

SEMINAR

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Coordinated regulation of Na⁺ extrusion and long- distance transport in plant



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To control net sodium (Na⁺) uptake, Arabidopsis plants utilize the plasma membrane Na⁺/H⁺ antiporter SOS1 that catalyzes Na⁺ efflux at the root and promotes Na⁺ loading into the xylem, and the channel-like HKT1;1 protein that mediates the reverse flux of Na⁺ unloading at the xylem. Together, these opposing transport systems govern the partition of Na⁺ within the plant, yet they must be finely co-regulated to prevent a futile cycle of xylem loading and unloading. Here we show that the Arabidopsis SOS3 protein acts as the molecular switch governing these Na⁺ fluxes by favoring the recruitment of SOS1 to the plasma membrane and its subsequent activation by the SOS2/SOS3 kinase complex under salt stress, while commanding HKT1;1 protein degradation upon acute salt stress. SOS3 achieves this novel role by direct and SOS2-independent binding to previously unrecognized functional domains of SOS1 and HKT1;1. These results evidence that roots first retain moderate amounts of salts to facilitate osmoregulation. When sodicity exceeds the stress set point, activation of SOS3 switches the balance towards Na⁺ export out of the root via the xylem. Thus, SOS3 functionally links and co-regulates the two major Na⁺ transport systems operating in vascular plants controlling plant tolerance to salinity.