

# 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 hypothesise 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.