

Pseudomonas syringae pathovars

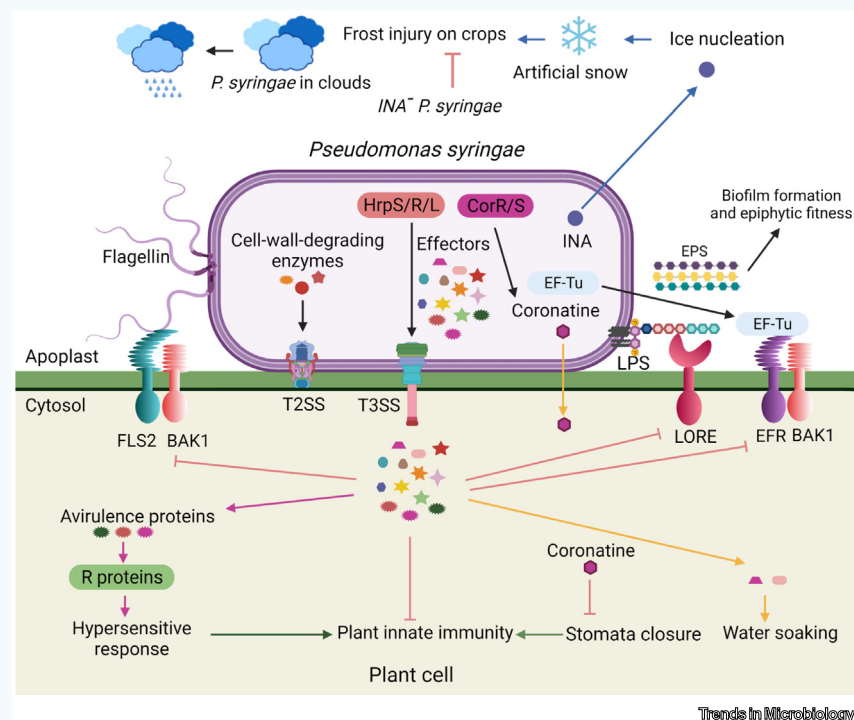
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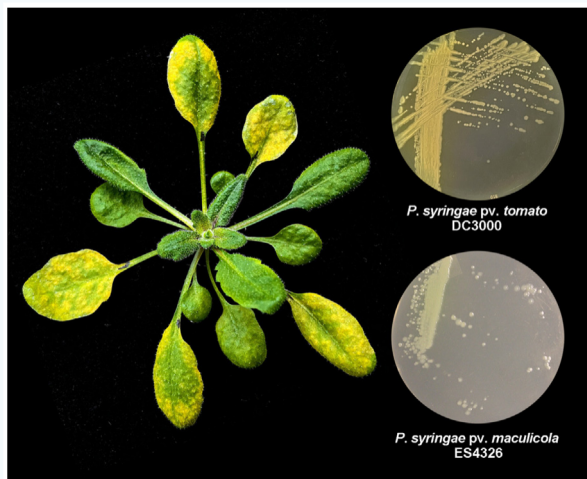
KEY FACTS:

P. syringae was first isolated from the lilac tree (*Syringa vulgaris*). Due to its production of the siderophore pyoverdinin on King's B medium, *P. syringae* has a yellow fluorescent appearance.

The complete genome sequence of the first *P. syringae*, *Pseudomonas syringae* pv. tomato (Pst) DC3000, was published in 2003. The genome size of Pst DC3000, which has a circular chromosome and two plasmids, is about 6.5 Mb.

Type III effectors (T3Es or Hops) that are delivered into host plant cells by the T3SS are required for *P. syringae* to cause diseases. The two major functions of T3Es are suppression of plant innate immunity and creation of an aqueous apoplastic space for pathogen growth. HopM1 and AvrE, two major T3Es in the Conserved Effector Locus (CEL) of the pathogenicity island, have been identified as the major causes for the water-soaking symptom during the infection.

Pseudomonas syringae is one of the best studied plant pathogens. *P. syringae* pathovars exhibit unique host specificities by infecting different kinds of plants, causing huge economic losses. Studies on the molecular interactions between *P. syringae* pathovars and plants have made huge contributions towards our understanding of the plant immune system and microbial pathogenicity. Flagellin, lipopolysaccharide (LPS), extracellular polysaccharides (EPS) and EF-Tu from *P. syringae* are recognized by their receptors/coreceptors FLS2-BAK1, LORE, and EFR-BAK1, respectively, to activate immunity triggered by pathogen-associated molecular patterns (PAMPs). *P. syringae* relies on cell-wall-degrading enzymes secreted through the type II secretion system (T2SS), type III effectors delivered into plant cells by the type III secretion system (T3SS), and toxins, such as coronatine, to cause diseases. Plants have developed resistance (R) proteins to recognize some of these effectors or avirulence proteins, inducing the hypersensitive response. Ice-nucleation-active (INA) proteins from *P. syringae*, which can initiate ice formation, have been used to produce artificial snow.



TAXONOMY AND CLASSIFICATION:

KINGDOM: Bacteria
 PHYLUM: Proteobacteria
 CLASS: Gammaproteobacteria
 ORDER: Pseudomonadales
 FAMILY: Pseudomonadaceae
 GENUS: *Pseudomonas*
 SPECIES: *Pseudomonas syringae*
 Gram-negative, yellow, fluorescent

Several phytotoxins, including coronatine, syringolin A, syringomycin, syringopeptin, phaseolotoxin, and tabtoxin, have been found in different *P. syringae* strains. The best characterized *P. syringae* toxin is coronatine, which mimics the active form of plant hormone jasmonic acid, jasmonoyl-isoleucine (JA-Ile). Coronatine antagonizes the function of salicylic acid in inducing stomata closure, triggering the reopening of stomata to allow pathogen invasion.

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Declaration of interests

No interests are declared.

Literature

1. Buell, C.R. et al. (2003) The complete genome sequence of the Arabidopsis and tomato pathogen *Pseudomonas syringae* pv. tomato DC3000. *Proc. Natl. Acad. Sci. U. S. A.* 100, 10181–10186
2. Patel, H.K. et al. (2014) The kiwifruit emerging pathogen *Pseudomonas syringae* pv. actinidiae does not produce AHLs but possesses three luxR solos. *PLoS One* 9, e87862
3. Melotto, M. et al. (2006) Plant stomata function in innate immunity against bacterial invasion. *Cell* 126, 969–980
4. Bent, A.F. et al. (1994) RPS2 of Arabidopsis thaliana: a leucine-rich repeat class of plant disease resistance genes. *Science* 265, 1856–1860
5. Mackey, D. et al. (2002) RIN4 interacts with *Pseudomonas syringae* type III effector molecules and is required for RPM1-mediated resistance in Arabidopsis. *Cell* 108, 743–754
6. Axtell, M.J. and Staskawicz, B.J. (2003) Initiation of RPS2-specified disease resistance in Arabidopsis is coupled to the AvrRpt2-directed elimination of RIN4. *Cell* 112, 369–377
7. Maki, L.R. et al. (1974) Ice nucleation induced by *Pseudomonas syringae*. *Appl. Microbiol.* 28, 456–459
8. Xin, X.F. et al. (2018) *Pseudomonas syringae*: what it takes to be a pathogen. *Nat. Rev. Microbiol.* 16, 316–328
9. Bender, C.L. et al. (1999) *Pseudomonas syringae* phytotoxins: mode of action, regulation, and biosynthesis by peptide and polyketide synthetases. *Microbiol. Mol. Biol. Rev.* 63, 266–292

Many strains of *P. syringae*, but not Pst DC3000, harbor ice-nucleation (INA) genes, which encode INA proteins. Once INA proteins translocate to the surface of the bacteria they can function as nuclei for ice formation. Therefore, the INA proteins have been used to make artificial snow. Conversely, *P. syringae* INA minus (INA[−]) mutants have been released into fields to protect crops from frost damage. It has been postulated that *P. syringae* plays a role in the earth's water cycle because it is frequently found in precipitates and in the center of hailstones.

DISEASE FACTS:

To date, over 50 pathovars of *P. syringae* have been discovered. *P. syringae* can cause diseases in many economically important crops, resulting in billions of dollars loss annually. For example, bacterial canker disease, caused by *P. syringae* pv. actinidiae, poses serious threats to the kiwifruit industry, leading to huge economic losses.

Pst DC3000 not only infects the economically important crop tomato, it also causes disease in the model plant *A. thaliana*. Both Pst DC3000 and *P. syringae* pv. maculicola (Psm) strain ES4326 are frequently used to study the molecular interactions between *P. syringae* and *A. thaliana*. Many milestone discoveries in molecular plant–pathogen interactions were made using the *P. syringae*–*A. thaliana* pathosystem.

Besides Pst DC3000 and Psm 4326, *P. syringae* pv. *syringae* (Pss) B728a, which causes brown spot disease on bean, serves as a model to study epiphytic fitness, while *P. syringae* pv. *phaseolicola* (Psp) 1448A, which is a causal agent of halo blight on bean, has been involved in the study of avr genes and non-host resistance.