Evaluation of the susceptibility of *Actinidia chinensis* var. *deliciosa* and *A. arguta* var. *arguta* to *Pseudomonas syringae* pv. *actinidiae*

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Introduction

Kiwifruit bacterial canker (KBC), caused by the bacterium *Pseudomonas syringae* pv. *actinidiae* (PSA), is currently the most destructive disease of kiwifruit plants. Conversely, *P. syringae* pv. *actinidifoliorum* (PFM), a genetically close bacterial strain, only causes necrotic spots in leaves, without leading to plant death.

Although there is some evidence on the role of several defence-related proteins on the genotypic variation to PSA susceptibility, how defence mechanisms are triggered in plants with different susceptibility to the pathogen is still unknown. This study **aimed** to identify metabolic and genetic responses triggered in two kiwifruit species with reported different susceptibility to bacterial canker (green kiwifruit – more susceptible, and kiwi berry – more tolerant) against a highly virulent (PSA) and a less virulent (PFM) bacterial strain.

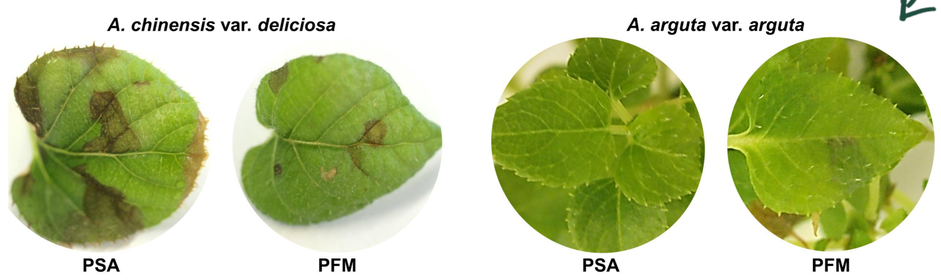
Methods

Micropropagated plants of *A. chinensis* var. *deliciosa* cv. 'Hayward' (green kiwifruit) and *A. arguta* var. *arguta* cv. 'Weiki' (kiwi berry) were inoculated with PSA or PFM, and disease development was monitored 1, 2, 7, 14 and 21 days post inoculation (dpi), through the evaluation of:

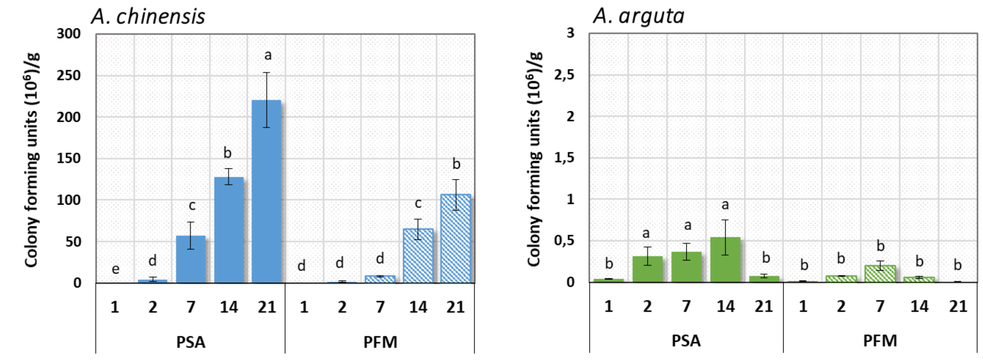
i. colony forming units in plant tissues (CFU),
ii. plant fitness (foliar symptoms, total chlorophylls and lipid peroxidation),
iii. expression of defence-related genes (*APX*, *SOD*, *CAT*, *LOX*, *SAM* and *Pto3*).

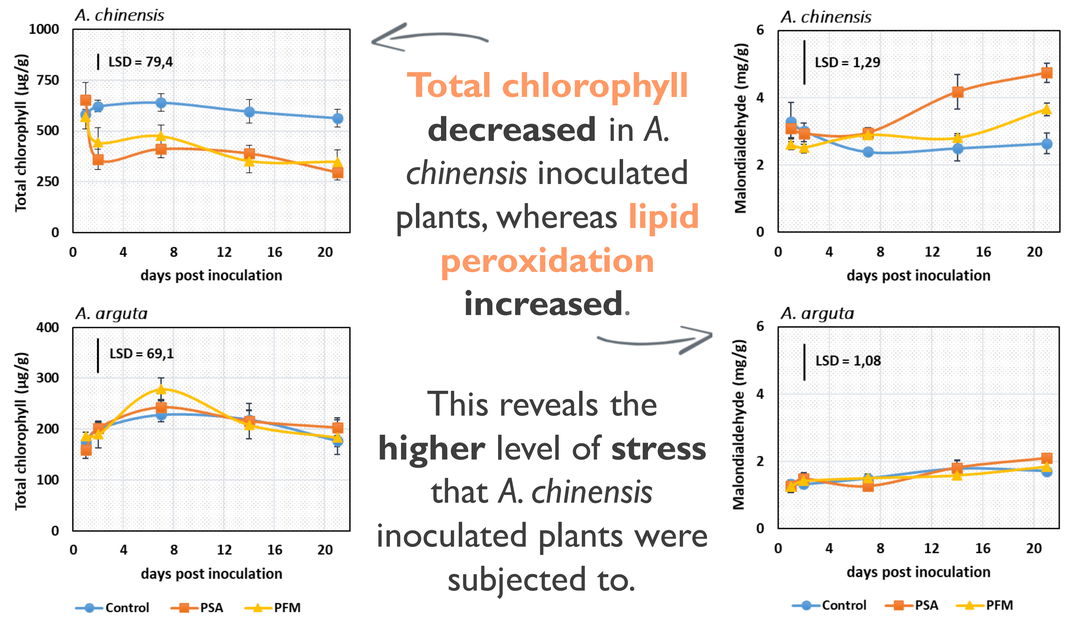
Results and Discussion

Disease symptoms appeared **sooner in *A. chinensis*** and progressed more extensively than in *A. arguta*. PSA induced symptoms were also more severe than with PFM.



In *A. chinensis* **colony forming units steadily increased** until the end of the experimental period, whereas in *A. arguta* bacterial density was **lower and**, decreased with time.





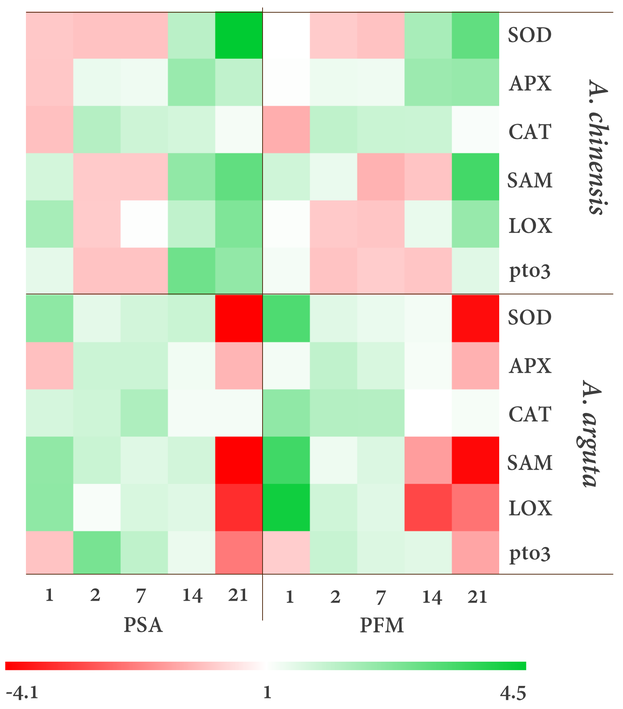
Total chlorophyll decreased in *A. chinensis* inoculated plants, whereas **lipid peroxidation increased**.

This reveals the **higher** level of **stress** that *A. chinensis* inoculated plants were subjected to.

Genes encoding antioxidant enzymes (*SOD, APX and CAT*) were upregulated sooner in *A. arguta*.

Genes involved in ethylene (*SAM*) and phenylpropanoids (*LOX*) pathways were downregulated in *A. arguta*.

Expression of gene *Pto3*, responsible for pathogen recognition, occurred 2 dpi in *A. arguta*, but only 14 dpi in *A. chinensis*.



Conclusions

Results confirm that *A. arguta* is more tolerant to PSA and PFM than *A. chinensis*, probably because it is able to recognise pathogen invasion and activate defence mechanisms from an earlier stage after infection. In addition, the more aggressive character of PSA, comparing with PFM, was demonstrated in controlled condition for the first time.

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