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Review

No hormone to rule them all: Interactions of plant hormones during the responses of plants to pathogens

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ABSTRACT

Plant hormones are essential regulators of plant growth and immunity. In the last few decades, a vast amount of information has been obtained detailing the role of different plant hormones in immunity, and how they work together to ultimately shape the outcomes of plant pathogen interactions. Here we provide an overview on the roles of the main classes of plant hormones in the regulation of plant immunity, highlighting their metabolic and signaling pathways and how plants and pathogens utilize these pathways to activate or suppress defence.

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1. Introduction

Plant hormones, also known as phytohormones, are naturally occurring small, organic molecules that are not only important for plant developmental processes, but also play an integral role as signaling molecules in defence and immune responses. Salicylic acid, jasmonic acid and ethylene are the traditional hormones associated with defence responses against pathogens, but in the past decade several pieces of evidence demonstrate that abscisic acid, gibberellic acid, cytokinin, auxin and brassinosteroids, typically associated with abiotic stress or developmental processes, are also key components of the immune response of plants. It is now clear that no single hormone controls plant immunity; rather, plant hormones tend to act interdependently, through complex antagonistic or synergistic interactions. The results of these interactions are changes in plant physiology that culminate in an appropriate defence response against pathogen attack, or in the case of successful pathogens, to changes that benefit the invading pathogenic organism. Biotrophic pathogens, or those that acquire nutrients from living cells, have different host physiological requirements than necrotrophic pathogens, which use toxins and cell wall degrading enzymes to cause cell death and obtain their nutrients from dead tissue. Not surprisingly, the host hormonal balance required for resistance to pathogens of different lifestyles is distinct, and pathogens have evolved several different strategies to shift this balance to their benefit.

In this review we discuss the role of the major classes of plant hormones in plant immunity, and whether they act as positive or negative regulators of defence responses. Given the vast literature on this topic, we focus mainly on examples of action of hormones in plant immunity on the model plant species Arabidopsis, while also citing hormone action in other plant species as possible and appropriate. To further contribute to the understanding of the roles of plant hormones in immunity, we also discuss hormone biosynthesis and signal transduction pathways, as well as their manipulation by pathogen effectors.

2. The master rings: key hormones in plant immunity

2.1. Salicylic acid

Salicylic acid (SA) is a phenolic compound with plant hormone activity, that is most recognized as an important endogenous signaling molecule in plant immunity. However, SA has also been documented to be indirectly involved in germination, flowering, mitochondrial electron transport and abiotic stress resistance, including thermotolerance [1–5]. The first indication that SA was associated with tolerance to biotic stress came from studies where application of SA to tobacco plants led to increased resistance against *TOBACCO MOSAIC VIRUS* (TMV) and increased accumulation of pathogenesis-related (PR) proteins [6]. This protective effect of SA was observed not only on tobacco, but on several other monocotyledonous and dicotyledonous plant species against a variety of biotrophic plant pathogens [7]. In addition, *in vitro* experiments demonstrated that this activity was due to plant-specific processes, rather than a direct killing activity of SA on pathogens [8]. SA levels were also found to accumulate at sites of pathogen infection, and a correlation was observed between SA accumulation and resistance to pathogenic attack. The similarity between the effects of SA application and pathogen attack on plant physiology led to the suggestion that SA was a signal for activation of defence against plant viruses [6]. These findings were later extended to other pathosystems and SA was determined a signal for defence to biotrophic pathogens in general [9].

SA is derived from the primary metabolite chorismate, by way of two major enzymatic pathways, one involving the phenylalanine ammonia lyase pathway, and another which involves a two-step process metabolized by the enzymes ISOCHORISMATE SYNTHASE (ICS), which converts chorismate to isochorismate, and ISOCHORISMATE PYRUVATE LYASE (IPL), which catalyzes the conversion of isochorismate into SA [10]. During the response to pathogens, plants preferentially employ the isochorismate pathway [11]. Once formed, SA accumulates both at the site of infection and systemically [12–14]. SA and/or a derivative of SA is typically required for innate immune responses (Pathogen-Associated Molecular Patterns (PAMP)-triggered immunity or PTI; Effector-Triggered Immunity or ETI) [15], localized resistance responses such as expression of *PATHOGENESIS-RELATED* (*PR*) genes and activation of programmed cell death, as well as for systemic acquired resistance (SAR), a form of broad-spectrum resistance to biotrophic pathogens that can act in both local and distal plant tissues [16,17]. In Arabidopsis, mutant plants lacking a functional ISOCHORISMATE SYNTHASE (ICS1) enzyme, *sid2*/*eds16*, fail to accumulate SA during pathogenic interactions, indicating that this enzyme is necessary for the majority of pathogen-induced SA biosynthesis [11,18]. While SA is biosynthesized in the chloroplasts, after biosynthesis most SA can be readily converted into a biologically inactive form, SA β-glucoside (SAG) [19], by a pathogen-inducible SA GLUCOSYLTRANSFERASE (SAGT) in the cytosol [20]. SAG biosynthesis is followed by transport to the vacuole [20–22], where it is stored until conversion back to biologically active SA [19]. SA can also be methylated into an inactive volatile form, methyl SA (MeSA), through the enzymes SA CARBOXYL METHYLTRANSFERASE (SAMT) and BENZOIC ACID/SA CARBOXYL METHYLTRANSFERASE (BSMT) [23,24].

The first studies to demonstrate the importance of SA in plant immunity used transgenic tobacco and Arabidopsis plants expressing the *nahG* transgene, encoding the bacterial SA-degrading enzyme salicylate hydroxylase [16,25]. *nahG* plants failed to accumulate SA and displayed increased susceptibility to biotrophic pathogens. Further, these plants failed to activate SAR, implicating SA accumulation in systemic resistance to pathogens [25]. Exogenous application of SA or SA analogues to *nahG* plants restored resistance both locally and systemically, as well as the expression of *PR-1*, a known marker of disease resistance to biotrophic pathogens [25]. In the early 1990′s, several genetic screens for Arabidopsis mutants impaired in SAR, showing increased susceptibility to pathogens or displaying altered responses to SA, led to the identification of different alleles of the *NON-EXPRESSOR OF PATHOGENESIS-RELATED GENES 1 (NPR1)*, now known to be a master regulator of SA-mediated defence responses [26–29]. *nahG* and *npr1* plants both showed increased susceptibility to biotrophic pathogens, including TMV, the oomycetes *Hyaloperonospora arabidopsidis* (*Hpa*, formerly *Peronospora parasitica*) and *Phytophthora parasitica*, as well as several bacterial pathogens such as *Pseudomonas syringae* pv. *tabaci* and *Pseudomonas syringae* pv. *maculicola* ES4326 [25–28], but decreased susceptibility to necrotrophic pathogens such as the fungi *Botrytis cinerea* and *Alternaria brassicicola* [30]. Collectively, these results established a model where SA is an important positive regulator of immunity to biotrophic pathogens, but a negative regulator of immunity to necrotrophic pathogens.

The identification of NPR1 was a first step in the elucidation of the SA signaling pathway. Cloning of the *NPR1* gene revealed that it encoded a protein with ankyrin repeats, as well as BTB/POZ repeats [31,32], domains known to mediate protein–protein interactions. Yeast two-hybrid screens identified proteins from the TGA family of bZIP transcription factors and the family of nuclear localized NIMIN1 proteins as NPR1-interacting proteins [33–37], implicating a function for NPR1 in the control of gene expression. Further stud-