Precise Location of the Cu(II)-inhibitory Binding Site in Higher Plant and Bacterial Photosynthetic Reaction Centers as Probed by Light-induced Absorption Changes*

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Inmaculada Yruela†, Miguel Alfonso§, Iñaki Ortiz de ZarateŠ, Guillermo Montoya¶, and Rafael Picorell||
From the Estación Experimental Aula Dei, Consejo Superior de Investigaciones Científicas, Apartado 202, 50080 Zaragoza, Spain

Light-dependent absorption change at 325 nm, ascribed to QA activity, was strongly reduced in the presence of Cu(II) in oxygen-evolving core complex. This change was much less affected in the presence of the herbicide 3-(3,4-dichlorophenyl)-1,1-dimethylurea (DCMU), indicating that the Cu(II)-binding site is different from that of the DCMU and that Cu(II) blocks QA reduction. Cu(II) does not eliminate the absorption change at 545 nm, ascribed to pheophytin reduction, in Na2S2O4-treated oxygen-evolving core and D1-D2-cytochrome b563 complexes. This indicates that Cu(II) does not affect the electron transport between P680 and pheophytin. Moreover, the activity of the bacterial reaction center probed by the absorption change at 790 nm was inhibited by Cu(II), but the signal at 530 nm, associated to the reduction of bacterioopheophytin in Na2S2O4-treated reaction center, was not inhibited. We conclude that Cu(II) impaired the photosynthetic electron transport between pheophytin and QA in both higher plants and photosynthetic bacteria. Cu(II) would bind to an amino acid(s) highly conserved in non-oxygenic and oxygenic reaction centers, which is(are) necessary for the electron transfer between pheophytin and QA. Based on the atomic structure of the bacterial reaction center several schemes of possible Cu(II) binding are shown.

The high pollution by heavy metals in the biosphere has led to an increasing attention to the effects of these toxic metals on living systems. Toxic levels of some of these heavy metals occur in natural and agricultural soils as a result of environmental pollution due to mining, smelting, manufacturing, agricultural, and waste disposal technologies (1). Among these metals, copper presents a high degree of toxicity (2). It is well known that Cu(II) inhibits the photosystem (PS) I electron transport in higher plants (3, 4), green algae (5), and cyanobacteria (6). However, the precise location of the Cu(II)-binding site and the underlying mechanism are a subject of debate. Recently, our group (7, 8) has made some progress on this matter. We have proved that oxygen evolution by PS II membranes was inhibited by Cu(II) when 2,6-dichlorobenzoquinone (DCBQ) or ferricyanide, but not silicomolybdate, were used as electron acceptors. This indicated that Cu(II) affected the reducing side of PS II. Moreover, by using trypsin-treated thylakoids we demonstrated that Cu(II)-inhibitory site is located before the Qb niche and close to the pheophytin-QA-iron domain of the PS II reaction center (RC). We have also characterized the Cu(II)-inhibitory mechanism based on measurements of oxygen evolution activity (8). This mechanism resulted non-competitive with respect to DCBQ and DCMU, and competitive with respect to protons. The non-competitive inhibition indicated that Cu(II)-binding site is different from that of the DCBQ and DCMU sites, the Qb niche. On the other hand, the competitive inhibition respect to protons may indicate that Cu(II) interacts with an essential amino acid group(s) that can be protonated or deprotonated in the inhibitory-binding site.

The endogenous electron acceptors of PS II include two "bound" plastoquinone (PQ) molecules, QA and Qb, and a pool of "free" PQ molecules that are present in the fluid lipid phase of the photosynthetic membranes (9-11). Upon illumination, the primary donor chlorophyll (P680) reduces QA via pheophytin, which in turn is re-oxidized by Qb. The Qb produced by the transfer of one electron from QA binds strongly to its binding site but Qb-, generated by a second turnover of QA-, is replaced by a "free" PQ molecule. Crystallization and x-ray studies on the RC from purple bacteria (12, 13) together with the isolation of PS II RC from higher plants (14) have made possible the comparison between the oxygenic and non-oxygenic reaction centers. A high sequence homology has been revealed between the L and M polypeptides of purple bacterial RC and the D1 and D2 polypeptides of the PS II RC, respectively (15-17). This high homology is more apparent in the primary donor and the quinone-iron domains. The photoreduction of QA and pheophytin is known to be accompanied by

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¶ To whom correspondence should be addressed. Tel.: 34-76-576511; Fax: 34-76-576520.

The abbreviations used are: PS, photosystem; D1 and D2, polypeptides of the photosystem II reaction center; DCBQ, 2,6-dichlorobenzoquinone; DCMU, 3-(3,4-dichlorophenyl)-1,1-dimethylurea; IR, infrared; L, light polypeptide of the bacterial reaction center; LDAO, laureyl dimethylamine N-oxide; M, medium polypeptide of the bacterial reaction center; MBS, 2-N-hydroxyethylpiperazine-N'-2-ethanesulfonic acid; OEC, oxygen evolving core complex; OGP, 1-a-n-octyl-β-D-glucopyranoside; P680, primary donor chlorophyll; PQ, plastoquinone; QA, primary quinone acceptor; QB, secondary quinone acceptor; RC, reaction center; Z, primary electron donor.

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a bleaching of the absorption bands at 325 and 545 nm, respectively, in oxygen evolving core complex (OECC) (11, 18, 19) and PS II RC (20). In contrast, the photoreduction of bacteriopheophytin can be followed by detecting the absorption change around 530 nm in bacterial RC (21, 22).

The goal of this work was to precisely locate the Cu(I)-inhibitory binding site. To this aim, we measured the light-dependent absorption changes at 325 and 545 nm in OECC and D1-D2-cytochrome b560 complex from higher plants, and at 790 and 530 nm in bacterial chromatophores and RC, respectively, in the presence of Cu(I). Several examples of possible Cu(I) binding to conserved amino acids in PS II and bacterial RCs are also given based on the crystal structure of purple bacterial RC.

MATERIALS AND METHODS

Biological Material—Sugar beet (Beta vulgaris L. cv. Monohill) was grown hydroponically in a growth chamber in half-Hoagland nutrient solution, under 325 μmol m⁻² s⁻¹ from fluorescent lamps at 25 °C, 80% humidity, and a 16-h light period.

Rhodospirillum rubrum S1 was grown semianerobically in 2-liter bottles at 32 °C in the medium described by Cohen-Bazire et al. (23). Illumination was provided by two 150-watt lamps, and the bacteria were harvested after 3–4 days at the end of the logarithmic growth phase.

Oxygen Evolving Core Complex (OECC) Isolation—Photosystem II membranes with high rate of oxygen evolution activity (i.e., 500 μmol of O₂ h⁻¹ mg chlorophyll⁻¹) using DCBQ (as electron acceptor) were prepared following the method of Berthold et al. (24) with some modifications as described in Ref. 7. Membranes were resuspended in 10 mM NaCl, 5 mM MgCl₂, and 50 mM MES-NaOH (pH 6.0), and centrifuged at 121,000 × g for 40 min. The pellet was solubilized with 35 mM OGP, 0.4 mM sucrose, 10 mM CaCl₂, 0.5 M NaCl, and 50 mM MES-NaOH (pH 6.0) at a chlorophyll concentration of 1.5 mg ml⁻¹. After centrifugation for 30 min at 4 °C, one part of the suspension was mixed with two parts of a solution containing 0.4 M sucrose, 0.5 M NaCl, 10 mM CaCl₂, and 50 mM MES-NaOH (pH 6.0), and centrifuged at 40,000 × g for 90 min. The supernatant was desalted by a 90-min dialysis against a solution containing 0.4 M sucrose, 10 mM CaCl₂, and 50 mM MES-NaOH (pH 6.0) using 555 and 700 nm as the reference wavelengths, respectively. The OECC sample was illuminated through an optical fiber and the D1-D2-cytochrome b560 complex was eluted with a light projector and those at 790 nm using an optical fiber. The filters used for the measurements at 530 nm with the bacterial RC were the same that used with the D1-D2-cytochrome b560 complex. For measurements at 790 nm, a blue-actinic light filters Schott BG-18 plus KG-3) was used and the photomultiplier was protected by a 5-mm Schott red BG-39 filter. The light-induced absorption changes at 530 nm were carried out with a light projector and those at 790 nm using an optical fiber. The filters used for the measurements at 530 nm with the bacterial RC were the same that used with the D1-D2-cytochrome b560 complex. For measurements at 790 nm, a blue-actinic light filters Schott BG-18 plus KG-3) was used and the photomultiplier was protected by a 5-mm Schott red BG-39 filter. The cuvette was covered with paraffin foil in the measurements made in the presence of Na₃S₂O₅.

RESULTS

To examine the Cu(I) effect on the Qₐ activity we measured the light-dependent absorbance change at 325 nm in OECC preparation in the presence of 80 μM CuCl₂ and compared with the control (no addition of inhibitor) (Fig. 1). This

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**Fig. 1. Optical absorption change kinetics induced by red actinic light at 325 nm in OECC preparation at room temperature in the presence of no inhibitor (A), 80 μM CuCl₂ (B), 10 μM DCMU (C), and 10 μM DCMU and 80 μM CuCl₂ (D).** The sample was suspended in 1 mM OGP, 0.4 mM sucrose, 10 mM NaCl, 10 mM CaCl₂, and 50 mM MES-NaOH (pH 6.5) at a chlorophyll concentration of 26.9 μg ml⁻¹. The sample was illuminated through an optical fiber and the light intensity at the surface of the cuvette was 1,750 μE m⁻² s⁻¹. Note that the y-axis scale is different in A and C as compared with B and D. The up-arrows and down-arrows indicate the light on and off, respectively.
concentration of CuCl₂ was shown to saturate the inhibition of oxygen evolution activity (7). Reduced Q₈ (Q₈⁻) exhibits a typical semiquinone optical spectrum dominated by absorbance in the 260–350 nm spectral region, showing a maximum at 325 nm (19). A marked reduction of the absorption change was observed in the presence of CuCl₂ (Fig. 1D) compared to the control experiment (Fig. 1A). In contrast, the signal was less affected in the presence of 10 µM DCMU (Fig. 1C). Data published by Schatz and van Gorkom (11) showed that different transitions such as (Q₈ → Q₈⁻), (Q₈ → Q₈⁻), (Z → Z⁻) and (S₀ → S₁) contributed to the absorption change at this wavelength. A similar signal intensity at 325 nm has been associated to Q₈⁻ and Q₈⁻, and both contributions represent about 85% respect to the total absorption change (11). The two other transitions are minority and both only represent about 15–20% (11). The smaller signal observed in the presence of DCMU (Fig. 1C) compared to the control (Fig. 1A) was due to the loss of the contribution by the Q₈⁻, that was eliminated in the presence of this herbicide. In this case, the signal decreased by about 45% respect to the control, which is consistent with published data (11). In the case of Cu(II) the signal decreased by about 81% respect to the control that corresponded well to the loss of the contributions by Q₈⁻ and Q₈⁻. These results thus indicate that Cu(II) blocks the reduction of Q₈ and consequently that of the Q₈. The residual signal obtained in Fig. 1D, which represents about 18% of the control, corresponds well to the contributions by the transitions (Z → Z⁻) and (S₀ → S₁) (11). Similar residual signal was observed in the presence of CuCl₂ in sample pre-treated with DCMU (Fig. 1D), indicating again that the Cu(II)-binding site is different to the DCMU-binding site and that it is located earlier in the electron transfer pathway. The concentrations of inhibitors used in the assays were 421- and 53-fold higher than that of the RC content in the OECC preparation for Cu(II) and DCMU, respectively, considering 50 chlorophyll/RC in OECC (29). The inhibitor/RC ratio used in these measurements was similar to that used in our previous works based on the Cu(II)-inhibition effect on the oxygen evolution activity by PSI II membranes (7).

The Cu(II) inhibition seems to be a reversible process. The optical absorption change at 325 nm, markedly reduced in the presence of Cu(II) (Fig. 2A) compared to the change with no addition of inhibitor (Fig. 1A), was recovered after the sample was centrifuged and washed twice with buffer to eliminate the Cu(II) (Fig. 2B). This result confirmed previous data reported by others (3).

It is well established that pheophytin presents a light-induced reversible absorption change around 540 nm in the D₁-D₂-cytochrome b₅₅₃ complex (20) and OECC (11). In the later complex, Q₈⁻ and Q₈⁻ also present a maximum at 545 nm, however no appreciable contributions of Z⁻ have been described (11). Fig. 3A shows the light-induced absorption change at 545 nm in OECC. A certain decrease of this change was observed in the presence of DCMU (Fig. 3B). Additional decrease but not complete elimination of the signal was observed with 80 µM CuCl₂ (Fig. 3C). From Fig. 3 (B and C) we calculated the intensity of the absorbance changes which corresponds to Q₈⁻ and Q₈⁻ and determined the absorbance ratio ΔA(Q₈⁻)/ΔA(Q₈⁻), resulting 0.32. This value corresponds well with that of 0.25 published by Schatz and van Gorkom (11). This result indicated that Cu(II) eliminated the contributions by Q₈⁻ and Q₈⁻ at this wavelength. The remaining absorption change must be due to the reduction of pheophytin. In that sense, the absorption change at 545 nm was measured in OECC preincubated with Na₂S₂O₄. This treatment induced chemically the reduction of both plastoquinone molecules, thus eliminating the contributions by Q₈⁻ and Q₈⁻. The signal intensity (Fig. 4A) was strongly reduced by the treatment with Na₂S₂O₄ (Fig. 4B) and after eliminating CuCl₂ by washing twice with buffer (B). The buffer content, chlorophyll concentration and illumination conditions are described in Fig. 1.

Fig. 2. Effect of Cu(II) on the light-induced absorption change at 325 nm by red actinic light at room temperature in OECC preparation pre-incubated with 80 µM CuCl₂ (A) and after eliminating CuCl₂ by washing twice with buffer (B). The buffer content, chlorophyll concentration and illumination conditions are described in Fig. 1.
Cu(II)-inhibitory Binding Site in Photosynthetic Reaction Center

Table I

|        | \( \Delta A_{325} \times 10^3 \) | \( \Delta A_{545} \times 10^3 \) |
|--------|-------------------------------|-------------------------------|
| No inhibitor | 1.35 ± 0.09                  | 1.68 ± 0.08                  |
| 80 \( \mu \text{M} \) CuCl\(_2\) | 0.25 ± 0.10                  | 0.44 ± 0.04                  |
| 10 \( \mu \text{M} \) DCMU      | 0.73 ± 0.10                  | 1.38 ± 0.02                  |
| 10 \( \mu \text{M} \) DCMU + 80 \( \mu \text{M} \) CuCl\(_2\) | 0.22 ± 0.07                  |

Fig. 3. Kinetics of the light-induced absorption changes at 545 nm by red actinic light in OECC preparation at room temperature in the presence of no inhibitor (A), 10 \( \mu \text{M} \) DCMU (B), and 80 \( \mu \text{M} \) CuCl\(_2\) (C). The buffer content and chlorophyll concentration were the same as in Fig. 1. The sample was illuminated through an optical fiber, and the light intensity at the surface of the cuvette was 1.050 \( \mu \text{E} \cdot \text{m}^{-2} \cdot \text{s}^{-1} \). Note that the y-axis scale is different in C.

Fig. 4. Kinetics of the absorption changes at 545 nm induced by red actinic light at room temperature in OECC preparation. Control (A) and preincubated with Na\(_2\)S\(_2\)O\(_4\) in the presence of no inhibitor (B), 80 \( \mu \text{M} \) CuCl\(_2\) (C), and 10 \( \mu \text{M} \) DCMU (D) are shown. The buffer content, chlorophyll concentration, and illumination conditions are described in Fig. 3. Note that the y-axis scale is different in A.

Fig. 5. Optical absorption change kinetics at 545 nm induced by red actinic light in D1-D2-cytochrome \( b_{593} \) complex at room temperature in the presence of no inhibitor (A) and 80 \( \mu \text{M} \) CuCl\(_2\) (B). The sample (\( A_{675.5} = 0.1 \)) was suspended in 50 mM Tris-HCl (pH 7.2) and illuminated with a light projector. The light intensity on the top of the cuvette was about 1,800 \( \mu \text{E} \cdot \text{m}^{-2} \cdot \text{s}^{-1} \).

Fig. 6. Effect of increasing concentrations of CuCl\(_2\) on the RC photochemical activity by measuring the absorption changes at 790 nm in chromatophores from R. rubrum S1. The sample was suspended in 10 mM Tris-HCl (pH 8.0) at an absorbance of 1.8 absorbance units at 860 nm. The sample was illuminated through an optical fiber, and the light intensity at the surface of the cuvette was 1.380 \( \mu \text{E} \cdot \text{m}^{-2} \cdot \text{s}^{-1} \).

transport in PS II at the level of \( Q_A \) and after the pheophytin site.

Since a high homology between PS II and bacterial RCs has been described (15-17), we have also studied the Cu(II) inhibition effect in chromatophores and RC preparation from R. rubrum. Fig. 6 depicts the effect of Cu(II) on the light-
induced absorption change at 790 nm in chromatophore preparations. This optical change due to a shift of the 800-nm band and associated with the RC activity (22) decreased with increasing amounts of CuCl₂. This result indicated that Cu(I) inhibits the bacterial RC activity and eliminated the oxidizing side of PS II as possible Cu(I)-inhibitory binding site, as suggested by Yruela et al. (7). From the inhibition curve, a value of 62.6 μM for the I₅₀ was calculated. Note that the I₅₀/RC ratio was 260, which corresponded well with that obtained for PS II membranes (I₅₀/RC = 228) (7). To calculate the RC concentration in chromatophores and PS II membranes, we considered 28 bacteriochlorophyll/RC (31) and 250 chlorophyll/RC (24, 29), respectively. Furthermore, Cu(I) has no effect on the absorption change at 530 nm, associated with the reduction of the secondary acceptors, QA and QB, and pheophytin in higher plant OECC and D1-D2-RC preparations, and in bacterial chromatophores and PS II samples. Our results indicated that Cu(I) inhibits the bacterial RC activity and eliminated the oxidizing side of PS II as possible Cu(I)-inhibitory binding site, as suggested by Yruela et al. (7). From the inhibition curve, a value of 62.6 μM for the I₅₀ was calculated. Note that the I₅₀/RC ratio was 260, which corresponded well with that obtained for PS II membranes (I₅₀/RC = 228) (7). To calculate the RC concentration in chromatophores and PS II membranes, we considered 28 bacteriochlorophyll/RC (31) and 250 chlorophyll/RC (24, 29), respectively. Furthermore, Cu(I) has no effect on the absorption change at 530 nm, associated with the reduction of bacteriopheophytin (21) in bacterial RC pretreated with Na₂S₂O₄ (Fig. 7), indicating that both PS II and bacterial RCs present similar behavior in respect to the Cu(I) inhibition effect.

**DISCUSSION**

Recently, we have probed that Cu(I) impaired the photosynthetic electron transport on the reducing side of PS II at the level of the pheophytin-Qₐ-iron domain of the RC (7), and we have characterized its inhibitory mechanism (8) mainly based on measurements of oxygen evolution activity. The aim of the present paper was to define precisely the location of the Cu(I)-binding site. We provide data on the inhibitory binding site in both types of RCs.

The presence of an essential amino acid(s) that can be protonated or deprotonated in the Cu(I)-inhibitory binding site in the PS II RC with which Cu(I) might interacts has been suggested by Yruela et al. (8). It is well established that His and Trp have a high affinity to bind copper. This is the basis for the immobilized metal-affinity chromatography for protein purification (32). This fact is also being exploited in protein engineering of metal-dependent enzyme activity (33). It has been shown that Cu(I) inhibits some proteases by coordinating to His residues in the active site (33). The presence of these amino acid residues close to Qₐ site has been described in spinach PS II (17, 34) and bacterial (35–37) RCs. Protein modeling based on the crystal structure of **Rhopodopseudomonas viridis** has suggested that some amino acid residues of Qₐ-binding site in spinach PS II RC have homologues in the bacterial RC (17). Among these, it has been found that D2-Trp²⁵⁴ is homologous to M-Trp²⁵⁰ and some His residues close to Qₐ site are present in both RCs (L-His²⁸⁹ and M-His³¹⁷ in **Rps. viridis**, and D1-His³¹⁵ and D2-His³¹⁵ in spinach). The x-ray crystal structure of the RC from **Rps. viridis** has shown that M-Trp²⁵⁰ residue is positioned within van der Waals contact distance of both bacteriopheophytin and Qₐ (35, 36). The location of this amino acid residue with respect to bacteriopheophytin and Qₐ has prompted speculation that it might be involved in promoting both tight binding of the quinone to the apoprotein (36) and fast electron transfer from bacteriopheophytin to Qₐ by enhancing the electronic overlap between both molecules (38). This amino acid residue has also been found in other bacteria (M-Trp²⁵⁰ in **Rhodobacter capsulatus** and M-Trp²⁵² in **Rb. sphaeroides** (37). Recently, works based in site directed mutagenesis in **Rb. capsulatus** have been done to dilucidate these questions and have probed that this amino acid residue affects the Qₐ binding and the speed of its reduction which would imply that
this residue is involved in the photosynthetic electron transport (39). The latter hypothesis has been proposed to explain the rapid rate of the electron transfer between bacteriopheophytin and Qₘ, considering that the indole group of M-Trp₂⁵₀ might act as a bridge between the orbitals of the primary electron donor, bacteriopheophytin, and the secondary electron acceptor, Qₐ (37).

From these data and our results, we conclude that the Cu(II)-inhibitory binding site is similar in oxygenic and non-oxygenic RCs and propose that Cu(II) might bind to a specific amino acid(s), His and/or Trp, located between (bacterial)phoophytin and Qₐ. The metal ion could bind in such a way that disrupts the local conformation of the (bacterial)phoophytin-Qₐ domain and thus inhibiting the photosynthetic electron transport. Fig. 8 shows some possible Cu(II)-inhibitory binding sites in the bacteriopheophytin-Qₐ domain of the RC based on the crystal structure of the RC from Rps. viridis (16), involving the conserved amino acid residues M-Asp²¹⁷, M-Thr²²⁰, M-Trp²⁵₀, and L-Glu¹⁰₄.

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