Pseudomonas syringae pv. *actinidiae* Takikdawa *et al.* 1989

Fig. 1. Drop of white exudate from an infected kiwifruit shoot (a); production of red exudate from an infected cane (b); an infected shoot wilting and dying in early summer (c); and small angular necrotic leaf spots caused by *Pseudomonas syringae* pv *actinidiae* (d). Photo credits J. L. Vanneste.

Disease: Bacterial canker of kiwifruit
Classification: D: Bacteria, C: Gammaproteobacteria, O: Pseudomonadales, F: Pseudomonadaceae

Bacterial canker of kiwifruit (Fig. 1), caused by the bacterium *Pseudomonas syringae* pv. *actinidiae* (Psa), affects different species of *Actinidia* including *A. deliciosa* and *A. chinensis*, the two species that constitute the majority of kiwifruit (green and yellow-fleshed) grown commercially around the world. This pathogen was first described in Japan in 1984. It was subsequently isolated in Korea and in Italy. Its economical impact can be significant, as is the case with the current outbreak in Latina (Italy).

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Distribution and Host Range:

This disease is present in Japan, Korea and Italy. Earlier reports of bacterial canker of kiwifruit from Iran and the USA refer to a different disease caused by the related pathogen *P. s.* pv. *syringae*. New Zealand and Australia are free of the disease.

Symptoms and Life Cycle:

Infections occur preferentially when the temperatures are relatively low, such as in spring and autumn.
In early spring the most visible symptom is the copious production of a red exudate from infected tissues. However, drops of white exudate, which contains the pathogenic bacteria, can also be found. This exudate allows the spread of the pathogen within and between orchards.
Buds on infected canes fail to develop or, if they do develop, they rapidly wilt and die. Later in the year, infected shoots wilt. The wilting starts at the extremity of the shoot and moves towards the parent cane. Leaves will first cup, wilt and then dry out. This probably reflects the plugging of the vascular system by the bacteria.
During summer the most visible symptoms are small angular necrotic spots on leaves, often surrounded by a yellow halo. This halo reveals production by the pathogen of phytotoxins such as phaseolotoxin or coronatine. However, not all strains of Psa produce those toxins.
During harvest and leaf fall, when environmental conditions are still warm and showery, the bacteria can move into these wounds and lay dormant until spring.

Control:

Typically with bacterial diseases, control is difficult and options are limited. Reducing inoculum through good orchard hygiene, and the prevention of infection during the suspected infection periods of spring and autumn, are the best recommendations available today. The risk of infection can be reduced by the use of copper containing products. However, overuse of copper can lead to strains that are copper-resistant. In Japan where the use of streptomycin is allowed for control of this pathogen, strains of Psa resistant to both copper and streptomycin have been found.

Further Reading:

Takikawa Y *et al*. (1989) *Annals of the Phytopathological Society of Japan* **55**, 437-444
Nakajima M *et al*. (2002) *Journal of General Plant Pathology* **68**, 68-74.
Rhees George J, Vanneste JL *et al*. *Plant Pathology* **59**, 453-464
Vanneste JL *et al* (2010) *New Zealand Plant Protection* **63**, In press.
http://www.batteriosi.it

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