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# STRUCTURAL DETERMINANTS OF KAT1 CHANNEL REGULATION BY 14-3-3 PROTEINS AND FUSICOCCIN

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Plants acquire $K^+$ ions for cell growth and movement by the concerted activation of $K^+$ channels and the $H^+$-ATPase. Here we present crystallographic and functional data showing that the $K^+$ inward rectifier KAT1 channel is regulated by 14-3-3 proteins and further modulated by the phytotoxin Fusicoccin, in analogy to the $H^+$-ATPase. We identified a 14-3-3 mode III binding site at the very C-terminus of KAT1 and co-crystallized it with tobacco 14-3-3 proteins to describe the protein complex at atomistic detail. Validation of this interaction by electrophysiology shows that 14-3-3 binding augments KAT1 conductance by increasing the maximal current and by positively shifting the voltage-dependency of gating. Fusicoccin potentiates the 14-3-3 proteins effect on KAT1 activity by stabilizing their interaction. Crystal structure of the ternary complex reveals a non-canonical binding site for the toxin that adopts a novel conformation, never described before. The structural insights underscore the adaptability of Fusicoccin, predicting more potential targets than so far anticipated. The data furthermore advocate a regulatory hub function of 14-3-3 proteins in $K^+$ uptake. They coordinate the essential up-regulation of $H^+$ export by the $H^+$-ATPase with $K^+$ influx by the inward rectifier for sustained $K^+$ uptake into plant cells.