The role of JAZ proteins in plant defence and hormone crosstalk.

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I certify that all material in this thesis which is not my own work has been identified and that no material has previously been submitted and approved For the award degree for this or any other University.

ABSTRACT

Phytohormones have a crucial role in plant defence responses. The role of each specific phytohormone is contingent on whether the pathogen is a bio- or a necrotroph. In the case of the hemibiotrophic *Pseudomonas syringae* pv *tomato* (DC3000) and the model plant *Arabidopsis thaliana*, the interaction of the three key plant hormones, salicylic acid (SA), jasmonic acid (JA) and abscisic acid (ABA), ultimately dictates the outcome of the infection. Post-invasion defence responses, chiefly regulated by the SA dependent pathway are compromised by the antagonistic action of JA and ABA. It is this he complex crosstalk between the hormone pathways that propels susceptibility or resistance. The Jasmonate ZIM domain (JAZ) transcription repressors are crucial part of the JA signaling cascade. JAZs form a co-receptor with F-BOX Coronatine Insensitive (COI) 1 that binds the bioactive form of JA, Jasmonic Isoleucine (JA-Ile). Subsequent to this JA perception is the specific degradation of JAZ proteins that allows transcription of early JA-related genes. Interestingly the bacterial phytotoxin coronatine, which is a JA-Ile mimic, increases bacterial virulence by exploiting the JA signaling pathway. In this project we establish the DC3000 induced susceptibility of *jaz10* and *jaz5/10* loss of function mutants that substantiates JAZ specificity and confirms coronatine-induced virulence. In addition, we demonstrate the different manifestation of symptom development caused by absence of key positive regulators of plant defence, SA and JAZ5/JAZ10 and uncover the severely compromised phenotype of *jaz5/jaz10/aao3/sid2-1.* Finally we produce the tools that will allow further research of JAZ protein interactions.

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