15 ABA-Induced Actin Reorganization in Guard Cells of *Commelina communis* is Mediated by Cytosolic Calcium Levels and by Protein Kinase and Protein Phosphatase Activitiesl

Jae-Ung Hwang and Youngsook Lee*

Division of Molecular Life Science, Pohang University of Science and Technology, Pohang, 790-784, Republic of Korea

In guard cells of open stomata under daylight, long actin filaments are arranged at the cortex, radiating out from the stomatal pore. Abscisic acid (ABA), a signal for stomatal closure, induces rapid depolymerization of cortical actin filaments and the slower formation of a new type of actin which is randomly oriented throughout the cell. This change in actin organization has been suggested to be important in signaling pathways involved in stomatal closing movement, since actin antagonists interfere with normal stomatal closing responses to ABA. Here we present evidence that the actin changes induced by ABA in guard cells of *Commelina communis* are mediated by cytosolic calcium levels and by protein phosphatase and protein kinase activities. Treatment of guard cells with $CaCl_2$ induced changes in actin organization similar to those induced by ABA, and removal of extracellular calcium with EGTA inhibited ABA-induced actin changes. These results suggest that Ca^{2+} acts as a signal mediator in actin reorganization during guard cell response to ABA. A protein kinase inhibitor, staurosporine, inhibited actin reorganization in guard cells treated with ABA or $CaCl_2$, and also increased the population of cells with long radial cortical actin filaments in untreated control cells. A protein phosphatase inhibitor, calyculin A, induced fragmentation of actin filaments in ABA- or $CaCl_2$-treated cells and in control cells, and inhibited the formation of randomly oriented long actin filaments induced by ABA or $CaCl_2$. These results suggest that protein kinase(s) and phosphatase(s) participate in actin remodeling in guard cells during ABA-induced stomatal closure.

Keywords: guard cell, actin filaments, protein kinase, protein phosphatase, Ca^{2+}, ABA