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Molecular Mechanisms Underlay the Enhancement of APS Reductase Expression and Sulfur Assimilation by Salt －Toward the Generation of Salt Tolerant Plants by Improving Sulfur Assimilation Capacity of Plants－

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Summary

Sulfur is one of the essential macronutrients for plants. Sulfur availability and its assimilatory capacity of plants greatly influence on yield and quality of crops. In plants, sulfur assimilation starts from sulfate uptake. Sulfates taken in plant cells are reduced to sulfide by several steps of reactions, including the reaction catalyzed by APS reductase (APR), and assimilated into cysteine. When plants are subjected to sulfur starvation (-S), transcript levels and the activities of sulfur assimilatory enzymes including APR are increased. This response should be an adaptation mechanism of plants for efficient utilization of limited sulfur source. Transcript levels and the activities of APR are also increased by salt (NaCl). These findings suggested that enhanced levels of APR activity by NaCl are due to the increased transcript levels, and the salt tolerance of plants can be increased by enhancement of sulfur assimilation. In this study, we analyzed the regulatory mechanisms of APR gene expression responded to –S and NaCl, to reveal the NaCl function in regulation of sulfur assimilation and future generation of salt tolerant plants by improving of sulfur assimilation capacity.

Among the three APRs existed in Arabidopsis, *APR2* and *APR3* are highly enhanced by -S and NaCl. Deletion constructs of every 300 bp in 2,474 bp and 2,184 bp upstream region of *APR2* and *APR3* were fused to luciferase gene, and introduced to Arabidopsis plants. Using these transgenic plants, we analyzed luciferase activities of plants under sulfur sufficient (+S), deficient (-S) and NaCl treated conditions. We succeeded to narrow down the upstream responsive regions of *APR2* and *APR3* to -S and NaCl into 300 bp. These results strongly suggested the identity of both responsive elements in *APR* promoters. The possibility that transcript levels of *APR*s are regulated through mRNA stability is also suggested. To determine the regulatory mechanisms of APR expression responsible to -S and NaCl, further deletion analysis to determine *cis*-acting elements are required. Possible identities of both -S and NaCl responsive regions also suggested the existence of crosstalk between -S and NaCl signal transduction. Verification of the crosstalk and its physiological meaning are interesting studies for future.