Pseudomonas syringae pv. actinidiae: the bacterial canker of kiwifruit

Costa G.¹ and Spinelli F.¹

¹: Università di Bologna, Dipartimento di Colture Arboree, viale Fanin 46 - 40127, Bologna, Italy
2: Plant & Food Research Ruakura East Street, Hamilton, 3214, New Zealand
3: Università di Bologna, Dipartimento di Morfofisiologia Veterinaria e Produzioni Animali, Via Tolara di Sopra, 50 Ozzano Dell’Emilia, Bologna, Italy

E-mail: spinelli@agrsci.unibo.it
History of the disease

• 1984 - Takikawa first isolation on Hayward plants in Japan
• 1989 - First taxonomical classification as a new subspecies *Pseudomonas syringae* pv *actinidiae*
• 1992 - First outbreak in Korea
• 1992 - First isolation in Italy (Latina area)
• 1992 - 2008 Serious outbreak in Italy
• 2008 - First record of infection on *A. chinensis*
• 2009 - Spread of the disease to the major growing areas in Italy and serious economical damages
• 2010 - First occurrence in New Zealand
Kiwifruits are economically important crops which are grown in several EUcountries (by order of importance in production: Italy, Greece, France, Portugal and Spain). In Japan and Korea, bacterial canker has become one of the most serious limiting factors for cultivating kiwifruit. In Italy, it is estimated that the economic due to P. syringae pv. actinidiae have reached 2,000,000 €.
Diffusion in Italy
Unsolved questions

- Epidemiology: how does Psa infect the plant?
- Epidemiology: how does the disease spread?
- Epidemiology: how do the environmental factors affect the disease development?
- Plant-pathogen interactions: which is the bacterial arsenal to hijack plant defences?
- Plant-pathogen interactions: how does the plant react to Psa?
- How can Psa be diagnosed?
- How can the disease be monitored?
- How can the diffusion of Psa be prevented?
- How can the disease be controlled?
Italian Projects


2012-PSA - Contenimento della batteriosi dell'actinidia (Pseudomonas syringae pv. Actinidiae)

2012-13 CRPV Progetto di ricerca sul cancro batterico dell'actinidia causato da Pseudomonas syringae pv. actinidiae (PSA)
Unsolved questions

• Epidemiology: how does Psa infect the plant?
• Epidemiology: how does the disease spread?
• Epidemiology: how do the environmental factors affect the disease development?
• Plant-pathogen interactions: what is the bacterial arsenal to hijack plant defences?
• Plant-pathogen interactions: how does the plant react to Psa?
• How can Psa be diagnosed?
• How can the disease be monitored?
• How can the diffusion of Psa be prevented?
• How can the disease be controlled?
Epidemiology

- STOMATA
- BROKEN TRICOMES
- WIND and HAIL DAMAGES
The role of trichomes
The role of stomata
Leaf spots
Lenticels
Lenticels
Dry Lenticels: $4 \times 10^8$ cfu ml$^{-1}$
Epidemiology

- STOMATA
- BROKEN THRICOMES
- WIND and HAIL DAMAGE

... ABSCISSION LESIONS:
  - LEAF SCAR
  - FLOWER SCARS
Bud Break

Bacterial Population (cfu ml$^{-1}$)

- Swollen:
  - Outside: $1.0 \times 10^2$
  - Inside: $1.0 \times 10^1$

- Opened:
  - Outside: $1.0 \times 10^3$
  - Inside: $1.0 \times 10^2$

- Opened with shoot:
  - Outside: $1.0 \times 10^4$
  - Inside: $1.0 \times 10^3$
Epidemiology

- STOMATA
- BROKEN THRICOMES
- WIND and HAIL DAMAGES

....

ABSCISSION LESIONS:
- LEAF SCAR
- FLOWER SCARS

FEMALE FLOWERS
Female flowers

Stigma
How does it moves on the stigma
Simultaneous monitoring of Psa and BCAs
How does it moves on the stigma?
Tracheids inside the stigmatic pedicel
Can PSA invade the plant *via* flowers?

**Hayward**

**Hort16A**
Can PSA invade the plant via flowers?
Can PSA invade the plant *via* flowers?
Epidemiology

- STOMATA
- BROKEN THRICOMES
- WIND and HAIL DAMAGES

ABSCISSION LESIONS:
- LEAF SCAR
- FLOWER SCARS

FEMALE FLOWERS

MALE FLOWERS
Male flowers
Can pollen infect flowers?

![Bar chart showing Psa population (Log cfu/flower part) for different parts of a flower: Stigma, Flower stalk, and Ovarium.](chart.png)
How does Psa move in the plant?
How does Psa move in the plant?

Movement inside xylem vessels
How does Psa move in the plant?
How to exploits this information and protocol

- Target control strategies on the different entry points
- Facilitate the breeding for resistance
- Screen products (i.e. elicitors)
- Elucidate the influence of environmental factors and cultural management
Psa arsenal to hijack plant defences

_Pseudomonas syringae pv. actinidiae_ Draft Genomes Comparison Reveal Strain-Specific Features Involved in Adaptation and Virulence to _Actinidia_ Species

Simone Marcelletti¹, Patrizia Ferrante¹, Milena Petriccione², Giuseppe Ferrao³, Marco Scorciachini¹*

¹ Research Centre for Fruit Trees, CRA, Roma, Italy, ² Research Unit for Fruit Trees, CRA, Caserta, Italy, ³ Department of Agricultural and Environmental Sciences, University of Udine, Udine, Italy

Abstract

A recent re-emerging bacterial canker disease incited by _Pseudomonas syringae pv. actinidiae_ (Psa) is causing severe economic losses to _Actinidia chinensis_ and _A. delicosa_ cultivations in southern Europe, New Zealand, Chile and South Korea. Little is known about the genetic features of this pathovar. We generated genome-wide illumina sequence data from two Psa strains causing outbreaks of bacterial canker on the _A. delicosa_ cv. Hayward in Japan (I-Psa, type-strain of the pathovar) and in Italy (I-Psa) in 1984 and 1992, respectively as well as from a _Psa_ strain (12-Psa) isolated at the beginning of the recent epidemic on _A. chinensis_ cv. Hort16A in Italy. All strains were isolated from typical leaf spot symptoms. The phylogenetic relationships revealed that _Psa_ is more closely related to _P. s. pv. theae_ than to _P. avellanae_ within genomospesies 8. Comparative genomic analyses revealed both relevant intrapathovar variations and putative pathovar-specific genomic regions in _Psa_. The genomic sequences of J-Psa and I-Psa were very similar. Conversely, the I2-Psa genome encodes four additional effector protein genes, lacks a 50 kb plasmid and the phaseolotoxin gene cluster, argK-tox but has acquired a 160 kb plasmid and putative prophage sequences. Several lines of evidence from the analysis of the genome sequences support the hypothesis that this strain did not evolve from the _Psa_ population that caused the epidemics in 1984–1992 and Italy but rather is the product of a recent independent evolution of the pathovar _actinidiae_ for infecting _Actinidia_ spp. All _Psa_ strains share the genetic potential for copper resistance, antibiotic detoxification, high affinity iron acquisition and
Psa arsenal to hijack plant defences

1. genes involved in the catabolism of aromatic plant compound S with possible antimicrobial effects
2. Genes interfering with NO metabolism
3. copA and copB genes involved in copper resistance
4. Psa induce ET emission in Actinidia spp. and unbalance the defence signalling pathways
5. Psa may induce stomata opening in A. chinensis

![Graph showing gas emission and stomatal conductance over time after infection](image-url)
Molecular diagnosis on Pollen, Leaves and shoots

LITERATURE:

Detection of Pseudomonas syringae pv. actinidiae, causal agent of bacterial canker of kiwifruit, from symptomless fruits and twigs, and from pollen

ANGELA GALLELLI, SIlVIA TALOCCI, ALESSIA L’AURORA and STEFANIA LORETTI
C.R.A. - Centro di Ricerca per la Patologia Vegetale, Via C.G. Bertero, 22, 00156 Roma, Italy
Near-infrared and other important wavelengths
Portable DA-meter

UNIBO
Remote sensing

Sistema di telerilevamento ASPIS

Università della Tuscia.
G.M. Balestra