Dynamic modeling resolves complex hormonal crosstalk in infected plants

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Abstract

Owing to their multi faceted interactions analysis of the combined output of plant hormones is always a challenge. Hormonal crosstalk plays a pivotal role in successful system protection or plant vulnerability. We developed a dynamical model and analysed the impact of individual plant hormones in an interactive way. We first established a network combining available interaction data and then used this molecular interaction network as a substrate for dynamic simulations on hormonal aspects of plant immunity. Our analysis revealed that plant hormones such as SA, GA and CK promote immunity against the infection of *Pst* DC3000 in *Arabidopsis*. On the other hand JA, Auxin and ABA promote vulnerability of *Arabidopsis*. These findings are in line with current literature, old and new experiments. Dynamic modelling can be applied to investigate antagonism and synergism between hormonal pathways in plants. It allows to study infections and host-pathogen interactions and in general the molecular events during organismic interactions.

1 Introduction

Plant hormones are shared weaponry in pathogen infections. Pathogenic attack causes hormonal imbalances in plants. Depending upon the trophic nature of pathogen, either SA (Salicylic Acid) or JA/ET (Jasmonic acid / Ethylene) mediated defense pathways are operative in plants [Gra9]. Antagonism between JA and SA and synergism between ET and JA has long been elucidated (reviewed by: [Rob11]. Furthermore, growth regulatory hormones such as auxin promote JA responses and suppress the SA pathway of resistance [Wan7]. Similarly, ABA (Abscisic Acid) antagonizes SA mediated defense signalling while SA abolishes ABA responses [Rob11]. On the contrary, GA (Gibberellic Acid) reinforces SA accumulation [Gra9]. It is worth mentioning that *Pst* DC3000 also promotes in-planta levels of auxin and ABA. Furthermore, it is injecting a JA mimicry to suppress SA mediated defense. Taking these pathways carefully into account, we established a plant immune defence network [Nas12] and performed dynamic simulations on various aspects of plant immunity. Here we highlight key connections in this network (Figure 1) and discuss the implications of various hormones in plant immune defence.

2 Results and Discussion

We analyzed the impact of phytohormones on immune defense using a Boolean model. Boolean network models have an advantage over ODE-based kinetic models in complex networks including immune and pathogen responses. In contrast to ODE models Boolean network models can also work when kinetic information is scarce and many nodes are involved [Sch11]. SQUAD (Standardized Qualitative Dynamical Systems; [Phi9]) is a powerful modelling package which combines Boolean and ODE models. It creates a system of exponential functions that allows interpolation between the step function of Boolean models according to the sum of activating and inhibitory input [Phi9]. To generate a network we integrated hormonal nodes with protein regulatory molecules in the cell according to the Boolean logic of their interactions (Figure 1; see [Nas12] for detailed Network topology). To model the impact of plant hormones on plant immunity, we performed SQUAD simulations by taking individual hormones as input activating nodes. The activation of PR-1 over time was used as an index of plant immunity (for detailed methodology see [Nas12]). We found in the simulations that the three hormones ET, SA and GA activate PR-1 (Figure 2 D, E, H). These hormones further enhance the signal of the activity of PR-1 in the presence of *Pst*, while JA, auxin and ABA diminish even the residual activity of PR-1 manifested by *Pst* alone (Figure 2 A). Contrary to the immunity promoting effect of SA and GA, we saw that auxin, JA and ABA mediate susceptibility of *Arabidopsis* against infection by *Pst* DC3000. Moreover, our modelling suggests a promoting role of cytokinin against infection by *Pst* DC3000 in *Arabidopsis* (Figure 2 G). These simulation results are in line with literature and own new experimental data and thus qualify analysis of Boolean models [Nas12] as a step forward to investigate plant regulatory and signalling networks. Boolean models specifically resolve here hormonal crosstalk during complex host-pathogen interactions in plants.

3 Conclusion

During host pathogen interactions in plants, *Pst* DC3000 uses effectors to modulate endogenous levels of phytohormones to mediate susceptibility of the *Arabidopsis* plant. Our simulations successfully predicted responses when hormone levels were changed and compared the uninfected plant to the plant infected with *Pst* DC3000. Modelling suggests that plant hormones such as SA and GA promote resistance against infection by *Pst* DC3000, while JA, auxin and ABA enhance susceptibility for infection of the plant. These predicted hormonal effects are similar to own and previous experimental results. Furthermore, our simulations suggested promoting effects of cytokinin on plant immunity against the infection by *Pst* DC3000 which again could be verified by experiment. Crosstalk of cytokinins as well as other hormones under different conditions in plants and other organisms can advantageously be explored with the methods of dynamic modelling outlined here.

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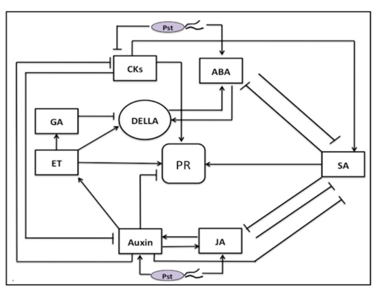

Figure 1: Logical connections in the plant pathogen hormone interaction network tested.
Infection with *Pst* DC3000 (shown with flagellae) in *Arabidopsis*. Connectivity among nodes is based either on activation (→) or inhibition (⊣).We give here only the most central backbone of interactions. For the detailed network topology modeled please see [Nas12]. PR-1 is a well-known key marker node for immunity against the infection of *Pst* in *Arabidopsis*.

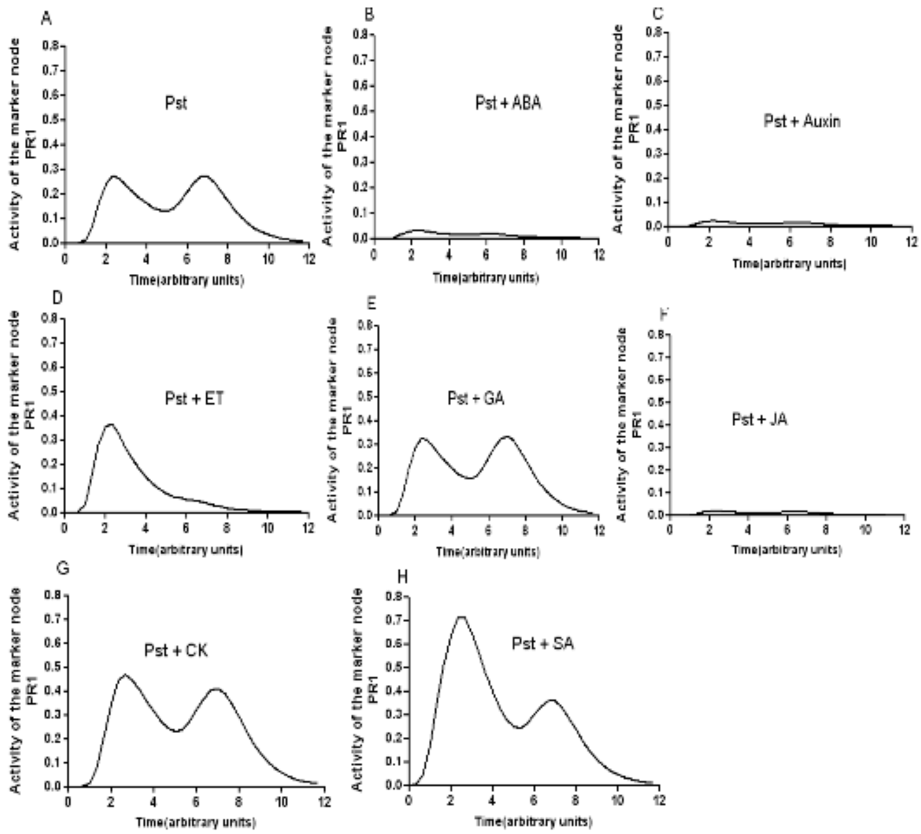

Figure 2: Modelling the impact of plant hormones on immune defense in *Arabidopsis* against by *Pst* DC3000.

Activity of PR-1 over arbitrary units of time (y-axis) is shown as immune output of the plant over time (x-axis). Activated nodes of *Pst* and plant hormones as activating input signal change the state of immunity in the host. Modelling of hormonal response for: A) Virulent *Pst* DC3000 infection and plant immune response B) *Pst* DC3000 infection after the application of plant hormone ABA C) auxin D) ethylene E) Gibberellic Acid F) Jasmonic Acid G) Cytokinin and H) Salicylic Acid. Experimental results qualitatively verified trajectories and shapes.