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Protein–protein interactions in carotenoid triggered quenching of phycobilisome fluorescence in *Synechocystis* sp. PCC 6803

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Abstract An inquiry into the effect of temperature on carotenoid triggered quenching of phycobilisome (PBS) fluorescence in a photosystem II-deficient mutant of *Synechocystis* sp. results in identification of two temperature-dependent processes: one is responsible for the quenching rate, and one determines the yield of PBS fluorescence. Non-Arrhenius behavior of the light-on quenching rate suggests that carotenoid-absorbed light triggers a process that bears a strong resemblance to soluble protein folding, showing temperature-dependent enthalpy of activated complex formation. The response of PBS fluorescence yield to hydration changing additives and to passing of the membrane lipid phase transition point indicates that the pool size of PBSs subject to quenching depends on the state of some membrane component.

Keywords: Cyanobacteria; Fluorescence; Non-photochemical quenching; Phycobilisome; Protein folding

1. Introduction

Phycobilisomes (PBSs) are the main light-harvesting antennae in cyanobacteria, positioned on the cytoplasmic surface of the thylakoid membrane. Chemically, they are water-soluble phycobiliprotein complexes consisting of an allophycocyanin (APC) core and lateral cylinders made up of a combination of phycocyanin (PC) and phycoerythrin. Pigments of the lateral cylinders transfer the absorbed energy to the APC core, which incorporates two terminal chromophoric energy emitters. These components transfer the energy further onto antenna chlorophylls (Chls) of photosystems I and II (PSI, PSII) located within the thylakoid membrane [1]. It has been shown that PBSs are capable of relatively rapid lateral diffusion on the surface of the thylakoid membrane, and that the bond between PBSs and PSII (and probably PSI) is unstable [2,3], whereas assembled PBSs retain their membrane association even in the absence of PSII or PSI reaction centers [4]. It has been proposed that PBSs have no integral membrane component and connect to the membrane via multiple weak interactions with lipid head groups [5]. Although individual protein-lipid interactions are weak, multiple interactions lead to a strong association with the membrane surface.

Photosynthetic organisms are able to control the balance between absorbed and utilized excitation energy. The mechanism of dissipation of excess absorbed energy depends on the structure of the photosynthetic apparatus. Higher plants dissipate excess energy via non-photochemical quenching (NPQ), observed as a fluorescence decrease of the light-harvesting complex, LHCII [6,7]. Cyanobacteria are deficient in Chl-binding LHC complexes, and therefore energy discharge takes place via PSI and PSII antenna Chls [8]. PBSs themselves lack the capacity to dissipate excess absorbed excitation energy without assistance, which leads to very high fluorescence levels in isolated PBSs [9]. A new pathway of dissipation of excess energy was recently found in cyanobacteria. Illumination of *Synechocystis* cells with strong blue or white light induced fluorescence quenching that was independent from the membrane lipids phase transition temperature as well as from electron transport inhibitors and from ΔpH [10]. It was discovered that the energy absorbed by PBSs may be quenched via carotenoids, which appears as blue light induced quenching of APC fluorescence. The phenomenon of cyanobacterial NPQ was first observed for the PSII-deficient mutant of the *Synechocystis* sp. PCC 6803 [11], and then for the wild strain and for other mutants of the same cyanobacterium [12,13]. Since cyanobacteria are devoid of the violaxanthin cycle, they must possess a different mechanism to dissipate excess energy. Recent discussions revolve around participation of cyanobacterial 35 kDa water-soluble orange carotenoid-binding protein (OCP) in NPQ of PBS fluorescence [12,14]. The structural model of OCP, at 2.1 Å resolution, consists of two domains [15]. An embedded carotenoid, 3′-hydroxyechinenone (hECN) spans both protein domains. Binding to the protein shortens the singlet S_1 state lifetime of hECN by changing the conformation of the carotenoid, making dissipation of absorbed energy more efficient [16]. The protein has a strong effect on the spectroscopic characteristics of the carotenoid: the absorption spectrum of hECN in OCP [16] accurately matches the action spectrum of NPQ in PSII-deficient *Synechocystis* sp. cells [11].

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Abbreviations: APC (PC), allo(phycocyanin); Chl, chlorophyll; F_{-BL} (F_{+BL}), fluorescence yield before (after) blue light treatment; hECN, 3′-hydroxyechinenone; LHC, light-harvesting complex; NPQ, non-photochemical quenching; OCP, water-soluble orange carotenoid-binding protein; PSII (PSI), photosystem II (photosystem I); PBS, phycobilisome

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Our study focused on factors that may affect the protein solution phase at the thylakoid membrane interface. Behavior of the protein solution phase depends on many factors, including the solvent, the temperature, and the pressure. Literary sources describe opposite effects of glycerol and of ionic strength on protein's solubility and on intermolecular forces; attractions increase with ionic strength, whereas repulsions increase with glycerol concentration [17]. To reveal the mechanisms of carotenoid-triggered fluorescence quenching of PBSs, the effects of these variables on quenching have been investigated for the PSII-deficient mutant of the cyanobacterium *Synechocystis* sp. PCC 6803. Non-Arrhenius temperature dependence of the light-on process rate constant and a strong response of quenching to glycerol and sucrose are discussed here in terms of protein–protein interaction at the membrane interface.

2. Materials and methods

2.1. Strains and growth conditions

Growth conditions of the PSII-deficient (*psbDI/C/DII*$^-$) strain of the cyanobacterium *Synechocystis* sp. PCC 6803 are described in [11]. The special character of this mutant enabled us to avoid the variable fluorescence of PSII and to register fluorescence changes of PBSs. All experiments were performed with 5-day cultures in the log phase of growth.

2.2. Fluorescence measurements

Time courses of fluorescence quenching in *psbDI/C/DII*$^-$ cells were measured with a PAM-101 fluorometer (Walz, Germany) [13]. Cell suspensions (10 μg ml^{-1} Chl) were placed in a 1 mm-cuvette in a Shimadzu TCC-240A thermo-electrically temperature controlled cell holder connected to PAM-101 with optic fibers. Fluorescence level F_{-BL} was detected in dark adapted cells; then the cells were additionally exposed to quenching light (500 nm, 1000 μE m^{-2}/s) for a period of time sufficient to achieve a completely quenched fluorescence level (F_{+BL}). The time course of fluorescence in each temperature point was taken as the mean of 3–5 individual measurements. We calculated the rate constant (k) for the light-on process through exponential approximation of experimental kinetics ($R^2 > 0,98$). Our estimates of the light-off process rate constant are quite rough, as the time course of the light-off process has an S shape, or some lag, and can not be accurately described by first-order kinetics, however, we calculated k for the dark reversibility from half reaction time, $k = \ln 2/t_{1/2}$.

Fluorescence emission spectra from 580 nm excitation light were recorded at 15 °C with a Shimadzu RF-5301PC instrument [13]. The spectrum of every sample was measured twice: before (F_{-BL}) and immediately after (F_{+BL}) a 4-min exposure to quenching light (500 nm; photon flux density ca. 1100 μE m^{-2}/s).

3. Results

3.1. Glycerol influence

To analyse the role of PBS-membrane associations in blue light induced quenching, we looked at the effect of different amounts of glycerol. It was previously shown that 30% glycerol has significant effect on both the PC rods and the terminal emitter, which are located on the PBS surface, as glycerol increases hydration at the particle surface [18]. Fig. 1 shows fluorescence spectra before and after blue-light illumination of *psbDI/C/DII*$^-$ cells treated with glycerol at various concentrations. Addition of 20% glycerol caused a small increase of carotenoid-induced quenching of PBS fluorescence, while addition of 30% glycerol caused a small increase both of F_{-BL} and

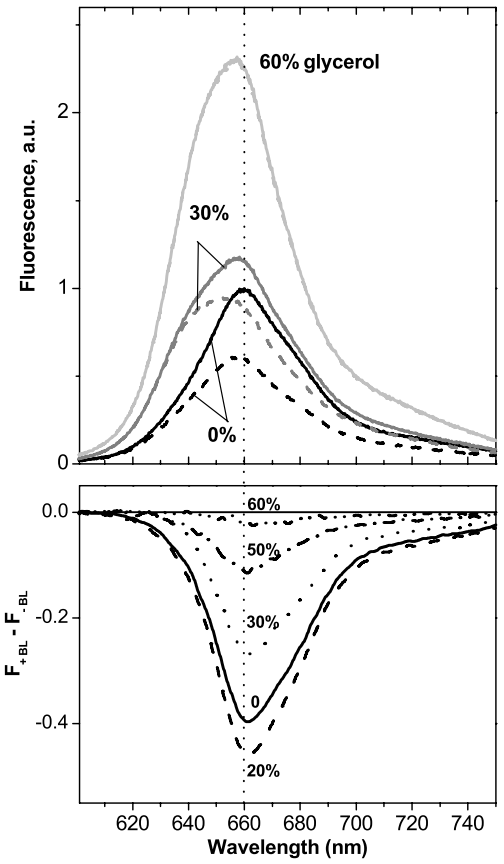

Fig. 1. Effect of glycerol on fluorescence emission spectra of *psbDI/C/DII*$^-$ cells before (solid) and after (dash) blue light illumination (A), and differential emission spectra ($F_{+BL} - F_{-BL}$) obtained at various concentration of glycerol (B). Concentration of glycerol (v/v %) shown in numeric labels.

F_{+BL}, and some decrease of quenching ($F_{-BL} - F_{+BL}$). An increase in dissociation of the PC rods from the PBSs was observed through the increased intensity of PC (650 nm) emission and a blue shift of PBS fluorescence band (Fig. 1, top). However, under glycerol concentration of up to 50%, carotenoid-absorbed light still induced a reversible fluorescence decrease with the constant APC maximum at 662 nm of the quenching spectra (Fig. 1, bottom). Thus, our data supports the assumption that it is the APC core fluorescence that is quenched in the blue light induced process [13,14]. High concentration of glycerol (>50%) completely inhibits light-induced quenching and doubles the level of initial fluorescence (Fig. 1). Taking into account the fact that blue light induced changes in the fluorescence yield were also absent in the *apcE*$^-$ mutant of *Synechocystis* [12,13], the loss of quenching we observed can be linked with PBSs uncoupling from thylakoid components. Similar reasoning was earlier employed to explain the similarity of the effect of high concentration glycerol treatment (60%) with that of deletion of *apcE* on energy transfer from the PBS to the pigments in the thylakoid membrane [19].

3.2. High osmotic pressure effect

It was previously shown that the NPQ seen in cells of the cyanobacterium *Synechocystis* sp. is strongly affected by a high osmotic strength media [20]. Sucrose – a substance causing a hyperosmotic stress [21] - affects reversibility of blue light in-

duced fluorescence quenching and the fluorescence level subjected to quenching (Fig. 2). Addition of sucrose to a final concentration of 0.3 M causes an about 40% decrease of the magnitude, but not of the rate (Fig. 2, inset), of blue light induced quenching, and blocks completely the dark recovery component of the time course. A higher amount of sucrose (0.7 M) cuts significantly both the magnitude (up to 75%) and the rate of quenching; no recovery was observed under these conditions. The final level of light-induced quenching (F_{+BL}) was independent from the presence of sucrose. Fluorescence emission spectra of the final level induced both by high amount of sucrose (not shown) and by blue light were identical. Similar behavior found under the effect of 1 M phosphate was interpreted as stabilization of PBSs on the membrane surface [20] via coupling of free PBSs to an intra-membrane complex. We think that PSI may be this intra-membrane complex in *psbDI/C/DII*$^-$ cells. Nevertheless PSI appears to be an unlikely agent of blue light induced carotenoid triggered quenching of PBS fluorescence, because there is no NPQ effect on energy migration from PBS to PSI in the *psbDI/C/DII*$^-$ cells (Rakhimberdieva, unpublished data).

3.3. Temperature dependence of quenching rate

Illumination of *psbDI/C/DII*$^-$ cells with blue light (500 nm) leads to a fast decrease of fluorescence characterized by mono-exponential kinetics (Fig. 3A), followed by a relatively fast and complete return of fluorescence to the initial level within about 15 min at 35 °C. Lowering the temperature slows down the rate of quenching and particularly its dark reversibility; very slow – but complete – reversibility was observed at 15 C and below. The temperature dependence of the rate constants are shown in Fig. 3B as an Eyring plot. The slope of the plot at any given temperature is the enthalpy of activation. Temperature independent activation enthalpy of 46 kJ/mol has been earlier reported for both induction and dark decay of the light-induced quenching process in the wild type of *Synechocystis* sp. [22]. We have calculated activation enthalpy to be 48 kJ/mol at 25 °C and 86.5 kJ/mol at 5 °C. High activation enthalpy in the whole temperature range seems to indicate that light-on quenching arises from large-scale protein conformational transitions, rather than from local rearrangements of pigments within some of protein domains, which have been shown for low temperature in plant LHCII [23]. The magnitude of activation enthalpy of dark recovery is still greater than that for the light-on process (>100 kJ/mol). The plot of the light-on (quenching) process (Fig. 3B, white circles) displays strong curvatures and is monotonous: experimental points can be well fitted polynomially (solid line) with $R^2 = 0.999$. Similar Non-Arrhenius temperature dependencies of rate constants are typical for soluble protein-folding processes [24,25], where the heat capacity increment correlates well with the hydrophobic surface exposure during the conformational transition [26].

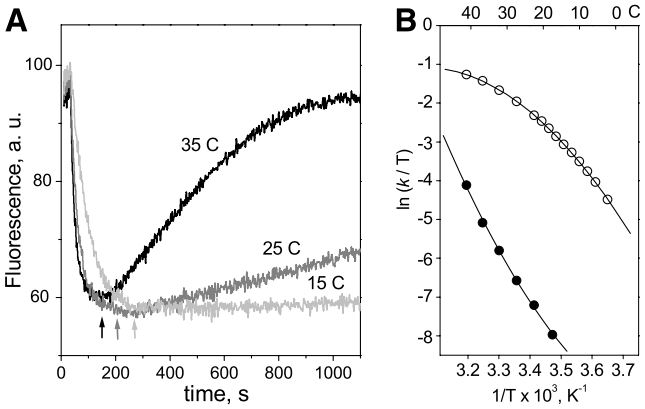

Fig. 3. Time courses (A) and Eyring plots of the rate (B) of PBS fluorescence quenching and its dark recovery in *psbDI/C/DII*$^-$ cells. Quenching was induced by blue light (500 nm, 1000 μE m^{-2}/s) at each particular temperature, and the quenching rate constant (k) was calculated as described in Methods for the light-on process (white circles) and for the light-off process (black circles); solid lines are second-order polynomial regressions for the experimental points, $R^2 > 0.99$.

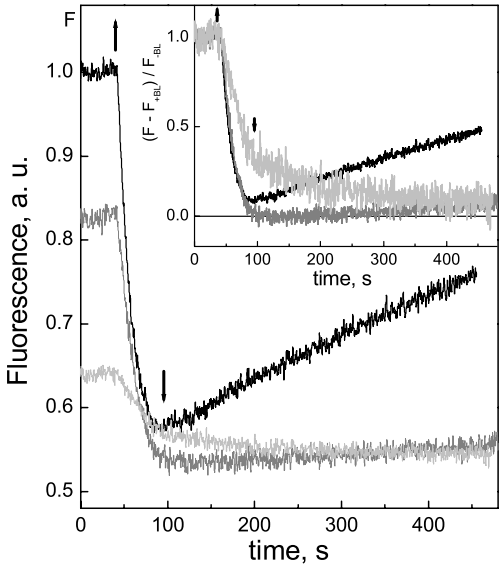

Fig. 2. Effect of 0.3 M (grey) and 0.7 M (light grey) sucrose on time courses of blue light induced quenching of PBS fluorescence and its dark recovery in *psbDI/C/DII*$^-$ cells; control shown in black. Inset: the same time courses normalized to the variable part of fluorescence.

3.4. Temperature dependence of fluorescence intensity

Not only the quenching rate, but both the initial (F_{-BL}) and the final (F_{+BL}) fluorescence levels are temperature dependent (Fig. 4A and B). As opposed to the smooth temperature dependence of the rate constants (Fig. 3B), the temperature dependence of the PBS fluorescence intensity (Fig. 4A) shows a break at 10 °C. This temperature point was shown to be the transition range of the membrane lipids of *Synechocystis* sp. [10]. Drastic changes of physiological activities in the cyanobacterial membranes are observed at phase transition temperatures [27]. The values of F_{-BL} and F_{+BL} of mutant cells show a similar temperature dependence and are characterized by an inflection point in the temperature range. The magnitude of variable fluorescence ($1 - F_{+BL}/F_{-BL}$) is roughly constant and shows no temperature dependence (Fig. 4C). This behavior of F_{-BL} and F_{+BL} suggests that PBS interaction with membrane components is responsible for the APC fluorescence yield, in spite of fact that the process of light-induced quenching of APC fluorescence is unrelated to membranes.

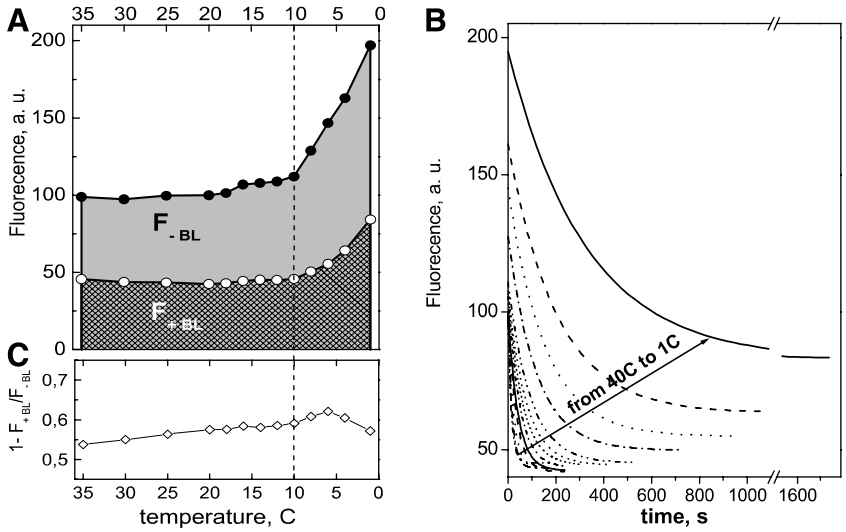

Fig. 4. Temperature dependence of PBS fluorescence yields before (F_{-BL}, black circles) and after (F_{+BL}, white circles) blue light illumination of *psbDI/C/DII*$^-$ cells (A), dependence of the relative magnitude of fluorescence quenching ($1 - F_{+BL}/F_{-BL}$) on temperature (C) and exponential approximations of experimental PBS fluorescence quenching time courses over the temperature range from 40 °C to 1 °C used to calculate *k* values in Fig. 3, as well as $F_{-BL}$and F_{+BL} (B).

4. Discussion

Although PBS fluorescence quenching does not depend on either the inhibitors of electron transport or on ΔpH [10,12], additives that change hydration have a strong effect on this quenching. This is a noticeable similarity between carotene-induced APC quenching and the processes in protein solutions. Temperature dependence of the rate of the blue light induced process amplifies the resemblance. The pattern is very characteristic of the temperature dependencies of soluble protein folding/unfolding rates. Simple (gas-phase) chemical reactions usually show linear Arrhenius behavior, whereas complex protein-folding reactions typically show curved plots. This is usually explained by invoking a change in heat capacity (DC_p) in the transition under study [26], which arises from the exposure of hydrophobic surface area to water, in the denatured state. The hydrophobic side chains are surrounded by 'icebergs' of water that melt with increasing temperature, thus making a large contribution to the heat capacity of the denatured state and a smaller one to the compact transition state for folding [28]. This type of temperature dependence was, for example, the basis for application of protein folding thermodynamics to modeling of the photocycle of the water-soluble photoactive yellow protein (PYP), which functions as the bacterial photoreceptor [25].

Osmotic regulation of exciton exchanges between donor holochromes (ApcD, ApcF, ApcE) of PBS cores and Chl *a* holochromes of PSI of thylakoid membranes was proposed [29]: PBSs in hyper-osmotic suspension deliver more excitation to PSI. We suggest that the PBS–PSI super-complex dissociation decreases in the hyper-osmotic cell suspension, which causes the highly fluorescent pool of uncoupled PBSs at the lipid surface to shrink. Only these mobile PBSs show blue light induced fluorescence quenching, since the PBS–PSI super-complex can only have a very low fluorescence yield because of the high rate of energy dissipation via P700 [30].

There is a noticeable pool of PBSs at the membrane surface in the *psbDI/C/DII*$^-$cells, which connects with the lipid surface only by PBS-lipid interactions. High fluorescence of this PBS pool is quenched via interaction of their APC core and the blue light activated OCP; the process follows the soluble protein folding/assembly pathway. However, the loss of carotenoid-triggered quenching of APC fluorescence under high concentrations of glycerol and the absence of this type of quenching in the *apcE*$^-$ mutant [12] appear to call for PBS fixation on the membrane interface to enable reaction with the OCP. Most likely, the role of PBS-lipid interactions is to localize and to concentrate the unbound PBSs at the lipid surface in order to facilitate protein–protein associations involved in the formation of the PBS–OCP complex. The pool size is probably driven by the state change of some membrane components via variation of the PBS–PSI super-complex dissociation.

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