O., Py, B., Bell, K., Rauscher, L., Rodriguez-Palenzuela, P., Toussaint, A., Holeva, M.C., He, S.Y., Douet, V., Boccara, M., Blanco, C., Toth, I., Anderson, B.D., Biehl, B.S., Mau, B., Flynn, S.M., Barras, F., Lindeberg, M., Birch, P.R.J., Tsuyumu, S., Shi, X., Hibbing, M., Yap, M.-N., Carpentier, M., Dassa, E., Umehara, M., Kim, J.F., Rusch, M., Soni, P., Mayhew, G.F., Fouts, D.E., Gill, S.R., Blattner, F.R., Keen, N.T. and Perna, N.T. (2011) Genome sequence of the plant pathogenic bacterium *Dickeya dadantii* 3937. *J. Bacteriol.* **193**, 2076–2077.

Green, S., Studholme, D.J., Laue, B.J., Dorati, F., Lovell, H., Arnold, D., Cottrell, J.E., Bridgett, S., Blaxter, M., Huitema, E., Thwaites, R., Sharp, P.M., Jackson, R.W. and Kamoun, S. (2010) Comparative genome analysis provides insights into the evolution and adaptation of *Pseudomonas syringae* pv. *aesculi* on *Aesculus hippocastanum*. *PLoS ONE*, **5**, e10224.

Griffith, C.S., Sutton, T.B. and Peterson, P.D. (2003) *Fire Blight: The Foundation of Phytobacteriology*. St. Paul, MN: APS Press.

Han, S.W., Sriariyanun, M., Lee, S.W., Sharma, M., Bahar, O., Bower, Z. and Ronald, P.C. (2011) Small protein-mediated quorum sensing in a Gram-negative bacterium. *PLoS ONE*, **6**, e29192.

Hann, D.R., Gimenez-Ibanez, S. and Rathjen, J.P. (2010) Bacterial virulence effectors and their activities. *Curr. Opin. Plant Biol.* **13**, 388–393.

He, Y.W., Wy, J., Cha, J.S. and Zhang, L.H. (2010) Rice bacterial blight pathogen *Xanthomonas oryzae* pv. *oryzae* produces multiple DSF-family signals in regulation of virulence factor production. *BMC Microbiol.* **10**, 187.

Hinton, J.C.D., Sidebotham, J.M., Hyman, L.J., Pérombelon, M.C.M. and Salmond, G.P.C. (1989) Isolation and characterisation of transposon-induced mutants of *Erwinia carotovora* subsp. *atroseptica* exhibiting reduced virulence. *Mol. Gen. Genet.* **217**, 141–148.

Hinton, J.C.D., Gill, D.R., Lalo, D., Plastow, G.S. and Salmond, G.P.C. (1990) Sequence of the *peh* gene of *Erwinia carotovora*: homology between *Erwinia* and plant enzymes. *Mol. Microbiol.* **4**, 1029–1036.

Hommais, F., Oger-Desfeux, C., Van Gijsegem, F., Castang, S., Ligori, S., Expert, D., Nasser, W. and Reverchon, S. (2008) PecS is a global regulator of the symptomatic phase in the phytopathogenic bacterium *Erwinia chrysanthemi* 3937. *J. Bacteriol.* **190**, 7508–7522.

Huynh, T.V., Dahlbeck, D. and Staskawicz, B.J. (1989) Bacterial blight of soybean: regulation of a pathogen gene determining host cultivar specificity. *Science*, **245**, 1374–1377.

Jeong, K.S., Lee, S.E., Han, J.W., Yang, S.U., Lee, B.M., Noh, T.H. and Cha, J.S. (2008) Virulence reduction and differing regulation of virulence genes in *rpf* mutants of *Xanthomonas oryzae* pv. *oryzae*. *Plant Pathol. J.* **24**, 143–151.

Joardar, V., Lindeberg, M., Jackson, R.W., Selengut, J., Dodson, R., Brinkac, L.M., Daugherty, S.C., Deboy, R., Durkin, A.S., Giglio, M.G., Madupu, R., Nelson, W.C., Rosovitz, M.J., Sullivan, S., Crabtree, J., Creasy, T., Davidsen, T., Haft, D.H., Zafar, N., Zhou, L., Halpin, R., Holley, T., Khouri, H., Feldblyum, T., White, O., Fraser, C.M., Chatterjee, A.K., Cartinhour, S., Schneider, D.J., Mansfield, J., Collmer, A. and Buell, C.R. (2005) Whole-genome sequence analysis of *Pseudomonas syringae* pv. *phaseolicola* 1448A reveals divergence among pathovars in genes involved in virulence and transposition. *J. Bacteriol.* **187**, 6488–6498.

Johnson, K.B. and Stockwell, V.O. (1998) Management of fire blight: a case study in microbial ecology. *Annu. Rev. Phytopathol.* **36**, 227–248.

Jones, S., Yu, B., Bainton, N.J., Birdsall, M., Bycroft, B.W., Chhabra, S.R., Cox, A.J.R., Reeves, P.J., Stephens, S., Winson, M.K., Salmond, G.P.C., Stewart, G.S.A.B. and Williams, P. (1993) The *Lux* autoinducer regulates the production of exoenzyme virulence determinants in *Erwinia carotovora* and *Pseudomonas aeruginosa*. *EMBO J.* **12**, 2477–2482.

Jovanovic, M., James, E.H., Burrows, P.C., Rego, F.G., Buck, M. and Schumacher, J. (2011) Regulation of the co-evolved HrpR and HrpS AAA+ proteins required for *Pseudomonas syringae* pathogenicity. *Nat. Commun.* **2**, 177.

Kazemi-Pour, N., Condemine, G. and Hugouvieux-Cotte-Pattatt, N. (2004) The secretome of the plant pathogenic bacterium *Erwinia chrysanthemi*. *Proteomics*, **4**, 3177–3186.

Kepseu, W.D., Sepulchre, J.-A., Reverchon, S. and Nasser, W. (2010) Toward a quantitative modelling of the synthesis of the pectate lyases, essential virulence factors in *Dickeya dadantii*. *J. Biol. Chem.* **285**, 28 565–28 576.

Kim, W.-S., Gardan, L., Geider, K. and Rhim, S.L. (1999) *Erwinia pyrifoliae* sp. nov., a novel pathogen affecting Asian pear trees (*Pyrus pyrifolia* Nakai). *Int. J. Syst. Bacteriol.* **49**, 899–906.

Koczan, J.M., McGrath, M.J., Zhao, Y. and Sundin, G.W. (2009) Contribution of *Erwinia amylovora* exopolysaccharides amylovoran and levan to biofilm formation: implications in pathogenicity. *Phytobacteriology*, **99**, 1237–1244.

Kotoujansky, A. (1987) Molecular genetics of pathogenesis by soft-rot erwinias. *Annu. Rev. Phytopathol.* **25**, 405–430.

Kpémoua, K., Boher, B., Nicole, M., Calatayud, P. and Geiger, J.P. (1996) Cytochemistry of defense responses in cassava infected by *Xanthomonas campestris* pv. *manihotis*. *Can. J. Microbiol.* **42**, 1131–1143.

Kvitko, B.H., Park, D.H., Velásquez, A.C., Wei, C.F., Russell, A.B., Martin, G.B., Schneider, D.J. and Collmer, A. (2009) Deletions in the repertoire of *Pseudomonas syringae* pv. *tomato* DC3000 type III secretion effector genes reveal functional overlap among effectors. *PLoS Pathog.* **5**, e1000388.

Lacroix, B., Tzfira, T., Vainstein, A. and Citovsky, V. (2006) A case of promiscuity: *Agrobacterium*'s endless hunt for new partners. *Trends Genet.* **22**, 29–37.

Lee, B.M., Park, Y.J., Park, D.S., Kang, H.W., Kim, J.G., Song, E.S., Park, I.C., Yoon, U.H., Hahn, J.H., Koo, B.S., Lee, G.B., Kim, H., Park, H.S., Yoon, K.O., Kim, J.H., Jung, C.H., Koh, N.H., Seo, J.S. and Go, S.J. (2005) The genome sequence of *Xanthomonas oryzae* pathovar *oryzae* KACC10331, the bacterial blight pathogen of rice. *Nucleic Acids Res.* **33**, 577–586.

Lee, S.W., Han, S.W., Bartley, L. and Ronald, P.C. (2006) Unique characteristic of *Xanthomonas oryzae* pv. *oryzae* AvrXa21 and implications for plant innate immunity. *Proc. Natl. Acad. Sci. USA*, **103**, 18 395–18 400.

Lee, S.W., Han, S.W., Sririyanum, M., Park, C.J., Seo, Y.S. and Ronald, P.C. (2009) A Type I-secreted, sulfated peptide triggers XA21-mediated innate immunity. *Science*, **306**, 850–853.

Lelliott, R.A. and Dickey, R.S. (1984) Genus VII. Erwinia. In: *Bergey's Manual of Systematic Bacteriology*, Vol. **1**. (Krieg, N.R.A. and Holt, J.G., eds), pp. 469–476. Baltimore, MD: Williams & Wilkins Co.

Lemanceau, P., Expert, D., Gaymard, F., Bakker, P.A.H.M. and Briat, J.-F. (2009) Role of iron in plant–microbe interactions. *Adv. Bot. Res.* **51**, 491–549.

Li, C.M., Brown, I., Mansfield, J., Stevens, C., Boureau, T., Romantschuk, M. and Taira, S. (2002) The Hrp pilus of *Pseudomonas syringae* elongates from its tip and acts as a conduit for translocation of the effector protein HrpZ. *EMBO J.* **21**, 1909–1915.

Li, Y., Peng, Q., Selimi, D., Wang, Q., Charkowski, A.O., Chen, X. and Yang, C.H. (2009) The plant phenolic compound p-coumaric acid represses gene expression in the *Dickeya dadantii* Type III secretion system. *Appl. Environ. Microbiol.* **75**, 1223–1228.

Lindeberg, M., Myers, C.R., Collmer, A. and Schneider, D.J. (2008) Roadmap to new virulence determinants in *Pseudomonas syringae*: insights from comparative genomics and genome organization. *Mol. Plant–Microbe Interact.* **21**, 685–700.

Liu, H., Coulthurst, S.J., Pritchard, L., Hedley, P.E., Ravensdale, M., Humphris, S., Burr, T., Takle, G., Brurberg, M.B., Birch, P.R.J., Salmond, G.P.C. and Toth, I.K. (2008) Quorum sensing coordinates brute force and stealth modes of infection in the plant pathogen *Pectobacterium atrosepticum*. *PLoS Pathog.* **4**, e1000093.

Liu, Y., Chatterjee, A. and Chatterjee, A.K. (1994) Nucleotide sequence, organization and expression of *rdgA* and *rdgB* genes that regulate pectin lyase production in the plant pathogenic bacterium *Erwinia carotovora* subsp. *carotovora* in response to DNA-damaging agents. *Mol. Microbiol.* **14**, 999–1010.

Liu, Y., Cui, Y., Mukherjee, A. and Chatterjee, A.K. (1998) Characterization of a novel RNA regulator of *Erwinia carotovora* ssp. *carotovora* that controls production of extracellular enzymes and secondary metabolites. *Mol. Microbiol.* **29**, 219–234.

Lopez, C., Jorge, V., Piégu, B., Mba, C., Cortes, D., Restrepo, S., Soto, M., Laudié, M., Berger, C., Cooke, R., Delseny, M., Tohme, J. and Verdier, V. (2004) A unigene catalogue of 5700 expressed genes in cassava (*Manihot esculenta*). *Plant Mol. Biol.* **56**, 541–554.

Lopez, C., Soto, M., Restrepo, S., Piégu, B., Cooke, R., Delseny, M., Tohme, J. and Verdier, V. (2005) Cassava gene expression profile in response to *Xanthomonas axonopodis* pv. *manihotis* infection using a cDNA microarray. *Plant Mol. Biol.* **57**, 393–410.

Lopez, M.M., Rosello, M., Llop, P., Ferrer, S., Christen, R. and Gardan, L. (2011) *Erwinia piriflorinigrans* sp. nov., a novel pathogen that causes necrosis of pear blossoms. *Int. J. Syst. Evol. Microbiol.* **61**, 561–567.

Lozano, J.C. (1986) Cassava bacterial blight: a manageable disease. *Plant Dis.* **70**, 1089–1093.

Ma, B., Hibbing, M.E., Kim, H.S., Reedy, R.M., Yedidia, I., Breuer, J., Breuer, J., Glasner, J.D., Perna, N.T., Kelman, A. and Charkowski, A.O. (2007) Host range and molecular phylogenies of the soft rot enterobacterial genera *Pectobacterium* and *Dickeya*. *Phytopathology*, **97**, 1150–1163.

Mattinen, L., Somervuo, P., Nykyri, J., Nissinen, R., Kouvonen, P., Corthals, G., Auvinen, P., Aittamaa, M., Valkonen, J.P. and Pirhonen, M. (2008) Microarray profiling of host-extract induced genes and characterization of the type VI secretion cluster in the potato pathogen *Pectobacterium atrosepticum*. *Microbiology*, **154**, 2387–2396.

Mew, T. (1989) An overview of the world bacterial blight situation. In: *Proceedings of the International Workshop on Bacterial Blight of Rice*, pp. 7–12. Los Banos: International Rice Research Institute.

Mew, T., Alvarez, A., Leach, J. and Swings, J. (1993) Focus on bacterial blight of rice. *Plant Dis.* **77**, 5–12.

Minsavage, G.V., Dahlbeck, D., Whalen, M.C., Kearney, B., Bonas, U., Staskawicz, B.J. and Stall, R.E. (1990) Gene-for-gene relationships specifying disease resistance

in *Xanthomonas campestris* pv. *vesicatoria*–pepper interactions. *Mol. Plant–Microbe Interact.* **3**, 41–47.

Mizukami, T. and Wakimoto, S. (1969) Epidemiology and control of bacterial leaf blight of rice. *Annu. Rev. Phytopathol.* **7**, 51–72.

Mole, B.M., Baltrus, D.A., Dangl, J.L. and Grant, S.R. (2007) Global virulence regulation networks in phytopathogenic bacteria. *Trends Microbiol.* **15**, 363–371.

Morris, C.E., Kinkel, L.L., Xiao, K., Prior, P. and Sands, D.C. (2007) Surprising niche for the plant pathogen *Pseudomonas syringae*. *Infect. Genet. Evol.* **7**, 84–92.

Mukherjee, A., Cui, Y., Ma, W., Liu, Y. and Chatterjee, A.K. (2000) *hexA* of *Erwinia carotovora* ssp. *carotovora strain* Ecc71 negatively regulates production of RpoS and rsmB RNA, a global regulator of extracellular proteins, plant virulence and the quorum-sensing signal, N-(3-oxohexanoyl)-L-homoserine lactone. *Environ. Microbiol.* **2**, 203–215.

Mulholland, V. and Salmond, G.P.C. (1995) Use of coliphage lambda and other bacteriophages for molecular genetic analysis of *Erwinia* and related Gram-negative bacteria. In: *Methods in Molecular Genetics*, Vol. **6B** (Adolph, S.D., ed.), pp. 24, 439–454. London: Academic Press.

Muniz, C.A., Jaillard, D., Lemaitre, B. and Boccard, F. (2007) *Erwinia carotovora* Evf antagonizes the elimination of bacteria in the gut of *Drosophila* larvae. *Environ. Microbiol.* **9**, 106–119.

Murillo, J., Bardaji, L. and Führer, E. (2010) La grasa de las judías, causada por la bacteria *Pseudomonas syringae* pv. *phaseolicola*. *Phytoma*, **224**, 27–32.

Murthy, V. and Devadath, S. (1984) Role of seed in survival and transmission of *Xanthomonas campestris* pv. *oryzae* causing bacterial blight of rice. *Phytopathology*, **110**, 15–19.

Ngugi, H.K., Lehman, B.L. and Madden, L.V. (2011) Multiple treatment meta-analysis of products evaluated for control of fire blight in the eastern United States. *Phytopathology*, **101**, 512–522.

Noda, T. and Kaku, H. (1999) Growth of *Xanthomonas oryzae* pv. *oryzae in planta* and in guttation fluid of rice. *Ann. Phytopathol. Soc. Jpn.* **65**, 9–14.

Norelli, J.L., Jones, A.L. and Aldwinckle, H.S. (2003) Fire blight management in the twenty-first century: using new technologies that enhance host resistance in apple. *Plant Dis.* **87**, 756–765.

O'Brien, H., Desveaux, D. and Guttman, D.S. (2011) Next-generation genomics of *Pseudomonas syringae*. *Curr. Opin. Microbiol.* **14**, 1–7.

Ochiai, H., Inoue, Y., Takeya, M., Sasaki, A. and Kaku, H. (2005) Genome sequence of *Xanthomonas oryzae* pv. *oryzae* suggests contribution of large numbers of effector genes and insertion sequences to its race diversity. *Jap. Agric. Res. Quart.* **39**, 275–287.

Oh, C.-S. and Beer, S.V. (2005) Molecular genetics of *Erwinia amylovora* involved in the development of fire blight. *FEMS Microbiol. Lett.* **253**, 185–192.

Ojeda, S. and Verdier, V. (2000) Detection of *Xanthomonas axonopodis* pv. *manihotis* in Cassava true seeds by nested-polymerase chain reaction assay (N-PCR). *Can. J. Plant Pathol.* **22**, 241–247.

Oliva, R., Win, J., Raffaele, S., Boutemy, L., Bozkurt, T.O., Chaparro-Garcia, A., Segretin, M.E., Stam, R., Schornack, S., Cano, L.M., van Damme, M., Huitema, E., Thines, M., Banfield, M.J. and Kamoun, S. (2010) Recent developments in effector biology of filamentous plant pathogens. *Cell. Microbiol.* **12**, 705–715.

Ou, S. (1972) *Rice Diseases*. Kew, Surrey: Commonwealth Mycological Institute.

Ou, S. (1985) *Rice Diseases*. Kew, Surrey: Commonwealth Agricultural Bureaux.

Park, C.J., Kazunari, N. and Ronald, P.C. (2010) Quantitative measurements of *Xanthomonas oryzae* pv. *oryzae* distribution in rice using fluorescent-labelling. *J. Plant Biol.* **15**, 595–599.

Parkinson, N., Stead, D., Bew, J., Heeney, J., Tsror (Lahkim), L. and Elphinstone, J. (2009) *Dickeya* species relatedness and clade structure determined by comparison of *recA* sequences. *Int. J. Syst. Evol. Microbiol.* **59**, 2388–2393.

Pérombelon, M.C.M. (2002) Potato diseases caused by soft rot erwinias: an overview of pathogenesis. *Plant Pathol.* **51**, 1–12.

Pérombelon, M.C.M. and Kelman, A. (1980) Ecology of the soft rot erwinias. *Annu. Rev. Phytopathol.* **18**, 361–387.

Pérombelon, M.C.M. and Salmond, G.P.C. (1995) Bacterial soft rots. In: *Pathogenesis and Host Specificity in Plant Diseases*, Vol. **1** (Prokaryotes) (Singh, U.S., Singh, R.P. and Kohmoto, K., eds), pp. 1–20. Oxford: Pergamon Press.

Pirhonen, M., Flego, D., Heikinheimo, R. and Palva, E.T. (1993) A small diffusible signal molecule is responsible for the global control of virulence and exoenzyme production in the plant pathogen *Erwinia carotovora*. *EMBO J.* **12**, 2467–2476.

Pitzschke, A. and Hirt, H. (2010) New insights into an old story: *Agrobacterium*-induced tumor formation in plants by plant transformation. *EMBO J.* **29**, 1021–1032.

Ponciano, G., Webb, K., Bai, J., Vera Cruz, C. and Leach, J.E. (2004) Molecular characterization of the *avrXa7* locus from *Xanthomonas oryzae* pv. *oryzae* field isolates. *Physiol. Mol. Plant Pathol.* **64**, 145–153.

Preston, G.M. (2000) *Pseudomonas syringae* pv. *tomato*: the right pathogen, of the right plant, at the right time. *Mol. Plant Pathol.* **1**, 263–275.

Rademaker, J.L., Louws, F.J., Schultz, M.H., Rossbach, U., Vauterin, L., Swings, J. and de Bruijn, F.J. (2005) A comprehensive species to strain taxonomic framework for *Xanthomonas*. *Phytopathology*, **9**, 1098–1111.

Rajeshwari, R. and Sonti, R.V. (2000) Stationary-phase variation due to transposition of novel insertion elements in *Xanthomonas oryzae* pv. *oryzae*. *J. Bacteriol.* **182**, 4797–4802.

Restrepo, S. and Verdier, V. (1997) Geographical differentiation of the population of *Xanthomonas axonopodis* pv. *manihotis* in Colombia. *Appl. Environ. Microbiol.* **63**, 4427–4434.

Restrepo, S., Duque, M.C. and Verdier, V. (2000a) Characterization of pathotypes among isolates of *Xanthomonas axonopodis* pv. *manihotis* in Colombia. *Plant Pathol.* **49**, 680–687.

Restrepo, S., Velez, C.M. and Verdier, V. (2000b) Measuring the genetic diversity of *Xanthomonas axonopodis* pv. *manihotis* within different fields in Colombia. *Phytopathology*, **90**, 683–690.

Robert-Baudouy, J. (1991) Molecular biology of Erwinia: from soft-rot to antileukaemics. *Trends Biotechnol.* **9**, 325–329.

Rodionov, D.A., Gelfand, M.S. and Hugouvieux-Cotte-Pattat, N. (2004) Comparative genomics of the KdgR regulon in *Erwinia chrysanthemi* 3937 and other gamma-proteobacteria. *Microbiology*, **150**, 3571–3590.

Rodríguez-Palenzuela, P., Matas, I.M., Murillo, J., López-Solanilla, E., Bardaji, L., Pérez-Martínez, I., Rodríguez-Moskera, M.E., Penyalver, R., López, M.M., Quesada, J.M., Biehl, B.S., Perna, N.T., Glasner, J.D., Cabot, E.L., Neeno-Eckwall, E. and Ramos, C. (2010) Annotation and overview of the *Pseudomonas savastanoi* pv. savastanoi NCPPB 3335 draft genome reveals the virulence gene complement of a tumour-inducing pathogen of woody hosts. *Environ. Microbiol.* **12**, 1604–1620.

Ronald, P.C. and Beutler, B. (2010) Plant and animal sensors of conserved microbial signatures. *Science*, **330**, 1061–1064.

Ryan, R.P. and Dow, J.M. (2011) Communication with a growing family: DSF signaling in bacteria. *Trends Microbiol.* **19**, 145–152.

Ryan, R.P., Fouhy, Y., Lucey, J.F., Crossman, L.C., Spiro, S., He, Y.-W., Zhang, L.-H., Heeb, S., Cámara, M., Williams, P. and Dow, J.M. (2006) Cell–cell signaling in *Xanthomonas campestris* involves an HD-GYP domain protein that functions in cyclic di-GMP turnover. *Proc. Natl. Acad. Sci. USA*, **103**, 6712–6717.

Ryan, R.P., Vorhölter, F.J., Potnis, N., Jones, J.B., Van Sluys, M.A., Bogdanove, A.J. and Dow, J.M. (2011) Pathogenomics of *Xanthomonas*: understanding bacterium–plant interactions. *Nat. Rev. Microbiol.* **9**, 344–355.

Salanoubat, M., Genin, S., Artiguenave, F., Gouzy, J., Mangenot, S., Arlat, M., Billault, A., Brottier, P., Camus, J.C., Cattolico, L., Chandler, M., Choisne, N., Claudel-Renard, C., Cunnac, S., Demange, N., Gaspin, C., Lavie, M., Moisan, A., Robert, C., Saurin, W., Schiex, T., Siguier, P., Thebault, P., Whalen, M., Wincker, P., Levy, M., Weissenbach, J. and Boucher, C.A. (2002) Genome sequence of the plant pathogen *Ralstonia solanacearum*. *Nature*, **415**, 497–502.

Salmond, G.P.C. (1992) Bacterial diseases of potatoes: from classical phytobacteriology to molecular pathogenicity. *Neth. J. Plant Pathol.* **98** (Suppl, 2), 115–126.

Salmond, G.P.C. (1994) Secretion of extracellular virulence factors of plant pathogenic bacteria. *Annu. Rev. Phytopathol.* **32**, 181–200.

Salzberg, S.L., Sommer, D.D., Schatz, M.C., Phillippy, A.M., Rabinowicz, P.D., Tsuge, S., Furutani, A., Ochiai, H., Delcher, A.L., Kelley, D., Madupu, R., Puiu, D., Radune, D., Shumway, M., Trapnell, C., Aparna, G., Jha, G., Pandey, A., Patil, P.B., Ishihara, H., Meyer, D.F., Szurek, B., Verdier, V., Koebnik, R., Dow, J.M., Ryan, R.P., Hirata, H., Tsuyumu, S., Won Lee, S., Seo, Y.S., Sriariyanum, M., Ronald, P.C., Sonti, R.V., Van Sluys, M.A., Leach, J.E., White, F.F. and Bogdanove, A.J. (2008) Genome sequence and rapid evolution of the rice pathogen *Xanthomonas oryzae* pv. *oryzae* PXO99A. *BMC Genomics*, **9**, 204.

Samson, R., Legendre, J.B., Christen, R., Fischer-Le Saux, M., Achouak, W. and Gardan, L. (2005) Transfer of *Pectobacterium chrysanthemi* (Burkholder *et al*., 1953) Brenner I. 1973 and *Brenneria paradisiaca* to the genus *Dickeya* gen. nov. as *Dickeya chrysanthemi* comb. nov and *Dickeya paradisiaca* comb. nov. and delineation of four novel species, *Dickeya dadantii* sp. nov., *Dickeya dianthicola* sp. nov., *Dickeya dieffenbachiae* sp. nov. and *Dickeya zeae* sp. nov. *Int. J. Syst. Evol. Microbiol.* **55**, 1415–1427.

Scholthof, K.-B.G., Adkins, S., Czosnek, H., Palukaitis, P., Jacquot, E., Hohn, T., Hohn, B., Saunders, K., Candresse, T., Ahlquist, P., Hemenway, C. and Foster, G.D. (2011) Top 10 plant viruses in molecular plant pathology. *Mol. Plant Pathol.* **12**, 938–954.

Schwessinger, B. and Ronald, P.C. (2012) Plant innate immunity: recognition of conserved microbial signatures. *Ann. Rev. Plant Biol.* **8**, 23.

Sebaihia, M., Bocsanczy, A.M., Biehl, B.S., Quail, M.A., Perna, N.T., Glasner, J.D., DeClerck, G.A., Cartinhour, S., Schneider, D.J., Bentley, S.D., Parkhill, J. and Beer, S.V. (2010) Complete genome sequence of the plant pathogen *Erwinia amylovora* strain ATCC 49946. *J. Bacteriol.* **192**, 2020–2021.

Segond, D., Dellagi, A., Lanquar, V., Rigault, M., Patrit, O., Thomine, S. and Expert, D. (2009) NRAMP genes function in *Arabidopsis thaliana* resistance to *Erwinia chrysanthemi* infection. *Plant J.* **58**, 195–207.

Shenge, K.C., Mabagala, R.B., Mortensen, C.N., Stephan, D. and Wydra, K. (2007) First report of bacterial speck of tomato caused by *Pseudomonas syringae* pv. *tomato* in Tanzania. *Plant Dis.* **91**, 462.

Simpson, A.J.G., Reinach, F.C., Arruda, P., Abreu, F.A, Acencios, M., Alvarenga, R., Alves, L.M.C., Araya, J.E., Baia, G.S., Baptlsta, C.S., Barros, M.H., Bonaccorsi, H.O., Bordin, S., Bové, J.S., Brione, M.R.S., Buenoll, M.R.P., Camargo, A.A., Camargo, L.E.A., Caffaro, D.M., Carrer, H., Colauto, N.B., Colombo, C., Costa, F.F., Costa, M.C.R., Costa-Neto, C.M.L., Coutinho, L., Cristofani, M., Dias-Neto, E., Docena, C., El-Dorry, H., Facincani, A.P., Ferreira, A.J.S., Ferreira, V.C.A., Ferro, J.A., Fraga, J.S., França, S.C., Franco, M.C., Frohme, M., Furlan, L.R., Garnier, M., Goldman, G.H., Goldman, M.H.S., Gomes, L.S., Gruber, A., Ho, P.L., Hoheisel, J.D., Junqueira, M.L., Kemper, E.L., Kitajima, J.P., Krieger, J.E., Kuramae, E.E., Laigret, F., Lambais, M.R., Leite, L.C.C., Lemos, E.G.M., Lemos, M.V.F., Lopes, S.A., Lopes, C.R., Machado, J.A., Machado, M.A., Madeira, A.M.B.N., Madeira, H.M.F., Marlno, C.L., Marques, M.V., Martlns, E.A.L., Martins, E.M.F., Matsukuma, L.Y., Mencke, C.F.M., Mlracca, E.C., Miyaki, C.Y., Monteiro-Vltorello, C.B., Moon, D.H., Nagai, M.A., Nascimento, A.L.T.O., Netto, L.E.S, Nhani Jr., A., Nobrega, F.G., Nunes, L.R., Oliveira, M.A., Oliveira, M.C., Oliveira, R.C., Palmieri, D.A., Paris, A., Peixoto, R.R., Pereira, G.A.G., Pereira Jr., H.A., Pesquero, J.B., Quaggio, R.B., Roberto, P.G., Rodrigues, V., Rosa, A.J.M., Rosa Jr., V.E., Sá, R.G., Santelli, R.V., Sawasaki, H.E., Silva, L.C.R., Silva, A.M., Silva, F.R., Silva Jr., W.A., Silveira, J.F., Silvestri, Siqueira, W.J., Souza, A.A., Souza, A.P., Terenzi, M.F., Truffi, D., Tsai, S.M., Tsuhako, M.H., Vallada H., Van Sluys, M.A., Verjovski-Almeida S., Vettore A.L., Zago M.A., Zatz M., Meidanis J. and Setubal, J.C. (2000) The genome sequence of plant pathogen *Xylella fastidiosa*. *Nature*, **406**, 151–159.

Singh, R.A., Das, B., Ahmed, K.V. and Pal, V. (1980) Chemical control of bacterial leaf blight of rice. *Trop. Pest Manage.* **26**, 21–25.

Sławiak, M., van Beckhoven, J.R.C.M., Speksnijder, A.G.C.L., Czajkowski, R., Grabe, G. and van der Wolf, J.M. (2009) Biochemical and genetical analysis reveal a new clade of biovar 3 *Dickeya* spp. strains isolated from potato in Europe. *Eur. J. Plant Pathol.* **125**, 245–261.

Smith, E.F. and Townsend, C.O. (1907) A plant tumor of bacterial origin. *Science*, **25**, 671–673.

Smith, I.M., Dunez, J., Lelliott, R.A., Phillips, D.H. and **Archer, S.A. (eds)** (1988) *European Handbook of Plant Diseases*. Oxford: Blackwell Scientific Publications.

Swings, J., Van den Mooter, M., Vauterin, L., Hoste, B., Gillis, M., Mew, T. and Kersters, K. (1990) 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. Bacteriol.* **40**, 309–311.

Toth, I., Perombelon, M.C.M. and Salmond, G.P.C. (1993) Bacteriophage fKP-mediated generalised transduction in *Erwinia carotovora* subspecies *carotovora*. *J. Gen. Microbiol.* **139**, 2705–2709.

Toth, I., Mulholland, V., Shih, Y.-L., Bentley, S., Pérombelon, M.C.M. and Salmond, G.P.C. (1997) Generalised transduction in the potato blackleg pathogen, *Erwinia carotovora* subspecies *atroseptica*, via phage fM1. *Microbiology*, **143**, 2433–2438.

Toth, I.K., Bell, K., Holeva, M.C. and Birch, P.R.J. (2003) Soft rot erwiniae: from genes to genomes. *Mol. Plant Pathol.* **4**, 17–30.

Toth, I.K., Newton, J.A., Hyman, L.J., Lees, A.K., Daykin, M., Williams, P. and Fray, R.J. (2004) Potato plants genetically modified to produce N-acylhomoserine lactones increase susceptibility to soft rot erwiniae. *Mol. Plant–Microbe Interact.* **17**, 880–888.

Toth, I.K., van der Wolf, J.M., Saddler, G., Lojkowska, E., Helias, V., Pirhonen, M., Tsror (Lahkim), L. and Elphinstone, J.G. (2011) *Dickeya* species: an emerging problem for potato production in Europe. *Plant Pathol.* **60**, 385–399.

Tsror (Lahkim), L., Erlich, O., Lebiush, S., Hazanovsky, M., Zig, U., Sławiak, M., Grabe, G., van der Wolf, J.M. and van de Haar, J.J. (2009) Assessment of recent outbreaks of *Dickeya* sp. (syn. *Erwinia chrysanthemi*) slow wilt in potato crops in Israel. *Eur. J. Plant Pathol.* **123**, 311–320.

Tzfira, T. and Citovsky, V. (2002) Partners-in-infection: host proteins involved in the transformation of plant cells by *Agrobacterium*. *Trends Cell Biol.* **12**, 121–129.

Vanneste, J.L. (ed.) (2000) *Fire Blight; the Disease and its Causative Agent* Erwinia amylovora. London: CAB International.

Vauterin, L., Rademaker, J. and Swings, J. (2000) Synopsis on the taxonomy of the genus *Xanthomonas*. *Phytopathology*, **7**, 677–682.

Venkatesh, B., Babujee, L., Liu, H., Hedley, P., Fujikawa, T., Birch, P.R.J., Toth, I.K. and Tsuyumu, S. (2006) The *Erwinia chrysanthemi* 3937 PhoQ sensor kinase regulates several virulence determinants. *J. Bacteriol.* **188**, 3088–3098.

Verdier, V., Dongo, P. and Boher, B. (1993) Assessment of genetic diversity among strains of *Xanthomonas campestris* pv. *manihotis*. *J. Gen. Microbiol.* **139**, 2591–2601.

Verdier, V., Mosquera, G. and Assigbétsé, K. (1998) Detection of the cassava bacterial blight pathogen, *Xanthomonas axonopodis* pv. *manihotis*, by polymerase chain reaction. *Plant Dis.* **82**, 79–83.

Verdier, V., Restrepo, S., Mosquera, G., Jorge, V. and Lopez, C. (2004) Recent progress in the characterization of molecular determinants in the *Xanthomonas axonopodis* pv. *manihotis*–cassava interaction. *Plant Mol. Biol.* **56**, 573–584.

Verdier, V., Vera Cruz, C. and Leach, J.E. (2011) Controlling rice bacterial blight in Africa: needs and prospects. *J. Biotechnol.* Available at http://www.ncbi.nlm.nih.gov/pubmed/21963588. [accessed on Sept 22, 2011].

Vorhölter, F.J., Schneiker, S., Goesmann, A., Krause, L., Bekel, T., Kaiser, O., Linke, B., Patschkowski, T., Rückert, C., Schmid, J., Sidhu, V.K., Sieber, V., Tauch, A., Watt, S.A., Weisshaar, B., Becker, A., Niehaus, K. and Pühler, A. (2008) The genome of *Xanthomonas campestris* pv. *campestris* B100 and its use for the reconstruction of metabolic pathways involved in xanthan biosynthesis. *J. Biotechnol.* **134**, 33–45.

Waters, C.M. and Bassler, B.L. (2005) Quorum sensing: cell-to-cell communication in bacteria. *Annu. Rev. Cell Dev. Biol.* **21**, 319–346.

Wharam, S., Mulholland, V. and Salmond, G.P.C. (1995) Conserved virulence factor regulation and secretion systems in bacterial pathogens of plants and animals. *Eur. J. Plant Pathol.* **101**, 1–13.

Whitehead, N., Barnard, A.M.L., Slater, H., Simpson, N.J.L. and Salmond, G.P.C. (2001) Quorum sensing in Gram-negative bacteria. *FEMS Microbiol. Rev.* **25**, 365–404.

Wydra, K., Zinsou, V., Jorge, V. and Verdier, V. (2004) Identification of pathotypes of *Xanthomonas axonopodis* pv. *manihotis* in Africa and detection of specific quantitative trait loci (QTL) for resistance to cassava bacterial blight. *Phytopathology*, **94**, 1084–1093.

Yamazaki, A., Li, J., Hutchins, W.C., Wang, L., Ma, J., Ibekwe, A.M. and Yang, C.-H. (2011) Commensal effect of pectate lyases secreted from *Dickeya dadantii* on proliferation of *Escherichia coli* O157:H7 EDL933 on lettuce leaves. *Appl. Environ. Microbiol.* **77**, 156–162.

Yang, C.-H., Gavilanes-Ruiz, M., Okinaka, Y., Vedel, R., Berthuy, I., Boccara, M., Chen, J.W., Perna, N.T. and Keen, N.T. (2002) hrp genes of *Erwinia chrysanthemi* 3937 are important virulence factors. *Mol. Plant–Microbe Interact.* **15**, 472–480.

Yang, S., Peng, Q., Zhang, Q., Zou, L., Li, Y., Robert, C., Pritchard, L., Liu, H., Hovey, R., Wang, Q., Birch, P.R.J., Toth, I.K. and Yang, C.-H. (2010) Genome-wide identification of HrpL-regulated genes in the necrotrophic phytopathogen *Dickeya dadantii* 3937. *PLoS ONE*, **5**, e13472.

Young, J.M., Park, D.C., Shearman, H.M. and Fargier, E. (2008) A multilocus sequence analysis of the genus *Xanthomonas*. *Syst. Appl. Microbiol.* **5**, 366–377.

Zaltsman, A., Krichevsky, A., Loyter, A. and Citovsky, V. (2010) *Agrobacterium* induces expression of a plant host F-box protein required for tumorigenicity. *Cell Host Microbe*, **7**, 197–209.

Zhang, J., Li, W., Xiang, T., Liu, Z., Laluk, K., Ding, X., Zou, Y., Gao, M., Zhang, X., Chen, S., Mengiste, T., Zhang, Y. and Zhou, J.M. (2010) Receptor-like cytoplasmic kinases integrate signaling from multiple plant immune receptors and are targeted by a *Pseudomonas syringae* effector. *Cell Host Microbe*. **7**, 290–301.

Zupan, J., Muth, T.R., Draper, O. and Zambryski, P.C. (2000) The transfer of DNA from *Agrobacterium tumefaciens* into plants: a feast of fundamental insights. *Plant J.* **23**, 11–28.

