MICROBIOLOGY

MICROBE PROFILE

Arnold and Preston, *Microbiology* 2019;165:251–253

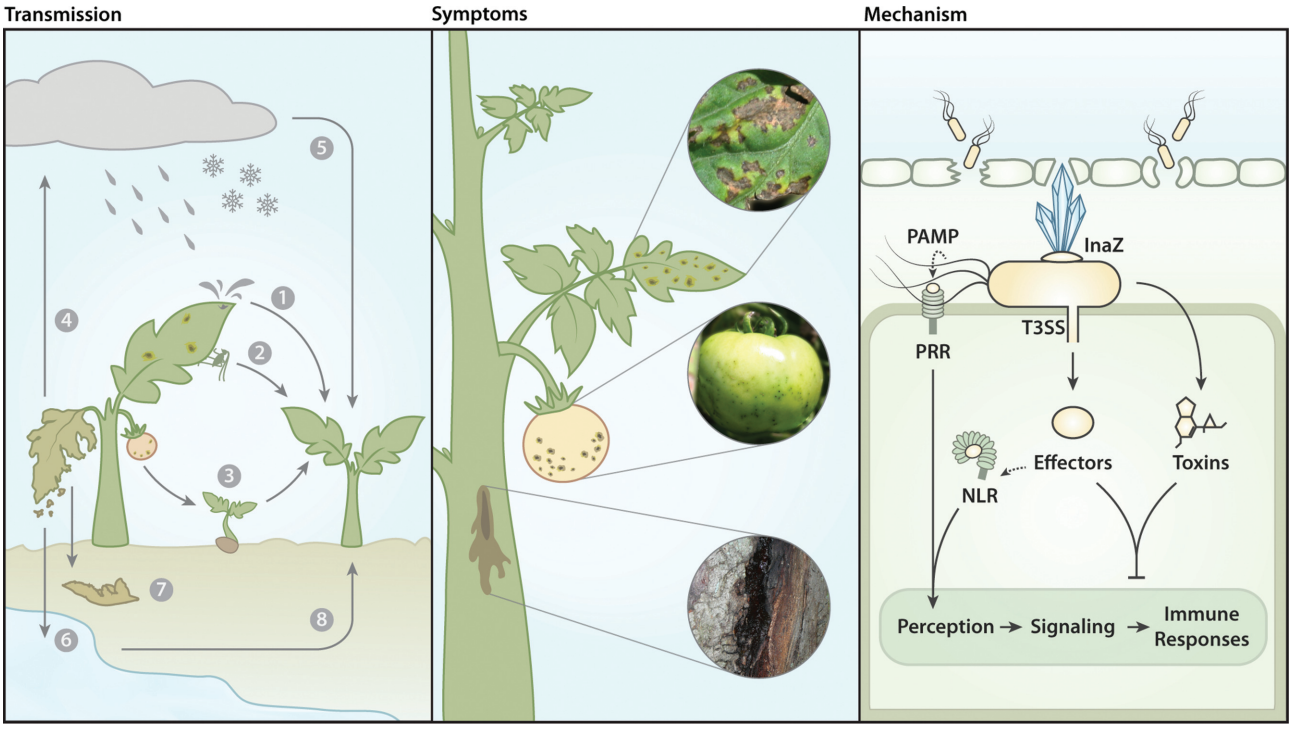
DOI 10.1099/mic.0.000715

MICROBIOLOGY SOCIETY

OPEN MICROBIOLOGY

Pseudomonas syringae: enterprising epiphyte and stealthy parasite

Dawn L. Arnold[1,*] and Gail M. Preston[2]



Graphical abstract

The lifecycle and pathogenicity mechanisms of *Pseudomonas syringae*. **Transmission**: *P. syringae* can be disseminated by rainsplash, aerosols and airborne plant particles (1), insect vectors (2) or as a seed-borne pathogen (3). If carried into the atmosphere (4), ice-nucleating *P. syringae* strains can contribute to ice nucleation in clouds and be disseminated in snow or rainfall (5). *P. syringae* can also be disseminated through terrestrial water systems (6, 8) and through plant debris in soil (7, 8). **Symptoms**: *P. syringae* infections are commonly characterized by chlorosis and necrosis of leaves, stem tips, buds and flowers (top); by necrotic lesions and delayed ripening or altered development of fruit (middle); and by cankers and galls of woody tissues (bottom). **Mechanism**: *P. syringae* enters plant tissues through wounds and natural openings such as stomata. Some strains of *P. syringae* can increase frost damage to plant tissues through ice nucleation promoted by proteins such as InaZ. Pathogen-associated molecular patterns (PAMPs) produced by *P. syringae*, such as flagellin, are recognized by plant pattern recognition receptors (PRRs), triggering the induction of plant immune responses. *P. syringae* counters plant immune responses through the production of toxins, and the secretion of effector proteins via a type III secretion system (T3SS). Effector proteins and toxins disable or subvert plant immune responses and alter plant metabolism and physiology to promote *P. syringae* infection. Effectors can also be directly or indirectly recognized by plant immune receptors, notably nucleotide-binding domain and leucine-rich repeat region (NLR)-containing proteins, thereby triggering plant immune responses. Picture: Nattapong Sanguankiattichai.

Abstract

Pseudomonas syringae is best known as a plant pathogenic bacterium that causes diseases in a multitude of hosts, and it has been used as a model organism to understand the biology of plant disease. Pathogenic and non-pathogenic isolates of *P. syringae* are also commonly found living as epiphytes and in the wider environment, including water sources such as rivers and precipitation. Ice-nucleating strains of *P. syringae* are associated with frost damage to crops. The genomes of numerous strains of *P. syringae* have been sequenced and molecular genetic studies have elucidated many aspects of this pathogen's interaction with its host plants.

TAXONOMY

Domain *Bacteria*, phylum *Proteobacteria*, class gamma subdivision, order *Pseudomonadales*, family *Pseudomonadaceae*, genus *Pseudomonas*, species *Pseudomonas syringae*. The '*P. syringae* complex' encompasses up to 10 *Pseudomonas* species and over 60 pathogenic variants (pathovars) with different host ranges.

PROPERTIES

P. syringae is a Gram-negative aerobic bacterium with rod-shaped cells, which are typically 1.5 um long and 0.7–1.2 um in diameter. The cells are motile, using at least one polar flagellum. The optimal temperature for growth ranges from 22–30 °C, and *P. syringae* is negative for oxidase and arginine dihydrolase activity. Many *P. syringae* strains produce the polysaccharide levan and elicit the hypersensitive response on tobacco, which are elements of the levan, oxidase, potato rot, arginine dihydrolase, tobacco hypersensitivity (LOPAT) test. The *P. syringae* species complex is subdivided into pathovars depending on the plant species they infect. Currently over 60 pathovars have been defined [1].

GENOME

The complete genome sequences of three pathovars of *P. syringae* were published between 2003 and 2005. These were pv. *tomato* strain DC3000 with a 6.4 Mb chromosome and two plasmids (74 and 67 kb); pv. *syringae* strain B728a, which has a genome size of 6.1 Mb; and pv. *phaseolicola* strain 1448A with a 5.9 Mb chromosome and two plasmids (131 and 51 kb) (http://www.pseudomonas-syringae.org/). With advances in DNA sequencing technologies, many more genome sequences are now available; for example, a recent study compared the genomes of 391 *P. syringae* strains [2]. The majority of *P. syringae* strains have a similar genome size of approximately 5–6 Mb [1] and contain a number of plasmids. A number of studies have compared and contrasted these genomes. For example, the gene gain and loss from 27 strains of 18 pathovars using whole-genome sequences of *P. syringae* has been investigated and it was found that the pan genome for all 27 lineages was 11 025 genes, with the core genome of 2595 genes [1].

PHYLOGENY

The *P. syringae* complex is currently divided into at least 13 phylogenetic groups (phylogroups), although this number is still debated. It is suggested that seven of these phylogroups can be considered 'primary' because they are monophyletic and distinct from the six 'secondary' phylogroups [1]. The features of these groups raise interesting questions about the biology and evolution of *P. syringae*. For example, *P. syringae* appears to undergo a moderately high rate of recombination over a large number of loci, both within and between phylogroups. However, a recent analysis of rates of recombination within and between phylogroups revealed a higher rate of recombination within primary phylogroups than between primary and secondary phylogroups [2]. Strains found in individual phylogroups do have common features, for example, strains in phylogroup 2 secrete a smaller number of effector proteins relative to phylogroups 1 and 3 and commonly produce a toxin known as syringomycin. Additional work needs to be done to define *P. syringae* as a species and to understand the processes that affect the evolution of *P. syringae* populations [1].

KEY FEATURES AND DISCOVERIES

P. syringae pathovars cause a range of diseases on a diverse array of plants, including diseases of annual crops, such as bacterial speck on tomato and halo blight on beans, as well as diseases of woody plants and trees, such as bleeding canker on horse-chestnut and a recent epidemic of kiwifruit canker. Symptoms include water-soaked lesions, chlorosis, blights and cankers. *P. syringae* colonizes a range of plant tissues, including leaves, seeds, seedlings, fruit and bark, and can persist in non-host environments [3].

Received 18 June 2018; Accepted 17 August 2018; Published 14 November 2018
Author affiliations: [1]Centre for Research in Bioscience, Faculty of Health and Applied Sciences, The University of the West of England, Frenchay Campus, Bristol BS16 1QY, UK; [2]Department of Plant Sciences, University of Oxford, Oxford OX1 3RB, UK.
***Correspondence:** Dawn L. Arnold, Dawn.Arnold@uwe.ac.uk
Keyword: *Pseudomonas syringae*.

Molecular genetic studies carried out on *P. syringae* strains have led to many significant discoveries regarding plant–pathogen interactions. For example, *P. syringae* pv. *phaseolicola* has been studied extensively and a number of discoveries have been made using it [4]. These included the first reports of mutants that lead to the loss of pathogenicity to bean and the ability to elicit the hypersensitive response. This work lead to the identification of hypersensitive reaction pathogenicity (*hrp*) genes, which were shown to form a bacterial type three secretion system (T3SS) that is used to deliver effector proteins into plant cells. Other areas where *P. syringae* has significantly contributed to the understanding of plant–pathogen interactions include the elucidation of race structures controlled by the interactions of effector genes in the bacteria and resistance genes in the host, the function of effector proteins in pathogenicity, and the evolution of pathogenicity. Recently, *P. syringae* has inspired new developments in synthetic biology, such as the development of 'genetic amplifiers' based on its T3SS regulatory system [5].

Many studies have been carried out on a selected group of *P. syringae* pathovars that infect a limited group of plants. However, more recently attention has turned to *P. syringae* in the wider environment. Work by Morris *et al.* [6] investigating the bacterium's ecology has found *P. syringae* in diverse environments, including rain and snowfall.

There is an increasing understanding of the role that reservoirs of populations of *P. syringae* play in disease outbreaks. An example of this is the recent epidemic of *P. syringae* pv. *actinidiae*, which had a devastating effect of the New Zealand kiwifruit industry. It has now been shown by isolating *Pseudomonas* from cultivated and wild kiwifruit across six provinces in China, that China was the likely origin of the pandemic lineage that was introduced into New Zealand [7].

OPEN QUESTIONS

- To what degree and how does *P. syringae* manipulate host plants to create a microenvironment that is conducive to bacterial growth?
- What is the frequency and significance of co-infections by multiple strains of *P. syringae* or by *P. syringae* and other micro-organisms?
- How do plant resistance mechanisms act to restrict the growth of *P. syringae*?
- Can we predict and prolong the durability of resistance?
- Where do outbreaks originate from? Can we predict the emergence of new epidemics?

Funding information
D. A. and G. P. acknowledge support from the Biotechnology and Biological Sciences Research Council, United Kingdom.

Conflicts of interest
The authors declare that there are no conflicts of interest.

References

1. Baltrus DA, McCann HC, Guttman DS. Evolution, genomics and epidemiology of *Pseudomonas syringae*: challenges in bacterial molecular plant pathology. *Mol Plant Pathol* 2017;18:152–168.
2. Dillion MM, Thakur S, Almeida RND, Guttman DS. Recombination of ecologically and evolutionarily significant loci maintains genetic cohesion in the *Pseudomonas syringae* species complex. *bioRxiv*. doi: doi.org/10.1101/227413.
3. Preston GM. Profiling the extended phenotype of plant pathogens: challenges in bacterial molecular plant pathology. *Mol Plant Pathol* 2017;18:443–456.
4. Arnold DL, Lovell HC, Jackson RW, Mansfield JW. *Pseudomonas syringae* pv. *phaseolicola*: from 'has bean' to supermodel. *Mol Plant Pathol* 2011;12:617–627.
5. Wang B, Barahona M, Buck M. Engineering modular and tunable genetic amplifiers for scaling transcriptional signals in cascaded gene networks. *Nucleic Acids Res* 2014;42:9484–9492.
6. Morris CE, Monteil CL, Berge O. The life history of *Pseudomonas syringae*: linking agriculture to earth system processes. *Annu Rev Phytopathol* 2013;51:85–104.
7. McCann HC, Li L, Liu Y, Li D, Pan H *et al.* Origin and evolution of the kiwifruit canker pandemic. *Genome Biol Evol* 2017;9:932–944.

Edited by: D. J. Kelly