

Diurnal Variations in the Kinetics of Delayed Luminescence from *Scenedesmus* Cells after Different Excitation Periods with Different Excitation Light in High- and Low-CO₂ Regimes*

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The photosynthetic capacity in *Scenedesmus* cells, measured by light-induced O₂ evolution, varies during the 24 h cell cycle.^{1,2} The delayed luminescence is the result of recombination reactions of photoinduced charge pairs in photosystem II (PS II), i.e. the positive charges of the S-states on the oxygen-evolving enzyme and the negative charges on the quinones QA and QB.^{3,4} The aim of this work was to examine whether there were any changes in the delayed luminescence decay kinetics that could be correlated with the photosynthetic capacity of the cells during cell development in the 24 h cell cycle.

Synchronously cultured cells of the green alga *Scenedesmus obtusiusculus* grown in solutions flushed with air containing 2.5% CO₂ (high-CO₂ cells) were harvested every second hour during the 15 h light and 9 h dark cell cycle. The cell samples were excited for 5 s or 30 s with white light. During the first 8 h of the cell cycle no transient peak in the delayed luminescence could be observed (Fig. 1). In cells sampled after 10–16 h, a transient peak in the delayed luminescence could be observed only after a 30 s excitation period. The early output of photons is low when a transient peak is formed later in the decay curve. Only a 30 s excitation period induced a transient

peak in low-CO₂ cells (cells equilibrated in normal air in light for 2 h) during most of the 24 h cell cycle (Fig. 1).

Light of wavelength 700 nm induced a transient peak in the delayed luminescence during the whole cell cycle except in cells sampled after 4–6 h (Fig. 2), whereas 680 nm light induced a shoulder or a weak transient peak except in cells sampled after 4–6 h. The formation of a transient peak in the delayed luminescence appears to be related to the photosynthetic activity. When the experimental conditions induce imbalances in the pools of intermediary compounds of the Calvin cycle, ATP formation will probably take place during subsequent darkness by reversal of some steps in the Calvin cycle (e.g. DPGA → PGA and RUBP → RUMP). The light-activated ATPase may hydrolyse ATP in darkness, creating a transthylakoidal ΔpH⁺ which increases the reversed electron flow to QA/QB and gives rise to luminescence as long as higher S-states occur on the enzyme catalyzing oxygen evolution. Inhibition of cyclic photophosphorylation by antimycin A or inhibition of ATP formation by the uncoupler CCCP abolishes the transient peak in delayed luminescence, as does N₂-flushing of the cells for 2 h in light.

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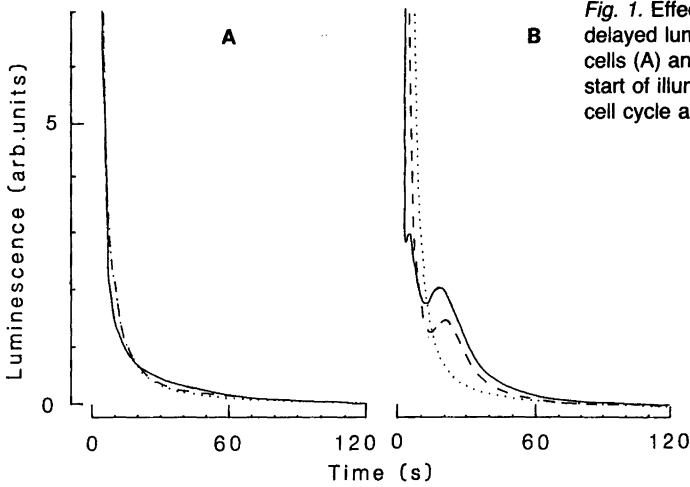


Fig. 1. Effects of white light excitation for 30 s on the delayed luminescence decay kinetics for high-CO₂ cells (A) and low-CO₂ cells (B) measured (—) at start of illuminated cell cycle, (---) after 2 h in the cell cycle and (···) after 4 h in the cell cycle.

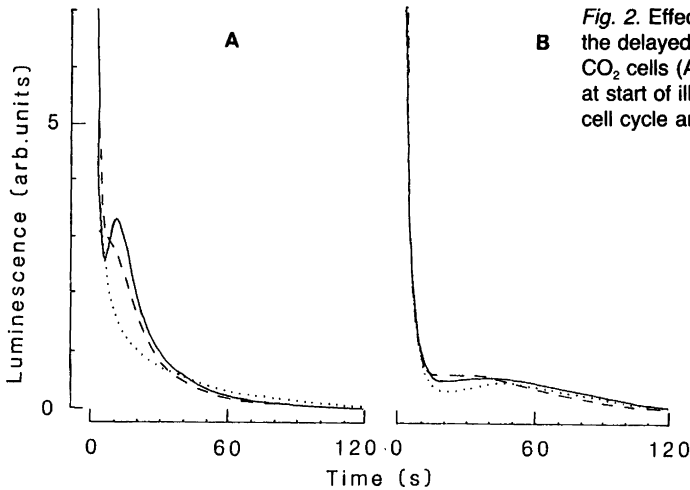


Fig. 2. Effects of 700 nm light excitation for 5 s on the delayed luminescence decay kinetics for high-CO₂ cells (A) and low-CO₂ cells (B) measured (—) at start of illuminated cell cycle, (---) after 2 h in the cell cycle and (···) after 4 h in the cell cycle.

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