Volume 56(1) 75-92, 2012
Acta Biologica Szegediensis
http://www.sci.u-szeged.hu/ABS

Ascorbate, as alternative electron donor to photosystem II, protects plants against photoinhibition and stimulates the photoproduction of hydrogen in green algae

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Oxygenic photosynthetic organisms produce organic materials by using light energy and water as terminal electron donor. The water splitting enzyme, *i.e.* oxygen evolving complex (OEC) is one of the most vulnerable components of the photosynthetic electron transport chain. It has been shown (Tóth et al. 2007, Biochim Biophys Acta 1767: 295-305) that if the OEC is damaged, alternative electron donors, present in large amounts, donate electrons to photosystem II (PSII) in vivo. Our studies carried out on higher plant leaves and algal cells have shown that this alternative electron donor molecule is ascorbate; the rate of electron donation depends on the ascorbate content of leaves: $t_{1/2}$ is approximately 25 ms in wild type Arabidopsis plants and about 55 ms in ascorbate-deficient mutants (Tóth et al. 2009 Plant Physiol. 149: 1568-1578).

When OEC is damaged by heat stress highly oxidising components (Tyr_Z^+ and $P680^+$) accumulate in PSII in the light, leading to the fast inactivation of PSII. We have demonstrated that under these conditions ascorbate has a protective function by providing electrons to PSII and slowing down the harmful accumulation of Tyr_Z^+ and $P680^+$ (Tóth et al. 2011 Plant Physiol. 156:382–392).

Based on these results, it was reasonable to assume that ascorbate, by replacing the water splitting enzyme and supporting the electron transport without oxygen evolution, can enhance the photoproduction of hydrogen in *Chlamydomonas reinhardtii* cells. Photoproduction of hydrogen is known to depend on the activity of PSII; however, the oxygen evolution associated with PSII activity strongly inhibits the hydrogenase. It has earlier been shown that Chlamydomonas cells are able to evolve considerable amounts of hydrogen under anaerobic conditions following their sulphur deprivation, which suppresses their PSII activity (Melis and Happe 2001; 127:740–748). Our experiments have shown that the addition of 10 mM ascorbate to sulphur-deprived cell culture accelerates significantly the linear electron transport via PSII to PSI and to the hydrogenase, leading to a three-fold increase in hydrogen production. Similar results were obtained by using diphenylcarbazide (DPC), an artificial electron donor to PSII. The stimulation of hydrogen production was sensitive to diuron and dibromothymoquinone (inhibitors of PSII and the cytochrome b_6f complex, respectively), which proves that the enhancement of the hydrogen evolution by ascorbate and DPC can indeed be accounted for by their functioning as alternative PSII electron donors.

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Characterisation of host-pathogen interaction during *Candida* infections

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Candida species are known as members of the normal human flora. However under certain circumstances these commensalist yeasts are able to transform themselves into opportunistic pathogens. *C. parapsilosis* is considered to be the second or third most common *Candida* species causing candidiasis after *C. albicans*. The response of the mammalian immune system given to the *C. albicans* is well-exemined, and based on our pervious work it is clear, that some *Candida* derived lipases play role as virulence factor. On the other hand little is known on the interaction between the immune system and other *Candida* species, like *C. parapsilosis*.

We previously showed that *C. parapsilosis* lipase knockout (LIP-) mutants were significantly deficient in their capacity to produce biofilm, to grow in lipid rich medium, and to survive in macrophages. In an attempt to understand this reduced virulence phenotype, we developed an *in vitro* model system using murine macrophage -like cell line J774.2. We examined the gene expression in J774.2 macrophages infected with wild type (WT) *C. parapsilosis* and LIP- cells. The complex response of murine macrophages to infection with *C. parapsilosis* was investigated at the level of gene expression using Agilent mouse microarray. 155 and 512 genes were identified as being differentially regulated at 3 and 8 hours post infection, respectively. Most of the upregulated genes encoded molecules that were involved in immune response and inflammation, transcription, signalling, apoptosis, cell cycle, electron transport and cell adhesion. Of particular interest were the upregulation of proinflammatory cytokines, typical of the classically activated macrophages such as TNF, IL-1 and IL-15, and also the upregulation of TNF-receptor family members such as *TNFRSF9* associated with Th1 T-helper cell responses. Additionally, the microarray data indicate significant differences between the response to *C. parapsilosis* infection and that of *C. albicans*.

Flow cytometry analysis proved, that elevated mRNA level of TNFRSF9 correlated to the elevated amount of protein on the surface of J774.2 macrophage cells upon *C. parapsilosis* WT infection. Similar pattern of TNFRSF9 (CD137) regulation could be observed in cells from whole human blood upon *C. parapsilosis* WT infection. To further examine the host pathogen interactions we established a human monocyte (THP-1) cell line infection model. THP-1 cells were infected with eight different *Candida* strains from the parapsilosis *sensu lato*