Several Aspects of Current Research Into the Role of Calcium in Plant Physiology

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Calcium plays a variety of significant roles in the life cycle of plants. This review describes a brief summary of several examples of such roles in an attempt to provide some common ground relevant to the roles of calcium, with emphasis on the coupling between various stimuli and their respective responses. The selected topics include the regulation of turgor pressure, tropic responses, the cell cycle, and cell motility.

Key words: Calcium — Calcium ion (Ca^{2+}) — Cell cycle — Cell motility — Gravitropism — Turgor pressure

There is no doubt that calcium plays a crucial role in the regulation of a wide spectrum of responses in plant cells. Modern research on the role of calcium in plants initially may have reflected the abundant studies of animal systems. However, now sufficient knowledge has accumulated from plant systems to demonstrate that the dynamics of calcium are an integral part of the mechanisms that control the plant life cycle. A landmark review by Hepler and Wayne was published in 1985, so now we would like to focus attention on several studies that have been reported subsequently, since these studies may provide a more general perspective on this field. We have made no attempt to cover all the literature that involves calcium in plants. Rather, it has been our aim to describe concisely recent progress in examinations of the function of cellular calcium in plant systems and the various new findings in this area.

Regulation of turgor pressure

Stomatal pores open or close in response to the complex combinations of environmental stimuli, such as light, hormones, and the level of CO_2. This behavior of stomata is modulated through changes in the turgor pressure of the two guard cells that surround each stomatal pore. The involvement of ion fluxes across the plasma membrane and the tonoplast, as well as the synthesis and degradation of carbon-
containing metabolites, has been proposed as a major component of the regulation of this behavior (Hedrich and Schroeder, 1989; Mansfield et al., 1990). In particular, movement of K\(^+\) across the plasma membrane makes a significant contribution to the regulation of the turgor pressure. In the past several years, the role of Ca\(^{2+}\) in stomatal closure has been investigated extensively in relation to the movement of K\(^+\). In Commelina, McAinsh et al. (1990) were the first to show directly that an increase in \([\text{Ca}^{2+}]_{\text{cyt}}\) occurred prior to the stomatal closure induced by ABA. They loaded the guard cells with a fluorescent indicator of Ca\(^{2+}\), fura-2, by microinjection and succeeded in measuring \([\text{Ca}^{2+}]_{\text{cyt}}\) by microphotometry. With respect to the application of fluorescent indicators of Ca\(^{2+}\) to plant cells, an excellent review of this field was recently published by Bush and Jones (1990). The increase in \([\text{Ca}^{2+}]_{\text{cyt}}\) can be expected to inactivate inwardly directed K\(^+\) channels in the plasma membrane and to increase conductance with a permeability of the plasma membrane to anions. Both of these phenomena can be detected by the patch-clamp technique in the guard cells of Vicia (Schroeder and Hagiwara, 1989). The former may suppress an influx of K\(^+\), and the latter may result in activation of outwardly directed K\(^+\) channels by depolarization of the plasma membrane. The consequent acceleration of an efflux of K\(^+\) results in loss of turgor and leads to stomatal closure.

Blatt et al. (1990) reported that the electrophysiological events described above could be induced by the intracellular release of IP\(_3\), from its inactive, photolabile, caged form, into the cytoplasm of the guard cells. They postulated that the phosphatidylinositol pathway is involved in regulation of the K\(^+\) channels by Ca\(^{2+}\). The intracellular release of IP\(_3\) upon photolysis of caged IP\(_3\) was found to bring about an increase in \([\text{Ca}^{2+}]_{\text{cyt}}\), which was then followed by stomatal closure in Commelina (Gilroy et al., 1990). Caged Ca\(^{2+}\) had the same effect. Since the closure of stomata could be observed even in the presence of La\(^{3+}\), a commonly used blocker of Ca\(^{2+}\) channels in the plasma membrane, Ca\(^{2+}\) was assumed that it was mobilized from an intracellular compartment.

Using similar methodology to that of McAinsh et al. (1990), Gilroy et al. (1991) loaded the guard cells of Commelina with another fluorescent indicator, namely, indo-1 and visualized changes in \([\text{Ca}^{2+}]_{\text{cyt}}\) that accompanied stomatal closure by fluorescence image analysis together with microphotometry. Under precisely controlled experimental conditions, they confirmed that treatment with ABA always induced stomatal closure. Some, but not all, of the guard cells exposed to ABA exhibited a transient increase in \([\text{Ca}^{2+}]_{\text{cyt}}\) prior to the closure of stomata. As the intracellular compartment involved in the mobilization of Ca\(^{2+}\), they suggested the vacuole and/or some endomembrane system in the vicinity of the nucleus. The heterogeneity in the responsiveness of the various stomata to ABA in terms of \([\text{Ca}^{2+}]_{\text{cyt}}\) was also mentioned by McAinsh et al. (1990). ABA seems to induce stomatal closure through a Ca\(^{2+}\)-dependent pathway or through a Ca\(^{2+}\)-independent pathway, depending on the individual stomata. Alternatively, a transient increase in \([\text{Ca}^{2+}]_{\text{cyt}}\) and stomatal closure are merely parallel phenomena, both of which can be induced by treatment with ABA.

Lamprothamnium is a brackish characean alga. The internodal cells maintain