Drought stress induces upregulation of specific components that function in ferredoxin-dependent cyclic electron transfer

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In the linear electron flow of photosynthesis electrons originating from water are transferred via thylakoid-membrane embedded protein complexes Photosystem II (PSII), cytochrome b_6f complex (Cyt b_6f) and Photosystem I (PSI) to $NADP^+$, which is reduced to NADPH. Concomitantly, formation of a proton gradient and production of ATP takes place. In cyclic electron transfer (CET) electrons are circulated via PSI, Cyt b_6f complex and plastoquinone pool back to PSI. CET produces ATP without accumulation of NADPH. There are at least two possible routes for CET in higher plants. In ferredoxin (FD) or ferredoxin-plastoquinone reductase (FQR) –dependent route electrons are transferred via PSI and FD back to plastoquinone pool. PGR5 and PGRL1 proteins are the only known components that specifically participate in this route. FQR is a hypothetical enzyme, and according to some studies ferredoxin-NADPH-reductase (FNR) may participate in the FQR-route. NDH-dependent route cycles electrons through PSI to $NADP^+$ via FD and FNR. Electrons are then transferred further from NADPH to the plastoquinone pool via the NDH-complex.

In the present study we have shown that drought stress induces up-regulation of FD-dependent CET specific genes *PGR5* and *PGRL1* both at transcriptional and translational levels in *Arabidopsis thaliana* (1). In contrast, expression of *NDH-H* gene remained at the same level in drought stress when compared to control (1). This may suggest that drought stress induces CET via FQR-dependent route, and indeed drought stress accelerated the rate of $P700^+$ re-reduction in darkness (1). It was also shown that the expression of FNR leaf isoforms was slightly up-regulated at the transcriptional level, and that similarly to high light (2) there was a clear release of FNR from the thylakoid membrane upon drought stress (1). In our previous studies we have shown that neither FNR isoform seems to play a specific role in CET reactions (3,4). Moreover, also responses of other potential CET related gene families were studied.

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3. Lintala et al (2007) Plant J. 49, 1041-1052.
4. Lintala et al. (2009) Plant J. 57, 1103-1115.