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

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**GAPC, cadmium, oxidative stress, Arabidopsis thaliana**

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.

Cd²⁺ 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 Cd²⁺-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 H₂O₂ and NO administration. The exposure of *Arabidopsis* seedlings to Cd²⁺ led to an accumulation of H₂O₂ 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 Cd²⁺ stress determined an increase of GAPDH activity. Scavenging of H₂O₂ and NO in Cd²⁺ 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 Cd²⁺-induced oxidative stress, is mediated by the levels of H₂O₂ 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.