

Breeding for resistances to ralstonia solanacearum

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Introduction

Ralstonia solanacearum, the causal agent of bacterial wilt, is one of the most devastating plant pathogenic bacteria ([Mansfield et al., 2012](#)) with a large host range encompassing more than 200 plant species which include major agricultural crops such as tomato, potato and banana ([Hayward, 1991](#) ; [Elphinstone, 2005](#)). *R. solanacearum* is a soil-borne bacterium that enters plant roots, invades xylem vessels and spreads rapidly to aerial parts of the plant through the vascular system where its high level of multiplication leads to wilting symptoms and, ultimately, plant death ([Genin, 2010](#)). In addition to its lethality, the ability of *R. solanacearum* to survive in soils for many years and to form latent infections within indigenous weeds contributes to the difficult eradication of the bacterium ([Hayward, 1991](#) ; [Wenneker et al., 1999](#)). The pathogen is found worldwide, primarily in tropical and subtropical regions ([Hayward, 1991](#)) but also in Europe and North America where cold-tolerant strains were introduced in the 1990s ([Janse et al., 2004](#) ; [Swanson et al., 2005](#)). The dissemination of *R. solanacearum* is a threat to crops and the pathogen is considered a quarantine bacterium.

Ralstonia solanacearum strains present an extensive genetic diversity and are divided in four phlotypes corresponding roughly to the strains' geographic origin: Asia (phlotype I), the Americas (II), Africa (III), and Indonesia (IV). Phlotype II has two subclusters: IIA and IIB ([Fegan and Prior, 2005](#)) and only strains belonging to phlotype IIB are responsible for bacterial wilt of potato in cold and temperate regions ([Janse et al., 2004](#)). Phlotypes are not related with host preference as strains from all

phylotypes are able to cause disease on potato, tomato, pepper, and eggplant ([Cellier and Prior, 2010](#) ; [Lebeau et al., 2011](#)).

Among the virulence determinants of *R. solanacearum*, the type three secretion system (TTSS), a molecular syringe whose structural and regulatory elements are encoded by *hrp* (hypersensitive response and pathogenicity) genes, is essential for pathogenicity ([Vasse et al., 2000](#)). TTSS injects effector proteins into plant cells to favor the bacterial infection by subverting and exploiting the host signaling pathways ([Poueymiro and Genin, 2009](#)). Effectors could promote nutrient leakage but mostly they are predicted to manipulate plant defenses ([Goel et al., 2008](#) ; [Deslandes and Rivas, 2012](#)).

There are two levels in plant immunity ([Jones and Dangl, 2006](#)). The first one uses cell surface pattern recognition receptors (PRRs) to detect pathogen-associated molecular patterns (PAMPs) and initiate PAMP-triggered immunity (PTI). The second involves nucleotide-binding leucine-rich repeat (NB-LRR) proteins, encoded by resistance (*R*) genes, which sense pathogen effectors and elicit a potent immune response called effector-triggered immunity (ETI). ETI is faster, longer and stronger than PTI and usually leads to a local cell death, the hypersensitive response (HR), which stops the spread of the pathogen ([Jones and Dangl, 2006](#)). Some effectors that enable the pathogen to overcome PTI are recognized by R proteins and the effector is thus termed an avirulence (Avr) protein ([Jones and Dangl, 2006](#)). Disease (susceptibility) results when one or both R/Avr partners are absent. Conversely, resistance ensues when R and cognate Avr effector are both present. The activation of plant defenses by an R protein is termed

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qualitative resistance and provides a complete disease resistant phenotype. An incomplete resistance (tolerance), leading to the reduction rather than the eradication of the disease, is called quantitative ([Young, 1996](#)).

Quantitative disease resistance (QDR) molecular characterization is still in its infancy. *R* genes ([Roux et al., 2010](#) , [2014](#) ; [Van der Linden et al., 2013](#)) can contribute to QDR as well as components such as kinases or transporters ([St Clair, 2010](#) ; [Roux et al., 2014](#)).

Continuous increase in food production is needed to face the world's population growth. One way to achieve this goal is by sustainably reducing crop losses to pathogens like *R. solanacearum*. This review provides a summary on current control strategies of bacterial wilt and highlights the difficulties of breeding for resistance to *R. solanacearum*. In addition, knowledge gained from studies in model plants on resistance to bacterial wilt will be discussed. Finally, the use of *R. solanacearum* effectors to decipher the molecular mechanisms of plant immunity and identify new sources of resistance will be explored.

Strategies for Control of *R. solanacearum*

Integrated strategies for control of *R. solanacearum* are complex because the bacterium is able to infect crops as a soil-borne, water-borne, or seed/tuber-borne organism. Therefore, to avoid the dissemination of *R. solanacearum* , it is recommended to plant healthy seeds in pathogen free soil combined with irrigation water known to be free of *R. solanacearum* ([French, 1994](#)). In infested soils, crop rotation (2-5 years), control of weed hosts and survey of water for irrigation can reduce the bacterial load ([Lopez and Biosca, 2005](#)). Chemical control, in addition to being potentially harmful <https://assignbuster.com/breeding-for-resistances-to-ralstonia-solanacearum/>

to the environment, was not proved to be efficient to eradicate *R. solanacearum* ([Saddler, 2005](#) ; [Denny, 2006](#)). This can be explained by the bacterium localization in the deeper soil layers or sheltered in xylem vessels of infected plants and weeds ([Wenneker et al., 1999](#)). In addition, a soil dependent effect has been observed and therefore soil disinfection is not universally applicable ([Saddler, 2005](#)). An alternative control strategy was to use biological control agent such as antagonistic bacteria or avirulent mutants of *R. solanacearum* but the promising results obtained under controlled conditions were not confirmed in the field ([Saddler, 2005](#)). The most extensively studied avirulent mutants of *R. solanacearum* are *hrp*⁻ mutant strains that no longer possess a functional TTSS ([Frey et al., 1994](#)). *hrp*⁻ mutant strains are still able to multiply, and, likely control bacterial wilt through competition with wild type strains for space and nutrients. Assays of protection by *hrp*⁻ mutants have been conducted on tomato and potato plants but, thus far, they did not help to reduce bacterial wilt in fields ([Saddler, 2005](#) ; [Denny, 2006](#)). In the absence of efficient strategies to eradicate *R. solanacearum* from infected soils and water, the use of resistant cultivars appears to be the best disease control strategy.

Breeding for Resistance to *R. solanacearum*

Resistance breeding to *R. solanacearum* in solanaceous crops appears to be regional or linked to climatic conditions ([Hayward, 1991](#)) and this limited success is due to all the constraints resistant cultivars must outsmart. First, the breeding must combine durable resistance with desirable agronomic traits. Second, resistant cultivars must be able to face the diversity of agro-ecological zones where the bacteria proliferates and the high genetic

variability of *R. solanacearum* strains. Third, breeding for highly resistant cultivars must be prioritized to avoid further *R. solanacearum* dissemination due to tolerant plants that shelter virulent bacteria without showing disease symptoms. Finally, the available sources of resistance were found to be polygenic and, despite the identification of QTLs (quantitative trait loci) controlling resistance to bacterial wilt in tomato ([Mangin et al., 1999](#) ; [Wang et al., 2000](#) , [2013](#) ; [Carmeille et al., 2006](#)), tobacco ([Qian et al., 2013](#)) and eggplant ([Lebeau et al., 2013](#)), the development of resistant crops is impeded by the difficulty in transferring into cultivars a high number of genes that, in addition, can be linked to undesirable traits ([Denny, 2006](#)).

In potato, high level resistance to bacterial wilt has been identified in *Solanum phureja* species ([Sequeira and Rowe, 1969](#)), however, the resistance was unstable across locations and its breakdown was triggered by high temperature and decrease in light intensity ([French and De Lindo, 1982](#)). In tomato, the polygenic resistance to bacterial wilt in the resistant cultivar Hawaii 7996 ([Grimault et al., 1995](#)) was suggested to be strain specific ([Wang et al., 2000](#)) and, more recently, it was hypothesized that QTLs in Hawaii 7996 may deploy a phylotype-specific resistance ([Carmeille et al., 2006](#)). These results exemplify the difficulty of obtaining a worldwide resistance to *R. solanacearum*.

Genetic Basis of Resistances in Model Plants

To date, studies on resistance to bacterial wilt in model plants were mainly conducted in *Arabidopsis thaliana* and *Medicago truncatula* . As *R. solanacearum* infects a large number of leguminous plants, such as peanut and common bean, a model pathosystem for the study of *R. solanacearum* - <https://assignbuster.com/breeding-for-resistances-to-ralstonia-solanacearum/>

legume interactions was developed with *M. truncatula* ([Vaillau et al., 2007](#)). A genetic analysis of recombinant inbred lines identify three QTLs involved in disease resistance and fine mapping showed that the major QTL include a cluster of seven putative *R* genes ([Vaillau et al., 2007](#) ; [Ben et al., 2013](#)). Future functional characterization is required to depict the molecular mechanisms underlying *M. truncatula* resistance to bacterial wilt and to appraise their potential application in breeding.

Several *R. solanacearum* strains are able to cause the wilting of *A. thaliana* plants ([Deslandes et al., 1998](#)) and insightful knowledge on the molecular basis of resistance to bacterial wilt was uncovered in this pathosystem. In *A. thaliana* accession Col-0, a polygenic mechanism encompassing three QTLs governs resistance to *R. solanacearum* strain 14. 25 ([Godiard et al., 2003](#)). In these loci, the LRR receptor-like kinase *ERECTA* involved in development of aerial organs was identified thus suggesting a cross-talk between resistance and developmental pathways ([Godiard et al., 2003](#)). Other studies established that resistance in *A. thaliana* to *R. solanacearum* could also be monogenic and identified dominant and recessive loci ([Deslandes et al., 1998](#) ; [Ho and Yang, 1999](#)). Further analysis determined that the recessive *RRS1-R* (Resistance to *Ralstonia Solanacearum* 1) gene encodes an atypical R protein harboring a C-terminal WRKY DNA-binding domain ([Deslandes et al., 2002](#)) hypothesized to act as a negative transcriptional regulator of plant defenses ([Noutoshi et al., 2005](#)). It's the recognition by *RRS1-R* of the bacterial effector PopP2 that triggers resistance in *A. thaliana* accession Nd-1 inoculated with *R. solanacearum* GMI1000 strain ([Deslandes et al., 2003](#)). *RRS1-R* physically associates with another R protein, *RPS4*

(Resistant to *Pseudomonas Syringae* 4), to cooperatively trigger immunity ([Narusaka et al., 2009](#) ; [Williams et al., 2014](#)). The transfer of the *RRS1/RPS4* pair of *R* genes from *A. thaliana* into tomato was able to confer immunity to *R. solanacearum* ([Narusaka et al., 2013](#)). This study demonstrates that interfamily transfer of *R* genes can provide a new strategy to develop pathogen-resistant crops ([Narusaka et al., 2013](#)). Surprisingly, PopP2 perception by RRS1 can also lead to *A. thaliana* tolerance to bacterial wilt ([Van der Linden et al., 2013](#)). In the accession Kil-0, after inoculation with *R. solanacearum* strain BCCF402, the plants showed no wilting symptoms despite high bacterial numbers ([Van der Linden et al., 2013](#)). This work is one of few examples describing that tolerance is not always a polygenic trait and that an *R* gene can be common for both *A. thaliana* resistance and tolerance ([Roux et al., 2010](#) ; [Van der Linden et al., 2013](#)). Van der Linden and co-workers pointed out the risk of *R. solanacearum* persistence in the field after deployment of RRS1 in transgenic crops. Thus, despite a promising interfamily transfer of RRS1/RPS4 in crops ([Narusaka et al., 2013](#)), the use of RRS1 as a source of resistance to bacterial wilt may not be a relevant choice.

Effector Assisted Identification of Resistances to *R. solanacearum*

Unraveling the molecular functions of effectors is insightful for a mechanistic understanding of the processes underlying plant immune responses ([Deslandes and Rivas, 2012](#)). Despite the fact that *R. solanacearum* is one of the most destructive phytopathogens, few of its effectors have been functionally characterized and the nature of their plant targets remains

largely unknown [for reviews see ([Peeters et al., 2013b](#) ; [Deslandes and Genin, 2014](#))]. Some *R . solanacearum* effectors have been shown to trigger an HR-like response in petunia ([Arlat et al., 1994](#)), *A. thaliana* ([Solé et al., 2012](#) ; [Williams et al., 2014](#)), tobacco ([Arlat et al., 1994](#) ; [Poueymiro et al., 2009](#) ; [Solé et al., 2012](#)) and eggplant ([Nahar et al., 2014](#)). Besides, some *R . solanacearum* effectors have been found to be under a strong diversifying positive selection, to contribute to the pathogenicity of the bacteria and to dampen plant defense responses ([Peeters et al., 2013b](#) ; [Deslandes and Genin, 2014](#)). Collectively these data demonstrate that further functional characterization of *R . solanacearum* effectors will help to identify pivotal components of plant immunity that are manipulated by, or able to perceive, effectors. In order to gather useful knowledge to transfer in breeding programs, future work on characterizing molecular interactions between effectors and plant components should take place in agronomical relevant plants. For example, research could be conducted on tomato and potato, two major crops, whose genome availability should facilitate functional studies.

The availability of genome sequences from eleven *R . solanacearum* strains, belonging to the four phylotypes, allowed to establish the core effector repertoire of the bacterium ([Peeters et al., 2013a](#)). Core effectors presumably represent ancestral effectors and can be considered as crucial for the interaction between *R . solanacearum* and its hosts ([Peeters et al., 2013a](#)). “ Effectoromics ” refers to a new high-throughput approach that uses effectors for probing plant germplasm to detect *R* genes and to improve their deployment in the field ([Vleeshouwers et al., 2011](#) ; [Vleeshouwers and Oliver, 2014](#)). This useful technique could be applied to identify *R .*

solanacearum effectors with avirulent function and hasten the discovery of their cognate *R* genes. Besides, by performing plant germplasm screening with core effectors, the chances to identify an *R* gene displaying a broad and durable spectrum of resistance should be increased. Furthermore, effector-assisted breeding also allows us to determine the potential of a new *R* gene for durable resistance after deployment in the field by checking its expanded recognition specificity toward various effector alleles found in the pathogen population ([Vleeshouwers and Oliver, 2014](#)). As *R . solanacearum* strains present an extensive genetic diversity, the efficiency of an effectomics approach could be increased by focusing on bacterial strains/plant germplasm systems that have co-evolved jointly in a same geographic area.

Perspectives

Thus far, resistances to bacterial wilt available in solanaceous crops behave differently under changing environmental conditions. In addition, *R . solanacearum* strains exhibit an extensive genetic diversity worldwide. It thus seems like a chimera to look for universal resistances to bacterial wilt. A more realistic approach would be to seek for sources of resistance adapted only to a given ecosystem. Moreover, an ecological approach of bacterial wilt would enable to establish, in natural ecosystems, the pathogen population profile and monitor its changes. This could potentially lead to the identification of new biological control agents, naturally occurring in the soil microflora in fields, which would be more efficient to compete with *R . solanacearum* ([Saddler, 2005](#)). Also, a precise knowledge of the endemic *R . solanacearum* strains encountered in cultivation areas would guide the deployment of the most relevant resistant cultivars.

The identification of new resistance sources could be accelerated by exploiting *R. solanacearum* effectors through an effectomics approach ([Vleeshouwers and Oliver, 2014](#)), as well as through the molecular characterization of their virulence and avirulence functions. Once identified, monogenic over polygenic resistances should be prioritized to facilitate their transfer into crops and provide a higher level of resistance more likely to favor the eradication of *R. solanacearum*.

After identifying new resistance sources, to increase the chance of success in achieving durable resistance to bacterial wilt, *R*-gene stacking in fields should be applied ([Dangl et al., 2013](#)). Moreover, to reduce the bacterial pressure and decrease resistance breakdown, deployment of resistant cultivars should be backed up with an integrated management strategy to decrease the bacteria survival in soil, water and in the rhizosphere of weedy and native non-host plants ([Lopez and Biosca, 2005](#)).

In conclusion, only the elaboration of a complex strategy for resistance and control of *R. solanacearum* will be able to fight off this multifaceted pathogen.

Conflict of Interest Statement

The author declares that the research was conducted in the absence of any commercial or financial relationships that could be construed as a potential conflict of interest.

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