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Confirmation and Functional Characterisation of Potassium Ion Channel Gene for Iron Toxicity Tolerance in Rice

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Abstract

Iron toxicity is a major nutrient disorder in rice caused by an excessive uptake of iron occurring in flooded soils due to low redox potential, which favours the formation of the soluble and plant available form, Fe^{2+} (ferrous). After uptake of Fe^{2+} via the rhizodermis, Fe will be translocated into the shoot. In plant cells Fe^{2+} catalyzes the formation of reactive oxygen species (ROS) via the Fenton reaction which is known to cause irreversible damage to biomolecules such as lipids, proteins and DNA resulting in a reduced plant growth and yield loss. In our previous study, QTL related to the shoot iron concentration in Fe-toxic conditions were identified with GWAS study. This led to the identification of a candidate gene for Fe-exclusion, i.e. a putative potassium (K) ion channel gene OsAKT1 (LOC_Os01g45990). We hypothesised that K and Fe might have antagonistic interactions in Fe toxic conditions. In order to proof this concept, experiments were conducted with two knockout mutant lines for the candidate genes named osakt1–1 and osakt1–2. Semi-quantitative RT-PCR confirmed that the target gene was not expressed in mutant lines. Mutants and wild-types were grown for 4 weeks in a hydroponic culture followed by a five day, 1000 ppm of $FeSO_4$*$7H_2O$ treatment to simulate an acute iron stress. The iron treatment led shoot Fe concentrations exceeding toxicity threshold, to visible symptom formation quantified as Leaf Bronzing Score (LBS), and negatively affected spectral reflectance indices such as the normalised difference vegetation index (NDVI) and photochemical reflectance index (PRI), lipid peroxidation, as well as the uptake of potassium. The knock out mutant osakt1–1 had significantly lower shoot K concentration but higher Fe concentration, along with significantly higher LBS, NDVI and PRI compared to the wild-types. Therefore, antagonistic translocation of Fe and K to the leaf sheath and leaf blade could be detected which underpins our hypothesis. Ongoing experiments aim at further elucidating the mechanism behind the antagonistic interaction of Fe and K, and the resulting susceptibility of osakt1 mutants, including the role of antioxidant enzymes (ROS-scavengers). Our results therefore help to explain the important role of K in Fe toxicity tolerance.

Keywords: Genotype tolerance, iron toxicity, potassium, rice, tolerance mechanism

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