PLANT PROGRAMMED CELL DEATH IS RELATED TO ROS-ACTIVATED K^+ EFFLUX, WHICH IS MEDIATED BY K^+ OUTWARDLY RECTIFYING CHANNEL GORK

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In animal cells, KCNH2 channel is directly activated by reactive oxygen species (ROS) and responsible for induction of programmed cell death (PCD) through the loss of K^+ and activation of K^+-regulated endonucleases/cell death proteases. Here, it was demonstrated that NaCl and pathogen-induced PCD symptoms, such as loss of electric membrane poten-

-1 (lacking major K^+ efflux channel) and after application of specific K^+ channel and ROS scavengers antagonists (wild type *Arabidopsis thaliana* WS plants). K^+ channel blockers and HR scavengers prevented stress-induced PCD in the wild type. Using CaspACE™ FITC-VAD-FMK In Situ Marker (Promega), it was found that cell death caspase-like protease activities (triggered by salt stress, elicitors and hydroxyl radicals) increase in K^+-dependent manner in *Arabidopsis thaliana* root cell cytoplasm. Moreover, DeadEnd™ Fluorometric TUNEL tests showed that stress/ROS-inducible endonuclease activity is also regulated by cytosolic K^+. *Gork1-1* plants demonstrated significant delay in activation of both caspase-like activities and endonucleases. Overall, results of this study strongly suggest that plants and animals share PCD cascade involving Shaker group of redox-sensitive K^+ outwardly rectifying channels.