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THE ROLE OF ABC1K7 AND ABC1K8, TWO ABC1K KINASES OF *ARABIDOPSIS THALIANA*

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The activity of *bc1 complex* kinases (ABC1K) belong to a large group of atypical protein kinases found in prokaryotes and eukaryotes. In bacteria and mitochondria, ABC1K kinases are involved in the respiratory pathway, being necessary for the synthesis of the coenzyme Q. In chloroplasts, ABC1K proteins play a role in prenylquinone synthesis and stress responses, but their precise function remains unclear.

A functional characterization was carried out for ABC1K7 and ABC1K8, two ABC1K proteins of the *Arabidopsis thaliana* plastome. The comparison of *abc1k7* and *abc1k8* mutants, *abc1k7/abc1k8* double mutant and wild-type plants revealed a reduction in plastidial iron-containing proteins of the Cytb₆f complex in the mutants. Iron uptake from soil is not hampered in mutant lines, suggesting that ABC1K7 and ABC1K8 affect iron distribution within the chloroplast. Moreover, mutant plants accumulated more ferritin and superoxide, and showed reduced tolerance to reactive oxygen species (ROS). Because ROS take part in abscisic acid (ABA) signaling, we investigated the relation between ABA and *ABC1K7* and *ABC1K8* and found that both genes were upregulated by ABA treatment, while expression of several ABA-responsive genes resulted affected in mutants. Moreover, analyzing ABA-mediated processes, we determined that germination was more affected by ABA treatment and osmotic and salt stress in the single and double mutants than in wild-type plants. Stomatal aperture was also reduced in the mutants under standard growth conditions and was not further reduced by exogenous ABA application. Furthermore, ABA-induced senescence symptoms were more severe in the leaves of the mutants compared to wild type leaves. Taken together, these data suggest that ABC1K7 and ABC1K8 probably act in signaling pathways that influence responses to ROS production and oxidative stress, such as ABA signaling, probably by influencing the cellular redox state and chloroplast lipid metabolism.