学位論文の要旨

Abstract of Thesis

研究科 School	Graduate School of Environmental and Life Science
専　攻 Division	Agricultural and Life Science
学生番号 Student No.	77501652
氏　名 Name	AKTER FAHMIDA

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 salicylic acid-induced stomatal closure

サリチル酸誘導気孔閉口における活性カルボニル種の関与

学位論文の要旨 Abstract of Thesis

Stomata are tiny pores on the leaf epidermis of higher plants that serve as a pathway both for the influx of CO_2 into the plants from atmosphere for optimal photosynthesis and for the transpirational water loss. A stomatal aperture is controlled by a pair of specialized guard cells surrounding the stoma. The guard cells sense and integrate multiple environmental and endogenous signals. Abscisic acid (ABA), methyl jasmonate (MeJA), and salicylic acid (SA) are involved in unique mechanisms to regulate stomatal apertures to mediate various defense responses. Salicylic acid is a phenolic defense hormone that regulates various plant physiological processes and confers drought tolerance in plants. An elevated SA level induces stomatal closure in many plant species. While ABA and MeJA induce NAD(P)H oxidase-mediated ROS production, SA induces SHAM sensitive peroxidase-mediated ROS production. A variety of stresses induce overproduction of ROS in plants and the accumulated ROS oxidizes lipids, 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) which may act as signaling molecules at low concentrations. RCS was reported to function downstream of ROS and because of its toxicity, accumulated RCS impairs growth of plants. RCS is known to mediate ABA and MeJA signaling in guard cells but the involvement of RCS in SA-induced stomatal closure remains to be clarified. Glutathione (GSH) is an essential metabolite that regulates plants growth, development, defense systems, signaling mechanisms, and gene expression via the redox state of GSH pools in plants. It has been reported that ABA- and MeJA-induced stomatal closure is accompanied by decreasing intracellular GSH, and that intracellular GSH regulates stomatal movement. But the role of GSH in SA signaling is still unknown.

In Chapter 2, I investigated the involvement of RCS in SA-induced stomatal closure in *Arabidopsis thaliana*. I found that SA significantly increased RCS production in guard cells. Application of SA significantly induced stomatal closure in Arabidopsis. Application of RCS scavengers, carnosine and pyridoxamine, inhibited SA-induced stomatal closure. These results indicate that RCS is involved in SA-induced stomatal closure *A. thaliana*.

Name	AKTER FAHMIDA

In Chapter 3, I investigated the involvement of RCS in SA signaling in guard cells using wild-type tobacco (*Nicotiana tabacum*) (WT), and transgenic tobacco overexpressing Arabidopsis (*Arabidopsis thaliana*) alkenal reductase (AER-OE). I found that SA significantly increased RCS production in guard cells of wild type (WT). Application of SA significantly induced stomatal closure in WT but not in the AER-OE. Overexpression of the RCS scavenging enzyme 2-alkenal reductase (AER) inhibited SA-induced stomatal closure and RCS accumulation. These results indicate that RCS mediates SA signaling in guard cells.

In Chapter 4, I investigated the regulation of SA signaling by GSH in guard cells using an *A. thaliana* mutant, *cad2-1*, which is deficient in the first GSH biosynthesis enzyme, γ-glutamylcysteine synthetase. Application of SA decreased stomatal apertures with decreasing intracellular GSH level in guard cells. Decreasing GSH by the *cad2-1* mutation and by a GSH-decreasing chemical, 1-chloro-2,4-dinitrobenzene, enhanced the SA-induced stomatal closure. A treatment with glutathione monoethyl ester restored the GSH level in the *cad2-1* guard cells and complemented the stomatal phenotype of the mutant. I concluded that GSH negatively modulates SA-induced stomatal closure in *A. thaliana*.

Salicylic acid induces ROS production in guard cells. During stress conditions, overproduction of ROS oxidizes lipids, resulting in the production of RCS. GSH allows the efficient scavenging ROS. My results from this thesis concluded that RCS is involved in SA-induced stomatal closure and that SA-induced stomatal closure is negatively regulated by GSH.