
Supplemental Table 1. Details of mutant lines used in this study. The principal characteristics of the lines are given, as well as the reference(s) in which the mutant and/or mutation was first reported or characterized.

- cat2* T-DNA knockout for CATALASE2.
Conditional photorespiratory oxidative stress mutant (Queval et al. 2007). Constitutive activation of salicylic acid pathway under oxidative stress conditions (Chaouch et al. 2010). Wild-type phenotype when grown under high CO₂.
- gr1* T-DNA knockout for GLUTATHIONE REDUCTASE1.
Wild-type phenotype in optimal conditions due to back-up NADPH-thioredoxin system for GSSG reduction (Marty et al. 2009), but *gr1* shows altered responses to oxidative stress (Mhamdi et al. 2010a).
- cad2* Deletion mutation in *GSH1* gene leading to loss of two amino acids in γ -GLUTAMYL-CYSTEINE SYNTHETASE. Identified in a screen for enhanced cadmium sensitivity, *cad2* has about 30% wild-type leaf glutathione contents (Howden et al. 1995; Cobbett et al. 1998). Wild-type phenotype in optimal growth conditions.
- rax1* Point mutation in *GSH1* gene leading to an amino acid substitution in γ -GLUTAMYL-CYSTEINE SYNTHETASE. Identified in a screen for altered high light responses, *rax1* has about 30-40% wild-type leaf glutathione contents (Ball et al. 2004). Wild-type phenotype in optimal growth conditions.
- pad2* Point mutation in *GSH1* gene leading to an amino acid substitution in γ -GLUTAMYL-CYSTEINE SYNTHETASE. Identified in a screen for decreased phytoalexin (camalexin) contents, *pad2* has about 20-25% wild-type leaf glutathione contents (Parisy et al. 2007). Wild-type phenotype in optimal growth conditions.
- sid2* Point mutation in ISOCHORISMATE SYNTHASE 1.
Identified in a screen for decreased induction of salicylic acid (Nawrath and Métraux 1999). Allowed ICS1 to be identified as major source of pathogen-induced salicylic acid production in Arabidopsis (Wildermuth et al. 2001). In response to bacterial challenge, salicylic acid stays close to basal uninduced Col-0 levels or below. Wild-type phenotype in optimal growth conditions.
- npr1* Point mutation in NONEXPRESSOR OF PATHOGENESIS RELATED GENES 1.
Identified in a screen for mutants that fail to induce pathogenesis-related genes (Cao et al. 1994). The NPR1 protein was subsequently shown to be thiol-regulated, with reduction allowing the protein to move into the nucleus from the

cytosol to induce *PR* genes (Mou et al. 2003). Wild-type phenotype in optimal growth conditions.

References to table

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