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REGULATION OF *ARABIDOPSIS GDH2* NUCLEAR GENE EXPRESSION DEPENDS ON FUNCTIONAL STATE OF MITOCHONDRIA AND CHLOROPLASTS

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The results of recent studies indicate that expression of some plant nuclear genes depends on functional state of mitochondria and chloroplasts. We have demonstrated that expression of *gdh2* gene encoding beta-subunit of mitochondrial glutamate dehydrogenase depends on a redox state of mitochondrial respiratory chain. Treatment of *Arabidopsis thaliana* cell culture with respiratory complex III inhibitor antimycin A or complex IV inhibitor KCN led to rapid increase of *gdh2* transcript content. Complex I inhibition by rotenone had no influence on the transcript level. We suggest that *gdh2* expression responds to changes of redox state of the respiratory chain segment located between complex I and complex III. We suppose that the revealed effect is not due to elevated generation of reactive oxygen species occurring upon the electron transport chain blockage, because cell treatment with hydrogen peroxide and paraquat did not lead to induction of *gdh2* expression. Experiments with *Arabidopsis* green seedlings have demonstrated that *gdh2* gene expression and GDH2 enzyme activity decrease strongly in the normal and high light conditions and increase in darkness. Resuming our experiments on different *Arabidopsis* organs and cell types we generalize that *gdh2* expression is maximal when both respiratory and photosynthetic electron transport chains are inhibited, and minimal when both of the electron transport chains are highly active. There are a number of hypotheses which would explain such a regularity. The first one proposes an energetic deficit as a regulatory factor initiating *gdh2* gene induction. We assume, however, that sugar starvation or ATP depletion cannot be the main factors in regulation of *gdh2* expression, because oxidative phosphorylation uncoupling by FCCP did not mimic the effects of antimycin A or prolonged dark treatment on the *gdh2* gene expression. The second hypothesis is developed for chloroplast-to-nucleus signaling and proposes that the regulatory signal can be initiated by redox state of plastoquinone pool and mediated by thylakoid membrane-bound protein kinases. We assume that similar mechanism would exist also in mitochondria-to-nucleus signaling, so that *gdh2* expression would depend on redox state of both ubiquinone and plastoquinone pools. This is confirmed by our experiments, in which the involvement of serine/threonine protein kinases in the antimycin-related *gdh2* induction was demonstrated as an ultimate step in transduction of the regulatory signal to the nucleus. Abscisic acid and/or pyridine nucleotides ratio changes can also participate in the retrograde regulation of *gdh2* gene expression. Thus, we have demonstrated that regulation of *gdh2* gene expression depends on mitochondrial and chloroplast functional state, and it can be considered as another example of retrograde regulation. The redox states of ubiquinone/plastoquinone pools are the most likely primary factors of this regulation type, which also involves serine/threonine protein kinases in signal transduction.

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