ELSEVIER

Biochimica et Biophysica Acta 1229 (1995) 208–214

BBA Biochimica et Biophysica Acta

The kinetics of photophosphorylation at clamped ΔpH indicate a random order of substrate binding

Gunther Kothen, Oliver Schwarz, Heinrich Strotmann *

Institut für Biochemie der Pflanzen, Heinrich Heine Universität, Universitätstraße 1, 40225 Düsseldorf, Germany

Received 16 September 1994; revised 14 December 1994; accepted 9 January 1995

Abstract

The rate of photophosphorylation of isolated chloroplast thylakoids was investigated at a clamped ΔpH of 2.5 ($\Delta\Psi = 0$). On variation of the concentration of ADP at different fixed concentrations of phosphate, straight lines were obtained in double-reciprocal plots which intersected in one point below the *x*-axis. Upon variation of the phosphate concentration at several fixed concentrations of ADP, similar kinetics were found. These results indicate a random order of binding of the two substrates. Calculation of the dissociation constants and Michaelis constants from these two sets of data according to a bisubstrate rapid equilibrium random model yielded satisfactory agreement. The kinetic constants were found to be unchanged by thiol modulation or demodulation of CF_0CF_1. The kinetics of inhibition of phosphorylation by the product ATP indicated a competitive effect with regard to ADP as well as phosphate. At a given substrate concentration, the inhibition by ATP was larger at lower than at higher concentration of the respective cosubstrate. These results fully confirm the proposed random mechanism.

Keywords: Photophosphorylation; Chloroplast; CF_0CF_1; ΔpH clamp; Enzyme kinetics

1. Introduction

Photophosphorylation of chloroplasts is driven by an electrochemical proton gradient across the thylakoid membrane which is built up by photosynthetic electron transport, and catalyzed by a reversible proton translocating ATPase. This enzyme, which is a constituent of the thylakoid membrane, consists of the membrane-integral sector CF_0 which acts as a proton channel, and the extrinsic hydrophilic part CF_1 which carries the catalytic centers [1]. While ATP is formed, protons are translocated through the ATPase complex. The H^+/ATP stoichiometry has been determined in the past to be 3 [2–4], but more recently a ratio of 4 [5] was reported.

The mechanism of coupling of proton flow with ATP formation has not been definitively understood. Probably energy coupling includes conformational changes of the protein as proposed by Boyer and his co-workers [6]. According to the so-called 'energy-linked binding change mechanism' the energy of the transmembrane proton gradient is invested to bind the substrates and release the product rather than to facilitate the formation of the phosphoanhydride bond. A variety of experimental results speak in favor of this hypothesis (review in Ref. [6]).

The kinetics of photophosphorylation and oxidative phosphorylation have often been investigated, but with partly diverging results. A difficult problem is the maintenance of the proton gradient, particularly at limiting electron transport. The extent of the transmembrane proton gradient is a function of the rate of proton influx driven by light-dependent electron transport on one hand, and the rates of nonproductive and coupled proton effluxes on the other hand. Increase of the concentrations of ADP and phosphate stimulates proton efflux due to acceleration of phosphorylation and thus causes a drop of the steady-state proton gradient. As emphasized by several authors [7–10], the validity of enzyme kinetic data obtained at non-constant driving force may be challenged.

Abbreviations: CF_0CF_1, proton translocating ATPase ('ATP synthase') of the thylakoid membrane of chloroplasts; Chl, chlorophyll; ΔpH, transthylakoidal pH difference; Tricine, *N*-[2-hydroxy-1,1-bis(hydroxymethyl)ethyl]glycine.

* Corresponding author. Fax: +49 211 3113706.

SSDI 0005-2728(95)00005-4

Recently, we have introduced a method to keep the proton gradient constant, independent of the experimental conditions ('ΔpH clamp') [10]. It is based on the idea that the gradient will be maintained if changes of the rate of proton efflux are balanced by exactly the same alterations of proton influx. In chloroplasts, the rate of proton influx can be easily changed by modulation of the intensity of the photosynthetic light. Kinetic changes of the proton gradient can be conveniently monitored by the fluorescence of 9-aminoacridine [11]. Our ΔpH clamp technique employs the fluorescence of 9-aminoacridine as measuring parameter in an electronic feedback circuit. As expected, the kinetics of photophosphorylation obtained at clamped proton gradient were rather different from those obtained at the same light intensity [10].

The binding order of the two substrates ADP and phosphate in phosphorylation is a characteristic attribute of the catalyst CF_0CF_1. On the basis of inhibition studies with thiophosphate and the thiophosphate analogue of ADP, adenosine 5'-*O*-(2-thiodiphosphate), a compulsory order with binding of ADP preceding the binding of phosphate was proposed for photophosphorylation [12]. Enzyme kinetic analyses on mitochondrial and bacterial vesicles, on the other hand, indicated a random mechanism for oxidative phosphorylation [13,14]. In the present study we have employed our ΔpH clamp technique to approach this problem again by kinetic experiments on isolated chloroplast thylakoids under energetically defined conditions.

2. Experimental

2.1. ΔpH clamp instrument

The ΔpH clamp instrument was described in Ref. [10]. As a modification, the electronic feedback controller was replaced by a computer-sustained device with appropriate software. An IBM-compatible personal computer was equipped with a 12-bit analog/digital and 12-bit digital/analog converter. The program, being an implementation of a proportional-differential controller, was written with the compiler MS-Quick-Basic. The digital controller affords faster and more sensitive regulation dynamics. Moreover, the modified instrument allows automatic correction for the artificial quench of the 9-aminoacridine fluorescence signal caused by ADP or ATP [10]. To this end, a quench curve was stored in the computer.

2.2. Methods

Chloroplast thylakoids were isolated from freshly harvested spinach leaves as described in Ref. [15]. The reaction medium consisted of 25 mM Tricine buffer (pH 8.0), 50 mM KCl, 5 mM $MgCl_2$, 50 μM pyocyanine, 50 nM valinomycin, 10 mM glucose and 20 U ml^{-1} hexokinase (salt-free, Sigma) and contained chloroplasts at a final chlorophyll concentration of 25 μg/ml. The final volume was 2.5 ml, the temperature was 20° C. When thiol-modulated thylakoids were employed, the medium contained additional 10 mM dithiothreitol.

For detection of ΔpH ($\Delta\Psi$ is cancelled by valinomycin/K^+) the basal fluorescence and then the maximal fluorescence following the addition of 5 μM 9-aminoacridine are stored in the computer. Subsequently the thylakoids are preilluminated for 2 min at full light intensity (≈ 400 W m^{-2} red light > 630 nm). After decay of the fluorescence signal, the controller unit is activated to find the light intensity necessary to establish the prechosen steady-state ΔpH of 2.5 units. After 2 min, phosphorylation is started by the addition of ADP and [^{32}P]phosphate at the indicated concentrations. This addition gave rise to an immediate increase of light intensity to keep the ΔpH constant. Four samples were taken within the following 30 s, immediately deprotonized by 0.5 M $HClO_4$ and stored on ice. The ADP-regenerating hexokinase system was included to avoid accumulation of ATP and exhaustion of ADP. However, in the experiments in which inhibition of

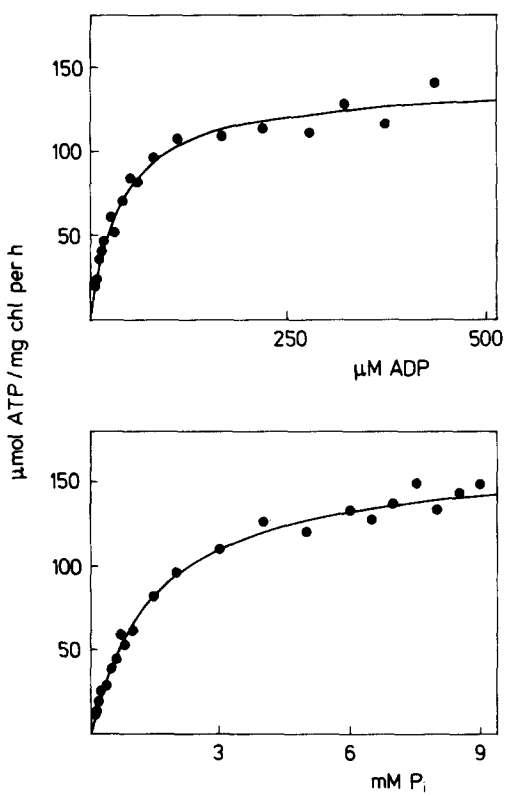


Fig. 1. Rate of photophosphorylation as function of the ADP concentration at a constant phosphate concentration of 10 mM (upper part) and as function of phosphate concentration at a constant ADP concentration of 0.5 mM (lower part). The ΔpH was clamped at 2.5 units, the chloroplasts were unmodulated. The results show original data of two individual experiments. The curves are computed hyperbolic fittings.

phosphorylation by ATP was assayed, hexokinase and glucose had to be omitted. In these experiments, rates were mathematically corrected by the method of Lee and Wilson [16]. The formed ^{32}P-labeled ATP or glucose 6-phosphate, respectively, was determined by separation of inorganic from organic [^{32}P]phosphates [17] followed by measurement of Cerenkov radiation.

For quantification of the 9-aminoacridine fluorescence signal, the equilibrium phosphate potential calibration method [18] was employed. In the calculation an H^+/ATP, stoichiometry of 4 [5] instead of 3 was used. This depressed the previously employed ΔpH scale [10,18,19] by about 0.6 units.

3. Results

3.1. Variation of substrate concentrations

Photophosphorylation of non-modulated or thiol-modulated thylakoids measured at clamped ΔpH as function of ADP concentration (5 to 500 μM) or phosphate concentration (50 μM to 10 mM) yielded hyperbolic curves. Fig. 1 shows two examples. ATP hydrolysis of thiol-modulated thylakoids measured as function of ATP concentration (5 to 500 μM) likewise yielded hyperbolic curves (not shown). These results indicate that the proton transport-coupled ATPase reaction in forward and backward direction apparently follows the Michaelis-Menten theory. In previous studies we have demonstrated that at clamped ΔpH the Michaelis constants for ADP [19,20] and for phosphate [10] were independent of the extent of the proton gradient in the range employed, although v_{max} increased exponentially with ΔpH. These results led to the conclusion that binding and dissociation of a substrate (at cosubstrate saturation) were fast processes in comparison with the rate-limiting reaction, which allowed us to apply the mathematics of a rapid equilibrium enzyme mechanism. It should be noted that an increase of the K_m for ADP was reported at high proton gradients [21], a result which was interpreted to indicate diffusion limitation. This ΔpH range, however, was instrumentally not available in our experiments.

In a bi-substrate rapid equilibrium enzyme reaction, the order of binding of the two substrates may be discriminated by analyzing reaction rates as a function of the concentration of one substrate at several fixed concentrations of the other substrate and vice versa. In this study we have employed thiol modulated and non-modulated thylakoids. For thiol modulation, the thylakoids were preilluminated in the presence of DTT, a treatment by which a disulfide group is reduced to a dithiol group in the γ subunit of CF_1 [22]. Subsequently, the rates of phosphorylation were assayed as function of substrate concentrations at a clamped ΔpH of 2.5.

Fig. 2a shows Lineweaver-Burk diagrams of phosphorylation by thiol-modulated thylakoids as function of the concentration of ADP at four different fixed concentrations of phosphate. In Fig. 2b the inverse experiment (variation of phosphate concentration at three fixed ADP concentrations) is shown. In both series the straight lines intersect at one point located below the x-axis. The symmetric kinetics obtained at variation of ADP or phosphate, respectively, exclude a compulsory order of substrate binding, but is a characteristic feature of a random mechanism. In an ordered rapid equilibrium mechanism the apparent Michaelis constant for the leading substrate would approach zero when the following substrate approaches saturation, and upon variation of the second substrate, the maximal velocity would be independent of the concentration of the leading substrate. Accordingly, the intersection points would be above rather than below the x-axis. Fig. 3 shows Lineweaver-Burk plots of phosphorylation by non-modulated chloroplasts as function of phosphate concentration at three different ADP concentrations. The principal similarity to the results of Fig. 2b is obvious.

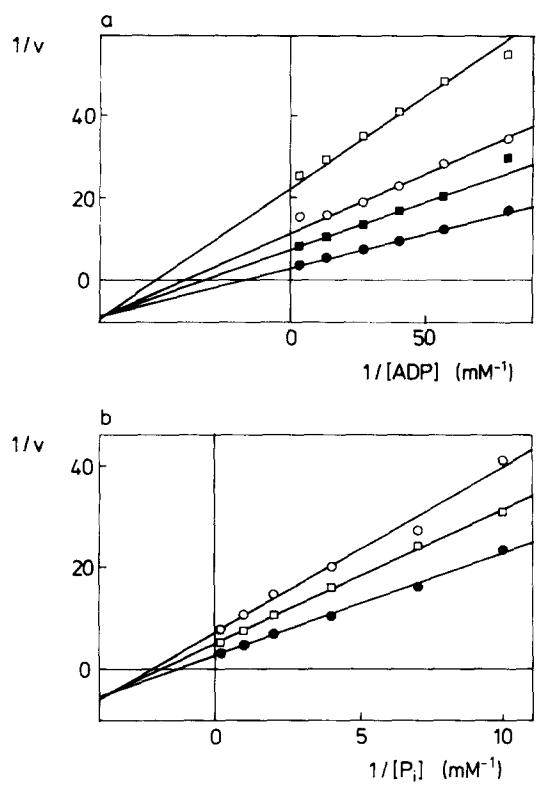
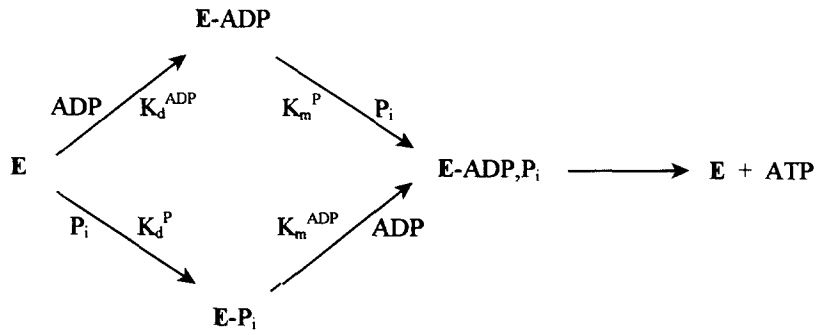


Fig. 2. Lineweaver-Burk plots of photophosphorylation at clamped ΔpH of 2.5 with thiol-modulated thylakoids (a) as function of ADP concentration at four different phosphate concentrations: 0.1 mM (open squares), 0.2 mM (open circles), 0.5 mM (filled squares) and 5 mM (filled circles), (b) as function of phosphate concentration at three different ADP concentrations: 27 μM (open circles), 54 μM (open squares) and 550 μM (filled circles). The points are mean values of six (a) and four (b) independent experiments, respectively. $1/v$ is given in (mg Chl) h (mmol ATP)$^{-1}$.

The bisubstrate rapid equilibrium random mechanism may be described by the following reaction scheme:



Because of the symmetric character of the reaction scheme the ratios K_m^{ADP}/K_d^{ADP} and K_m^P/K_d^P must be identical. If this ratio is designated α, $K_m^{ADP} = \alpha K_d^{ADP}$ and $K_m^P = \alpha K_d^P$.

The rate equation may be expressed as follows:

$$v = \frac{[ADP][P_i]v_{max}}{\alpha\left(K_d^{ADP}[P_i] + K_d^{ADP}K_d^P + K_d^P[ADP]\right) + [ADP][P_i]}$$

The apparent Michaelis constants $K_m'^{ADP}$ and $K_m'^P$ at given concentrations of the cosubstrates $[P_i]$ or [ADP], respectively, are obtained by

$$K_m'^{ADP} = \alpha K_d^{ADP}\left([P_i] + K_d^P\right)/\left([P_i] + \alpha K_d^P\right)$$

and

$$K_m'^P = \alpha K_d^P\left([ADP] + K_d^{ADP}\right)/\left([ADP] + \alpha K_d^{ADP}\right)$$

These equations indicate that the apparent Michaelis constant extrapolates to the dissociation constant at zero concentration of the cosubstrate. The apparent Michaelis constant, on the other hand, approaches the true Michaelis constant when the enzyme is saturated with the second substrate, i.e., when $[P_i] \gg K_d^P$ or $[ADP] \gg K_d^{ADP}$, respectively. The parameters K_d^{ADP}, $K_m^{ADP} = \alpha K_d^{ADP}$, K_d^P, $K_m^P = \alpha K_d^P$ and v_{max} can be determined from secondary plots. To this end, the slopes (s) and the intercepts on the y-axis (i) of the Lineweaver-Burk diagrams are plotted as function of the reciprocal of the respective cosubstrate concentration. These plots again yield straight lines with slopes s_s and s_i and y-axis intercepts i_s and i_i, respectively (Table 1). When ADP is the variable substrate, the constants are obtained as follows: $K_d^{ADP} = s_s/s_i$, $K_m^{ADP} = i_s/i_i$, $K_d^P = s_s/i_s$, $K_m^P = s_i/i_i$, $V_{max} = 1/i_i$ [23]. Correspondingly, the five parameters can be obtained from secondary plots of the experiments with P_i as the variable substrate. The constants obtained from the three sets of experiments are listed in Table 1. For thiol-modulated chloroplasts the parameters computed from the two different experimental series are in good agreement. Moreover, the dissociation and the Michaelis constants obtained for non-modulated chloroplasts match with those obtained with modulated chloroplasts. The maximal velocity of photophosphorylation at ΔpH of 2.5, on the other hand, is lower by a factor of 4–5 with non-modulated than modulated thylakoids. The Michaelis constant for ADP (47–65 μM) is in the range of K_m values determined at constant high light intensity in cyclic photophosphorylation [12,24] or at constant proton gradient [19]. The actual substrate, however, probably is the ADP-Mg complex rather than free ADP [25,26]. Under our conditions (12.5 to 275 μM ADP in the presence of 5 mM Mg^{2+}), the calculated complex concentration is nearly constantly lower by 8 to 9% than the total ADP concentration if a complex dissociation constant of $4.57 \cdot 10^{-4}$ (own determination, unpublished) is used, and less than 3% lower if a dissociation constant of $1.25 \cdot 10^{-4}$ [26] is used. Hence, the K_m for the ADP-Mg complex may be slightly lower [26]. The K_m value for phosphate (0.8–0.9 mM) is somewhat higher than that determined at uncontrolled proton gradient [12,26], but agrees well with the values determined at clamped ΔpH [10,27].

Figs. 2 and 3 show that the apparent K_m values for ADP increase with increasing concentration of phosphate

Table 1
Kinetic parameters calculated from the results of Figs. 2 and 3

	[ADP] varied; reduced enzyme	[P_i] varied	
		reduced enzyme	oxidized enzyme
K_d^{ADP} [μM]	16	17	14
K_m^{ADP} [μM]	65	50	47
K_d^P [mM]	0.21	0.28	0.27
K_m^P [mM]	0.82	0.86	0.88
v_{max} [μmol(mg Chl)$^{-1}$ h^{-1}]	417	400	91
K_m^{ADP}/K_d^{ADP}	4.1	2.9	3.3
K_m^P/K_d^P	3.9	3.1	3.3

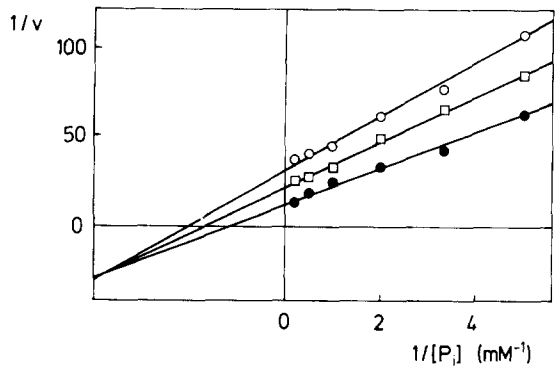


Fig. 3. Lineweaver-Burk plots of photophosphorylation with non-modulated thylakoids at clamped ΔpH of 2.5 as function of phosphate concentration at 25 μM (open circles), 50 μM (open squares) and 500 μM (filled squares) ADP. The values are means of 4 independent experiments. $1/v$ is given in (mg Chl) h (mmol ATP)$^{-1}$.

and vice versa, indicating that under the conditions employed the affinity for one substrate decreases as a consequence of binding of the other substrate. The ratio, α, between the Michaelis constants and the dissociation constants is in the range of 3–4 (Table 1).

3.2. Product inhibition

Another means to discriminate between a compulsory and a random order of substrate binding is by the kinetics of product inhibition. Here, we examined the effect of ATP on photophosphorylation by thiol-modulated thylakoids at the same clamped ΔpH of 2.5. In order to recognize the inhibition type, the concentrations of ADP or phosphate, respectively, were varied at a low and a high concentration of the respective cosubstrate in absence and presence of 470 μM ATP. For obvious reasons, the ADP-regenerating system which was routinely present in the previous experiments was omitted and the rates were corrected for the amount of substrate consumed during the reaction [16]. No correction was necessary for the back reaction (ATP hydrolysis), which was found to be insignificant at the employed ΔpH. The data (Fig. 4) clearly show competitive inhibition of phosphorylation by ATP with respect to both ADP and phosphate, which strongly confirms a random mechanism. In a compulsory order of substrate binding, the reaction product would be competitive with regard to the leading substrate, but not to the following substrate. The random mechanism is furthermore supported by the finding that the competitive effect is much more pronounced at low than at high concentration of the cosubstrate (Fig. 4). Competitive inhibition of photophosphorylation by ATP with regard to ADP was found previously. However, the measurements were carried out at high light intensity and high phosphate concentration [24]. Accordingly, the apparent inhibition constant obtained was rather high (mM range).

Thus, the theoretical evaluation of the above results

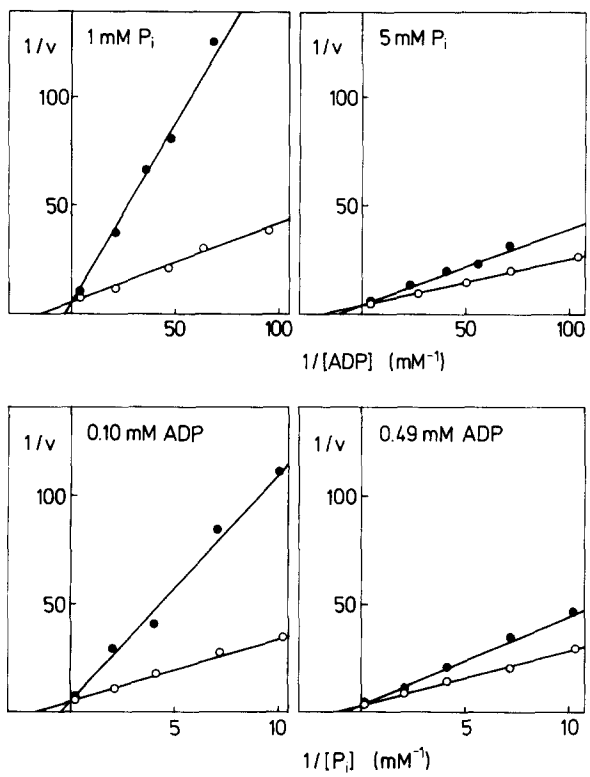


Fig. 4. Lineweaver-Burk plots of photophosphorylation with thiol-modulated thylakoids at clamped ΔpH of 2.5 as function of ADP concentration (upper part) and phosphate concentration (lower part), respectively, in the absence (open circles) and presence of 470 μM ATP (filled circles). The concentrations of phosphate (upper part) and ADP (lower part), respectively are indicated. The values are means of four to six independent experiments. $1/v$ is given in (mg Chl) h (mmol ATP)$^{-1}$.

assumes ATP to be a competitive inhibitor with regard to both substrates, ADP and phosphate. If the inhibition constant is K_i^{ATP}, the extended rate equation yields:

$$v = \{K_i^{ATP}[ADP][P_i]v_{max}\} / \{\alpha K_d^{ADP}(K_i^{ATP}[P_i] + K_d^P K_i^{ATP} + K_d^P[ATP]) + K_i^{ATP}[ADP]([P_i] + \alpha K_d^P)\}$$

If the apparent Michaelis constant for ADP at a given concentration $[P_i]$ is designated as above $K_m'^{ADP}$ in the absence and $K_m''^{ADP}$ in the presence of product at a concentration [ATP], the inhibitor constant K_i^{ATP} is calculated by the equation:

$$K_i^{ATP} = \frac{K_m'^{ADP} K_d^P [ATP]}{(K_m''^{ADP} - K_m'^{ADP})([P_i] + K_d^P)}.$$

Table 2
Inhibitor constant for ATP calculated from the results of Fig. 4

	[ADP] varied		[P$_i$] varied	
	1 mM P$_i$	5 mM P$_i$	0.1 mM ADP	0.49 mM ADP
K_i^{ATP} [μM]	27	37	27	32

For the calculation the following mean values obtained from Table 1 were employed: $K_d^{ADP} = 16$ μM, $K_d^P = 245$ μM.

In the inverse experiment (variation of the concentration of substrate B), the inhibitor constant is analogously obtained.

The values for $K_m'^{ADP}$, $K_m''^{ADP}$, $K_m'^{P}$ and $K_m''^{P}$ were obtained from Fig. 4, K_d^{ADP} and K_d^{P} were mean values taken from Table 1. The inhibition constant thus calculated for ATP obtained from the results of the four experiments of Fig. 4 is between 27 and 37 μM (Table 2). The K_m^{ATP} in ATP hydrolysis ('light-triggered ATPase') determined by the ΔpH clamp technique was found to be about 75 μM and independent of ΔpH [20].

4. Discussion

The symmetric kinetic patterns obtained upon mutual variation of the two substrates ADP and phosphate as well as the competitive kinetics of product inhibition indicate a random mechanism of chloroplast photophosphorylation at a clamped ΔpH of 2.5. The application of a rapid equilibrium random model for the calculation of the kinetic parameters yielded satisfactory quantitative agreement between the different series (Table 1). On the basis of similar studies, a random order of substrate binding was also concluded for oxidative phosphorylation of submitochondrial particles [13] and inside-out plasma membrane vesicles of *Paracoccus denitrificans* [14]. However, an ordered sequence, with ADP preceding the binding of phosphate, was proposed for photophosphorylation on the basis of inhibitor studies with analogues of phosphate and ADP [12]. Thiophosphate was found to be a competitive inhibitor with regard to inorganic phosphate, but an uncompetitive inhibitor with respect to ADP. The β-thiophosphate analogue of ADP, which is not a substrate in photophosphorylation [28], was a competitive inhibitor with regard to ADP at high phosphate concentration, but a non-competitive inhibitor with regard to ADP at low phosphate concentration and a non-competitive inhibitor with regard to phosphate [12]. The complex kinetics, which may partially be due to the fact that the proton gradient was not controlled, actually require additional assumptions [12] to fulfill an ordered mechanism. Thus, this conclusion must be regarded with some reservation.

The above model, which employs only one catalytic site, is simplified. It is known that at least two active sites participate in catalysis [6]. However, as the first site is saturated at very low substrate concentrations [29,30] and uni-site catalysis is quantitatively insignificant, the application of a bisubstrate Michaelis-Menten model may be justified for the 'physiological' substrate concentration ranges employed . The results confirm such a model with good approximation. The proposed random mechanism is compatible with the results on ^{18}O exchange [6]. Early reports that chloroplast ATPase catalyzed ATP-P_i exchange, but very low ATP-ADP exchange [31], spoke in favor of an ordered mechanism with ADP as the leading substrate. However, the subsequent finding that the ATP-P_i exchange is not an exchange reaction in strict sense but the result of ATP-driven rephosphorylation of free ADP [7], has invalidated this argument. The lack of extensive ATP-ADP exchange may be due to the fact that at low ΔpH, micromolar concentrations of ADP effectively deactivate CF_0CF_1 [1].

The proton gradient is not only the driving force of phosphorylation but also an activator of the proton translocating ATPase [1,18,32]. The ΔpH profiles of the two processes are different; furthermore, the ΔpH profiles of activation are strongly dependent on thiol modulation [32], and on the presence of effectors like ADP and phosphate [18]. The oxidized enzyme shows 50% activity at ΔpH values well above 3, but after thiol modulation the activation curve is shifted towards lower values by at least one ΔpH unit [32]. As the ΔpH curve of the catalytic reaction is intermediate, photophosphorylation at moderate proton gradients is limited by enzyme activity if the enzyme is oxidized, but limited by the driving force if the enzyme is in the reduced form [32].

At light and substrate saturation, we found, in accordance with other authors [32–34], phosphorylation rates in the range of 1400 μmol ATP/mg Chl per h, both at modulated and non-modulated CF_0CF_1. The rates at saturation of ADP and phosphate and a ΔpH of 2.5 thus account for about 30% of the absolute maximal rate in the case of reduced enzyme, but less than 10% in the case of the oxidized enzyme. Nevertheless, the kinetic constants (K_m, K_d) were largely identical (Table 1), suggesting that thiol modulation does not change the catalytic properties of CF_0CF_1. It is well known that the thiol-modulated enzyme, but not the demodulated enzyme is capable of catalyzing coupled ATP hydrolysis ('light-triggered ATPase'), whereas both forms can catalyze ATP formation. This fact is probably not due to a mechanistic difference (e.g., different H^+/ATP stoichiometries) between the two enzyme forms, but may be explained by the opposite energetic and kinetic effects of the proton gradient. ΔpH inhibits ATP hydrolysis energetically on one hand, but activates the enzyme on the other hand. Only when CF_0CF_1 is in the reduced form is the gradient necessary to support enzyme activity low enough to permit appreciable rates of ATP hydrolysis energetically [35].

Although the results reported here do not allow conclusions about the energy-linked reaction step(s) in proton transport-coupled phosphorylation, this important question should be briefly addressed. The ΔpH independency of the Michaelis constants [10,19,36] initially called into question whether the rate-limiting ΔpH-dependent step really can be the binding of the substrates and the release of the product ATP as proposed by the 'energy-linked binding change mechanism' [6]. However, the subsequent finding that the dissociation constants for ADP and P_i decrease with the increase of ΔpH and that the ΔpH dependency of the dissociation constants was the same as the ΔpH depen-

dency of overall ATP formation [20], indicated that the increase of affinity for the first of the two substrates that bind to the active center is at least one relevant ΔpH-linked reaction in photophosphorylation.

Acknowledgements

This work was supported by Deutsche Forschungsgemeinschaft (Sonderforschungsbereich 189) and by Fonds der Chemischen Industrie.

References

[1] Strotmann, H. and Bickel-Sandkötter, S. (1984) Annu. Rev. Plant Physiol. 35, 97–120.
[2] Junge, W., Rumberg, B. and Schröder, H. (1970) Eur. J. Biochem. 14, 575–581.
[3] Portis, A.R. and McCarty, R.E. (1974) J. Biol Chem. 249, 6250–6254.
[4] Rathenow, M. and Rumberg, B. (1980) Ber. Bunsenges. Phys. Chem. 84, 1059–1062.
[5] Rumberg, B., Schubert, K., Strelow, F. and Tran-Anh, T. (1990) in Current Research in Photosynthesis (Baltscheffsky, M., ed.), Vol. III, pp. 125–128, Kluwer, Dordrecht.
[6] Boyer, P.D. (1993) Biochim. Biophys. Acta 1140, 215–250.
[7] Davenport, J.W. and McCarty R.E. (1986) Biochim Biophys. Acta 851, 136–145.
[8] Quick, W.P. and Mills, J.D. (1987) Biochim. Biophys. Acta 893, 197–207.
[9] Quick, W.P. and Mills, J.D. (1988) Biochim. Biophys. Acta 932, 232–239.
[10] Strotmann, H., Thelen, R., Müller, W. and Baum, W. (1990) Eur. J. Biochem. 193, 879–886.
[11] Schuldiner, S., Rottenberg, H. and Avron, M. (1972) Eur. J. Biochem. 25, 64–70.
[12] Selman, B.R. and Selman-Reiner, S. (1981) J. Biol. Chem. 256, 1722–1726.
[13] Kayalar, C. Rosing, J. and Boyer, P.D. (1977) J. Biol. Chem. 252, 2486–2491.
[14] Pérez, J.A. and Ferguson, S.J. (1990) Biochemistry 29, 10503–10518.
[15] Strotmann, H. and Bickel-Sandkötter, S. (1977) Biochim. Biophys. Acta 460, 126–135.
[16] Lee, H.J. and Wilson, I.B. (1971) Biochim. Biophys. Acta 242, 519–522.
[17] Sugino, Y. and Miyoshi, Y. (1964) J. Biol. Chem. 239, 2360–2364.
[18] Lohse, D., Thelen, R. and Strotmann, H. (1989) Biochim. Biophys. Acta 976, 85–93.
[19] Heinen, G. and Strotmann, H. (1989) Z. Naturforsch. 44c, 473–479.
[20] Kothen, G., Schwarz, O. and Strotmann, H (1992) in Research in Photosynthesis (Murata, N., ed.), Vol. II, pp. 661–668, Kluwer, Dordrecht.
[21] Bizouarn, T., De Kouchkovsky, Y. and Haraux, F. (1989) Biochim. Biophys. Acta 974, 104–113.
[22] Schumann, J., Richter, M.L. and McCarty, R.E. (1985) J. Biol. Chem. 260, 11817–11823.
[23] Segel, I.H. (1975) Enzyme Kinetics. John Wiley & Sons, New York.
[24] Franek, U. and Strotmann, H. (1981) FEBS Lett. 126, 5–8.
[25] Komatsu, M. and Murakami, S. (1976) Biochim. Biophys. Acta 423, 103–110.
[26] Zhou, J.M. and Boyer, P.D. (1992) Biochemistry 31, 3166–3171.
[27] Strelow, F., Tran-Anh, T. and Rumberg, B. (1990) in Current Research in Photosynthesis (Baltscheffsky, M., ed.), Vol. III, pp. 125–128, Kluwer, Dordrecht.
[28] Strotmann, H., Bickel-Sandkötter, S., Edelmann, K., Eckstein, F., Schlimme, E., Boos, K.S. and Lüstorff, J. (1979) Biochim. Biophys. Acta 545, 122–130.
[29] Gräber, P. and Labahn, A. (1992) J. Bioenerg. Biomembr. 24, 493–497.
[30] Stroop, S.D. and Boyer, P.D. (1985) Biochemistry 24, 2304–2310.
[31] Carmeli, C. and Racker, E. (1973) J. Biol. Chem. 248, 8281–8287.
[32] Junesch, U. and Gräber, P. (1987) Biochim. Biophys. Acta 893, 275–288.
[33] Avron, M. (1960) Biochim. Biophys. Acta 40, 257–272.
[34] Gräber, P., Schlodder, E. and Witt, H.T. (1977) Biochim. Biophys. Acta 461, 426–440.
[35] Kleefeld, S., Lohse, D., Mansy, A.R. and Strotmann, H. (1990) Biochim. Biophys. Acta 1019, 11–18.
[36] Muneyuki, E., Kagawa, Y. and Hirata, H. (1989) J. Biol. Chem. 264, 6092–6096.