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A theoretical study on the cross-talk of stress regulatory pathways in root cells

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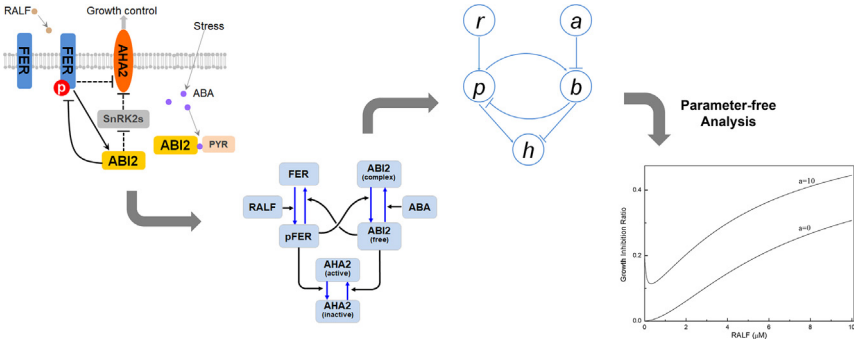
HIGHLIGHTS

- The cross-talk between two growth regulatory pathways in root cells introduces non-trivial response to stresses.
- An analytic parameter-free approach is introduced to investigate the response tendencies.
- A simple constraint on the non-linear regulatory exponents leads to the non-monotonic growth inhibition.

GRAPHICAL ABSTRACT



ABSTRACT

The plants developed more dedicated regulatory pathways than the animals did to response various environment stresses, since they could not run away. The cross-talk among the pathways generally introduce non-trivial regulatory behaviors, from which the plants may benefit. For better understanding the regulatory mechanism due to cross-talk, we study in this work two entangled stress regulatory pathways in root cells. A quantitative model of the regulatory network is constructed in the simplest fashion. An analytic parameter-free approach is then employed to analyse the response tendencies. It leads us to a simple constraint on the non-linear regulatory exponents. Under the constraint, a transition to the non-monotonic growth inhibition happens at finite concentration of ABA, due to which the plants could survive from cold/heat stress. The parameter-free tendency analysis would also be applied to further experiments, especially in the case of insufficient data for multi-parameter fitting.

1. Introduction

A plant must accurately perceive and respond to the constantly changing environment in order to survive and complete its life cycle. At the molecular level, each environmental signal can be a physical or chemical factor detected or sensed by a plant cell through a network of proteins and small molecules working together to translate a 'signal' into a 'cellular response'. This process, often referred to as signal transduction, entails specific sensors and sensing components corresponding to distinct signals [1–10].

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The phytohormone abscisic acid (ABA) relevant signaling pathway plays a central role in the whole stress-answering network of plant [11–16]. The experiments reported the ABA relevant pathway induces hypersensitive response to the cold/heat stress, which frequently lead to plant injury or even death [17]. The cross talk from other plant hormones, such as rapid alkalinization factor (RALF), save the hypersensitivity by modulating the response [17,18]. RALF itself is also a growth inhibitor in the root cells [19–21]. A question arises that how the cross-talk between two growth inhibitors, ABA and RALF, could lead to moderate response. This is a typical question on the non-trivival regulatory behaviors due to cross-talk among pathways.

It is commonly realized that the plants utilize the cross-talk between different signaling pathways for sensitive and moderate stress-answering [22]. For example, the interaction between signaling pathways of sugar and ABA is crucial to the regulation of sugar metabolism in plants [23–25]. The cross-talk between ABA and ethylene pathways leads to interesting behavior on stomatal closure in guard cells [26]. The cross-talks among auxin and other phytohormones have important impacts on the regulation of lateral root development in *Arabidopsis thaliana* [27–31]. There have been several theoretical studies on the cross-talks among regulatory pathways, which based on simulations of the dynamics of the complicated networks [2, 3, 32–34]. There is, however, still not a clear understanding of the mechanism behind the moderated responses due to two interacting inhibitors, such as ABA and RALF. In this work, we perform a theoretical study on the ABA and RALF relevant pathways. An analytic parameter-free approach based on the topological structure of the network is employed. A constraint on the non-linear regulatory exponents is discovered. Under this constraint, a transition to the moderated response happens when both ABA and RALF are present.

The paper is organized as follows. In Sec. 2, we introduce the model of the regulatory network. In Sec. (1), we analyse the model by the parameter-free approach and show the results. A short conclusion is presented in Sec. (2) follows.

2. The model for the regulatory network

2.1. The two growth inhibition pathways and the cross-talk between them

The plant hormones ABA and RALF are well known for the growth inhibition on root cells, which are mediated by ABI2 in cytoplasm and FER on membrane respectively [35, 19, 16]. Recent biology experiment discovered the cross-talk between the two pathways, due to the interaction between ABI2 and pFER [36,37,17]. Fig. 1(A) shows the molecular mechanism of the two regulatory pathways and also the bridges between them. The hormone ABA is mediated by the ABI2 [11–16]. The free ABI2 in cell inhibits SnRK2s [38–41], while SnRK2s suppresses cell growth via AHA2 [42]. ABA works as a linker between free ABI2 and Pyrabactin (Pyr). The ABI2 captured by ABA and Pyr can no more control SnRK2s. As a consequence, ABA inhibits the cell growth by suppressing the free ABI2 in cell. In the other pathway, the hormone RALF is mediated by the membrane receptor FER [43]. Once bound by RALF, FER can be phosphorylated to pFER, which suppresses cell growth via AHA2 [19, 44]. The interaction between the pathways is confirmed by experiment that pFER can promote the level of the free ABI2 and the free ABI2 dephosphorylates pFER [36, 45, 46, 37]. Current experiment, however, can not crystalize the complex interactions among AHA2, pFER, SnRK2s, and the free ABI2, which may be mediated by unknown agents and hence are marked by dash lines in Fig. 1(A).

By controlling the level of ABA and RALF, the plant experiment measured the length of roots, which is translated into growth inhibition rate. As expected, solely applying single hormone increases the growth inhibition rate. It is unexpected that the experiment reported decreasing growth inhibition rate for introducing RALF in the system with presence of ABA, as shown by the scatter symbols in Fig. 2 [17]. It is somehow puzzling that the additional growth inhibitor (RALF) stimulates the cell growth. A simple reasoning arises by noting the interaction between pFER and free ABI2. pFER changes the balance between free ABI2 and ABI2-ABA-Pyr complex. The promoted free ABI2 then stimulate the cell growth by suppressing SnRK2s. We are more confident with this story when we learn from the biologists that in the absence of ABA, all the ABI2 are free of Pyrabactin. The pFER-ABI2 promotion is hence saturated in this case. It explains why the pFER-ABI2 pathway seems not working in absence of ABA [17].

2.2. The simplest quantitative model

To clarify the above insights on the cross-talk between the two pathways, we construct a simplest model of the regulatory network, as shown in Fig.1(B). To avoid unnecessary complexity, the following assumptions are made:

(1) The total concentrations of ABI2, FER and AHA2 are constant, while each of them switches in two states, i.e.

$$\begin{aligned} [\mathrm{FER}] + [\mathrm{pFER}] &= C_{\mathrm{FER}}, \\ [\mathrm{ABI2_{free}}] + [\mathrm{ABI2_{complex}}] &= C_{\mathrm{ABI2}}, \\ [\mathrm{AHA2_{inactive}}] + [\mathrm{AHA2_{active}}] &= C_{\mathrm{AHA2}}. \end{aligned} \tag{1}$$

(2) The phosphorylation of FER is promoted by [RALF], which is an external input. Taking the other phosphorylase into consideration, the phosphorylating rate can be modelled as $k_0 + k_1[\mathrm{RALF}]$. The reaction flux is given by

$$J_{\mathrm{FER}\to\mathrm{pFER}} = (k_0 + k_1[\mathrm{RALF}])[\mathrm{FER}] \tag{2}$$

(3) The dephosphorylation of pFER is promoted by ABI2(free), while other dephosphorylase may also help the reaction. It gives

$$J_{\mathrm{pFER}\to\mathrm{FER}} = (k_7 + k_2[\mathrm{ABI2_{free}}])[\mathrm{pFER}] \tag{3}$$

(4) In cellular condition, Pyrabactin are abundant for the formation of ABI2-ABA-Pyr complex. Meanwhile, [ABA] is an external input, which can be assumed as a constant. The formation rate of ABI2(complex) is modelled as k_4[ABA]. The reaction flux is then given by

$$J_{\mathrm{ABI2_{free}}\to\mathrm{ABI2_{complex}}} = k_4[\mathrm{ABA}][\mathrm{ABI2_{free}}] \tag{4}$$

(5) The reaction from ABI2(complex) to ABI2(free) is mediated by pFER via a rate k_3[pFER], which gives

$$J_{\mathrm{ABI2_{complex}}\to\mathrm{ABI2_{free}}} = k_3[\mathrm{pFER}][\mathrm{ABI2_{complex}}] \tag{5}$$

(6) The active/inactive switch of AHA2 are affected by pFER and ABI2(free). It is already known that several intermediate molecules are involved to mediate the effects(17; 19), while the explicit regulatory mechanism is still not clear. We will show later the linear regulation assumption would be oversimplified. Losing no generality, we model the switching rate in a non-linear fashion as

$$\begin{aligned} J_{\mathrm{AHA2_{active}}\to\mathrm{AHA2_{inactive}}} &= k_5[\mathrm{pFER}]^m[\mathrm{AHA2_{active}}], \\ J_{\mathrm{AHA2_{inactive}}\to\mathrm{AHA2_{active}}} &= k_6[\mathrm{ABI2_{free}}]^n[\mathrm{AHA2_{inactive}}], \end{aligned} \tag{6}$$

where m and n is the effective exponent for the non-linear regulation [47].

Noting that the signals are transmitted in the regulatory network via the concentration of the substrates, for the sake of the regulatory sensitivity, the concentration should not be saturated in the reaction [48, 49]. Bearing this in mind, we adopt in the above formula the mass-action law, which is a well approximation of the full Michaelis-Menten kinetics in the condition of abundant enzyme and unsaturated substrate [50, 51, 52, 53].