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Crosstalk between salicylic acid and Jasmonate/ethylene signaling pathway in plant

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Abstract
Defense responses are an additional energy consuming step in plant development. Ideally, a plant recognizes distinct pathogens and employs specific pathways against them. Classical phytohormones, such as auxin, cytokinin (CK), brassinosteroid (BR), abscisic acid (ABA), salicylic acid (SA), ethylene (ET), jasmonate (JA) and the recently identified strigolactones (SLs), coordinate effective defense responses by activating complex network by defense gene expression. The SA-and JA/ET-mediated signaling pathways were thought to be the backbone of plant immune responses against biotic invaders. In general, the SA plays a central role in local and systemic-acquired resistance (SAR) against biotrophic pathogens, while JA/ET-mediated defense response contributes mainly against necrotrophic pathogens. Increasing evidence indicates that the SA- and JA/ ET-mediated defense response pathways are mutually antagonistic to each other.

Keywords: Defense, crosstalk, salicylic acid, JA/ET-Signalling, transcription factors

1. Introduction
Plants are sessile organisms, so they are not free to move under frequent attack of a broad spectrum of pathogens, including Insects, viruses, bacteria and fungi, in their habitat. On the basis of infection strategies, pathogens are classified as either biotrophic or necrotrophic pathogen. Biotrophic pathogens such as Pseudomonas syringae are host-specific (Glazebrook, 2005) [1]. They first penetrate into epidermal cells and multiply themselves in the intercellular spaces by feeding on living host tissue. Besides, necrotrophic pathogens produce toxic metabolites by which they kill host plant cells and then feed on the remains. It is demonstrated that ethylene and jasmonate play a major defense response against necrotrophs. The jasmonate- or ethylene-insensitive Arabidopsis mutants show enhanced susceptibility to the necrotrophic *Botrytis cinerea*.
Arabidopsis mutants are most commonly used to investigate the crosstalk of defence phytohormones, their biosynthesis and regulatory mechanisms governing defence against pathogens (Ádám *et al*., 2018; Gaffney *et al*., 1993; Wildermuth *et al*., 2001) [2–4]. Many intermediate signal molecules, such as pathogen-associated molecular patterns (PAMPs)-triggered immunity (PTI) and effector-triggered immunity (ETI), are activated by salicylic acid (SA). SA triggered defense response in Arabidopsis plants infested with biotrophic *P. syringae* and significantly compromised resistance against necrotrophic pathogen by suppression of the JA /ET signaling pathway (Thomma *et al*., 1999) [5].
The roles and models of classical phytohormones, such as auxin, cytokinin (CK), brassinosteroid (BR) and abscisic acid (ABA) have been comprehensively discussed in many reviews (Choudhary *et al*., 2012; Ha *et al*., 2012; Ku *et al*., 2018; Ton *et al*., 2009; Yu *et al*., 2018) [6–10]. Here, we discuss and explore the most up-to-date signalling crosstalk of phytohormones, with particular emphasis on transcriptional regulation.
Defense responses are an additional energy consuming step in plant development. Ideally, a plant recognizes distinct pathogens and employs specific pathways against them. These specific pathways interact with other signalling pathways and thus provide the potential energy allocation in plants. For instance, the SA- and ET/JA-mediated defense signalling pathways activated in response to pathogen infection act in both synergistically and antagonistically. It has been reported that SA and JA at low concentration showed synergetic expression of both SA target gene PR1 and JA target gene PDF1.2 whereas at higher concentration of SA and JA produces the antagonistic expression of these genes (Mur *et al*., 2006) [11].

2. SA Inhibits JA/ET-Signalling

Much attention has been paid to the antagonistic effect of SA on the JA/ET mediated pathways. Pseudomonas syringae, which is a biotrophic pathogen, induces SA pathway, repression of the JA/ET-pathway, leads to increased susceptibility to the Alternaria brassicae, which is a necrotrophic pathogen(Spoel *et al*., 2003) [12]. However, the molecular mechanism behind the antagonistic effect of SA repressing the JA/ET-pathway is largely still unclear. It is already reported that the of JA biosynthesis and repression of JA/ET-signaling pathways by SA is independent. Although many genes of JA biosynthesis such as LOX2, OPR3, AOS and AOC are repressed by SA, the exogenous application of SA represses PDF1.2 (JA-induces marker gene) expression to the same level in the AOS mutant as in the wild-type plants. It may be due to the repression by SA occurring downstream of JA perception (Leon-Reyes *et al*., 2010) [13].

3. Antagonistic effect of SA at the Gene Transcriptional level.

Recent studies suggest that SA represses the JA/ET-signalling pathway mainly at the transcription level. SA induces many negative regulators which interfere with some transcription factors such as the ERF branch which are regulated by JA/ET-pathways. Li and his colleagues reported that expression of SA-induced WRKY70 transcription factor suppresses the JA-induced PDF1.2 expression (Li *et al*., 2004, 2006) [14, 15]. It was suggested that the WRKY binding site in the promoters of SA repressed JA/ET-responsive gene was over-repressed. Thus, SA induced WRKY70 may bind to the promoter of JA/ET-responsive genes and inhibit their expression. However, in some wrky70 knock-out mutants SA was still able to repress JA-responsive marker genes. This result indicates that WRKY70 is only sufficient but not necessary for repression of the JA/ET pathway by SA. It is very important and demanding that issues with SA and JA/ET-pathways need to be further clarified to develop resistance in host plants.

4. TGA transcription factors and SA-JA/ET-signalling pathway

TGA transcription factors such as TGA2, TGA5 and TGA6 are positive regulators of SA mediated signalling pathways. However, class II TGA transcription factors have both positive and negative roles in the JA/ET signalling pathways. Zander *et al*. showed that in the young axenic-cultured triple Arabidopsis mutant the expression of PDF1.2 (which is a JA-induced marker gene) expression was blocked even after a combined treatment of ACC (an ET precursor) and JA (Zander *et al*., 2010) [16]. It was suggested that the expression of the PDF1.2 gene was not induced by either of ACC (an ET precursor) and JA. In myc2 mutant there was a hyper-induction of PDF1.2 expression after a combined ACC and JA treatment. In this case the expression of PDF1.2 was not repressed by SA treatment in the myc2 tga256 mutants. Similarly, expression of PDF1.2 was not repressed by SA treatment in tga2356 mutant (Leon-Reyes *et al*., 2010) [13].

Some ACC-responsive genes are dependent on TGA transcription factors. Half of the ACC-responsive genes, which are dependent on TGA transcription factor, are induced by ET and these ET-induced ACC responsive-TGA dependent genes are targeted by SA in the SA-JA/ET crosstalk (Zander *et al*., 2014) [17]. Zander *et al*. said that TGA transcription factors are mainly targeting the ERF-branch which is regulated by JA/ET-pathways. TGA transcription factor regulates JA/ET pathway by binding directly to the promoter of ORA59, which is a master regulator of the ERF-branch (Zander *et al*., 2014) [18]. The binding activity of TGA TF is enhanced by ET. Thus targeting TGA TFs may provide an essential regulatory mode to activation and SA-mediated repression of JA/ET-mediated pathways. Therefore, SA may regulate the transcriptional activity of the class II TGAs to manipulate the JA/ET-signaling pathway.

5. SA-induced glutaredoxin

The glutaredoxin (GRX) are small ubiquitous redox enzymes that are essential for maintaining a stable cellular redox state (Gutsche *et al*., 2015) [19]. It was shown that SA induces the expression of specific glutaredoxin (GRX) ROXY19 in Arabidopsis. ROXY19 interacts with the TGA factors and represses their expression as a result the expression of ORA59 and PDF1.2 was repressed (Gutsche *et al*., 2015) [18]. However, van der Does *et al*. reported that the expression of ORA59 protein was eliminated by SA application (Van der Does *et al*., 2013) [20]. These suggest that SA may induce degradation of transcription activator of ET/JA-signaling pathway by which SA represses ET/JA-signaling pathway.

The NPR1 has been known as an essential master regulator of SA-JA/ET crosstalk. NPR1 is essentially required for SA mediated induction of WRKY70 and ROXY19 expression, which repress the ET/JA-signaling pathway. NPR1 is present as an oligomer in cytosol. It must be switched from oligomer to monomer in order to translocate into nucleus. In nucleus NPR1 monomeric form induces SA mediated response. Surprisingly, translocation of NPR1 into the nucleus is not required for SA-JA/ET crosstalk. The expression of recombinant NPR1 protein that is retained into cytosol was sufficient to repress the expression of JA-induced PDF1.2 genes by SA application. Still the molecular mechanism of how NPR1 exerts its function in SA-JA/ET crosstalk is unknown.

6. JA Negatively Regulates SA Biosynthesis

JA represses the SA response antagonistically. COI1 and MYC are JA receptor and JA responsive transcription factors, respectively. Deletion of both COI1 and MYC results into increased accumulation of SA. Enhanced SA gives resistance to the biotrophic pathogens (Spoel & Dong, 2008) [21]. Pathogens are well known to adapt into adverse/ resistant host plant conditions due to high mutation rate (Fernández-Calvo *et al*., 2011; Laurie-Berry *et al*., 2006) [22, 23]. Pathogens can manipulate this SA-JA/ET crosstalk for their own benefits.

7. Perspectives: Developing Better Host Plant resistance

The biosynthesis, metabolism and signaling transduction of SA, JA and ET have been elucidated so far. But, do we really fully understand the actual signaling crosstalk of these defense phytohormones? Probably not. In nature, defense phytohormones work together in an ecological context to manage different invading pathogens(Berens *et al*., 2017; Robert-Seilaniantz *et al*., 2011; Verma *et al*., 2016) [24–26]. The mechanism behind the crosstalk of SA-JA/ET mediated pathways is poorly understood due to the high level of complexity and it requires further study. Interestingly, the SA receptors such as NPR3 and NPR4 have been shown to promote the degradation of JA transcriptional repressor JAZs

and activate the JA signalling. Both positive and negative regulatory factors of the signaling pathways might be the target for modulating defence hormonal crosstalk.

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