Diffusion of Phycobilisomes on the Thylakoid Membranes of the Cyanobacterium Synechococcus 7942

EFFECTS OF PHYCOBILISOME SIZE, TEMPERATURE, AND MEMBRANE LIPID COMPOSITION*

Mary Sarcina‡, Mark J. Tobin§, and Conrad W. Mullineaux¶

From the ‡Department of Biology, University College London, Darwin Building, Gower Street, London WC1E 6BT, United Kingdom and §Central Laboratory of the Research Councils, Daresbury Laboratory, Daresbury, Warrington, Cheshire WA4 4AD, United Kingdom

Phycobilisomes are the major accessory light-harvesting complexes of cyanobacteria. They consist of large highly ordered aggregates of phycobiliproteins and linker proteins attached to the cytoplasmic surface of the thylakoid membrane (for review see Refs. 1 and 2). The structures of some phycobiliproteins and phycobilisome subcomplexes are known to high resolution (1, 2). However, in structural terms we know little about the association of phycobilisomes with reaction centers and with the thylakoid membrane. Phycobilisomes have often been assumed to act as the light-harvesting antennae for photosystem II alone. However, energy transfer studies on a photosystem II-free mutant (3) and wild-type cells (4) indicate that phycobilisomes can also interact and efficiently transfer energy to photosystem I. Up to approximately half of the light energy absorbed by phycobilisomes may be transferred to photosystem I, depending on the adaptation of the cells (3). Specific components of the phycobilisome cores are required for energy transfer to photosystem I as they are for photosystem II (5). These results imply that phycobilisomes can interact structurally with photosystem I as well as with photosystem II.

The association of the phycobilisomes with the membrane does not depend on the presence of reaction centers. In vivo, phycobilisomes are assembled and are membrane-associated even when no reaction centers are present (6). The nature of the association of phycobilisomes with membranes is unclear. Phycobilisomes are largely composed of hydrophilic protein subunits (1, 2). The ApcE protein, a high molecular weight component of the phycobilisome core, seems to play a crucial role in phycobilisome-membrane association (7, 8). However, sequence analysis did not reveal any likely membrane-spanning domain (9), although phase partitioning suggests a hydrophobic region on the protein (10). It has been suggested that membrane association may be mediated by an acyl group attached to ApcE (11). However, the deletion of the "PB-loop", the main candidate for a membrane-association domain, had no obvious effect on phycobilisome assembly or function (12). Phycobilisomes in vitro dissociate into their component subunits, unless they are maintained in quite specific high ionic strength buffers (13). Thus, it is difficult to explore the nature of phycobilisome-membrane coupling in vitro by changing the ionic strength of the medium. Phycobilisomes remain functionally coupled to membranes or reaction centers in vitro in a high phosphate medium (14), and detergent treatment is generally required to separate intact phycobilisomes from thylakoid membranes (15).

We have previously used fluorescence recovery after photobleaching (FRAP)† to probe the mobility of phycobilisomes in vivo in the elongated cyanobacterium Dactylococcopsis salina (16). The technique involves the use of a highly focused confocal laser spot to bleach the fluorescent pigments in a line across the cell. Subsequent changes in the bleaching pattern indicate diffusion of the pigment-protein complexes and may be used to calculate the diffusion coefficient (16). Surprisingly, we found that phycobilisomes diffused rapidly on the membrane surface, whereas photosystem II was immobile (16). The result implies

Received for publication, July 26, 2001, and in revised form, September 18, 2001
Published, JBC Papers in Press, October 4, 2001, DOI 10.1074/jbc.M107111200

* This work was supported by a Biotechnology and Biological Sciences Research Council research grant (to C. W. M.) The costs of publication of this article were defrayed in part by the payment of page charges. This article must therefore be hereby marked “advertisement” in accordance with 18 U.S.C. Section 1734 solely to indicate this fact.

† To whom correspondence should be addressed. Tel.: 44-20-7679-2326; Fax: 44-20-7679-7096; E-mail: c.mullineaux@ucl.ac.uk.

‡ From the Department of Biology, University College London, Darwin Building, Gower Street, London WC1E 6BT, United Kingdom and Central Laboratory of the Research Councils, Daresbury Laboratory, Daresbury, Warrington, Cheshire WA4 4AD, United Kingdom.

¶ To whom correspondence should be addressed. Tel.: 44-20-7679-2326; Fax: 44-20-7679-7096; E-mail: c.mullineaux@ucl.ac.uk.

‡ From the Department of Biology, University College London, Darwin Building, Gower Street, London WC1E 6BT, United Kingdom and Central Laboratory of the Research Councils, Daresbury Laboratory, Daresbury, Warrington, Cheshire WA4 4AD, United Kingdom.
that there are no stable phycobilisome-reaction center complexes (16, 17).

Here we report studies of the mobility of phycobilisomes in the cyanobacterium *Synechococcus* sp. PCC7942. This organism has thylakoid membranes arranged as regular concentric cylinder aligned along the long axis of the cell (18), a favorable configuration for our FRAP technique (16, 17). *Synechococcus* 7942 is well characterized and transformable, making it an excellent model system for our studies. The availability of mutants allows us to probe the factors affecting the mobility of the phycobilisomes. We find that phycobilisomes diffuse slightly faster in a mutant in which the phycobilisome rod elements are missing (19), indicating that the size of the phycobilisomes plays a role in determining their rate of diffusion.

The temperature dependence of the phycobilisome diffusion coefficient suggests that the phycobilisomes have no integral membrane domain. In a mutant in which the unsaturation of fatty acids in the thylakoid membrane lipids is increased (20), resulting in increased thylakoid membrane fluidity, there is a large and unexpected decrease in phycobilisome mobility, which suggests that lipids play a crucial role in controlling phycobilisome-reaction center interaction. We discuss the possible physiological role(s) of phycobilisome mobility.

**EXPERIMENTAL PROCEDURES**

**Growth of Cells—** *Synechococcus* sp. PCC7942 was grown in BG11 medium (21) supplemented with 10 mM NaHCO₃. For the R2HECAT mutant (19), the growth medium was supplemented with chloramphenicol at 5 µg/ml, and for the desA transformant (20), the medium was supplemented with kanamycin at 50 µg/ml. Liquid cultures were grown in an orbital shaking incubator at 30 °C with white illumination at approximately 10 microeinsteins m⁻² s⁻¹. For use in FRAP measurements, the cells were elongated by treatment with thiobendazole, a treatment that resulted in increased mean cell length without any detectable alteration in photosynthetic function (22).

**FRAP Measurements—** FRAP experiments were carried out at Central Laboratory of the Research Councils Daresbury Laboratory using the scanning confocal microscope Sylopt with a 633-nm helium-neon laser or a 442-nm helium-cadmium laser (16). Fluorescence was selected using a Schott RG665 red glass filter, transmitting light at above approximately 665 nm. Under these conditions, excitation with 442-nm light allowed observation of fluorescence predominantly from photosystem II, and excitation with 633-nm light allows observation of fluorescence predominantly from phycobilisome cores (16). Cells were spread on 1.5% agar containing BG11 growth medium covered with a glass coverslip and placed on a temperature-controlled stage under the microscope objective lens. A ×40 oil immersion lens (numerical aperture 1.3) was used with 20 µm pinholes to create a confocal spot with full-width at half-maximum dimensions of approximately 0.9 µm in the Z-direction and 0.3 µm in the XY-plane.

Cells aligned in the Y-direction were selected. The confocal spot was scanned for approximately 1 s in the X-direction across the middle of the cell to create the bleach. The confocal spot was then scanned in the XY-plane to record a sequence of images of the cell at 3-s intervals.

**Data Analysis—** Images were analyzed using Optimas software (Optimas Corporation). A one-dimensional bleaching profile was extracted by integrating across the cell in the X-direction. The base-line fluorescence from the unbleached cell was subtracted, and the bleaching profile was then fitted to a Gaussian curve using SigmaPlot (Jandel Scientific) to obtain a measurement of the bleach depth. Diffusion coefficients were obtained by plotting maximum bleach depth versus time using Equation 1,

\[
C(t) = C_0 [R(t)^2 + S D t]^{-1/2}
\]

where \(C(t)\) is the bleach depth at time \(t\), \(C_0\) is the initial bleach depth, \(R_0\) is the initial half-width \((1/e)^2\) of the bleach, and \(D\) is the diffusion coefficient. The equation describes one-dimensional diffusion in the case where the initial bleaching profile has a Gaussian form (16).

**Spectroscopy—** Absorption spectra were recorded in an Aminco DW2000 spectrophotometer. Fluorescence emission spectra were recorded at 77 K in a Perkin-Elmer LS550 luminescence spectrometer fitted with a liquid nitrogen sample housing. Cells (5 µm chlorophyll in BG11 medium) were injected into 4-mm diameter silica tubes and dark-adapted for 10 min at 30 °C before freezing in liquid nitrogen. The excitation and emission slit-widths were 5 nm.

**RESULTS**

**Mobility of Phycobilisomes and Photosystem II in Synechococcus 7942—** The chlorophylls and phycobilin pigments of cyanobacteria can be selectively excited, allowing the phycobilisomes or the photosystem II reaction centers to be visualized in fluorescence images (16). Figs. 1 and 2 show FRAP images sequences showing the mobility of photosystem II and phycobilisomes, respectively. In the case of photosystem II (Fig. 1), we could detect no diffusion on the timescale of the measurement. The same result was observed in *D. salina* (16). However, phycobilisomes diffused rapidly (Fig. 2). At 30 °C, the average diffusion coefficient for phycobilisomes was \((3.1 \pm 1.0) \times 10^{-10} \mathrm{~cm}^2 \mathrm{s}^{-1}\). The diffusion coefficient is comparable, although slightly faster than that previously measured in *Dactylococcus* (16). *Synechococcus* cells were much smaller than those of *Dactylococcus*, therefore, the experiment was carried out on a smaller scale with a bleach width of approximately 1–2 µm rather than 5–7 µm in *Dactylococcus* (16). The recovery of fluorescence was much faster in *Synechococcus* with 90% recovery typically occurring in approximately 5 s rather than 1–2 min as in *Dactylococcus*. However, the different time scales reflect the much smaller scale of the *Synechococcus* experiment rather than any major difference in the diffusion coefficient.

**Effect of Phycobilisome Size—** We have explored the effect of the phycobilisome size by measuring the phycobilisome diffusion coefficient in a mutant lacking the phycobilisome rod elements. The mutant R2HECAT (19) lacks the cpeA, cpeB, cpeD, cpeE, cpeF, cpeH, and cpeI genes coding for the α- and β-subunits of phycocyanin and rod-linker polypeptides (19). However, the phycobilisome cores are still assembled and functional (19). The phycobilisome cores have a molecular mass of 1200–1300 kDa (19) and dimensions of approximately 22 × 11 × 12 nm (23). The intact phycobilisomes of wild-type cells are hexadiscoidal structures with a typical molecular mass of approximately 6000 kDa and typically a longest diameter of approximately 60 nm (23).

At 30 °C, the mean diffusion coefficient for the phycobilisome cores in R2HECAT was \((7.1 \pm 0.8) \times 10^{-10} \mathrm{~cm}^2 \mathrm{s}^{-1}\). This result compares with a mean diffusion coefficient of \((3.1 \pm 1.0) \times 10^{-10} \mathrm{~cm}^2 \mathrm{s}^{-1}\) in the wild-type. This is a significant difference (Student's t test, \(p = 0.0062\)). Thus, phycobilisome diffusion at growth temperature is faster by a factor of 2.3 ± 0.7 in the R2HECAT mutant.
Effect of Temperature—The temperature dependence of the diffusion coefficient for phycobilisomes provides a way to explore the interaction of the phycobilisomes with the membrane. Upon cooling below normal growth temperature, all biological membranes undergo a lipid phase transition from the liquid-crystalline to the gel phase. In the thylakoid membranes of *Synechococcus* 7942, this transition occurs at approximately 10 °C below growth temperature, i.e. at around 20 °C (24). We have used FRAP with a lipid-soluble fluorescent marker to show that phase transition decreases the lipid diffusion coefficient by a factor of approximately 6 upon cooling from 30 to 20 °C or below.2 Phase transition would be expected to have a major effect on the lateral mobility of integral membrane proteins. However, we found that temperature does not have a strong influence on the diffusion coefficient of phycobilisomes (Fig. 3). Upon cooling from 30 to 10 °C, there is no significant change in the phycobilisome diffusion coefficient. At the intermediate temperature of 20 °C, phycobilisome diffusion is slightly faster by a factor of 2.1 ± 0.7 (Fig. 3).

Effect of Lipid Desaturation—Mutants in which the thylakoid membrane lipid composition is altered provide a further opportunity to explore the interaction between phycobilisomes and membranes. We have used desA⁺, a transformant of *Synechococcus* 7942 that contains desA, the Δ12 fatty acid desaturase gene from *Synechocystis* 6803 (20). As a result, desA⁺ cells contain approximately 40% 16:2/16:0 and 18:2/16:0 glycerolipids, whereas both species are absent in the wild-type (20). Fatty acid desaturation would be expected to increase the fluidity of the membrane. We have used FRAP with a fluorescent lipid-soluble marker to show that the lipid diffusion coefficient at 30 °C is approximately six times faster in desA⁺ cells than it is in the wild-type.2 The effect of desA on phycobilisome diffusion is shown in Figs. 3 and 4. Unexpectedly, we found that phycobilisome diffusion was far slower in desA⁺ than in the wild-type (Figs. 3 and 4). At 30 °C, the mean phycobilisome diffusion coefficient in desA⁺ was (2.5 ± 1.2) 10⁻¹² cm² s⁻¹, slower than in the wild-type by a factor of 120 ± 70 (Fig. 3). Cooling the cells to 20 or 10 °C led to an increase in the phycobilisome diffusion coefficient in desA⁺ (Fig. 3).

Light-harvesting Function in desA⁺—Absorption spectra for wild-type and desA⁺ cells indicated no significant changes in the content of chlorophyll and phycobilins (data not shown). Fluorescence emission spectra recorded with chlorophyll excitation (435 nm) provide an indication of the photosystem II/photosystem I ratio (25). Again, there were no significant differences between wild-type and desA⁺ cells. However, desA⁺ differed significantly from the wild-type in fluorescence emission spectra recorded with phycocyanin (600 nm) excitation (Fig. 5). As compared with the wild-type, desA⁺ showed a blue shift in the phycocyanin fluorescence peak at approximately 650 nm and a marked increase in fluorescence at 680 nm. This peak comes both from photosystem II and from long wavelength pigments in the phycobilisome core. A specific increase in this peak with no comparable increase in the photosystem II peak at 695 nm suggests a decrease in the efficiency of energy transfer from phycobilisomes to reaction centers (5).

**DISCUSSION**

**Mobility of Phycobilisomes and Photosystem II**—Our results show that phycobilisomes in *Synechococcus* 7942 diffuse rap-
Phycobilisome Diffusion

46833

idly, whereas the photosystem II reaction centers are immobile (Figs. 1 and 2). We previously found the same pattern in another cyanobacterium, D. salina (16). In qualitative terms we have found the same pattern in other cyanobacteria, including Synechocystis 6803 (data not shown), although the small size and unfavorable membrane conformation of Synechocystis means that we cannot measure diffusion coefficients accurately in this cyanobacterium. The results indicate that the association between phycobilisomes and reaction centers is transient and unstable (16, 17, 26), and that this is generally the case in cyanobacteria. The phycobilisome diffusion coefficients measured in Synechococcus (Fig. 3) and Dactylococcopsis (16) are not very different; the mean diffusion coefficient at growth temperature is faster by a factor of approximately 2 in Synechococcus.

Diffusion of Intact Phycobilisomes or Detached Rod Elements—We have interpreted our FRAP results in terms of the movement of intact phycobilisomes (16, 17, 26), because we excite the phycobilisomes with short wavelength light predominantly absorbed by phycocyanin in the phycobilisome rods and observe long-wavelength fluorescence predominantly from the phycobilisome cores (16). However, the spectral overlap makes it hard to completely exclude an alternative possibility that the phycobilisome cores are immobile and that the diffusion we see is of rod elements, which may not be stably coupled to the phycobilisome cores in vivo. We have further investigated this problem using a mutant (R2HECAT), which lacks phycobilisome rods but contains stably assembled and functional phycobilisome cores (19). We find that the phycobilisomes are mobile in this mutant. Because the rod elements are lacking, the cores must be moving. Thus, the diffusion we observe in the wild-type is most probably that of intact fully assembled phycobilisomes.

Effect of Phycobilisome Size—The diffusion of many membrane-associated proteins is strongly influenced by the interactions in the cytoplasmic domain. There are several proteins in which truncation of the cytoplasmic domain leads to an increased lateral diffusion coefficient (27). Both specific and nonspecific interactions with cytosolic components can contribute to a drag, which reduces lateral mobility (27). Cytosolic crowding is likely to have a major influence on the diffusion rate of any component of the cytoplasmic domain with the diffusion of large molecules being impeded more than that of small molecules (28). Phycobilisomes are large protein complexes predominantly in the cytoplasm. They are present in high concentrations covering much of the cytoplasmic surface of the thylakoid membrane (29). The R2HECAT mutant (19) gives us the opportunity to investigate the extent to which phycobilisome mass, crowding, and drag in the cytoplasmic phase influence the rate of diffusion of the phycobilisomes. Because the mutant contains only phycobilisome cores, the phycobilisomes are effectively approximately three times smaller in terms of longest diameter and five times smaller in terms of mass (23). We find that at growth temperature the phycobilisomes diffuse slightly faster in R2HECAT than in the wild-type; the mean diffusion coefficient is faster by a factor of 2.3 ± 0.7. Thus, we can observe a small but significant effect of reducing the phycobilisome size.

How Do Phycobilisomes Interact with the Membrane?—Phycobilisomes interact with the membrane whether or not reaction centers are present. Phycobilisomes are assembled and are membrane-associated even in the absence of photosystem II (30), photosystem I (31), or both reaction centers (6). In the latter case, detergent treatment is still required to isolate intact phycobilisomes, indicating a strong interaction with the membrane (6). Thus, when phycobilisomes diffuse, we imagine them decoupling from a reaction center but remaining attached to the membrane surface. The phycobilisome will then diffuse freely on the membrane surface before coupling to another reaction center (16, 17). However, the nature of the interaction of the phycobilisomes with the membrane is unclear. The ApcE protein or “anchor polypeptide” of the phycobilisome core is implicated in the interaction with the membrane (7–9). Proposals for the association of ApcE with the membrane have included an integral membrane domain (7) or a covalently attached acyl group (11). However, we found that cooling below the phase transition temperature of the membrane had no significant effect on the mobility of phycobilisomes (Fig. 3). Under the same conditions, the diffusion coefficient of a lipid-soluble fluorescent marker was reduced by a factor of six.2 This finding strongly suggests that there is no integral membrane component of any kind in the phycobilisome. Instead, we propose that phycobilisomes interact with lipid head groups at the membrane surface. A precedent for this kind of interaction is spectrin, a component of the erythrocyte cytoskeleton. Spectrin is proposed to interact with the membrane via multiple weak interactions with lipid head groups (32). As with phycobilisomes, spectrin can diffuse rapidly on the membrane surface, and the diffusion coefficient is not strongly affected by cooling to the phase transition temperature of the membrane (32). Although the individual protein-lipid interactions are weak, multiple interactions lead to a strong association with the membrane surface (32).

Effect of Lipid Desaturation: A Role for Lipids in Controlling Phycobilisome-Reaction Center Coupling—We have measured the rates of phycobilisome diffusion in cells of the desA mutant, which contains approximately 40% of glycerolipids with diunsaturated acyl chains, species that are absent in the wild-type (20). This process results in a lower phase transition temperature and a more fluid thylakoid membrane. Unexpectedly, we found that phycobilisome diffusion was slower in desA, by a factor of approximately 120 at 30 °C (Figs. 3 and 4). This cannot be a direct consequence of the membrane fluidity change, because the membrane is more fluid in desA+. Therefore, the most likely explanation is that the interaction with the reaction centers is stabilized in desA+. We know that photosystem II is immobile (Fig. 1). Therefore, if the binding of phycobilisomes to photosystem II is stabilized, the diffusion coefficient for phycobilisomes will be reduced. Changes in low temperature fluorescence emission spectra (Fig. 5) also suggest an altered phycobilisome-reaction center interaction in desA++. How could lipid desaturation alter phycobilisome-reaction center interaction? Perhaps specific lipids or the general lipid environment of the membrane play a crucial role in mediating phycobilisome-reaction center interaction. It is also possible that the effect is indirect, the change in phycobilisome-reaction center coupling may be a physiological response to some alteration in conditions in the desA+ cells. Whichever possibility is correct, it is clear that the lipid composition of the membrane strongly influences the interaction of phycobilisomes with reaction centers. It would be very interesting to observe the diffusion of phycobilisomes in mutants lacking photosystem II or photosystem I reaction centers. We would predict that phycobilisomes should diffuse faster in such mutants. Unfortunately, such mutants are not available in Synechococcus 7942, which is an obligate phototroph (33). Mutants lacking photosystem II and/or photosystem I have been made in Synechocystis 6803 (34) and Synechococcus 7002 (35), but the small cell size of these organisms precludes quantitative FRAP measurements.2

Physiological Role(s) of Phycobilisome Mobility

Phycobilisome mobility is characteristic of all of the cyanobacteria that we have examined including D. salina (16),
Phycobilisome Mobility Is Required for Regulation of Light-Harvesting through State Transitions—The physiological adaptation mechanism known as state transitions involves the redistribution of phycobilisomes between photosystem II and photosystem I. This mechanism presumably requires movement of the phycobilisomes (26). However, state transitions occur on a time scale of a few seconds to approximately a minute. At the diffusion rates we observe, we can estimate that a phycobilisome could diffuse from photosystem II to photosystem I in approximately 15 ms, assuming a typical concentration of reaction centers in the membrane exists (17). Thus, it is likely that the rate at which state transitions occur is controlled by the signal transduction pathway rather than by the diffusion of the complexes. We could predict that state transitions could still occur whether the diffusion rate of phycobilisomes were hundreds of times slower. In fact, we found that state transitions occur normally in the desA* mutant (data not shown), although phycobilisome diffusion is approximately 120 times slower than in the wild-type (Fig. 3).

Phycobilisome Mobility Is Required for Synthesis and Turnover of Thylakoid Membrane Components—Phycobilisomes are large complexes that normally occupy the cytoplasmic surface of the thylakoid membrane (29). It could be argued that phycobilisome mobility is necessary to allow the access of ribosomes, proteases, and regulatory enzymes to the membrane surface in order to allow synthesis, turnover, and regulation of thylakoid membrane components. One prediction of this idea would be that the turnover of the D1 polypeptide should be slower in the desA* mutant in which phycobilisome mobility is greatly reduced. However, it appears that D1 turnover is actually faster in desA* than in the wild-type (36).

Phycobilisome Mobility Increases the Efficiency of Light-harvesting—Phycobilisomes are mobile on the same time scale as the secondary electron transport reactions. Could phycobilisomes decouple from photochemically “closed” reaction centers and reassociate with open reaction centers, thus minimizing the wasteful transfer of excitons to closed reaction centers? In this model, phycobilisome mobility would be a way to allow a limited pool of phycobilisomes to act as efficient light-harvesting antennae for a much larger pool of reaction centers.

The altered phycobilisome mobility in the desA* and R2HECAT mutants will provide us with an approach to test these ideas.

Acknowledgments—We thank Petter Gustafsson (University of Umeå, Sweden) for the gift of the R2HECAT mutant and Norio Murata (National Institute for Basic Biology, Okazaki, Japan) for the gift of the desA* transformant.

REFERENCES
1. Grossman, A. R., Schaefer, M. R., Chiang, G. G., and Collier, J. L. (1993) Microbiol. Rev. 57, 725–749
2. MacColl, R. (1998) J. Struct. Biol. 124, 311–334
3. Mullineaux, C. W. (1994) Biochim. Biophys. Acta 1184, 71–77
4. Mullineaux, C. W. (1992) Biochim. Biophys. Acta 1095, 285–292
5. Ashby, M. K., and Mullineaux, C. W. (1999) Photosynth. Res. 61, 169–179
6. Yu, J., Wu, Q., Mao, H., Zhao, N., and Vermaas, W. F. J. (1999) JUBMB Life 48, 455–630
7. Redlinger, T., and Gantt, E. (1982) Proc. Natl. Acad. Sci. U. S. A. 79, 5542–5546
8. Capuano, V., Bruaux, A., Tandeau de Marsac, N., and Houmard, J. (1991) J. Biol. Chem. 266, 7239–7247
9. Houmard, J., Capuano, V., Colombano, M. V., Coursin, T., and Tandeau de Marsac, N. (1990) Proc. Natl. Acad. Sci. U. S. A. 87, 2152–2156
10. Siidler, W. A. (1994) in The Molecular Biology of Cyanobacteria (Bryant, D. A., ed.) pp. 139–216, Kluwer Academic Publishers, Norwell, MA
11. Bald, D., Krup, J., and Rognes, M. (1996) Photosynth. Res. 49, 103–118
12. Ajlanli, G., and Vormette, C. (1996) Eur. J. Biochem. 237, 154–159
13. Katoh, T. (1988) Methods Enzymol. 167, 313–318
14. Clement-Metral, J., Gantt, E., and Redlinger, T. (1985) Arch. Biochem. Biophys. 238, 10–17
15. Glazer, A. N. (1988) Methods Enzymol. 167, 304–312
16. Mullineaux, C. W., Tobin, M. J., and Jones, G. R. (1997) Nature 390, 421–424
17. Mullineaux, C. W. (1999) Aust. J. Plant Physiol. 26, 671–677
18. Sherman, D. M., Trosen, T. A., and Sherman, L. A. (1994) Plant Physiol. (Bethesda) 106, 251–262
19. Bhalerao, R. P., Gilbro, T., and Gustafsson, P. (1995) Photosynth. Res. 45, 61–70
20. Gombos, Z., Kanervo, E., Tsvetkov, N., Sakamoto, T., Aro, E.-M., and Murata, N. (1997) Plant Physiol. (Bethesda) 115, 551–559
21. Castenholz, R. W. (1988) Methods Enzymol. 167, 68–83
22. Szirmai, M., and Mullineaux, C. W. (2000) FEBS Microbiol. Rev. 191, 25–29
23. Glazer, A. N. (1984) Biochim. Biophys. Acta 768, 29–51
24. Murata, N. (1989) J. Bioenerg. Biomemb. 21, 61–78
25. Manz, N. H., Novel, N., Mullineaux, C. W., Newman, J., Bailey, S., and Robinson, C. (2000) FEBS Lett. 479, 72–77
26. van Thor, J. J., Mullineaux, C. W., Matthis, H. C. P., and Hellingwerf, K. J. (1998) Bot. Acta 111, 430–443
27. Zhang, F., Lee, G. M., and Jacobson, K. (1993) Bioessays 15, 579–588
28. Ellis, R. J. (2001) Curr. Opin. Struct. Biol. 11, 114–119
29. Mustardy, L., Cunningham, F. X., and Gantt, E. (1992) Proc. Natl. Acad. Sci. U. S. A. 89, 10021–10025
30. Bittersmann, E., and Vermaas, W. (1991) Biochim. Biophys. Acta 1098, 105–116
31. Shen, G., Brousse, S., and Vermaas, W. F. J. (1993) Plant Cell 5, 1853–1863
32. O‘Toole, P. J., Wolfe, C., Ladha, S., and Cherry, R. J. (1999) Biochim. Biophys. Acta 1419, 64–70
33. Zhang, C.-C., Jeanjean, R., and Jaset, F. (1998) FEBS Microbiol. Lett. 161, 285–292
34. Vermaas, W. F. J. (1994) Biochim. Biophys. Acta 1187, 181–186
35. Shen, G., and Bryant, D. A. (1995) Photosynth. Res. 44, 41–53
36. Sippola, K., Kanervo, E., Murata, N., and Aro, E.-M. (1996) Eur. J. Biochem. 251, 641–648
Diffusion of Phycobilisomes on the Thylakoid Membranes of the Cyanobacterium Synechococcus 7942: EFFECTS OF PHYCOBILISOME SIZE, TEMPERATURE, AND MEMBRANE LIPID COMPOSITION
Mary Sarcina, Mark J. Tobin and Conrad W. Mullineaux

J. Biol. Chem. 2001, 276:46830-46834.
doi: 10.1074/jbc.M107111200 originally published online October 4, 2001

Access the most updated version of this article at doi: 10.1074/jbc.M107111200

Alerts:
• When this article is cited
• When a correction for this article is posted

Click here to choose from all of JBC's e-mail alerts

This article cites 35 references, 8 of which can be accessed free at http://www.jbc.org/content/276/50/46830.full.html#ref-list-1