Deficiency of TROL protein causes changes in chloroplast morphogenesis

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Keywords: FNR, metabolic signaling, photosynthesis

TROL (thylakoid rhodanese-like) is an intrinsic thylakoid membrane protein involved in tethering of a flavoenzime ferredoxin:$NADP^+$ oxidoreductase (FNR). FNR catalyses the final electron transfer of oxygenic photosynthesis from ferredoxin to $NADP^+$. The primary use of generated NADPH is in Benson-Calvin cycle, where it reduces 1,3-diphosho glycerate, forming glyceraldehyde-3-phosphate. Electron flow to the Benson-Calvin cycle is likely being controlled by this reaction, since the involved enzyme glyceraldehyde-3-phosphate dehydrogenase (GAPDH) is being activated via thioredoxin.

The interaction of FNR with TROL is accomplished via a C-terminal domain similar to a Tic62 protein family repeats. Inactivation of TROL in a model plant *Arabidopsis thaliana* causes reduction in relative electron transport rate (ETR) at light intensities exceeding 250 $\mu mol_{PHOTONS}\ m^{-2}s^{-1}$. Simultaneously, the amount of nonphotochemical quenching (NPQ) increases, indicating enhanced dissipation of absorbed light energy as heat. TROL deficient plants exhibit phenotype less prone to photooxidative damage, while accumulating lower amounts of anthocyanins. It appears that without the TROL photosynthetic machinery directs lesser amount of trapped energy to photochemical reactions, possibly because of inefficient synthesis of NADPH by the FNR. Analysis of global gene expression by using Affymetrix chip technology revealed 237 genes with modulated expression (q-value <0.001). These changes prompted us to investigate cellular ultrastructure, with a special emphasis on chloroplast morphogenesis.

The ultrastructure revealed about twice as many smaller chloroplasts with irregular morphology in TROL deficient plants as in wild-type (Figure 1.). These smaller chloroplasts contained no starch grains and resembled plastids found in younger tissues. Photosynthetic membranes were not abundant and they appeared to have more non-appressed regions. In every case, envelope membranes were continuous and seemed normally developed. Mitochondria appeared to be unaffected by the mutation in size and fine structure.

We conclude that changes in photosynthetic performance, caused by the deletion of TROL, can influence the metabolic state of chloroplasts. Inefficient synthesis of NADPH probably induces changes in a cascade of plastid-to-nucleus retrograde signaling, leading to alterations in nuclear gene expression. This, in turn, causes changes in chloroplast morphogenesis.

M.A. Pabst, G. Zellnig (Eds.): MC2009, Vol. 2: Life Sciences,
DOI: 10.3217/978-3-85125-062-6-222, © Verlag der TU Graz 2009

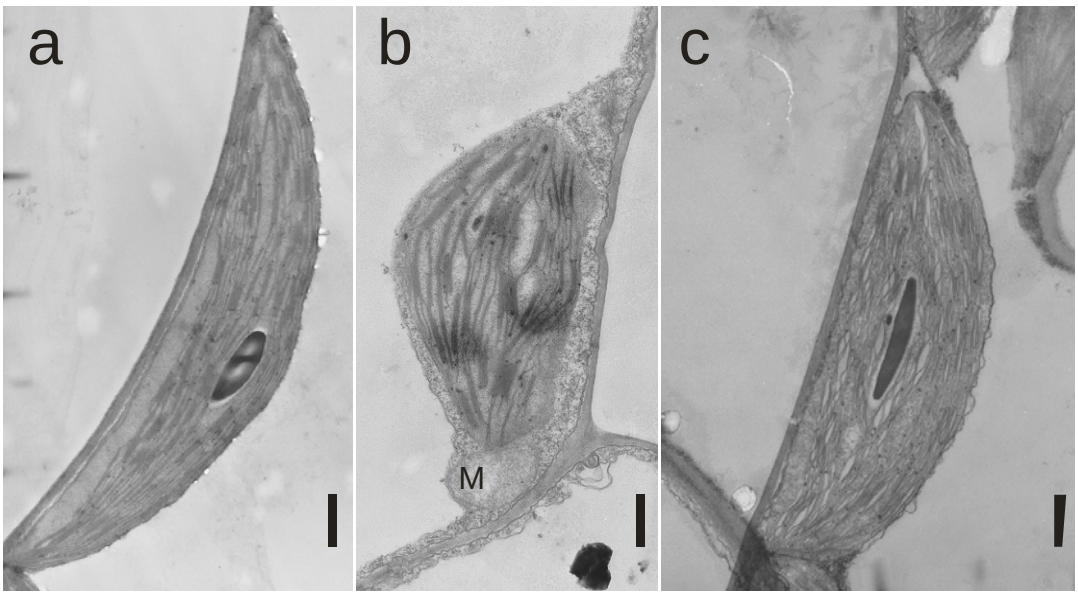

Figure 1. Chloroplast ultrastructure of plants grown under 80 $\mu mol_{PHOTONS}$ $m^{-2}s^{-1}$. a) chloroplast found in the wild-type, b) chloroplast found in TROL deficient plants, c) chloroplast found in plants where TROL accumulation was suppressed by antisense RNA expression. M - mitochondria. Scale bar = 1 μm.

	chloroplast area (in μm)
WT	9.33±1.17
KO	3.71±0.61
AS	6.01±1.23

Table 1. The surface of chloroplasts found in the wild-type (WT), TROL deficient plants (KO), and in plants where TROL accumulation was suppressed by antisense RNA expression (AS). Data are represented as mean +/- SEM

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