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Review

Chloroplast calcium and ROS signaling networks potentially facilitate the primed state for stomatal closure under multiple stresses

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ABSTRACT

Plant signaling networks have evolved adaptive mechanisms that enable the rapid response of stomata to the multiple abiotic and biotic stresses. These signaling networks are not simply chaotic but constrained and spread throughout the cellular compartments to achieve the Ca^{2+}-sensitivity priming of guard cells. The irreplaceable role of the chloroplast as a photosynthetic apparatus in higher plant is understood; however, the contribution of chloroplast signaling dynamics to Ca^{2+}-sensitivity priming is sometimes ignored. As a key booster of Ca^{2+}-sensitivity priming, the primed state of guard cells is potentially related to chloroplast Ca^{2+} and reactive oxygen species (ROS) signaling. This review will discuss the generation of chloroplast Ca^{2+} and ROS signaling during stomatal closure under various stimuli. Here, we provide a model demonstrating the involvement of chloroplast Ca^{2+} and the ROS signaling pathway in facilitating the primed state when plants sense a stressful environment, while addressing the roles of phospholipase D, phosphatidic acid, stromal acidification and PQ pool in the signaling networks. In addition, we summarize the recent studies on the mutants of the plant calcium sensing receptor, which could further support and develop our model.

Contents

1. Introduction

When plants are exposed to unfavorable environmental conditions, stomatal movement is one of the immediate plant responses. To facilitate stomatal movement, a pairs of guard cells that comprise the stomata have evolved robust mechanisms to sense and respond to various endogenous and environmental

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stimuli. The "Ca^{2+}-sensitivity priming" model has been recently appreciated as an important mechanism for driving stomatal movements under multiple stimuli (Hubbard et al., 2012; Laanemets et al., 2013). This model describes the requirement of both the repetitive cytosolic calcium (Ca^{2+}_{cyt}) transients and the primed state of guard cells for achieving Ca^{2+}-sensitivity priming that subsequently activates anion channels and stomatal closure. Arabidopsis lines expressing the cameleon reporter have revealed such Ca^{2+}_{cyt} transients under a wide variety of signals, including ABA, extracellular Ca^{2+}, cold, hydrogen peroxide (H_2O_2) and CO_2, as well as pathogenic elicitors (Allen et al., 2000; Pei et al., 2000; Klusener et al., 2002; Young et al., 2006; Siegel et al., 2009; Ye et al., 2013). The primed state has been suggested to regulate the sensitivity of the guard cell response to repetitive Ca^{2+} transients (Hubbard et al., 2012).

However, the frequency of Ca^{2+}_{cyt} transients does not affect stomatal closure (Allen et al., 2001; Young et al., 2006). Despite Ca^{2+}_{cyt} transients being widely believed to close the stomata under a wide range of environmental stimuli, this role is not universal; for example, ABA-induced Ca^{2+}_{cyt} transients are not able to close stomata in the Arabidopsis *plda1 pldd* double mutant (Uraji et al., 2012). In addition, ABA-induced stomatal closure at resting Ca^{2+}_{cyt} levels without Ca^{2+}_{cyt} transients was observed in *Vicia faba* guard cells (Levchenko et al., 2005), suggesting that the primed state is perhaps more crucial for controlling stomatal behavior than for other plant responses. Unfortunately, the primed state of guard cells is largely unknown. Interestingly, increasing evidence regarding chloroplast stromal calcium (Ca^{2+}_{str}) transient and reactive oxygen species (ROS) generation in chloroplasts may reveal the primed state under stress condition.

In this review, we deeply investigate the potential mechanism for facilitating Ca^{2+}_{str} transient and chloroplast ROS burst in guard cells under multiple stresses based on recent advances. We further establish a PQ pool-dependent model describing the mechanism for achieving the primed state through chloroplast Ca^{2+} and ROS signaling. In particular, we highlight the phenotype of mutants of the plant calcium sensing receptor (CAS) as an ideal material for supporting and developing the model that we proposed.

2. The nature of Ca^{2+}_{str} transients

The nature of Ca^{2+}_{str} transients is an emerging field. In plants, chloroplasts are capable of both Ca^{2+} uptake and release (Johnson et al., 2006), which are the important intracellular Ca^{2+} stores that sequester Ca^{2+} in the millimolar range (Larkum, 1968; Yamagishi et al., 1981). Light promotes Ca^{2+} uptake into chloroplasts (Xiong et al., 2006), and the driving force for Ca^{2+} import across the illuminated chloroplast envelope is probably potential stimulated (Roh et al., 1998; Pottosin et al., 2005). However, the free Ca^{2+} in chloroplast stroma remains stable in the nanomolar range in resting cells (Johnson et al., 1995; Sai and Johnson, 2002) because the vast majority of chloroplast Ca^{2+} accumulates in the thylakoid lumen or other unknown Ca^{2+}-binding sites in chloroplast under light conditions (Ettinger et al., 1999; Sai and Johnson, 2002) (Fig. 1).

Even though an accurate mechanism for Ca^{2+} translocation into the thylakoid lumen has yet to be reported, the Ca^{2+}/H^+ antiporter activity coupled with the photosynthetic electron transport (PET)-driven proton gradient across the thylakoid membrane and the cation channel on the thylakoid membrane (Pottosin and Schonknecht, 1996; Ettinger et al., 1999) are involved in this process. In addition, Ca^{2+} binding to thylakoid proteins is a potential mechanism for the unknown Ca^{2+} stores of chloroplasts (Asada, 1999) and is likely related to light-dependent phosphorylation by STN kinases (Stael et al., 2012).

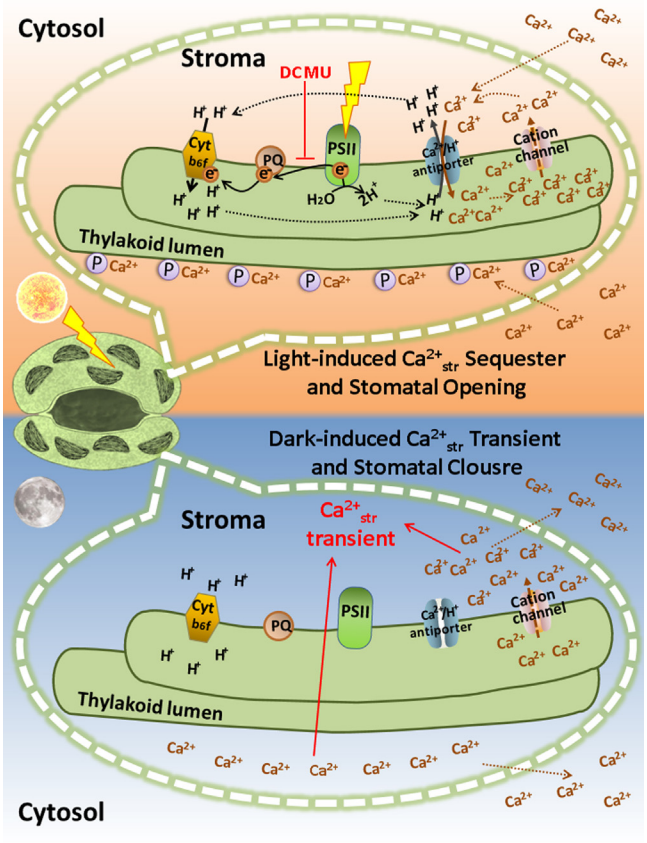

Fig. 1. Schematic representation of chloroplastic Ca^{2+} homeostasis and stomatal movement during the light or dark reaction. The exposure of guard cells to normal light drives photosynthetic electron transport (PET), resulting in a proton gradient across the thylakoid membrane. The PSII- and Cytb6f-based proton gradient promotes the thylakoid membrane Ca^{2+}/H^+ antiporter and sequesters Ca^{2+}_{str}, originally from Ca^{2+} uptake from the cytosol or Ca^{2+} release from cation channels, into the thylakoid lumen. In addition, the light-dependent phosphorylation of thylakoid proteins might enable Ca^{2+}_{str} binding to the thylakoid membrane. Finally, Ca^{2+}_{str} remains at low levels, contributing to stomatal opening. This process can be inhibited by 3-(3,4-dichlorophenyl)-1,1-dimethylurea (DCMU), a blocker of electron transfer between photosystem II (PSII) and the plastoquinone (PQ) pool. In contrast, PET and the proton gradient are eliminated when guard cells undergo light-to-dark transition. The dephosphorylation of thylakoid proteins and the cation channel release Ca^{2+} into stroma, leading to Ca^{2+}_{str} transients and stomatal closure. The increased Ca^{2+}_{str} is subsequently effluxed into the cytosol.

An analysis of dark-induced Ca^{2+}_{str} transients could reveal a novel mechanism of Ca^{2+}_{str} transients from the thylakoid lumen and the unknown Ca^{2+} binding sites. Interestingly, 3-(3,4-dichlorophenyl)-1,1-dimethylurea (DCMU), which prevents electron flow from photosystem II (PSII) to plastoquinone (PQ) and slows down the PSII- and cytochrome b6f complex (Cytb6f)-based generation of a proton gradient in the thylakoid lumen (Hihara et al., 2003), has no effect on the Ca^{2+}_{str} transient magnitude after light-to-dark transition but increases the Ca^{2+}_{str} level under light due to the inactive Ca^{2+}/H^+ antiporter (Johnson et al., 1995; Sai and Johnson, 2002), suggesting two independent systems for Ca^{2+} discharge. Thus, Ca^{2+} efflux from the thylakoid lumen *via* the cation channel due to the release of the proton gradient and the dissociation of Ca^{2+} by the dephosphorylation of thylakoid proteins was proposed to contribute to dark-induced Ca^{2+}_{str} transients (Fig. 1). Subsequent to the Ca^{2+}_{str} transients, a consecutive decrease in the Ca^{2+}_{str} level was observed (Sai and Johnson, 2002; Nomura et al., 2012), suggesting another unrevealed mechanism for Ca^{2+} efflux across the envelope.