学位論文の要旨

Abstract of Thesis

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学位論文題目 Title of Thesis（学位論文題目が英語の場合は和訳を付記）

Involvement of reactive carbonyl species in stomatal movements
（気孔運動への活性カルボニル種の関与）

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The phytohormones abscisic acid (ABA) and methyl jasmonate (MeJA) regulates many processes of plant growth and development and help plants to tolerate adverse environmental conditions. Plants synthesize the hormone ABA in response to drought, inducing stomatal closure, thus reducing water loss. MeJA induces stomatal closure similar to ABA. Salicylic acid (SA) is a phenolic phytohormone that regulates numerous plant physiological processes like seed germination, cell growth and ion uptake, and pathogen defense response. SA induces stomatal closure similar to ABA and MeJA. ABA- and MeJA-induced stomatal closure is accompanied by production of reactive oxygen species (ROS) mediated by plasma membrane NAD(P)H oxidases while SA-induced stomatal closure is accompanied by peroxidase-mediated extracellular ROS production and intracellular ROS accumulation. However, in spite of the importance of ROS in guard cell signaling, the mechanism of how the generating ROS is transduced into downstream reactions in guard cells is unknown. A variety of stresses induce overproduction of ROS in plants and the accumulated ROS oxidizes lipids, especially polyunsaturated fatty acids (PUFA), resulting in production of reactive compounds including aldehydes, ketones, and hydroxyl acids. Aldehydes and ketones containing α,β-unsaturated carbonyl structure are termed as reactive carbonyl species (RCS). Accumulated RCS impairs growth of plants because RCS is toxic to plants. Alkenal reductase (AER), aldehyde dehydrogenase (ALDH), aldo-keto reductase (AKR), and aldehyde reductase (ALR) detoxify RCS and overexpressing AER, ALDH, AKR, and ALR improves stress tolerance in plants. However, information about signal transduction mediated by RCS is limited.

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In Chapter 2, I investigated the involvement of RCS in ABA signaling pathway in guard cells using wild-type tobacco (*Nicotiana tabacum*) (WT), and transgenic tobacco overexpressing Arabidopsis (*Arabidopsis thaliana*) alkenal reductase (AER-OE), *A. thaliana* ecotype Columbia-0 and scavengers of RCS. I found that treatment with ABA and H_2O_2 significantly increased the contents of RCS in epidermal tissues of wild type (WT). Both overexpression of the RCS scavenging enzyme 2-alkenal reductase (AER) and application of RCS scavengers, carnosine and pyridoxamine, inhibited ABA- and H_2O_2-induced stomatal closure and RCS accumulation but not ABA-induced H_2O_2 production. Acrolein- and HNE-induced stomatal closure was inhibited in AER-OE plants and also by the application of carnosine and pyridoxamine. Moreover, acrolein significantly activated plasma membrane Ca^{2+}-permeable channels and more effectively initiated $[Ca^{2+}]_{cyt}$ elevation and induced stomatal closure than H_2O_2 did. Furthermore, acrolein suppressed K^+_{in} currents in guard cell of WT. These results indicate that production of RCS following ROS production and regulation of $[Ca^{2+}]_{cyt}$ elevation by RCS are important mechanisms for ABA-induced stomatal closure.

In Chapter 3, I examined the involvement of RCS in MeJA-induced stomatal closure using wild-type tobacco (*Nicotiana tabacum*) (WT), and transgenic tobacco overexpressing Arabidopsis (*Arabidopsis thaliana*) alkenal reductase (AER-OE), and scavengers of RCS. I found that MeJA induced stomatal closure in the WT but not in the AER-OE. MeJA-induced stomatal closure in WT was inhibited by RCS scavengers, carnosine and pyridoxamine. Moreover, MeJA induced H_2O_2 production in guard cells of both WT and AER-OE, and H_2O_2 production was not suppressed by the application of RCS scavengers. These results suggest that RCS is involved in MeJA-induced stomatal closure and function downstream of ROS production.

In Chapter 4, I investigated the involvement of RCS in SA-induced stomatal closure using wild-type tobacco (*Nicotiana tabacum*) (WT), and transgenic tobacco overexpressing Arabidopsis (*Arabidopsis thaliana*) alkenal reductase (AER-OE), and scavengers of RCS. SA induced stomatal closure in the WT but not in the AER-OE. SA-induced stomatal closure in WT was inhibited by RCS scavengers, carnosine and pyridoxamine. Moreover, SA induced H_2O_2 accumulation in guard cells of both WT and AER-OE. These results suggest that RCS mediates SA-induced stomatal closure and function downstream of ROS accumulation.

In Chapter 5, I examined the effects of acrolein on light-induced stomatal opening in *Arabidopsis thaliana*. Acrolein inhibited light-induced stomatal opening in a dose-dependent manner. Acrolein at 100 µM inhibited plasma membrane inward-rectifying potassium (K_{in}) channels in guard cells. Acrolein at 100 µM inhibited K_{in} channel KAT1 expressed in a heterologous system using *Xenopus leaves* oocytes. These results suggest that acrolein inhibits light-induced stomatal opening through inhibition of K_{in} channels in guard cells.