

Silicon-transporters-mediated silicon deposition at root endodermis decreases bypass flow and consequently alleviates salt toxicity in rice

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Abstract

Silicon (Si) can alleviate Na⁺ toxicity by decreasing bypass flow in salt-stressed rice (*Oryza sativa* L.), however, the underlying mechanisms remain veiled. Here, we demonstrate how Si decreases bypass flow and alleviates salt toxicity at physiological and molecular levels by using two rice mutants (*lsi1* and *lsi2*, defective in OsLsi1 and OsLsi2, respectively) and their wild types (WTs). Under salt stress, Si promoted plant growth and decreased Na⁺ root-to-shoot translocation in WTs, but not in mutants. Simultaneously, both quantitative estimation and fluorescent visualization of trisodium-8-hydroxy-1,3,6-pyrenetrisulphonic (PTS, an apoplastic tracer) show Si blocked bypass flow in WTs, but not in mutants. Energy-dispersive X-ray microanalysis (EDX) shows Si was deposited at root endodermis in WTs, but not in mutants. Moreover, root split experiment using *lsi1* WT shows shoot Si accumulation down-regulated the expression of Si transport genes (*OsLsi1* and *OsLsi2*) in root and accelerated Si deposition at root endodermis. In summary, our results reveal that 1) Si deposition at root endodermis reduces bypass flow, thereby alleviating salt toxicity in rice, and 2) the deposition of Si, which could be an active and physiologically-regulated process, is mediated by the cooperation of OsLsi1 and OsLsi2 and regulated by shoot Si accumulation.

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