COMMENTS ON THE POSSIBLE ROLES OF BICARBONATE AND CHLORIDE IONS IN PHOTOSYSTEM II

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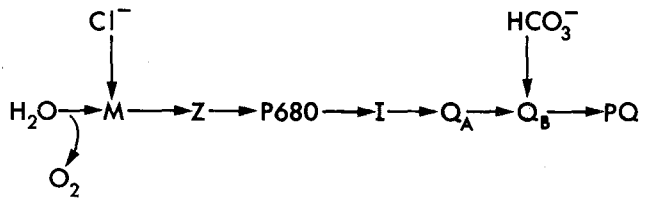
I. INTRODUCTION

Green plants possess the ability to oxidize water to molecular oxygen (O_2); this unique reaction is carried out by photosystem II (PSII), the focus of the present symposium. In what follows, we shall attempt to review data (a) on a unique role of bicarbonate (HCO_3^-) on the electron acceptor side of PSII; and (b) on the recent application of chlorine (^{35}Cl)-NMR to thylakoids showing the possible correlation of Cl^- ion with the O_2 evolving mechanism of photosynthesis; the function of Cl^- may be to stabilize a positive charge on the Mn-containing O_2 evolving enzyme.

Before discussing the roles of HCO_3^- and Cl^- in PS II, the framework of electron flow in PS II is presented for easy reference:



Scheme 1

Here, P680 is the reaction center chlorophyll a (Chl) of

[1]Supported by a NSF grant PCM 78-24532; the research on our Cl^- work was supported by a NSF grant PCM 79-11148 to H. S. Gutowsky.

ISBN 0-12-372360-4

PSII, I is the intermediate electron acceptor that includes pheophytin, Q_A is the first and Q_B the second quinone electron acceptor, the latter accepting 2 electrons in a sequence, PQ is a plastoquinone molecule of the plastoquinone pool, Z is the electron donor to $P680^+$, and M represents the charge accumulating species. The possible major sites of action of both Cl^- and HCO_3^- are suggested to be at the M and Q_B levels, respectively.

II. BICARBONATE

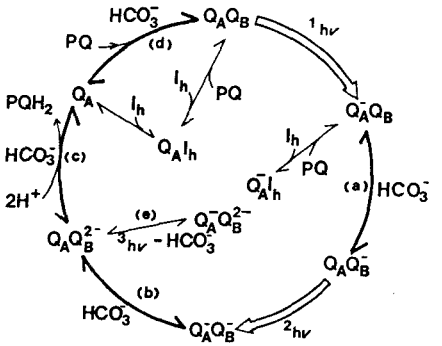
Warburg and Krippahl (1) discovered the stimulation of the Hill reaction by CO_2, and used it to suggest (2) that the source of O_2 in photosynthesis is CO_2, not H_2O. Although there is a general agreement that the ultimate source of O_2 is H_2O, a possible role of HCO_3^- as an immediate source of O_2, or, as a catalyst in some other step of O_2 evolution cannot be excluded (3,4). However, during the last several years, an alternative explanation has been obtained for the effect of HCO_3^-. A HCO_3^- effect on the electron acceptor side of PSII was first shown (5) as follows: HCO_3^- restored Chl fluorescence transient in HCO_3^- depleted thylakoids that had been previously inactivated on the O_2-evolving side. Furthermore, HCO_3^- depletion qualitatively mimicked the addition of diuron, which blocks electron flow between Q_A and Q_B, but not that of inhibitors on the water side. All of our results (6-8) clearly show that HCO_3^- affects electron flow at the level of quinones (Q_A, Q_B, PQ, see scheme 1) which mediate electron transfer between the PSII reaction center and the cytochrome b_6/f complex.

A. Current Hypothesis and Experimental Support

Bicarbonate is suggested to play a key role as a native component of the quinone-binding protein (the Q_B- or a nearby-protein) which is necessary for electron flow from Q_A^- to Q_B or Q_B^-, and for the exchange of Q_B^{2-} with a PQ molecule (see scheme 2). Its most important function may lie in providing the proper conformation of this protein for the exchange of Q_B^{2-} with a PQ molecule (reactions (c) and (d)).

In the following scheme, I_h = inhibitor/herbicide; other symbols are the same as in scheme 1. All the possible steps (a)-(d) where HCO_3^- may be required are shown in this scheme (cf. 9-11). (The main reactions are shown on the outer circle, whereas the interaction(s) with I_h (*e.g.*, a herbicide), *i.e.* the replacement of Q_B with I_h is shown inside the circle.)

It has been shown (12,13) that reactions (a) and (b) are slowed down by the absence of HCO_3^-. Chl fluorescence decay



Scheme 2

(a measure of Q_A^- to Q_A reaction) and X-320 decay (partially a measure of Q_A^- to Q_A) were slowed down from ∿200μs to several ms. Furthermore, reaction (c) was slowed down from a $t_{1/2}$ of ∿1ms to ∿150ms (14); with a dark time of ∿30ms between flashes, only 3 electrons could accumulate on the acceptor side, presumably blocking the centers in the $Q_A^-Q_B^{2-}$ state (see step (e) in scheme 2). However, with longer dark times, the electrons can slowly leak to the PQ pool, or to the donor side. No significant effect of HCO_3^- could be observed on the initial charge separation as the same fluorescence maximum is observed with or without HCO_3^- (7,15). Recently, it has been shown that the HCO_3^- effect is on the electron acceptor side even at low light intensities and in repetitive flashes of light (16).

The concept that the major HCO_3^- effect is in the Q_A, Q_B, PQ region is also supported by measurements on partial reactions. No HCO_3^- effect was observed on the kinetics of O_2 evolution (8), recovery of Z from Z^+ (as monitored by ESR signal II_{vf}), and the recovery of P680 from $P680^+$ (as monitored by fluorescence rise) (12); and no effect was seen in the electron flow from H_2O to Q_A (as monitored by O_2 evolution with silicomolybdate as an electron acceptor, with diuron present (17), or with ferricyanide as an electron acceptor in trypsin treated thylakoids) (18). However, a dramatic HCO_3^- effect was observed in electron flow from H_2O to Q_B or PQ (as monitored by electron flow to oxidized diaminodurene, with DBMIB present to block electron flow beyond PQ) (17). Furthermore, the HCO_3^- effect was absent in PSI reactions as measured by electron flow from reduced diaminodurene to methylviologen (with diuron present) (17), or from reduced duroquinone to methylviologen (S. Izawa, personal communication).

The relation of the HCO_3^- to the protein (Q_B- or a nearby-protein) to which herbicides bind was first suggested (19) when the binding of ^{14}C-atrazine was found to be drastically reduced by HCO_3^- depletion; the binding was restored when HCO_3^- was supplied. Beginning with the early work of Renger (20), a picture has emerged (21,22) that herbicides bind to the Q_B^- or

a nearby-protein. Measurements of the Hill reaction rates as a function of [HCO_3^-] with and without herbicide suggested a competitive binding (18), in apparent contradiction to ref. (19). This descrepancy was solved when measurements were made in the same samples of (a) the Hill reaction measurements with and without a fixed [ioxynil (I_h)] as a function of varying [HCO_3^-] and (b) the binding of ^{14}C-ioxynil with and without a fixed [HCO_3^-] as a function of varying [^{14}C-ioxynil] (23). The earlier data (18,19) were confirmed and a simple explanation emerged: when HCO_3^- is the limiting factor, less herbicide binds to HCO_3^- -free membranes, and thus more is available to inhibit the Hill reaction of HCO_3^- -sufficient membranes. These results on the interaction of herbicides and HCO_3^- can be explained by assuming that the absence of HCO_3^- causes a conformational change in the Q_B protein region such that the binding of I_h is also reduced (see scheme 2).

B. Active Species

The question of the active species involved in the bicarbonate effect has been around for quite a while (3,7). There could be different chemical species involved in the diffusion, the binding, and the actual biochemical action. The effect has a maximum at pH 6.4 (17), the pKa of HCO_3^-/CO_2 system, suggesting that both CO_2 and HCO_3^- may be involved (24). A possible role of the HCO_3^- species was suggested from the competitive nature of anions like formate and acetate (7,25). The diffusing species is, however, CO_2. At 5°C, where the equilibriation between CO_2 and HCO_3^- is slowed down, the lag observed in the initiation of the electron flow in HCO_3^- -depleted thylakoids is much longer with HCO_3^- than with CO_2 injection (26). However, if carbonic anhydrase is added, the lag with CO_2 is increased; these results were especially evident when formate was absent from the reaction medium (27). Furthermore, the difference in the kinetics of electron flow when CO_2 or HCO_3^- is injected is retained even when the surface charges of the membrane are shielded by the addition of the divalent or trivalent cations (28). Thus, the existence of a negatively charged barrier between the outside of the thylakoid membrane and the HCO_3^- binding site is proposed (7).

Although the diffusing species is CO_2, the biochemically active species must include HCO_3^-. Under equilibrium conditions the rate of electron flow in HCO_3^- -depleted membranes increases when [HCO_3^-] is increased with [CO_2] constant, but not when [CO_2] is increased with [HCO_3^-] constant, suggesting that HCO_3^- is, at least, one of the active species in the reconstitution process (D. Blubaugh and Govindjee, unpublished). This view is supported by the competitive action of formate and acetate (7,25).

C. Specificity

There is no known substitute for HCO_3^- in the reactivation of electron flow in HCO_3^--depleted thylakoids. Phosphate, pyrophosphate, arsenate, nitrate, trimethylacetate, p-hydroxybenzoate, glycine, and tricine (25), HCO_3^- analogs like HSO_3^- or HPO_3^{2-}, and CO_2 analogs like CS_2 (15), Cl^-, and formate (HCO_2^-) do not restore electron flow in HCO_3^--depleted thylakoids. It appears that the binding of HCO_3^- is tight since it takes special efforts (low pH and formate treatments) to remove it from the membrane. In addition, HCO_3^- is highly specific in another sense: about 1 HCO_3^- molecule is bound per 480 Chl molecules (29).

III. CHLORIDE

The role of Cl^- in photosynthesis was also discovered by Warburg (30); its role in photosynthetic electron transport from H_2O to $NADP^+$ is however restricted to the O_2-evolving (or electron donor) side of PSII (31-33). This role is to be contrasted with that of HCO_3^- which has been shown to function on the electron acceptor side of PSII, although HCO_3^- can, with a very low efficiency, replace Cl^- on the donor side. There is another difference: whereas HCO_3^- is a very specific anion for the acceptor side, Cl^- can be replaced, to some extent, by Br^- and NO_3^- on the donor side. It is, however, assumed that in vivo it is Cl^- that functions in that capacity.

Although the requirement of Cl^- on the O_2 evolving side is established, its mechanism of action is unknown (see S. Izawa, these proceedings). We will, however, review the experiments in which halophytic plants were used to show that Cl^- indeed functions on the electron donor side in PSII. We will also summarize the conclusions obtained through the application of ^{35}Cl-NMR studies to thylakoids from halophytes (34,35).

A. Site of Chloride Action in Halophytes

Thylakoids from certain salt-tolerant plants require high (>250 mM) [Cl^-] for maximal rate of O_2 evolution (36). Both the rate of O_2 evolution and the variable ($F_{max} - F_o$) Chl fluorescence intensity increase as the [Cl^-]is raised in the halophytes, *Avicennia germinans* and *Aster tripolium* (34). In the absence of added Cl^-, the variable Chl fluorescence was very low, but it was restored to its maximal value by the addition of electron donors to PSII (hydroxylamine, ascorbate-catechol or diphenylcarbazide); these experiments demonstrated that the

site of Cl^- action was before the electron donation sites of the added donors. The most likely site of Cl^- action is before Z and thus close to M (see scheme 1). Furthermore, high concentrations of NO_3^- and Br^- could largely replace Cl^-. This experimental system, requiring high concentrations of Cl^- for O_2 evolution, was chosen because of its suitability for ^{35}Cl-NMR investigations.

B. ^{35}Cl-NMR and O_2 Evolution Measurements

In thylakoids the quadrupolar relaxation of bound Cl^-, not the paramagnetic relaxation or the ^1H-^{35}Cl dipolar interactions, is the dominant linebroadening process. This quadrupolar relaxation is suitable for studying the binding of ^{35}Cl ions to the thylakoids (35,37).

The first measurements of ^{35}Cl-NMR spectra, made on thylakoids from *Avicennia germinans* and *Aster tripolium* (pH 7.3), showed that the ^{35}Cl absorption line was clearly broader than that in aqueous solution of equivalent $[Cl^-]$ (35). The broadening of the Cl^- peak was taken to indicate Cl^- binding to the thylakoid membrane. The observed approximate correlation between the pH dependence of the ^{35}Cl-NMR linewidth and the rate of O_2 evolution between pH 6-8 suggests a relationship between Cl^- binding and the O_2 evolution activity (34). It is of note, however, that in some cases a decrease in pH from 7.0 to 6.0, that led to a decrease in Cl^- binding, did not affect the O_2 evolution activity (Fig. 6A, ref. 34). Two additional observations were made: (a) removal of the very loosely bound Mn^{2+}, by washing with ethylenediaminetetraacetic acid, did not affect the ^{35}Cl-NMR linewidths, and (b) heat treatment of the thylakoids at 48°C for 3.5 minutes, which abolished O_2 evolution completely, decreased the ^{35}Cl-NMR linewidth and its pH dependence, noted above. The linewidth of heat treated thylakoids was still broader than that in solution, so some Cl^- must still remain bound to the membrane. These results suggest: (a) ^{35}Cl-NMR may be used to study the role of Cl^- in O_2 evolution since it is specific to Cl^- and the width is indicative of Cl^--binding; and (b) Cl^- binding to the O_2 evolving center may be estimated by subtracting the amount of Cl^- bound after specific heat treatment from that of the untreated sample.

C. Cl^- Binding Parameters from ^{35}Cl-NMR Measurements

The following information regarding ^{35}Cl-binding to thylakoids from halophytes has now been obtained.

1. Fast Chemical Exchange: Two observations suggest that there is a fast chemical exchange of Cl^- between the membrane sites and the bulk phase. (a) There is a single Lorentzian line

for most thylakoids (only when thylakoids are stacked and Cl^- may be trapped, is there an additional broad line). (b) As the temperature is increased, ^{35}Cl-NMR linewidth narrows suggesting that $T_{2(bound)}$ (the transverse relaxation time of the bound Cl^-) dominates the measurements. From these observations, the value of the linewidth, and certain other assumptions, the exchange rate was estimated to be >1,000 sec^{-1} (35).

2. Cl-Binding Constant (K_b): This was obtained (35) as follows: The inverse of the linewidth ($\Delta\nu$) for bound Cl^-, y (= $(\Delta\nu observed - \Delta\nu free)^{-1}$), was plotted as a function of $[Cl^-]$. The K_b was calculated by dividing the slope of the curve by the intercept of the plot since, for the fast exchange limit,

$$y = \frac{(\Delta\nu bound - \Delta\nu free)^{-1}}{[sites]}\,[Cl^-] + \frac{(\Delta\nu bound - \Delta\nu free)^{-1}}{K_b\,[sites]}.$$

Such a calculation yielded a value of 1.13 M^{-1} (for K_b in halophytes) which is very similar to the value for Cl^- binding to site B on hemoglobin (38).

3. Cl-Binding Energy: Using the relation $K_b \simeq A\, e^{\frac{-\Delta E\ binding}{kT}}$ (where k is Boltzman constant, A is another constant, and T = absolute temperature) and, by measuring K_b at two temperatures, one can calculate ΔE from the relationship (35):

$$\Delta E = k\,\frac{\ln (K_{b2}/K_{b1})}{T_{(1)}^{-1} - T_{(2)}^{-1}}$$, where $T_{(1)}$ and $T_{(2)}$ are the two

temperatures and K_{b1} and K_{b2} are binding constants at $T_{(1)}$ and $T_{(2)}$, respectively. Using this method, a value of ~9Kcal/mole for ΔE was obtained (35). This value shows that the character of Cl-binding is weakly ionic.

D. Estimate of the Number of Cl^- Sites per PSII.

It is not easy to obtain a firm number from the existing data. However, we are able to make an estimate with some assumptions (see below). This estimate for halophytes, for Cl^- sites that may be related to the O_2 evolution activity, is 20 -40 Cl^-/400 Chl a; its order of magnitude compares favorably with a value of 10 Cl^-/ 600 Chl, obtained in spinach by measuring ^{36}Cl binding (39). The details of the method and the assumptions involved in obtaining the estimate for Cl^-/PSII follow. The inverse of $\Delta\nu observed - \Delta\nu free$ (= y) as a

function of (thylakoid concentration)$^{-1}$, in terms of [Chl]$^{-1}$, was plotted for a fixed [Cl^-], 0.6 M. The number of Cl^- binding sites is unknown, but it must be related to [Chl] by a fixed ratio: [Sites] = [Chl]/f. The measured slope of the curve y as a function of [Chl]$^{-1}$ can be shown (35) to equal $f \cdot (\Delta\nu bound)^{-1} \cdot ([Cl^-] + 1/Kb)$. Since $\Delta\nu$bound is expected to be about 10 KHz (based on a comparison with the stronger binding of Cl^- to a site on hemoglobin (38)),and since the other parameters are known ([Cl^-] = 0.6 M; K_b = 1.13 M^{-1}), one can estimate f to be about 4 (35). If we assume that PSII contains 400 Chl molecules, then there are 100 Cl^- sites per PSII. However, under complete inhibition of O_2 evolution activity, we had only a 20-40% reduction of the linebroadening; thus, the number of Cl^- binding sites associated with O_2 evolution is 20-40 Cl^-/400 Chl in halophytes (35). This number remains to be determined for other plants from ^{35}Cl-NMR data.

The Cl^- associated with the O_2 evolution may be distributed between two pools. In spinach, the two Michaelis-Menten constants, K_M, are $\sim$1 mM and 8 mM (32) and the corresponding ones for halophytes (35) are 5-10 mM and 20-25 mM (pH 7.8) or 1 mM and 12 mM (pH 7.0); these latter numbers are not too different from those of spinach.

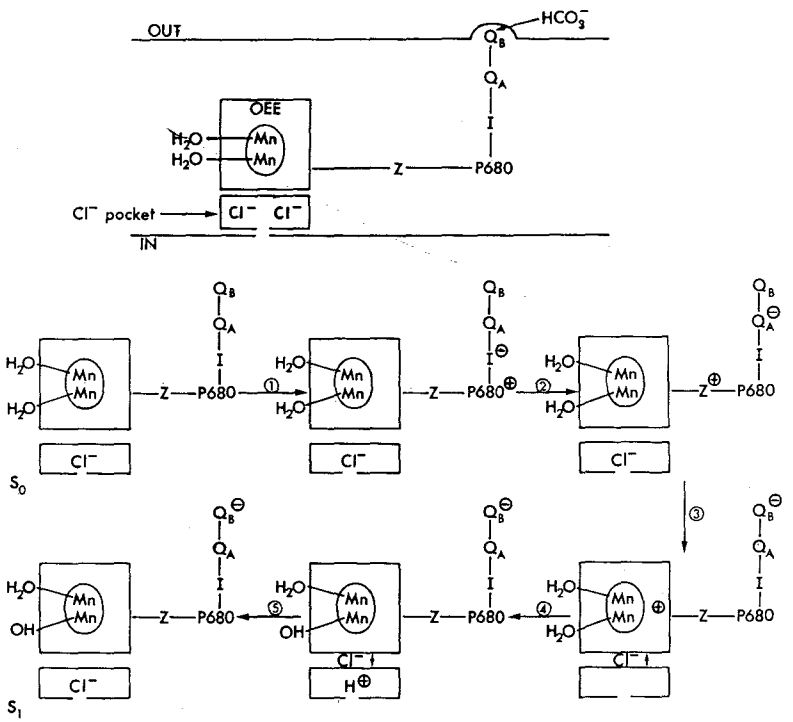
E. A Model of Anion Specificity

Our current hypothesis (34,35) is that the Cl^- related to O_2 evolution is bound to the Mn-containing oxygen evolving enzyme (40); its location is on the inner side of the thylakoid membrane and the Cl-binding site is in a "pocket". The binding energy of Cl^- ions is $\sim$9 Kcal/mole, and the binding is reversible (the exchange rate is >1,000 sec^{-1}). Cl^- ions must be bound to some positively charged species. F^- ions inhibit O_2 evolution, in this picture, by replacing Cl^- (suggested from O_2 data (34), and from NMR data, Baianu, I.C., unpublished). This inhibition occurs, in our view, because F^- binds more tightly than Cl^- since its ionic field is about 1.8 times that of Cl^-; this does not permit weaker reversible binding, which we postulate, is necessary for the operation of the "S" states. The situation with OH^- is more complex. Even though OH^- has approximately the same ionic field (1.6 times that of Cl^-) as F^-, it inhibits O_2 evolution at much lower concentrations (3 μM versus 100 mM) suggesting that OH^- and F^- must have different actions. The OH^- may substantially modify the charge distribution in the thylakoids and/or the binding sites.

In contrast, SO_4^{2-} and PO_4^{2-}, which have 1.3 to 1.7 times the ionic field of Cl^-, do not inhibit O_2 evolution in the presence of Cl^-, unlike F^- and OH^-. PO_4^{2-} and SO_4^{2-} do not replace Cl^- as they are too large (their anion volume is in the range of 55Å^3)

and, thus, cannot enter the "Cl^- pocket" (the anion volume of Cl^- is 25Å^3). The order of effectiveness of those anions that can replace Cl^- is Br^-, NO_3^-, and I^-, with Br^- being the most effective (32,34). This order of effectiveness is paralleled by both an increasing volume (30Å^3 to 40Å^3) and a decreasing ionic field. Since the Cl^- is most effective in stimulating O_2 evolution, we have suggested that its molecular volume and ionic field are optimum for the function it performs. We believe that Cl^- stabilizes the positive charges accumulated on the electron donor side of PSII. We further suggest that its binding and unbinding facilitates the operation of the O_2 clock, and this is why a reversible binding and an appropriate binding energy are so crucial to the function of Cl^- *in vivo*.

Our current scheme (scheme 3), though speculative, describes the binding and release of Cl^- during the S-state transitions (only the $S_0 \rightarrow S_1$ transition is shown). It should be self explanatory. We propose that there are several Cl^- ions that are present in a pocket (also see ref. 39) and can exchange with Cl^- near the O_2 evolving enzyme. This is expected to be on



Scheme 3

TABLE I. Chloride Binding and Unbinding at the O_2 Evolving Enzyme (from and to a "Chloride Pocket") during the S-State Transitions: A Working Model.

Reactions	$S_0 \rightarrow S_1$	$S_1 \rightarrow S_2$	$S_2 \rightarrow S_3$	$S_3 \rightarrow S_0$
Cl^- Binding	Yes	Yes	Yes	Yes (2x)
Cl^- Unbinding	Yes	No	Yes	Yes (2x)
Change in Positive Charge on the O_2 evolving enzyme	1 charge formed and 1 removed	1 charge formed but not removed	1 charge formed and 1 removed	1 charge formed but 2 removed
Proton Release [see review in 40]	$1H^+$	None	$1H^+$	$2H^+$
Possible "State" of Water Molecules in PSII	$(2H_2O)$ ↓ -1e (OH,H_2O)	(OH,H_2O) ↓ no change	(OH,H_2O) ↓ -1e (OH,OH)	(OH,OH) ↓ -2e (O_2)

the inner side of the membrane (see top of scheme 3), and, is shown, for brevity with only one or two ions. [For comparison, the postulated site of HCO_3^- on the Q_B-protein is also shown in the top portion of the scheme.] Postulated action of Cl^- on the other S-state transitions, along with the proton release, is shown schematically in Table I. No attempt has been made to propose a model for the other changes occurring in PS II.

IV. CONCLUDING REMARKS

The role of two anions, HCO_3^- and Cl^-, in photosystem II has been reviewed here: the former appears to function on the electron acceptor side, whereas the latter functions on the electron donor side of PSII. An NMR technique has been used for the first time to learn more about the Cl^- binding to the thylakoid membrane, and its relationship to the molecular mechanism of O_2 evolution.

Our collected impression is that the major function of HCO_3^- is to change the conformation of the Q_B (or a nearby-) protein to allow the efficient exchange of Q_B^{2-} with a PQ molecule (for a theoretical model of HCO_3^- action, see (41)). On the other hand, the major function of Cl^- is to stabilize the positive charges on the electron donor (water) side of PSII, possibly via a reversible binding to the Mn-containing oxygen evolving enzyme.

ACKNOWLEDGEMENTS

We are specially grateful to W.F.J. Vermaas, for collaboraion in the bicarbonate research reviewed here. We thank T. Ogawa, A.W. Rutherford, G. Renger and Rajni Govindjee for discussions during the preparation of this manuscript. Thanks are also due to I. Terashima, T. Ogawa, A. Gallagher, M. Ikeuchi and H. Koike for help with the drawing of figures. Our special thanks are due to Agnès Rutherford for typing the manuscript, and to M. Kimimura for typing the camera-ready copy.

REFERENCES

1. Warburg, O. and Krippahl, G. (1958) Z. Naturforsch. 13B, 509-514.
2. Warburg, O., Krippahl, G., Gewitz, H.S. and Völker, W. (1959) Z. Naturforsch. 14B, 712-724.
3. Stemler, A. (1982) in Photosynthesis II. Development, Carbon Metabolism and Plant Productivity (Govindjee, ed.) pp. 513-539, Academic Press, New York.
4. Metzner, H., Fischer, K. and Bazlen, O. (1979) Biochim. Biophys. Acta 548, 287-295.
5. Wydrzynski, T. and Govindjee (1975) Biochim. Biophys. Acta 387, 403-408.
6. Govindjee and van Rensen, J.J.S. (1978) Biochim. Biophys. Acta 505, 183-213.
7. Vermaas, W.F.J. and Govindjee (1982) Proc. Indian Natl. Acad. Sci. B47, 581-605.
8. Vermaas, W.F.J. and Govindjee (1982) reference 3, pp. 541-558.
9. Velthuys, B.R. (1981) FEBS Lett. 126, 277-281.
10. Wraight, C.A. (1981) Israel J. Chem. 21, 348-354.
11. Cramer, W.A. and Crofts, A.R. (1982) in Photosynthesis I. Energy Conversion by Plants and Bacteria (Govindjee, ed.) pp. 537-588, Academic Press, New York.
12. Jursinic, P., Warden, J. and Govindjee (1976) Biochim. Biophys. Acta 440, 322-330.
13. Siggel, U., Khanna, R., Renger, G. and Govindjee (1977) Biochim. Biophys. Acta 462, 196-207.
14. Govindjee, Pulles, M.P.J., Govindjee, R., van Gorkom, H.J. and Duysens, L.N.M. (1976) Biochim. Biophys. Acta 449, 602-605.
15. Vermaas, W.F.J. and Govindjee (1982) Biochim. Biophys. Acta 680, 202-209.

16. Jursinic, P. and Stemler, A. (1982) Biochim. Biophys. Acta 681, 419-428.
17. Khanna, R., Govindjee and Wydrzynski, T. (1977) Biochim. Biophys. Acta 462, 208-214.
18. van Rensen, J.J.S. and Vermaas, W.F.J. (1981) Physiol. Plant. 51, 106-110.
19. Khanna, R., Pfister, K., Keresztes, A., van Rensen, J.J. S. and Govindjee (1981) Biochim. Biophys. Acta 634, 105-116.
20. Renger, G. (1976) Biochim. Biophys. Acta 440, 287-300.
21. Trebst, A. and Draber, W. (1979) in Advances in Pesticides Science, Part 2 (Geissbühler, H., ed.) pp. 223-234, Pergamon Press Oxford.
22. Pfister, K., Steinback, K.E., Gardner, G. and Arntzen, C.J. (1981) Proc. Natl. Acad. Sci. U.S.A. 78, 981-985.
23. Vermaas, W.F.J., van Rensen, J.J.S. and Govindjee (1982) Biochim. Biophys. Acta 681, 242-247.
24. Vermaas, W.F.J. and van Rensen, J.J.S. (1981) Biochim. Biophys. Acta 636, 168-174.
25. Good, N. (1963) Plant Physiol. 38, 298-304.
26. Sarojini, G. and Govindjee (1981) Biochim. Biophys. Acta 634, 340-343.
27. Sarojini, G. and Govindjee (1981) in Photosynthesis II. Photosynthetic Electron Transport and Photophosphorylation (Akoyunoglou, G., ed.) pp. 143-150, Balaban International Science Services, Philadelphia.
28. Sarojini, G. and Govindjee (1981) Abstracts, 9th Annual Meeting of the American Society for Photobiology, MPM C8, p. 86.
29. Stemler, A. (1977) Biochim. Biophys. Acta 460, 511-522.
30. Warburg, O. and Lüttgens, W. (1944) Naturwissenschaften, 32, 301.
31. Izawa, S., Heath, R.L. and Hind, G. (1969) Biochim. Biophys. Acta 180, 388-398.
32. Kelley, P.M. and Izawa, S. (1978) Biochim. Biophys. Acta 502, 198-210.
33. Muallem, A., Farineau, J., Laine-Boszormenyi, M. and Izawa, S. (1981) reference 27, pp. 435-443.
34. Critchley, C., Baianu, I.C., Govindjee and Gutowsky, H.S. (1982) Biochim. Biophys. Acta 682, 436-445.
35. Baianu, I.C., Critchley, C., Gutowsky, H.S. and Govindjee (1983) Proc. Natl. Acad. Sci. U.S.A., submitted.
36. Critchley, C. (1982) Nature (London) 298, 483-485.
37. Dwek, R.A. (1973) NMR in Biochemistry, Clarendon Press, Oxford.
38. Chiancone, E., Norne, J.E., Forsen, S., Antoniai, E. and Wyman, J. (1972) J. Mol. Biol. 70, 675-688.

39. Theg, S.M. and Homann, P.H. (1982) Biochim. Biophys. Acta 679, 221-224.
40. Wydrzynski, T. (1982) Reference 11, pp. 469-506.
41. Shipman, L. (1981) J. Theoret. Biol. 90, 123-148.