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ARABIDOPSIS THALIANA GLYCERALDEHYDE-3-PHOSPHATE DEHYDROGENASE AS AN OXIDATIVE STRESS SENSOR

VESCOVI M.*, COSTA A.*, ZAFFAGNINI M.**, TROST P.**, LO SCHIAVO F.*

*) Department of Biology, Padova University, Via U. Bassi 58/B, 35131 Padova (Italy)
**) Laboratory of Molecular Plant Physiology, Department of Experimental Evolutionary Biology, University of Bologna, Via Irnerio 42, I-40126 Bologna (Italy)

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Glyceraldehyde-3-phosphate dehydrogenase (GAPDH) is a well known enzyme mainly involved in the glycolytic process. In mammalian cells, the GAPDH has been demonstrated also to play a role in the induction of apoptotic events. In particular, stimuli inducing oxidative stress have shown to induce nitrosylation of the GAPDH catalytic cysteine, leading to the enzyme inactivation and its relocalization into the nucleus, where it participates in the induction of apoptotic processes. In plants, both cytoplasmic and chloroplast GAPDH isoforms have been described, but up to now there are no evidences of their involvement in the induction of plant cell death events, even if nitrosylation of the same cysteine has been reported.

Cd^{2+} is a common environmental pollutant able to induce oxidative stress in plant cells with production of both reactive oxygen species and nitric oxide, leading to the induction of a senescence-like programme in cell cultures.

In order to investigate the possible involvement of plant GAPDHs in the Cd^{2+}-induced oxidative stress sensing, we focused on the *Arabidopsis* GAPC-1, one the two cytosolic GAPDH isoforms.

By performing *in vitro* analyses, using recombinant GAPC-1, we observed a reversible enzyme inactivation mediated by H_2O_2 and NO administration. The exposure of *Arabidopsis* seedlings to Cd^{2+} led to an accumulation of H_2O_2 and NO in roots where also an enhanced *GAPC-1* transcription and GAPC-1-YFP chimeric protein was detected, followed by its nuclear relocalization. In the *gapc-2* null mutant, where only the GAPC-1 enzyme is present, the Cd^{2+} stress determined an increase of GAPDH activity. Scavenging of H_2O_2 and NO in Cd^{2+} treated seedlings prevented the GAPC-1 accumulation.

Together these results support the hypothesis that the regulation of expression and activity of GAPC-1, in response to Cd^{2+}-induced oxidative stress, is mediated by the levels of H_2O_2 and NO in the cell that are directly sensed by the GAPC-1 enzyme.

We therefore propose that the GAPC-1 can be considered as an oxidative stress sensor.