A-25

AtMRP5, an *Arabidopsis* ATP-binding cassette transporter that binds sulfonylurea, is required for root growth under NaCl stress and stomatal movement.

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In this study, we have identified cDNA encoding a novel mammalian MRP-like protein (designated *AtMRP5*) in *Arabidopsis*. Experiments with promoter--glucuronidase fusion constructs disclose that AtMRP5 is expressed mainly in the elongation zone of roots actively growing root hairs, vascular bundles in cotyledons and leaves, and interestingly, pollen grains and abscission zones of siliques. Biochemical analyses reveal specific binding of expressed AtMRP5 to the MRP inhibitor, [3H]glibenclamide, with a *K*d of 7.2 nM. Sulfonylurea binding displays features typical of ligand-receptor binding, including saturation curves and displacement by other sulfonylureas with different affinities. To determine the role of AtMRP5 *in planta*, we identified an *Arabidopsis thaliana* mutant *(atmrp5-2)* using reverse genetics, in which the *AtMRP5* gene was disrupted by T-DNA insertion. Both wild-type and *atmrp5-2* mutant grew relatively well and appeared healthy on MS (1X) medium. In root bending assays using MS medium supplemented with 100 mM NaCl, root growth of *atmrp5-2* was substantially inhibited in contrast to almost normal growth of wild-type seedlings. NaCl stress-inhibited root growth was concentration-dependent, with *I50* values of approximately 100 mM for wild-type and 40 mM for *atmrp5-2* mutant plants, respectively. This hypersensitive response of *atmrp5-2* mutant was not observed during mannitol treatment. The root growth of wild-type measured in MS medium supplemented with the MRP inhibitor, glibenclamide, was blocked by Na+ in a very similar manner to that of *atmrp5-2* in NaCl alone. Na+ -dependent reduction of root growth of wild-type in the presence of glibenclamide was partially restored by diazoxide, a known K+ channel opener that reverses the inhibitory effects of sulfonylureas in animal cells. Furthermore, *atmrp5-2* displayed insensitivity to glibenclamide-induced stomatal opening and was impaired in ABA-induced stomatal closing. The results collectively indicate that the *atmrp5-2* mutation does not induce a defective osmotic stress response. Rather, the defect is restricted to Na+ tolerance. Therefore, AtMRP5 is required for Na+ tolerance and may function as a sulfonylurea and KCO-sensitive ion channel or a channel regulator.