

# The role of redox-regulated TGA transcription factors in R-gene-mediated resistance

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The two closely related Arabidopsis basic leucine zipper (bZIP) transcription factors TGA1 and TGA4 are required for the establishment of effector-triggered immunity (ETI) after infection with *Pseudomonas syringae* pv. *tomato avrRPS4* (Pst avrRPS4) in a salicylic acid (SA) dependent manner. The *tga14* mutant plants show an increased growth of the pathogen in comparison to Col-0 wildtype plants. To evaluate the role of SA with respect to the susceptible phenotype, we crossed the *tga14* mutant plant with the *sid2-2* mutant plant, which cannot accumulate SA after pathogen attack. The triple ko plants *tga14/sid2-2* show no increased susceptibility in comparison to the susceptible single ko plant *sid2-2* after infection with Pst avrRPS4. From this we conclude an activity of TGA1 and TGA4 downstream of the SA signaling pathway. TGA1 and TGA4 transcription factors have two conserved cysteine residues at the positions 260/266 and 256/262, which are described as possible redox-regulated switches with regard to increased SA levels. Possible mediators of a redox-regulation of cysteine residues are glutaredoxins. Glutaredoxins are potential oxidoreductases and can transfer electrons from glutathione (GSH) to oxidized cysteine residues. After microarray analysis we identified ROXY9, a member of the glutaredoxins, as a possible target gene of TGA1 and TGA4. The mutant plants *tga14* show a dramatic decrease of ROXY9 expression. With Yeast-Two-Hybrid assays we can show a general interaction of TGA transcription factors with different glutaredoxins. To investigate a possible role of ROXY9 in ETI we infected different RNAi-lines of ROXY9 with Pst avrRPS4. The RNAi-plants show a *tga14*-like susceptible phenotype. From this we assume a connection of TGA1/TGA4 and ROXY9 with respect to ETI after Pst avrRPS4. To discover the function of the redox-regulated cysteine residues we complemented *tga14* mutant plants on the one hand with the wildtype TGA1 or TGA4 and on the other hand with mutated TGA1 or TGA4, which mimics a constitutive reduction of the cysteine residues. The constitutive reduced forms of TGA1 and TGA4 can complement the ROXY9 expression whereas the wildtype TGA1 and TGA4 cannot rescue the decreased expression.

These results hypothesize an autoregulatory loop of TGA1/TGA4 and ROXY9 with regard to expression and protein activation. The transcription factors TGA1 and TGA4 control the gene expression of ROXY9 and the ROXY9 protein modifies the conserved cysteine residues of TGA1 and TGA4 to activate them. In *tga14* mutant plants the lack of ROXY9 avoids an activation of the wildtype TGA1 and TGA4 and a complementation is not established.