The ABA-Deficiency Suppressor Locus HAS2 Encodes the PPR Protein LOI1/MEF11 Involved in Mitochondrial RNA Editing

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Résumé en anglais	The *hot ABA-deficiency suppressor2* (*has2*) mutation increases drought tolerance and the ABA sensitivity of stomata closure and seed germination. Here we report that the *HAS2* locus encodes the MITOCHONDRIAL EDITING FACTOR11 (MEF11), also known as LOVASTATIN INSENSITIVE1. *has2/mef11* mutants exhibited phenotypes very similar to the ABA-hypersensitive mutant, *hai1-1 pp2ca-1 hab1-1 abi1-2*, which is impaired in four genes encoding type 2C protein phosphatases (PP2C) that act as upstream negative regulators of the ABA signaling cascade. Like *pp2c, mef11* plants were more resistant to progressive water stress and seed germination was more sensitive to paclobutrazol (a gibberellin biosynthesis inhibitor) as well as mannitol and NaCl, compared with the wild-type plants. Phenotypic alterations in *mef11* were associated with the lack of editing of transcripts for the mitochondrial *cytochrome c maturation FN_2* (*ccmFN_2*) gene, which encodes a cytochrome c-heme lyase subunit involved in cytochrome c biogenesis. Although the abundance of electron transfer chain complexes was not affected, their dysfunction could be deduced from increased respiration and altered production of hydrogen peroxide and nitric oxide in *mef11* seeds. As minor defects in mitochondrial respiration affect ABA signaling, this suggests an essential role for ABA in mitochondrial retrograde regulation.
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