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The *Rolled Fine Striped* (RFS) CHD3/Mi-2 chromatin remodeling factor epigenetically regulates genes involved in oxidative stress during leaf development in rice

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[Introduction]

The *rfs-1* gamma-ray allele and the *rfs-2* T-DNA insertion allele of *RFS* failed to complement each other and their mutants had similar phenotypes, producing extremely incurved leaves due to defective development of vascular cells on the adaxial side. Map-based cloning showed that the *rfs-1* mutant harbors a 9-bp deletion in a gene encoding a predicted CHD3/Mi-2 chromatin remodeling factor belonged to the SNF2-ATP-dependent chromatin remodeling family.

[Materials and Methods]

The *rfs-1* mutant, FL233 (Iwata et al., 1984), was induced from the rice japonica cultivar Norin-8 in a gamma-ray mutagenesis and the *rfs-2* mutant line, T-DNA-tagged mutant PFG 3D-02766, was obtained from the Crop Biotech Institute at Kyung Hee University, Republic of Korea. An *rfs-1* plant was crossed with a Korean japonica rice cultivar 'Seolakbyeo' and progressed to the F6generation. 'Seolakbyeo' was used as the parental wild-type plant in this study. Plants were grown in a paddy field during the rice growing period or in the growth chamber.

[Results and Discussions]

The *rfs-1* mutant had reduced leaf width, leaves that rolled to the adaxial side, and variegated leaves, which appeared in the 2-month-old plants. To investigate whether the chlorotic and cell death phenotypes that were observed in the abaxial side of *rfs-1* leaves were caused by the accumulation of ROS, we stained for hydrogen peroxide (H_2O_2) and superoxide radical (O_2^-) using 3,3'-diaminobenzidine (DAB) and nitroblue tetrazolium (NBT), respectively. The leaf blades of 2-month-old *rfs-1* plants exhibiting the chlorotic phenotype stained more strongly than the wild-type plants. The transcript levels of five ROS-related genes including *CATC*, *APX8*, a putative copper/zinc superoxide dismutase (Cu/Zn-SOD) gene, a putative superoxide dismutase (SOD) gene, and *Prx IIE2* (peroxiredoxin IIE2) were dramatically decreased in the *rfs-2* mutants. RFS affects modification of histone proteins that directly bind to ROS-related genes to maintain ROS homeostasis.

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