Short Communication

Photoinhibition and Recovery of Photosynthesis in *Anacystis nidulans**

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It is well documented that photoinhibition of photosynthesis in visible light may occur under conditions where the photosynthetic antenna absorbs light in excess of the requirements of photosynthesis.[1] The general consensus is that the primary site of photoinhibition is at, or close to, the reaction centre of PS II,[1] but PS I may also be affected.[2] Kyle *et al.*[3] have recently provided evidence that one site of inhibition is at the Q_B-protein on the reducing side of PS II, but there is still no consensus on the molecular mechanism of photoinhibition. We showed in an earlier work[4] that the cyanobacterium *Anacystis nidulans* responds to photoinhibitory treatment with changes in function of the photosynthetic apparatus that are typical for higher plants.[5] By studying photoinhibition of photosynthesis and its recovery in *A. nidulans* in the presence and absence of transcription and translation inhibitors we[4] have obtained evidence for the operation of a repair process that efficiently restores photosynthesis in dim light. From the results of our work[4] we suggested that the extent of photoinhibitory damage observed is the net result of a balance between the photo-damage and the operation of a repair process.

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Materials and methods

The cyanobacterium *A. nidulans* 625 (*Synechococcus* 6301) was grown in batch cultures in an inorganic medium[6] as described earlier.[7] The cells were grown under aerobic conditions at a temperature of 38 °C, and the cultures used for experiments were always in the logarithmic phase of growth. The extinction coefficients used for determinations of chlorophyll and phycocyanin were those of Stevens and Myers.[8] Cell densities and growth were determined from the absorbance at 750 nm.[9] The photoinhibitory treatment was carried out in a glass water bath thermostatted to 38 °C. Photoinhibitory light was supplied by a halogen lamp (Osram, Power Star®, HQI-IS 400 W, Berlin, FRG). Cells were kept in tubes (10 ml) and the photon flux density was regulated by varying the distance of the lamp from the samples.

Photosynthetic oxygen evolution was measured using a Clark-type oxygen electrode. Prior to measurements, 10 mM HCO_3^- was added to the solution to avoid CO_2 deficiency at high light intensity. The extent of photoinhibition was determined by calculating the rate of light-limited photosynthesis, expressed as a percentage of a control value measured immediately before photoinhibitory treatment. The light source in this case was an Atlas projector lamp (A1, 215 24 V/150W).

Results and discussion

Photoinhibitory treatment of *A. nidulans* led to rapid inhibition of the photosynthetic O_2-evolution, followed by attainment of a quasi steady-state level (Fig. 1). The initial phase of photoinhibition was much more pronounced at a PAR (photosynthetically active radiation) of 1000 μmol m^{-2} s^{-1} than at 500 and 250 μmol m^{-2} s^{-1}. The quasi steady-state level was reached within about 30 min and the level decreased roughly proportionally with increasing PAR. When transferred to dim light (PAR 5 μmol m^{-2} s^{-1}) after a 30 min photoinhibitory treatment at a PAR of 500 μmol m^{-2} s^{-1}, *A. nidulans* reactivated its photosynthetic O_2-evolution to 80–95 % of the control activity within 1 h (Fig. 2). In the presence of the translational inhibitor streptomycin, added at the onset of reactivation after a 30 min photoinhibitory treatment, no reactivation occurred (Fig. 2). This clearly shows that protein synthesis is required for reactivation of photosynthesis after photoinhibition. This protein synthesis was insensitive to rifampicin, a transcription inhibitor (Fig. 2). This is an unexpected result and it implies either that the proteins are translated by stable mRNA(s) or that rifampicin does not inhibit the transcription of the operon(s) involved.
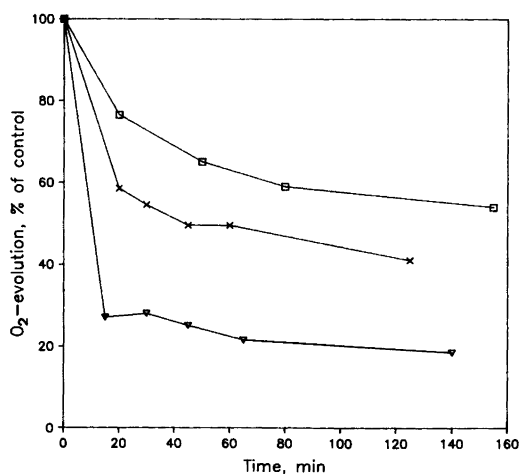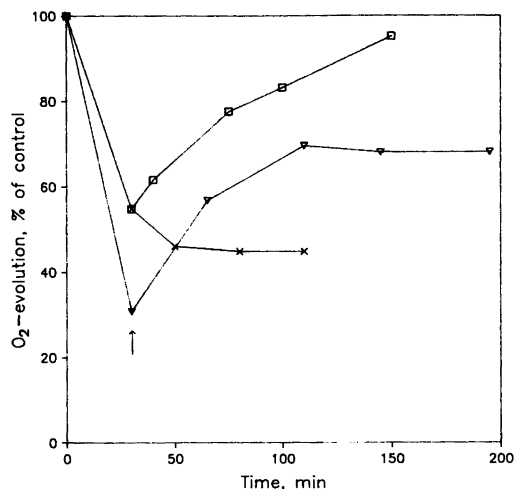

Fig. 2. Photosynthetic oxygen evolution (% of control) during photoinhibition and recovery vs. time. The arrow indicates the time of transfer to recovery conditions (PAR 5 μmol m^{-2} s^{-1}). Streptomycin and rifampicin were added to 250 μg ml^{-1}. $-\Box-$ Control; $-\times-$ + Strep.; $-\nabla-$ +Rif.

In order to obtain more information about this reactivation process, experiments were done with *A. nidulans* grown under low and high PAR (10 and 230 μmol m^{-2} s^{-1}), respectively. When samples of cells from the two lighting experiments were treated for 90 min in a gradient of PAR ranging from 10 to 600 μmol m^{-2} s^{-1}, cells grown in dim light became photoinhibited at much lower light levels than did cells grown in bright light (Fig. 3a). When a similar experiment was performed with the translation inhibitor streptomycin present during the incubation in light, a different pattern emerged (Fig. 3b): cells grown in both dim and bright light became severely inhibited even at relatively low PAR. In the presence of streptomycin, the light-dependence of inhibition was very similar for the two categories of cells. These results are consistent with the hypothesis that different capacities of the repair process determine the different susceptibilities to photoinhibition of photosynthesis of the two categories of cells. It was found that the rate of recovery was initially about 2.6 times higher for cells grown in bright light than for cells grown in dim light (data not shown). To further demonstrate the different rates of repair of the photoinhibitory damage to *A. nidulans* cells grown in
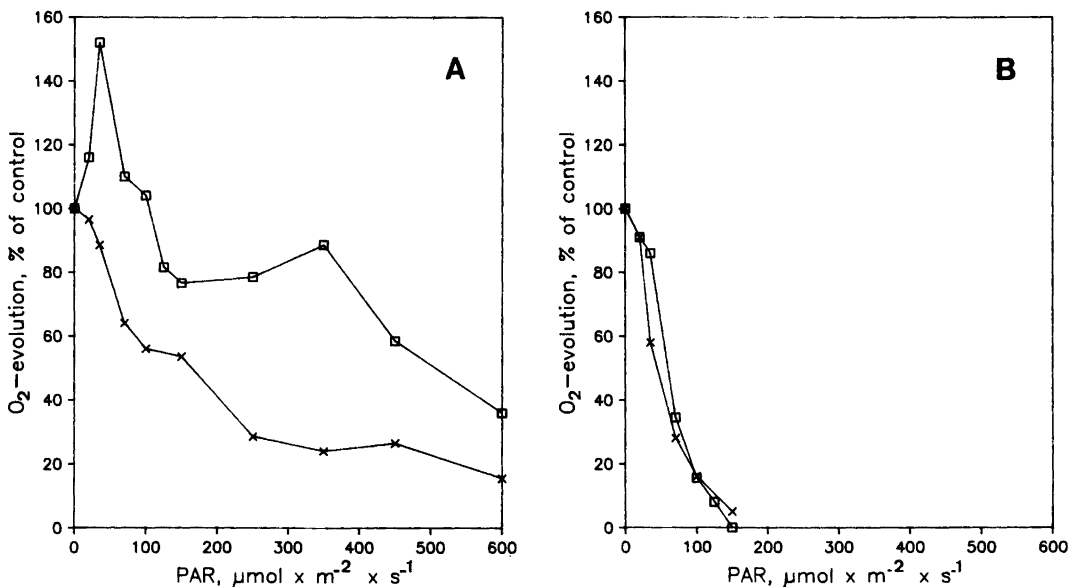

Fig. 1. Photosynthetic oxygen evolution (% of control) versus time of photoinhibitory treatment at indicated PAR. Control rate was 123 μmol (mg Chl)$^{-1}$ h^{-1}. Cells were grown at 50 μmol m^{-2} h^{-1}. $-\Box-$ 250 μmol m^{-2} s^{-1}; $-\times-$ 500 μmol m^{-2} s^{-1}; $-\nabla-$ 1000 μmol m^{-2} s^{-1}.

Fig. 3. Photosynthetic oxygen evolution (% of control) of cells grown in dim light (10 μmol m^{-2} s^{-1}) and bright light (120 μmol m^{-2} s^{-1}) during photoinhibition, without (A) and with (B) the translation inhibitor streptomycin (250 μg ml^{-1}). The time of photoinhibitory treatment was 90 min. $-\square-$ 120 μmol m^{-2} s^{-1}; $-\times-$ 10 μmol m^{-2} s^{-1}.

dim and in bright light they were photoinhibited so as to retain 60–70 % of their photosynthetic capacity. This was achieved by exposing cells grown in dim and in bright light to 130 and 450 μmol m^{-2} s^{-1}, respectively, for 90 min. Streptomycin was then added and it was found that the rate of photoinhibition became roughly 3 times higher for the cells grown in bright light than for those grown in dim light (Fig. 4). The results reported here strongly indicate that net photoinhibitory
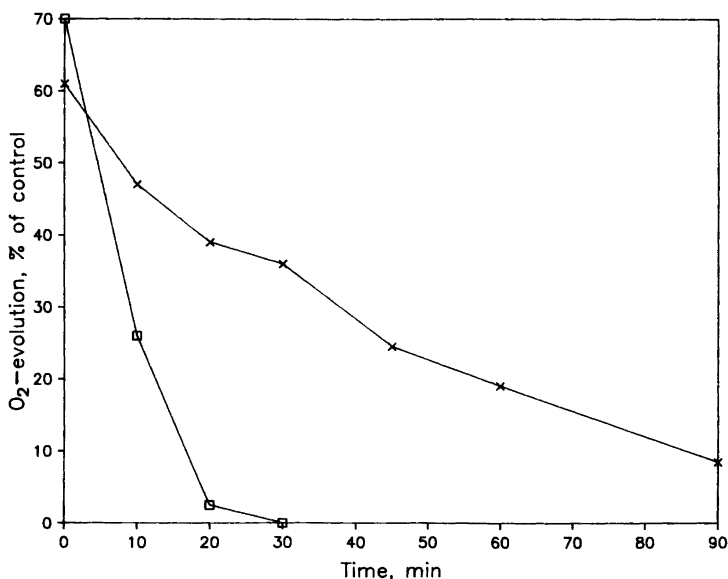

Fig. 4. Photosynthetic oxygen evolution (% of control) during photoinhibition when streptomycin was added after partial photoinhibition (ca. 40 %) of cells grown in dim light (10 μmol m^{-2} s^{-1}) and bright light (120 μmol m^{-2} s^{-1}). $-\square-$ 120 μmol m^{-2} s^{-1}; $-\times-$ 10 μmol m^{-2} s^{-1}.

damage results from a balance between the photoinhibitory process and the operation of the repair mechanism.

References

1. Powles, S. B. *Annu. Rev. Plant Physiol. 56* (1984) 15.
2. Satoh, K. and Fork, D. C. *Plant Physiol. 70* (1982) 1004.
3. Kyle, D. J., Ohad, I. and Arntzen, C. J. *Proc. Natl. Acad. Sci. U.S.A. 81* (1984) 4070.
4. Samuelsson, G., Lönneborg, A., Rosenqvist, E., Gustafsson, P. and Öquist, G. *Plant Physiol. 79* (1985) 992.
5. Ögren, E. and Öquist, G. *Physiol. Plant. 62* (1984) 193.
6. Siva, B., Rao, K., Brand, J. J. and Myers, J. *Plant Physiol., 59* (1977) 965.
7. Öquist, G. *Physiol. Plant. 22* (1969) 516.
8. Stevens, C. L. R. and Myers, J. *J. Phycol. 12* (1976) 99.
9. Samuelsson, G., Lönneborg, A., Gustafsson, P. and Öquist, G. *Plant Physiol. In press.*