

Lutein Can Act as a Switchable Charge Transfer Quencher in the CP26 Light-harvesting Complex*

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Energy-dependent quenching of excitons in photosystem II of plants, or qE, has been positively correlated with the transient production of carotenoid radical cation species. Zeaxanthin was shown to be the donor species in the CP29 antenna complex. We report transient absorbance analyses of CP24 and CP26 complexes that bind lutein and zeaxanthin in the L1 and L2 domains, respectively. For CP24 complexes, the transient absorbance difference profiles give a reconstructed transient absorbance spectrum with a single peak centered at ~980 nm, consistent with zeaxanthin radical cation formation. In contrast, CP26 gives constants for the decay components probed at 940 and 980 nm of 144 and 194 ps, a transient absorbance spectrum that has a main peak at 980 nm, and a substantial shoulder at 940 nm. This suggests the presence of two charge transfer quenching sites in CP26 involving zeaxanthin radical cation and lutein radical cation species. We also show that lutein radical cation formation in CP26 is dependent on binding of zeaxanthin to the L2 domain, implying that zeaxanthin acts as an allosteric effector of charge transfer quenching involving lutein in the L1 domain.

Regulation of light capture during photosynthesis occurs primarily within the antenna of photosystem II, the peripheral portion of which is comprised of trimeric light-harvesting complex (LHC) II⁴ (1) and the monomeric minor LHCs CP24, CP26, and CP29 (2). Regulation of light capture is critical for

plant fitness (3) and is achieved predominantly by a process termed energy-dependent quenching, or qE (4), one of a composite of processes involved in the non-photochemical quenching (NPQ) of excess absorbed light energy (5–7). Several characteristics distinguish qE from the other components of NPQ. First, qE is reversible on the seconds-to-minutes time scale, a feat that is thought to reflect rapid changes in the thylakoid lumen pH, which transmit information to the antenna where the molecular mechanism of qE is modulated. Secondly, the *npq1* and *npq4* mutant strains of *Arabidopsis thaliana*, which lack the capacity for generating zeaxanthin (Z) (8) and the PsbS protein (9), respectively, exhibit very little qE, consistent with Z and PsbS being necessary for qE.

The peripheral antenna is generally thought to be the location of the molecular mechanism of qE, although within precisely which of the LHCs, as well as by what molecular mechanism(s), are issues currently under intense investigation (10–13). Assigning where and by what mechanism(s) qE occurs is hindered by the fact that qE is, by definition, a phenomenon requiring an intact system, complicating purely deconstructive approaches for studying this mechanism. Nonetheless, several recent studies have combined various approaches to correlate the phenomenology of qE with various components and molecular mechanisms. For example, it was recently shown that excitation energy transfer from singlet-excited chlorophyll (Chl) to the S₁ state of lutein (Lut) occurs both within isolated LHCI trimeric complexes (13), previously hypothesized to be a site for qE (14), and *in vivo* in a qE-dependent manner (13). Evidence for charge transfer (CT) quenching of Chl excited states, a mechanism previously predicted from quantum chemical calculations (15, 16), within a Chl-Z heterodimer ((Chl-Z)) complex has been positively correlated with all of the phenomenological aspects of qE using isolated thylakoids (17, 18). Evidence for CT quenching, *e.g.* transient Z radical cation (Z^{•+}) formation (17, 18), has been demonstrated in a composite mixture of CP24, CP26, and CP29 complexes (17), and recently, in isolated CP29 complexes (19). Estimates suggest that CT quenching within all three minor complexes can account for a significant fraction of qE in isolated thylakoids (17), although it should be emphasized that simultaneous operation of multiple mechanisms throughout the antenna during qE is not excluded.

The molecular architecture of the CT site in CP29 has recently been elucidated (19). Based on homology with the LHCI complex, eight Chl and two carotenoid (Car) binding sites have been assigned to CP29, all of which reside within two protein domains

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⁴ The abbreviations used are: LHC, light-harvesting complex; qE, energy-dependent quenching; NPQ, non-photochemical quenching; CT, charge transfer; Chl, chlorophyll; Lut, lutein; Z, zeaxanthin; V, violaxanthin; TA, transient absorbance; NIR, near infrared region; Car, carotenoid; HPLC, high pressure liquid chromatography.

referred to as L1 and L2 (20). Lut and the xanthophyll cycle carotenoids (Z and violaxanthin, or V) preferentially bind to the L1 and L2 sites, respectively (21). Chls *a* that bind to sites A5 and B5 (corresponding to Chl 5 and Chl 12, respectively, in LHCI (14)) are excitonically coupled (20), their interaction has been shown to be necessary for regulation of fluorescence lifetime (22, 23) and Chl triplet quenching in the homologous protein Lhca4 (24), and they have been shown to be involved in CT quenching in a (Chl-Z) complex within the L2 domain (19).

The CP26 complex provides an opportunity to assess whether other Car species can be involved in CT quenching. Lut is bound at the L1 site, which also holds a pair of Chl binding sites (A2 and B2 corresponding to Chl 2 and Chl 7, respectively in LHCI (14)), both close to the Car and to each other (25), the features found for the L2 site and Chls A5 and B5 in CP29 (19).

In this report, we describe transient absorbance (TA) analyses of CP24 and CP26 complexes with various Cars bound to sites L1 and L2 by excitation of the complexes at the Chl Q_y transition and probing for transient species within the near infrared region (NIR), as in Refs. 17–19. The results suggest that although a single CT quenching site involving a $Z^{\cdot+}$ species operates within CP24, presumably within the L2 domain, two CT sites exist within CP26, one of which involves a $Z^{\cdot+}$ species within the L2 domain and the other a Lut radical cation ($Lut^{\cdot+}$) within the L1 domain. We discuss these results in the context of the different CT site architectures of the CP29 and CP26 complexes.

MATERIALS AND METHODS

Isolation of Antenna LHCs with Specific Xanthophylls—The genes for CP24 and CP26 polypeptides from *A. thaliana* were expressed in *Escherichia coli*, and the apoproteins were isolated followed by *in vitro* reconstitution with Chls (*a* and *b*), neoxanthin, Lut, and either V or Z. Pigments were extracted from the isolated antenna complexes with 80% acetone and then separated and quantified by HPLC as described in Ref. 26 and by fitting analysis of the spectrum of the acetone extract with the spectra of individual pigments as described in Ref. 27.

Transient Absorbance Setup—A femtosecond TA laser system that was recently described in detail was used to perform NIR TA analyses (17). Briefly, excitation pulses were centered at 650 nm, and the probe region used was 880–1080 nm. CP24 and CP26 complexes were resuspended in buffer solution (5 mM HEPES and 0.06% α -dodecylmaltocide at pH 7.6) to an OD of ~ 0.3 /mm (all samples were adjusted to the same OD at 650 nm) for TA analyses. A sample cell with a path length of 1 mm was chilled by a circulating water bath (VWR Scientific 1160, PolyScientific, Niles, IL), which was set at 7 °C during the data acquisition to prevent sample degradation.

RESULTS

Carotenoid Compositions of Isolated CP24 and CP26 Apoproteins—Isolated recombinant apoproteins of CP24 and CP26 overexpressed in *E. coli* were reconstituted *in vitro* with Chls (*a* and *b*), neoxanthin, Lut, and either V or Z, or in some instances, only with the individual Cars (below). Table 1 shows that, as determined by HPLC analyses, the CP24 complexes reconstituted in the presence of V (referred to herein as CP24_V)

TABLE 1

Pigment compositions of reconstituted CP24 and CP26 complexes
Viola, violaxanthin (V); Zea, zeaxanthin (Z); Neo, neoxanthin; Lut, lutein; Car, carotenoids.

Sample	# of Chls	Chl <i>a</i> /Chl <i>b</i>	Chl/Car	Neo	Viola	Lut	Zea	# of Car
CP26 _V	9	2.2	3.5	0.8	0.2	1.6	0.0	2.6
CP26 _Z	9	2.0	4.2	0.0	0.0	1.1	1.1	2.1
CP24 _V	10	1.5	4.4	0.0	0.6	1.6	0.0	2.2
CP24 _Z	10	1.6	4.7	0.0	0.0	0.8	1.4	2.1

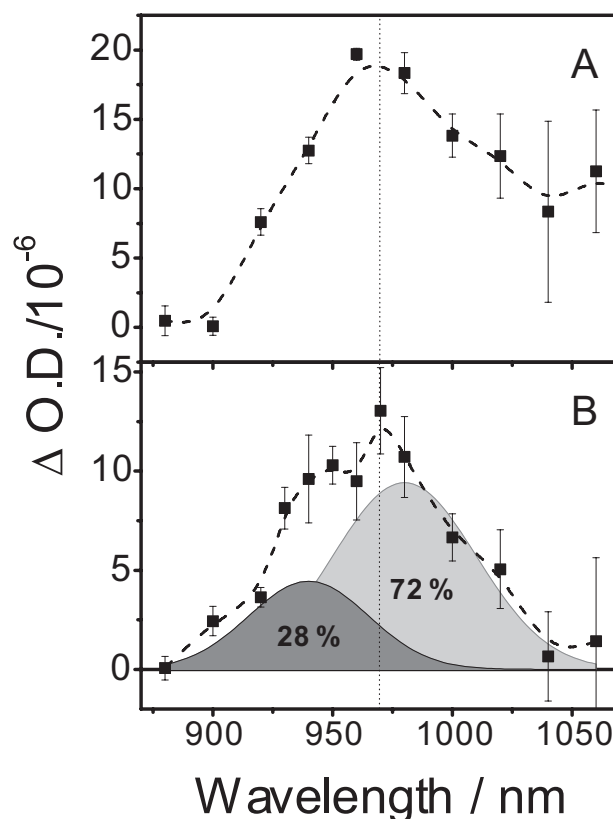


FIGURE 1. NIR TA spectrum for CP24 and CP26 complexes. A range of NIR TA kinetic profiles following excitation at 650 nm was generated in isolated CP24 and CP26 complexes that bind either V or Z by probing from 880 to 1080 nm. Reconstructed spectra for CP24 (A) and CP26 (B) samples were generated by estimating the maximum amplitude of the difference profiles (*i.e.* the average of time points 13–17 ps) at ~ 15 ps. Error bars represent the S.E. of the mean of five time points.

contained 0.6 V and 1.6 Lut molecules per 10 Chls, respectively. In contrast, the CP24_Z complexes consisted of 1.4 Z and 0.8 Lut molecules, respectively. The CP26_V complexes were comprised of 0.2 V and 1.6 Lut molecules, respectively, whereas the CP26_Z complexes consisted of 1.1 Z and 1.1 Lut molecules, respectively. The minor complexes preferentially bind Lut and Z (or V) in the L1 and L2 domains, respectively (27).

Variable Car^{•+} Formation in CP24 and CP26 Complexes—The Car radical cation ($Car^{\cdot+}$) species exhibits strong absorption in the NIR (28). Shown in Fig. 1 are TA difference spectra for CP24 (panel A) and CP26 (panel B) that were obtained, essentially as described in Refs. 17 and 19, from a series of NIR TA kinetic traces by excitation of the CP24_Z/CP24_V and CP26_Z/CP26_V complexes at the Chl Q_y transition and probing for transient $Car^{\cdot+}$ species from 880 to 1080 nm. The CP24 spectrum is characterized by broad absorption and a central peak at ~ 970 –980 nm, very similar to the TA spectrum

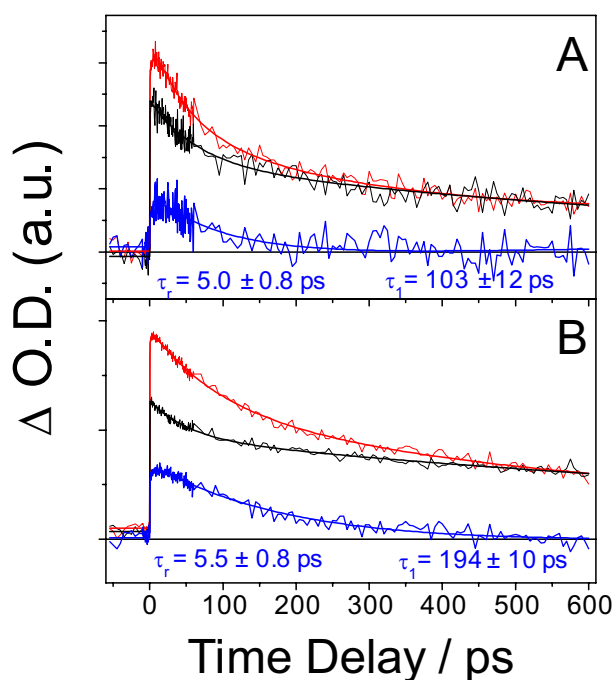


FIGURE 2. NIR TA kinetic profiles for CP24 and CP26 complexes probed at 980 nm. NIR TA profiles were obtained using isolated, monomeric CP24 (A) and CP26 (B) complexes by excitation at 650 nm and probing at 980 nm. The red and black traces represent TA kinetic profiles for the corresponding complexes bound by Z and V, respectively, and represent an average of more than 10 kinetic sweeps. Difference kinetic traces (blue) correspond to subtraction of the V-kinetic profiles from the Z-kinetics. a.u., arbitrary units.

obtained for CP29 and in which the 980 nm peak was assigned to transient Z^+ formation during CT quenching (17, 19, 29). In contrast to the single peak observed in the NIR TA spectra for CP24 and CP29 (19), the spectrum for CP26 exhibits, in addition to a peak at 980 nm, a shoulder at ~ 950 nm. Although these combined results are consistent with the occurrence of CT quenching involving a Z^+ species in the L2 domain in all three minor complexes, as has been previously suggested (17, 19), the two peaks observed in the CP26 TA spectrum are consistent with the transient formation of multiple $\text{Car}^{+\cdot}$ species, possibly indicative of an auxiliary CT site. Recall that the L1 site of CP26_Z is occupied by Lut, hinting that the alternative CT site may involve a $\text{Lut}^{+\cdot}$, previously shown to absorb at 940–950 nm (28). The area under the ~ 940 nm band is $\sim 28\%$ of the total absorbance, suggesting a somewhat lower yield of Lut cation, because Lut and Z bind to CP26 in a 1:1 ratio (Table 1), whereas the extinction coefficient of $\text{Lut}^{+\cdot}$ is 70% that of Z^+ (30, 31).

Transient Z^+ Formation in CP24 and CP26—CT quenching dynamics can be inferred from NIR TA kinetic analyses (17–19). Fig. 2A shows that the 980 nm TA kinetic profile for the CP24_V sample (black trace) is characterized solely by decay components, essentially as was observed in CP29 bound by V (19) and attributed to the dynamics of Chl excited state absorbance (17–19). In contrast, the 980 nm TA kinetic profile for CP24_Z complexes (red trace) exhibits multiexponential rise components followed by biphasic decay. The TA difference profile (blue trace), obtained by subtracting the 980 nm TA kinetics of the V-complexes from those of the Z counterparts, shows biphasic rise components, similar to those observed and kinetically modeled for CP29 (32). The faster and slower rise

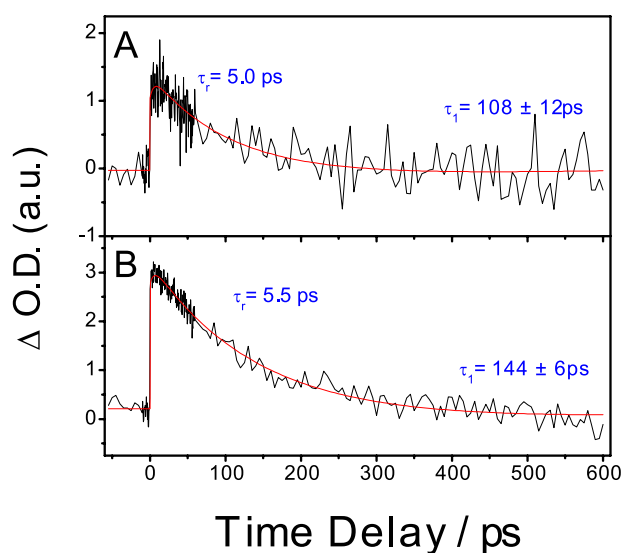


FIGURE 3. 940 nm TA difference profiles for isolated CP24 (A) and CP26 (B) complexes. NIR TA difference profiles were obtained as in Fig. 2 except that the probe wavelength was 940 nm. a.u., arbitrary units.

components displayed time constants of <500 fs and ~ 5 ps, respectively. The kinetics of the sub-ps rise are attributable to either very fast intracomplex energy transfer to the CT quenching site or to excitation of the CT site directly, as discussed in Ref. 32. The slower ~ 5 ps rise component can be assigned to the dynamics of intracomplex energy transfer to the CT trap site (32). The 980 nm signal in CP24 decays as a single exponential with a time constant of 103 ps, when compared with the 238-ps decay seen in CP29 at this wavelength (19). These results imply that the dynamics of energy transfer to the CT site are insensitive to the intrinsic variability of the L2 domains of CP24 and CP29, whereas the charge recombination of the respective charge-separated states is sensitive to the protein environment. The multiexponential rise components (<500 fs and 5 ps, respectively) of the 980 nm TA difference profile for CP26_Z (Fig. 2B) are similar to those observed for both CP24_Z and CP29_Z (19), whereas the TA signal decays with a time constant of 194 ps, intermediate between the CP24_Z and CP29_Z values.

If the $\text{Car}^{+\cdot}$ band at 940 nm is formed independently of the Z^+ 980 nm band, we expect different kinetics in the two spectral regions. Fig. 3A shows that the time constants of the biexponential rise components of the 940 nm TA difference profile for CP24 were <500 fs and 5 ps, respectively, and that the signal decays with a time constant of 108 ps. These CP24 TA dynamics are, within the noise level, the same as those observed at 980 nm (Fig. 2A), implying that the same $\text{Car}^{+\cdot}$ (i.e. Z^+) species is probed at the two wavelengths. Because all CP24 complexes bind two carotenoids, it is possible that some of the Z^+ signal comes from CP24 complexes containing Z in both L1 and L2 sites, although the kinetics would have to be identical in the two sites, which seems unlikely. However, the dominant (70% under this assumption) species is 1Lut/1Z, and we do not see a $\text{Lut}^{+\cdot}$ signal in this sample. The biphasic rise components of the 940 nm TA difference profile for CP26 are <500 fs and 5 ps (Fig. 3B), respectively, as was observed at 980 nm; however, the 940 nm signal decayed with a time constant of 144 ps, in contrast to 194 ps

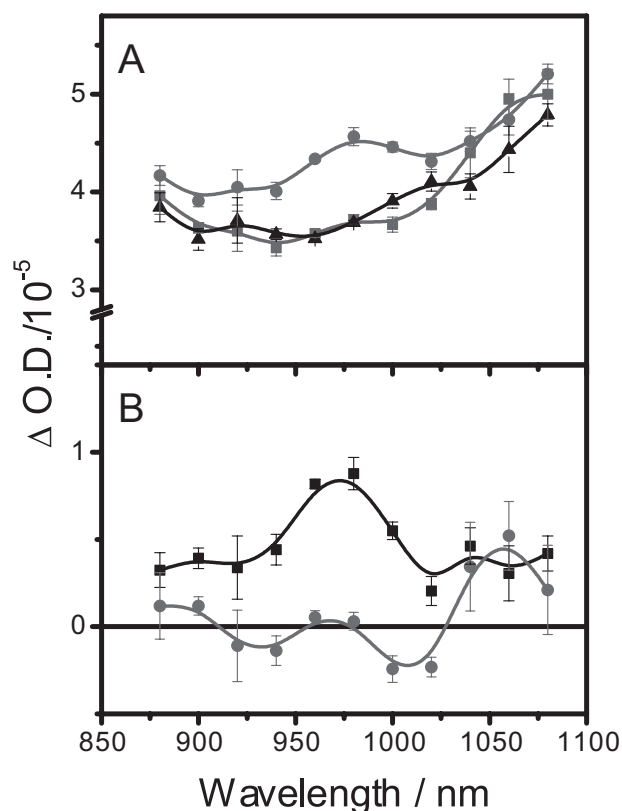


FIGURE 4. Transient spectra for CP26 complexes reconstituted with single carotenoid species. A range of NIR TA kinetic profiles (*i.e.* obtained by probing from 880 to 1080 nm) was generated in isolated CP26 complexes that were reconstituted solely with V, Z, or Lut. A, spectra for complexes that bind Z (circles), V (triangles), and Lut (squares) were obtained as in Fig. 1. The V spectrum was multiplied by 1.5 for comparison. B, difference spectra for Z-complexes minus V-complexes (squares) and L-complexes minus V-complexes (circles). Error bars represent the S.E. of five time points.

at 980 nm (Fig. 2B), supporting the idea of independent CT quenching sites in CP26 with Z in the L2 site and Lut in the L1 site.

Alternatively, the differential decay dynamics observed for CP26 at 940 and 980 nm, as well as the two-peak TA spectrum, could reflect protein environmentally induced variability in Z^{*+} formation within distinct populations of complexes that bind Z in both the L1 and the L2 domains. Fig. 4A shows NIR TA spectra that were obtained for CP26 complexes reconstituted solely with a single type of Car, *i.e.* V, Z, or Lut in both sites L1 and L2 (27). The pigment compositions of the complex are given in Table 2. The TA spectrum for CP26 reconstituted with only V does not exhibit absorption above the baseline throughout the region where Car^{*+} species typically absorb, confirming that V does not participate in CT quenching when bound to either L1 or L2 domains of CP26. Similarly, the TA spectrum for CP26 reconstituted with only Lut also shows no evidence for Car^{*+} formation, implying that CT quenching involving a Lut^{*+} is not supported when Lut occupies the L1 and L2 domains simultaneously. Occupancy of L2 by V, which competes for binding within this domain, apparently does not support a conformation of the protein that facilitates Lut^{*+} formation in the L1 site, as suggested by the lack of evidence for Car^{*+} formation in the spectrum for CP26 reconstituted with both

TABLE 2

Pigment compositions of reconstituted CP26 complexes with single xanthophyll species

Viola., violaxanthin (V); Zea., zeaxanthin (Z); Neo, neoxanthin; Lut, lutein; Car, carotenoids. Superscripts denote binding of carotenoid at both sites L1 and L2.

Sample	# of Chls	Chl a/Chl b	Neo	Viola	Lut	Zea	# of Car
CP26-Lut ²	9	2.7	0.0	0.0	1.8	0.0	1.8
CP26-V ²	9	2.5	0.0	2.0	0.0	0.0	2.0
CP26-Z ²	9	2.7	0.0	0.0	0.0	1.5	1.5

V and Lut (data not shown). In contrast, Car^{*+} formation is clearly evident in the spectrum for CP26 reconstituted solely with Z, where broad absorption characterized by a single peak centered at ~ 980 nm is observed. Furthermore, Fig. 4B shows that the difference spectrum, obtained by subtracting the spectrum of the complexes that bind V only from that of the complexes that were reconstituted solely with Z, exhibits a single peak at ~ 980 nm, consistent with previously reported Z^{*+} absorption characteristics during CT quenching (17–19). These results imply either that Z^{*+} formation occurs in both sites L1 and L2 and the respective Z^{*+} species possess similar absorption maxima or that Z^{*+} formation occurs only in the L2 site (*i.e.* CT quenching involving a Z^{*+} does not occur in the L1 site when bound by Z).

Overall these results imply that the 940 nm band in CP26_Z arises from Lut^{*+} , not Z^{*+} in the L1 site. Binding of Z to the L2 site has been shown to produce a conformational change in CP26 (33).

DISCUSSION

Z Is an Allosteric Modulator of CT Quenching within a (Chl-Lut) in CP26—Z has been proposed to have multiple roles in dissipating excess absorbed light energy (1, 11, 12, 17–19, 34). On the one hand, Z has been suggested to be directly involved in the quenching of Chl excited states during qE via CT quenching (18), evidence for which, *e.g.* transient Z^{*+} formation, was obtained in a composite mixture of all three minor complexes (17) and more recently in isolated CP29 (19, 32) and in isolated CP24 and CP26 complexes (this work). It has also been suggested that transfer of energy from singlet-excited Chl to the S_1 state of Z may occur, thereby allowing the direct quenching of Chl excited states (35, 36), although no evidence for this mechanism has been obtained. On the other hand, others have proposed indirect roles for Z in dissipating excess absorbed energy (12, 37, 38). It has been suggested, for example, that binding of Z to LHCII trimeric complexes induces conformational changes that result in the formation of excitonically coupled pairs of Chl dimers that form low-lying energy traps (12, 37, 38). According to this scenario, Z acts as a sort of allosteric modulator of energy quenching. Similarly, binding of Z to CP26 complexes has also been demonstrated to result in protein conformational changes that correlate with quenching of Chl excited states (33). Our demonstration of CT quenching likely involving a (Chl-Lut) complex in the L1 site of CP26 when Z alone binds to the L2 site suggests that the conformational changes induced by binding of Z activate CT quenching in the L1 site. Given that Z in the L2 site of CP26 also participates in the formation of a (Chl-Z) quenching complex (Figs. 1 and 3), these combined results suggest dual roles (*i.e.* both direct and indi-

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rect) for Z in CT quenching in CP26. 1) It directly participates in CT quenching in the L2 site; and 2) it acts as an allosteric modulator of the (Chl-Lut) quenching complex in the L1 site.

CT Site Architecture of the L1 Domain of CP26—We now turn to the question of why the L1 site in CP26 can support Lut radical cation formation, whereas the L1 site in CP29 does not (19). In CP29, both Chl A5 and Chl B5 were necessary for Z⁺ formation, leading us to suggest that a Chl dimer was required (19). The L1 site in CP29 has a single Chl (A2) appropriately located (20) for CT, whereas in CP26, the homology model suggests that Chl B2 is close to Chl A2 (25, 27), enabling Chl dimer formation⁵ similar to that between Chls A5 and B5 in CP29 (19). Thus our CP26 results are consistent with the proposal in Ref. 19 that an excitonically coupled Chl dimer is an essential component of the CT quenching mechanism in the minor LHCs.

Direct Roles for Lutein in qE—Z and Lut have both been suggested to be involved in qE (39, 40). Mutant strains of *A. thaliana* such as *lut2* that lack the ability to synthesize Lut exhibit low levels of qE relative to wild type (40). Moreover, the *npq1* mutant of *A. thaliana*, a strain that lacks detectable levels of Z, still exhibits measurable levels of qE (9), and this residual qE was eliminated in an *npq1 lut2* double mutant (41). These results are consistent with direct and/or indirect roles for Lut in qE, although by precisely what mechanism(s) could not be determined (40, 41). Consistent with an allosteric role of Z in Lut CT induction, a Lut-only mutant is unable to perform qE, but it should be noted that this mutant does not contain CP26 (42). To reconcile the qE phenotype of *npq1*, one possibility is that, *in vivo*, the interaction of CP26 with PsbS in the thylakoid membrane partially substitutes for the allosteric effect induced *in vitro* by Z binding to site L2 (21). In contrast, Lut was recently shown to be directly involved in the quenching of Chl excited states within LHClI via transfer of energy from singlet-excited Chl to the S₁ state of Lut, a mechanism that was correlated with *in vivo* qE phenomenology (13). Herein we demonstrate that Lut plays yet another direct role in qE via CT quenching involving a (Chl-Lut) complex within the L1 site of CP26.

Conclusions—CP26 exhibits Car⁺ formation in both the L1 and the L2 sites. The L1 site involves Lut, and the L2 site involves Z. The homology model structure of CP26 provides a rationale for the difference from CP29 with a potential excitonic Chl dimer lying close to the L1 site, in contrast to CP29 where only a single Chl is close to L1.

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⁵ Three Chls *a* (A1, A2, and B2) are proposed to contribute to the energetically lowest excitonic level in LHClI (43, 44). They are close to the L1 site (20). However, to generate a CT state, the Chls should be located cofacial near the middle of the Car (in this case, Lut in the L1 site) (15–16). In CP26, the putative heterodimer (Chl-Lut) includes Chl A2 (38). The estimated coupling strength of 20 cm⁻¹ between Chls A2 and A1 is significantly smaller than that (120 cm⁻¹) between Chl A2 and B2 in LHClI. We therefore suggest that the L1 domain contains a Chl pair, A2 and B2, in CP26 capable of forming a CT complex with Lut.

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**Lutein Can Act as a Switchable Charge Transfer Quencher in the CP26
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