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Auto-inhibited Ca^{2+}-ATPases ACA8 and ACA10 mediate wound-induced long-distance Ca^{2+}waves and ROS-dependent guard cell responsiveness in Arabidopsis

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The plasma membrane-localized auto-inhibited Ca^{2+}-ATPases ACA8 and ACA10 play a crucial role in cytosolic Ca^{2+} homeostasis and Ca^{2+}-dependent signaling in *Arabidopsis*. In our study, we demonstrate that both pumps contribute to maintain low basal cytosolic free Ca^{2+} concentration ($[Ca^{2+}]_{cyt}$) under resting conditions, as indicated by elevated basal $[Ca^{2+}]_{cyt}$ in *aca8* and *aca10* null mutant seedlings expressing the FRET-based Ca^{2+} sensor YC3.6. Moreover, using the red-shifted R-GECO1 Ca^{2+} indicator, we show that upon leaf and root wounding, wt seedlings exhibit rapid, long-distance Ca^{2+} wave propagation, whereas *both aca* mutants display impaired signal transmission, highlighting their key role also in systemic Ca^{2+} wave propagation. In contrast, by using the ROS-sensitive dye CM-H_2DCFDA, we detect intense ROS accumulation after wounding in *aca8 and aca10* mutants, with an intensity comparable to wt, suggesting that ACA8 and ACA10 are dispensable for wound-induced systemic ROS accumulation. Surprisingly, after injury and exogenous H_2O_2 treatment, stomatal closure is impaired in *aca* mutants, despite normal ROS production in guard cells, indicating that ACA8 and ACA10 are required to translate ROS signals into functional Ca^{2+} responses in stomatal regulation. Overall, our results support a model in which ACA8 and ACA10 act as key regulators of Ca^{2+} homeostasis and systemic signal propagation after wounding, thereby ensuring proper systemic communication and stomatal responsiveness.