Thermodynamic Investigation into the Mechanism of the Chlorophyll Fluorescence Quenching in Isolated Photosystem II Light-harvesting Complexes*

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Chlorophyll fluorescence quenching can be stimulated in vitro in purified photosystem II antenna complexes. It has been shown to resemble nonphotochemical quenching observed in isolated chloroplasts and leaves in several important respects, providing a model system for study of the mechanism of photoprotective energy dissipation. The effect of temperature on the rate of quenching in trimeric and monomeric antenna complexes revealed the presence of two temperature-dependent processes with different activation energies, one between −15 and 35 °C and another between −40 and 60 °C. The temperature of the transition between the two phases was higher for trimers than for monomers. Throughout this temperature range, the quenching was almost completely reversible, the protein CD was unchanged, and pigment binding was maintained. The activation energy for the low temperature phase was consistent with local rearrangements of pigments within some of the protein domains, whereas the higher temperature phase seemed to arise from large scale conformational transitions. For both phases, there was a strong linear correlation between the quenching rate and the appearance of an absorption band at 685 nm. In addition, quenching was correlated with a loss of CD at −495 nm from Lutein 1 and at 680 nm from chlorophylls a1 and a2, the terminal emitters. The results obtained indicate that quenching of chlorophyll fluorescence in antenna complexes is brought about by perturbation of the lutein 1/chlorophyll a1/chlorophyll a2 locus, forming a poorly fluorescing chlorophyll associate, either a dimer or an excimer.

The light-harvesting antenna of photosystem II in higher plants consists of the major trimeric complex LH2 and three minor monomeric complexes, CP29, CP26, and CP24 (1). These proteins function not only to harvest sunlight and deliver excitation energy to the photosystem II core complex but also to provide sites for photoprotection (2). Photoprotection of the photosynthetic apparatus of higher plants under conditions of excess absorbed radiation is achieved by the induction of a process that dissipates light energy from the light-harvesting complexes. This de-excitation of the excited singlet states of chlorophyll is called nonphotochemical quenching. The major fraction of nonphotochemical chlorophyll fluorescence quenching, qE, is regulated by the size of the thylakoid ΔpH (2), which controls both the protonation of amino acid side chains on proteins of the light-harvesting system and the activity of violaxanthin de-epoxidase. This enzyme catalyzes the synthesis of zeaxanthin from violaxanthin. Kinetic analysis of qE indicates that it arises from a change in the equilibrium between two discrete states of the light harvesting system, an unquenched U state and a quenched Q state (3). These states have fluorescence lifetimes of 0.4 and 1.6 ns, respectively (4). This equilibrium is controlled in an allosteric manner by protonation and zeaxanthin binding (5–8). The PsbS protein has an obligatory role in qE (9) and may provide the binding sites for zeaxanthin (10) and protons (11). It has been suggested that PsbS is a regulatory subunit of the photosystem II antenna, possibly interacting with LH2, and thereby controlling the conversion between U and Q (3). The concentration of PsbS controls the amplitude of qE (12, 13).

Purified light-harvesting complexes have been used to investigate the mechanism of nonphotochemical chlorophyll fluorescence quenching. Reconstitution of CP29 and CP26 allows incorporation of zeaxanthin into the Lut 1 and Lut 2 sites and results in a decrease in fluorescence yield (14, 15). Similarly, addition of exogenous zeaxanthin can induce fluorescence quenching in LH2, CP29, and CP26 (16). In both cases, the decrease in fluorescence is much less than that found for qE. In contrast, the induction of quenching under reduced detergent concentration, which promotes protein-protein interactions and formation of oligomers, leads to a large decrease in fluorescence. It was proposed that the Q state is stabilized by such interaction, pulling the equilibrium strongly in favor of this conformation. Alternatively, the enhanced quenching in oligomers may arise from new pigment interactions (17).

It was found that the endogenous (18) or exogenous (5, 16, 19) content of the xanthophyll cycle carotenoids violaxanthin and zeaxanthin could control quenching in vitro. In several ways, the quenching observed in vitro bears a striking similarity to that seen for qE in vivo (2). Particularly significant are the absorption changes associated with fluorescence quenching in isolated light harvesting complexes (5, 16, 20) and qE (21, 22). These point to an alteration in a subpopulation of chlorophyll and xanthophyll molecules. However, the molecular mechanism of quenching remains unresolved. It has been suggested that non-fluorescent chlorophyll a excimers or dimers are formed, which can be effective excitation quenchers in the complex (23). Another suggestion is that fluorescence quenching arises from a charge transfer complex of two chlorophyll
molecules (24). Alternatively, a direct role in quenching of chlorophyll excited states by either zeaxanthin or lutein (14, 26, 27) has been proposed.

Analysis of the rate of qE formation in leaves and chloroplasts revealed that the fluorescence decay followed second-order kinetics suggesting that quenching arose from a binary reaction of two chlorophyll molecules (8, 18). A binary reaction was also evident in vitro, where the kinetics of the decrease in fluorescence of LHCII, CP26, and CP29 robustly fitted a second-order kinetic model with respect to time after quenching initiation (16, 18, 19). Using this kinetic model, we show in this article that a thermodynamic analysis of fluorescence quenching in isolated light-harvesting complexes. We show that the quenching reaction has low activation energy consistent with an alteration in the intramolecular conformation and interaction of pigments. Quenching rate is shown to be tightly associated with the formation of a chlorophyll species that absorbs at 685 nm. In addition, we show that there is a decrease in the chlorophyll a CD signal around 680 nm and a change in the Lutein 1 CD signal consistent with an alteration within the Lutein 1 domain during quenching.

**MATERIALS AND METHODS**

Light-harvesting complexes were purified from spinach leaves as described previously (28). LHCII monomers were prepared by treatment of isoelectric focusing isolated trimers with phospholipase A as described previously (20). Investigation of chlorophyll fluorescence quenching followed a well-documented protocol: the samples were dissolved in 200 μM DM, and diluted into detergent-free medium at a specific pH and temperature to give a final DM concentration of ~6 μM (5, 6). To investigate the effect of temperature, the detergent-free medium was equilibrated at the desired temperature before sample addition. Chlorophyll fluorescence yield was measured using a PAM 101 fluorometer (Walz, Germany) as described previously (6) and displayed on a chart recorder. Data points were obtained by digitization of the chart recorder traces using Ungraph (Cambridge Soft). Kinetic analysis was carried out as described previously (18, 19), and the data were fitted to the simple hyperbolic decay $F = 1/(kt + 1/F_0) + F_\infty$, where $k$ is the second-order rate constant, $F_0$ is the amplitude of quenchable fluorescence, and $F_\infty$ is the amplitude of unquenchable fluorescence.

Measurements of the absorption change occurring during quenching at different temperatures were carried out using a Cary 500 UV-visible spectrophotometer (Varian), at a spectral resolution of 1 nm, with the sample essentially being completely quenched immediately after mixing (data not shown).

**RESULTS**

**Temperature Dependence of Fluorescence Quenching**—The rate of fluorescence quenching induced by dilution of samples of light-harvesting complexes into detergent-free solution is temperature dependent. Fig. 1 shows an Arrhenius plot of the rate of quenching for LHCII trimers, LHCII monomers, and CP26. For each complex, the quenching reaction is described by two distinct linear phases. In the first phase, the rate of quenching was almost temperature independent. However, the second phase was strongly dependent on the temperature, as evidenced by the large increase in the gradient of the Arrhenius plot. The extrapolated break point between these phases was 31.9 °C for CP26, 32.1 °C for LHCII monomers, and 38.5 °C for LHCII trimers. Above 60 °C, the rate of quenching became too fast to measure, with the sample essentially being completely quenched immediately after mixing (data not shown).

The gradient of the Arrhenius plot is a measure of the activation energy of the quenching reaction. The activation energies for the initial reaction were similar for all the complexes, being 8.36, 6.46, and 6.30 kJ/mole for CP26, LHCII trimers, and LHCII monomers, respectively. Above 35 °C for CP26 and LHCII monomers and 40 °C for LHCII trimers, the activation energy of quenching reaction increases 10-fold to 70–90 kJ/mole.

**Changes in CD Associated with Quenching**—Fig. 2 shows the CD spectra of LHCII and CP26 before and after quenching at 35 °C. In the unquenched state, the CD profile of LHCII in the red region had a characteristic shape with a strong positive band at 668–669 nm and negative bands at 653 and 682 nm (Fig. 2A). The spectrum of CP26 was similar to that of LHCII except that the negative chlorophyll b band around 652 nm was greatly reduced (Fig. 2B). It was found that in both complexes, quenching of chlorophyll fluorescence was associated with a decrease in the CD signal, particularly from the red-most band. Difference CD spectra calculated as quenched minus unquenched showed the loss of a chlorophyll a signal at 680 nm, seen as a positive band in the difference spectrum (Fig. 2, A and B, bottom). In addition to the change at ~680 nm, there was also the loss of a band at ~652 nm, suggesting an alteration in the chlorophyll b environment during quenching.

Fig. 2, C and D, shows protein CD spectra for LHCII and CP26, respectively. The CD spectra of both CP26 and LHCII are dominated by the classic pair of minima at 211 and 222 nm arising from the α-helical secondary structure. There is also a broad positive maximum at ~260 nm from the presence of a low proportion of random coiled structure. It was evident from the difference spectra of both complexes (Fig. 2, C and D, bottom) that there were no detectable changes in the protein CD during quenching.

**Temperature Dependence of CD**—Fig. 3 shows the amplitude of CD in the quenched state observed at 211 nm (protein) and 680 nm (chlorophyll) with increasing temperature for both trimeric LHCII (Fig. 3, A and C) and CP26 (Fig. 3, B and D). There was little or no change in the protein CD in either LHCII trimers or CP26 up to 60 °C (Fig. 3, A and B). When the temperature was raised above 60 °C, there was a strong decrease in the CD signal at 211 nm, most likely representing the unfolding of the helical secondary structure of the protein upon denaturation (see below). In contrast, there are characteristic changes in the chlorophyll CD (Fig. 3, C and D). Between 4 and 80 °C, there was a decrease in the amplitude of CD for both complexes, with the LHCII trimers showing a more pronounced decrease.
40 °C in LHCII trimers and between 4 and 32 °C in CP26, the chlorophyll CD at 680 nm showed negligible temperature dependence. Between 40 and 60 °C (LHCII trimers) and between 32 and 60 °C (CP26), there was a large decrease in the CD signal, showing a change in the interaction of the chlorophylls within the complexes. The temperature range over which there was little change in the chlorophyll CD signal was the same range in which there is little change in the rate of quenching. For both LHCII and CP26, the temperature at which the rate of chlorophyll fluorescence quenching begins to increase is the same temperature at which the CD signal at 680 nm begins to decrease.

CD of Carotenoids—Measurements on the CD changes of the carotenoids within the complexes were complicated by spectral overlap with chlorophylls (Fig. 4). However, the quenched minus unquenched difference spectrum revealed a number of features arising from LHCII carotenoids: a positive band at 495 nm together with negative bands at 445, 469, and 485 nm. These changes were also observed in CP26 during quenching in vitro (data not shown). The loss of the CD at 495 nm was found to respond to changes in the temperature in a manner similar to that seen for chlorophyll CD, with two distinct phases again resolved (Fig. 5): the amplitude of the CD change at 495 nm was only slightly temperature dependent between 4 and 40 °C, but the amplitude increased when the temperature was raised above 40 °C. This pattern of temperature dependence was again similar to that shown by the rate of quenching.

Absorption Changes—Absorbance changes around 685 nm have been found in all cases of in vitro quenching (5, 16, 20) and a similar nonphotochemical chlorophyll fluorescence quenching-related absorbance change has been observed in isolated chloroplasts (21, 22). To determine whether the changes in quenching rate at different temperatures were associated with alteration in this absorption change, absorption spectra were recorded before and after quenching, and $A_{685}$ was measured. As shown in Fig. 6A, $A_{685}$ exhibited little change up to 35 °C, but increased strongly above this temperature. The temperature dependences of the absorption change and of the rate of quenching were the same. In fact, there was a strong linear correlation ($r^2 > 0.98$) between the rate of quenching and the extent of $A_{685}$ (Fig. 6B).

Temperature Dependence of Reversibility and Complex Stability—In the range 5 to 60 °C, the fluorescence quenching rate and associated changes in CD were almost completely reversible by addition of detergent, for both CP26 and LHCII (Fig. 7, A and B). Thus, the effect of temperature was caused not by a
denaturing effect but only an enhancement of the dynamic behavior. If the temperature was increased beyond 55–60 °C, then denaturation did occur—the fluorescence quenching became irreversible, and the protein CD was lost (Fig. 7C). At this temperature, pigment binding was also disrupted, and fluorescence emission from chlorophyll b was observed (Fig. 7D).

**DISCUSSION**

*Activation Energy of Quenching*—In the temperature range below 35 °C, the rate of quenching in isolated LHCCI and CP26 was almost temperature independent. An activation energy of just 6–8 kJ/mol indicates that quenching arises from small changes in conformation (e.g. arising from alterations in the van der Waals interactions within the complex). Such changes may comprise small alterations in the orientation and/or positions of chlorophylls and/or carotenoids within individual subunits of LHCCI. Above 35–40 °C, there were dramatic increases in the rate of quenching and an increase in activation energy for all complexes. Because absorption and CD changes showed exactly the same temperature dependence as the quenching rate, it is concluded that the mechanism of quenching is the same both above and below this transition temperature. The second phase has an activation energy of between 70 and 90 kJ/mol, indicating larger alterations within the complex that allow it to adopt readily the quenched conformation. These changes might include the formation of H-bonds and alterations in charge interactions associated with larger conformational changes or, alternatively, a “melting” of bound lipid and/or detergent. Under the conditions used for induction of quenching, it is known that the complexes form oligomers and, in some cases, large macromolecular aggregates. The transition temperature may be associated with increased tendency for formation of such multimeric states. Because quenching is normally induced by lowering the detergent concentration, this observation provides support for the notion that the transition is associated with a disruption of protein/detergent interactions. It has previously been found that monomers of LHCCI have a greater tendency to adopt the quenched state compared with trimers (16), and it is therefore significant that the transition temperature was almost 10 °C higher for trimers compared with monomers.

The activation energy of qE in isolated spinach chloroplasts is similar to that found for the higher temperature phase (29). Here, it is possible that the three-dimensional organization of the photosystem II antenna within the thylakoid membrane stabilizes the unquenched state, increasing the energy required to activate the quenching process. Hence, LHCCI is locked into the unquenched state by its interaction with associated proteins, lipid, and xanthophylls. In both cases, the external interactions of LHCCI subunits control the internal configuration and the quenching state of the complex. In the case of qE, quenching occurs only by means of a “catalyst”—perhaps by protonation and zeaxanthin binding to the PsbS subunit.

*Stability of Light-harvesting Complexes*—The present data show that the changes in pigment configurations associated...
with quenching are fully reversible up to \(-50\)\textdegreeCelsius\(\). Up to this temperature range, the \(\alpha\)-helical structure of the complexes is retained, and pigment interactions are preserved, as evidenced by the lack of fluorescence emission from chlorophyll \(b\) and the unperturbed chlorophyll CD. Beyond 60\textdegreeCelsius\(,\) the complexes become denatured—helical structure is lost, chlorophyll \(b\) emission is observed, the chlorophyll CD is lost, and quenching becomes strong and irreversible. This points to a high degree of stability of the light harvesting complexes, as observed previously (30).

**Identity of the Quenching Species**—We have shown here that quenching in CP26 and LHCII is associated with spectral changes in chlorophylls and xanthophylls. More specifically, it has been demonstrated that over a 20-fold change in the rate of quenching, throughout a 50\textdegreeCelsius\(\) temperature range, strong correlation was observed with these spectral changes. In line with previous observations, quenching was associated with the appearance of an absorption band at 685 nm. There was an inverse relationship between the OD at 685 nm and CD changes around 680 nm in both CP26 and LHCII. However, it is apparent that although we see a loss of the CD signal around 680 nm associated with quenching, this is not associated with any new CD band in the red region. Thus, during quenching, the interaction of the chlorophylls responsible for the negative CD signal at 680 nm changes in such a way as to produce a species that has no CD signature but is visible as a new band in the OD spectra.

It could be suggested that in the quenched state, there is more freedom for those interacting chlorophylls, which give rise to the 680 CD signal in the unquenched state. However, this could not account for the appearance of the absorption band at 685 nm. A second possibility is that there is a new specific orientation of the interacting chlorophylls. In this case, formation of an almost perpendicular association of two chlorophyll molecules would not have a CD signal but could display a red-shifted absorption at 685 nm. Chlorophyll associates in various solvents or in liposomes have been shown to absorb at \(\sim685\) nm (31, 32).

The CD at 680 nm arises from the terminal emitter chlorophylls, \(a_1\) or \(a_2\) (33, 34), suggesting that quenching is associated with alterations within this domain. Any change in conformation of this domain would be expected to perturb Lutein 1, which lies \(\sim4\) \AA\ from chlorophyll \(a_1\) and \(a_2\) (35) (Fig. 8A). Indeed, differences in the CD spectrum in the Soret region were found, indicating some change in carotenoid conformation. Although it is difficult to determine what is responsible for the two short wavelength bands that appeared in the difference CD because of complications with overlap from chlorophyll, the long wavelength band at 495 nm most likely belongs to Lutein 1 (36). The structural model for LHCII reveals that the maximum separation between chlorophylls \(a_1\) and \(a_2\) is \(\sim8.5\) \AA\ (35). Additionally, the chlorophyll \(b1\) site has also been shown in LHCII to be occupied by chlorophyll \(a\) (33). This chlorophyll could form a second dimer within the Lutein 1 domain with chlorophyll \(a1\), with a chlorophyll-chlorophyll ring separation of less than 5.0 \AA\. It is very important to note that chlorophyll \(a\) dimers have been shown to be quenchers when their separation is less than 12 \AA\ (37).

**The Mechanism of Quenching Induction**—We propose a model for the mechanism of quenching based upon a change in pigment organization within the Lutein 1 domain. Because of the low activation energy, this change is suggested to be caused by only a small change in the tertiary structure of the LHCII protein (Fig. 8B). For this model, we assume that the pigment configuration in the structural model of LHCII (35) is that of the quenched state, because the two-dimensional crystals of LHCII used for crystallography are highly quenched.\(^2\) We suggest that that the configuration of pigments in this domain in the unquenched state prevents possible dissipative interactions between closely packed chlorophylls. We suggest that the position of Lutein 1 relative to chlorophylls \(a2\) and \(a1\) is particularly important. The change to quenched state is associated with the concerted loss of the chlorophyll CD signal at \(\sim680\) nm, Lutein 1 CD at 495 nm, and the formation of a new chlorophyll species absorbing at \(\sim685\) nm. The loss of chlorophyll CD signal indicates a change in the orientation of the terminal emitter chlorophyll, whereas the loss of Lutein 1 CD signal indicates an alteration in the interaction of this lutein with other pigments within the domain. Previously, it was shown by linear dichroism that there is a difference in lutein orientation in quenched LHCII aggregates compared with unquenched trimers (38). The change in the position of Lutein 1 is suggested to allow a new type of perpendicular association.

\(^2\)D. Klug and W. Kuhlbrandt, personal communications.
between chlorophylls a1 and a2, forming a quenching dimer/encrimer (Fig. 8D). An alternative model is that the creation of an increased chlorophyll-carotenoid interaction in the altered Lutein 1 domain is responsible for quenching. This idea would encompass suggestions about how carotenoids may directly quench chlorophyll excited singlet states (26, 27, 39, 40).

Nonphotochemical Quenching in Vivo—The similarity between the type of quenching studied here and the in vivo process of nonphotochemical quenching, qE, has been well documented (2, 5, 6, 16, 19). The mechanism for quenching responsible for qE has proved controversial. The requirement for protonation and the involvement of zeaxanthin has led to proposals quite different from the model presented in Fig. 8. Similarly, the obligatory requirement for the PsbS protein has called into question whether qE directly involves any of the antenna complexes. However, observations that PsbS does not bind chlorophyll (41) but that it provides qE-related protonation sites (11) suggests that this protein has an activating role in quenching. Similarly, it seems that PsbS also provides the binding site for zeaxanthin (10), generating the ΔA335 that is characteristic of qE (42). Thus, we propose that the conformational state of one or more antenna complexes is controlled by its interaction with PsbS. Because the antenna complexes in vivo are in a macro-organized oligomeric state (43), protein-protein interactions will govern the transitions between their unquenched and quenched states. Indeed such interactions may be necessary for the large increases in energy dissipation found in vivo as well as in vitro, explaining the high activation energy observed in vivo and in this study. Recent data have indicated that mutation of an LHCII protein in Chlamydomonas leads to inhibition of qE (44), suggesting that these proteins may also provide the sites of quenching in higher plants. This rationale is entirely consistent with the “LHCII-aggregation” hypothesis proposed more than 10 years ago (25).

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