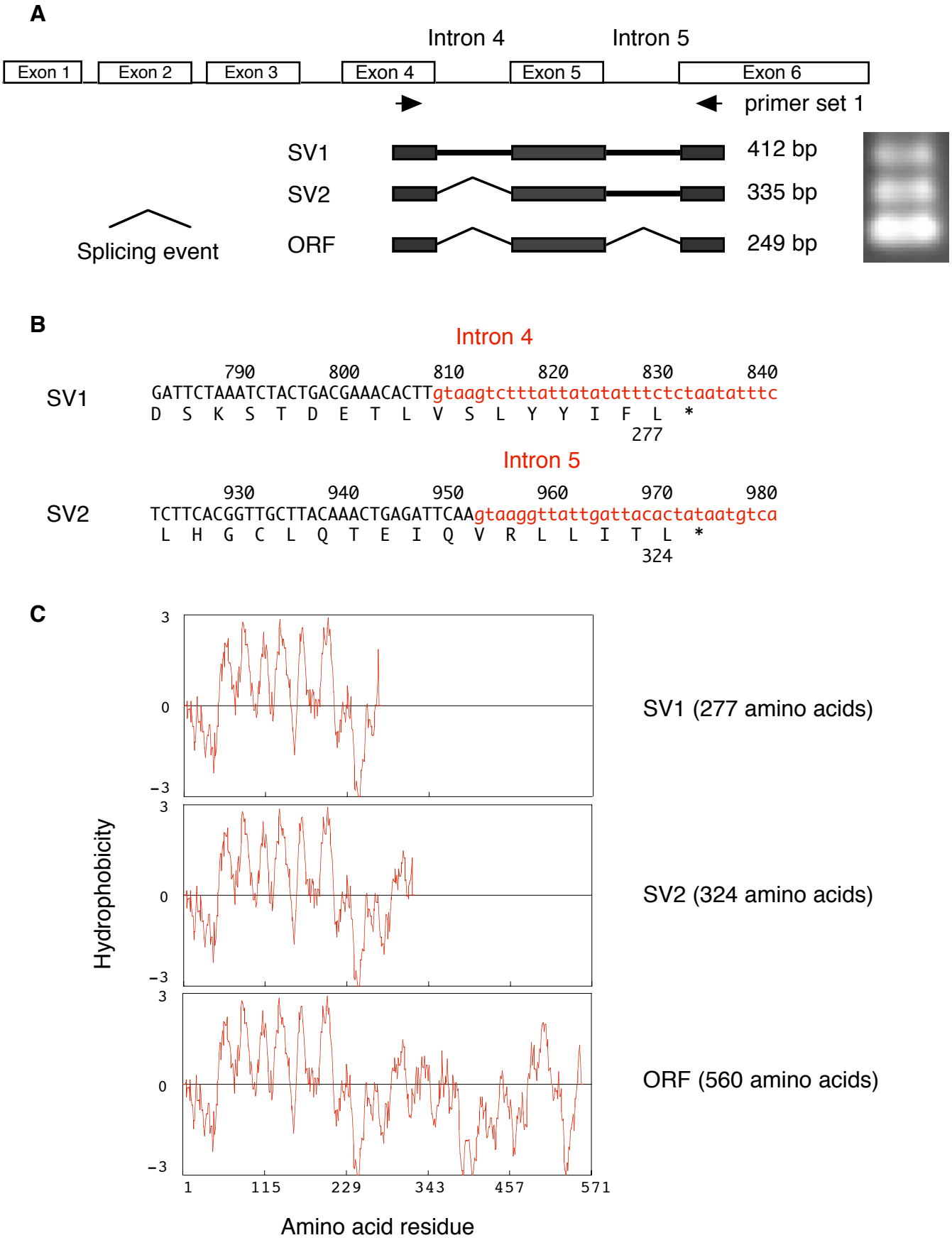

Fig. S1. Genomic structure and hydrophobicity profiles of deduced amino acids of AtALMT12. **(A)** Exons and introns of the *AtALMT12* locus. Positions of primer set #1 (arrows) giving rise to three amplification products (249, 335, 412 bp), representing the putative splicing variants, SV1 and SV2. **(B)** Partial sequence of the amplification products presumably from SV1 and SV2. Appearance of termination codons on introns is expected to reduce the peptide length from 560 to 277 or 324 amino acid residues. **(C)** Hydrophobic profiles of AtALMT12 proteins derived from SV1, SV2, and the complete coding sequence, as predicted by the Kyte-Doolittle algorithm. All these peptides have six putative trans-membrane domains.

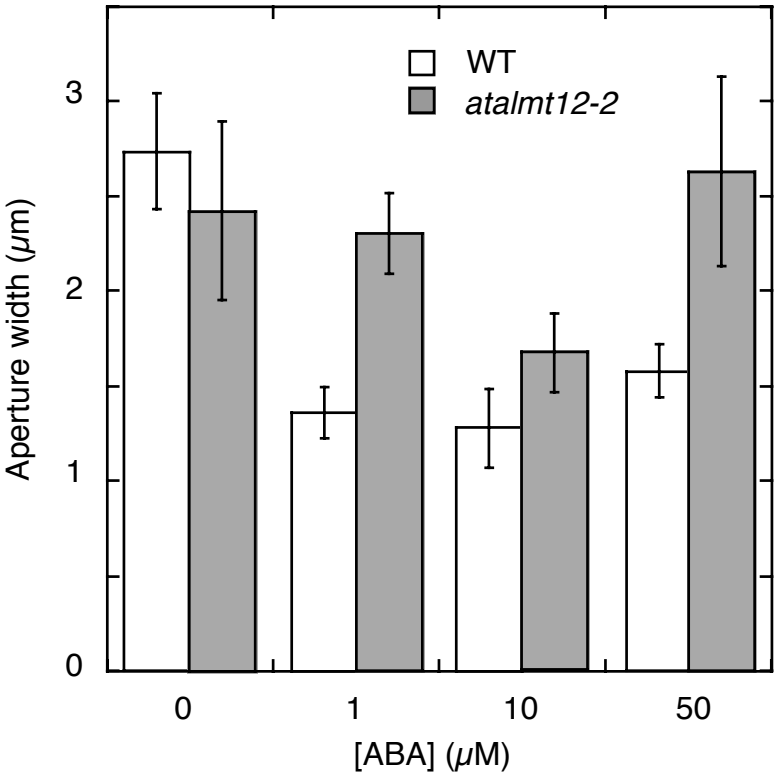

Fig. S2. Phenotypes of *atalmt12-2*.
Abscisic acid (ABA)-induced stomatal closure (n=5 experiments for 0, 1 and 10 μM ABA, n=3 experiments for 50 μM ABA, mean ± SEM). Twenty stomata were measured for each experiment.

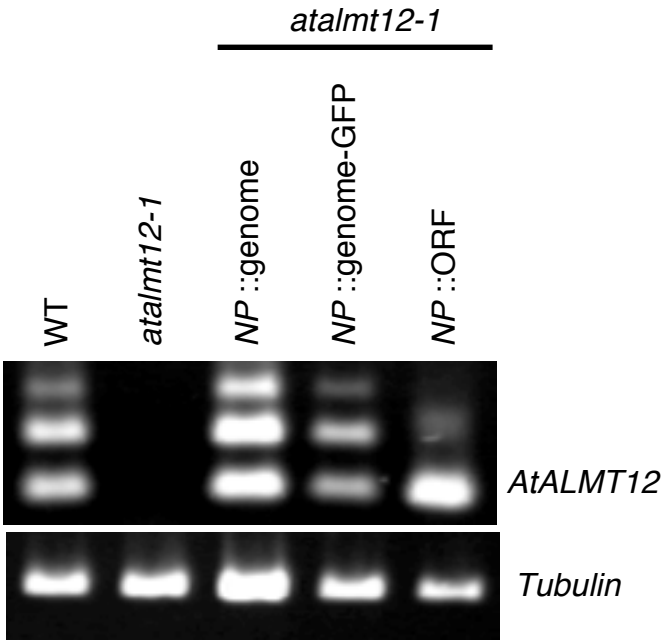

Fig. S3. *AtALMT12* expression in wild type, a knock-down mutant (*atalmt12-1*), and in the complementation lines in the *atalmt12-1* background. RT-PCR was done using the *AtALMT12* primer set #1 or β*-tubulin* primers.

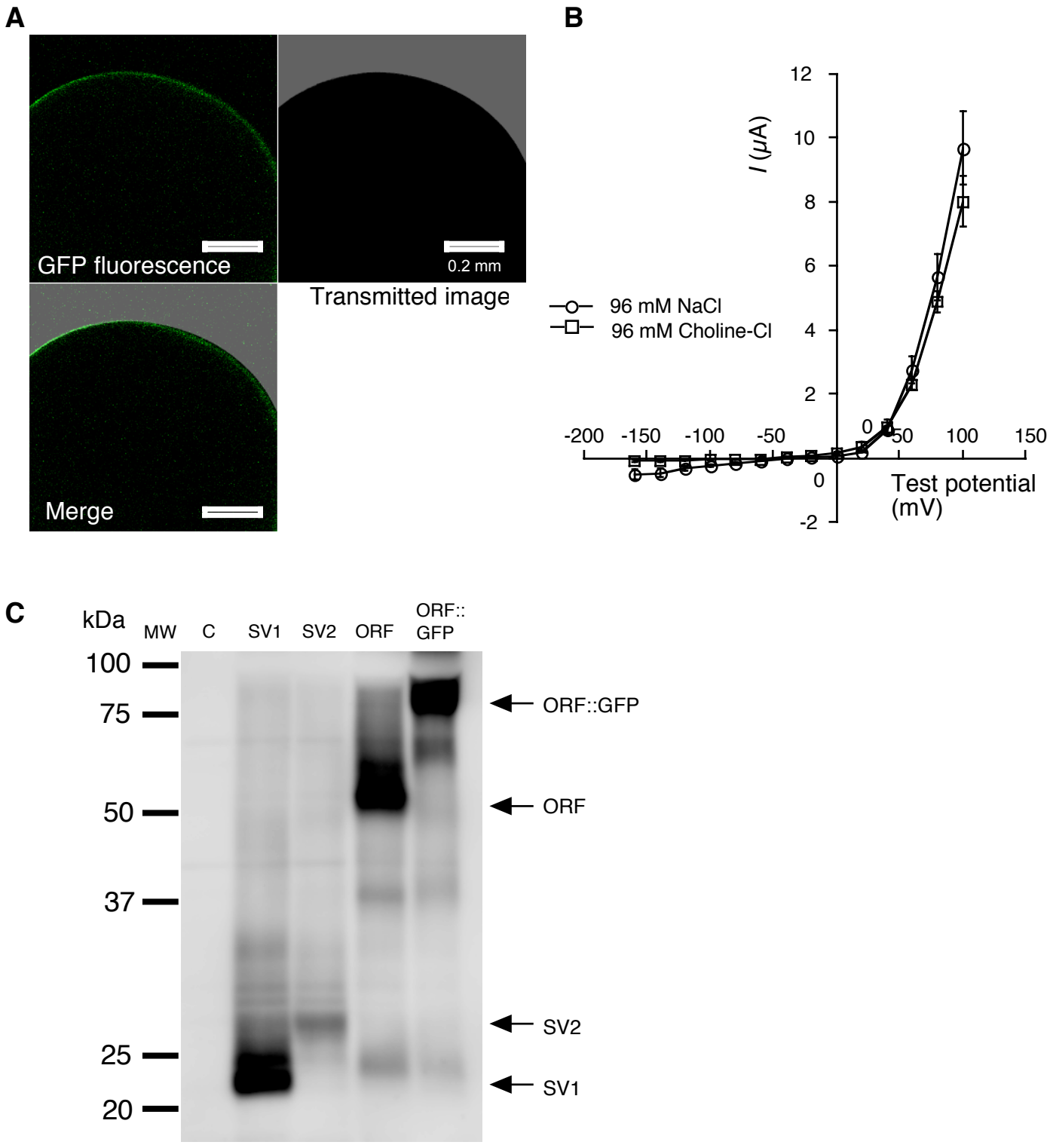

Fig. S4. AtALMT12 expression analyses in *Xenopus laevis* oocytes. **(A)** Confocal images of an *AtALMT12* (ORF)*::GFP* expressing oocyte one day after injection. The fluorescence signal was observed on the plasma membrane. **(B)** Current voltage relationships in *AtALMT12*-expressing oocytes recorded two different chloride salts. Data are mean ± SEM (n=3). **(C)** Immunoblot analysis of AtALMT12. Crude-membrane proteins of oocytes (10 µg) were separated by SDS-PAGE and blotted onto polyvinylidene difluoride membrane. Arrows indicate the positions of the various AtALMT12 bands.