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Emerson enhancement, photosynthetic control and protein phosphorylation in isolated maize mesophyll chloroplasts; dependence upon carbon metabolism

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Intact chloroplasts isolated from maize mesophyll have been used to investigate the relationship between the metabolic demand for ATP and the control of light-harvesting and electron transport. It was found that the rate of reduction of glycerate 3-phosphate can be either inhibited or stimulated by the addition of pyruvate, depending on their relative concentrations, pointing to optimisation of the ΔpH and the rate of electron transport. Pyruvate addition was associated with a stimulation of the phosphorylation of the light-harvesting chlorophyll protein complex, even when O_2 evolution was stimulated. Under a variety of conditions, the level of phosphorylation was inversely correlated with the ΔpH. Emerson enhancement of the reduction of glycerate 3-phosphate is increased when pyruvate is added; the increased rate of O_2 evolution brought about by far-red light is associated with an increase in the ΔpH. It is suggested that the ΔpH transmits the metabolic demand for ATP to the thylakoids, which respond by altering the relative rates of excitation of Photosystems I and II.

Introduction

Control of the rates of excitation of Photosystems I and II has long been recognised as necessary if photosynthesis is to proceed with high quantum efficiency. The difference between the absorption properties of PS II and PS I enabled Emerson to discover 'enhancement': the ability of far-red light absorbed preferentially by PS I to enhance the quantum efficiency of photosynthesis driven by light exciting PS II at a faster rate than PS I. Similarly, the changes in the extent of enhancement upon prolonged illumination with light preferentially exciting either photosystem, led to the discovery of the 'state transition' [1]; a mechanism by which imbalanced rates of excitation could be corrected. This observation of state transitions by changes in chlorophyll fluorescence and O_2 evolution, resulted in various hypotheses for the molecular mechanism by which the distribution of excitation energy is controlled. A major advance came with the discovery of the light-induced phosphorylation of the major light-harvesting complex associated with PS II, LHC-II [2]. The subsequent demonstration of ATP-induced decrease in chlorophyll fluorescence, that indicated a decrease in the absorption cross-section of PS II, established the basis for the state transition [3,4]. Furthermore, the dependency of protein kinase activity on the redox state of the inter-system electron transfer chain [5–8] provided a simple explanation of how the thylakoid can 'sense' and 'correct' for spectral imbalance; over-excitation of PS II will induce reductive activation of the thylakoid protein kinase, phosphorylating the light-harvesting complexes of PS II resulting in a decrease in the rate of excitation transfer to the reaction centre [9–11].

One prediction of this model is that there will be an optimum redox state of the electron transfer system which will be reached as the control mechanism works. The idea of an optimum redox state is, of course, reminiscent of the notion of 'redox poise' required for the cycling of electrons around PS I through the cytochrome *b–f* complex [12]. Therefore, it was proposed that the phosphorylation of LHC-II, has a role in stimulating the extra H^+ translocation that will result

Abbreviations: PGA, glycerate 3-phosphate; LHC-II, Light-harvesting complex associated with Photosystem II; PS I, Photosystem I; PS II, Photosystem II; *qE*, energy-dependent quenching of chlorophyll fluorescence.

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from optimising redox poise [13,14]. The result of decreasing the rate of electron transport through PS II would be similar to the well-known poising effect observed on adding small amounts of the PS I inhibitor DCMU to chloroplasts [15]. Furthermore, the increased transfer of excitation to PS I after phosphorylation (Refs. 16, 17 but see Ref. 18) would also directly stimulate cyclic electron transport around PS I. It has been suggested that PS I cyclic electron transport, as judged by the effects of antimycin A, is necessary for in vivo photosynthesis [19–21]. The requirement for PS I cyclic electron transfer has been rationalised in terms of the ATP deficit that would result from linear electron transport with an $H^+/2e$ ratio of 4. The involvement of a protonmotive Q-cycle, which would raise the $H^+/2e$ to 6.0, is still controversial, although work indicates that it is an obligatory event in the operation of the cytochrome *b–f* complex [22]. However, it is clear that electron transfer involving cytochrome *b*-563 and giving rise to the slow rise of the 518 nm bandshift in flash experiments is predominant when an extra demand for ATP is present, for example, in maize mesophyll chloroplasts in the presence of pyruvate [23].

The link between ATP demand and redox poise through cyclic electron flow suggests a link between protein phosphorylation and metabolism; it is the metabolic processes in the stroma that determine the requirements for ATP and NADPH (for a review see Ref. 13). The first evidence to support this hypothesis came from the observation that the maximum level of LHC-II phosphorylation in intact maize mesophyll chloroplasts occurred in the presence of pyruvate [24]. The effect of pyruvate was traced to lower ΔpH in its presence due to ATP consumption via pyruvate-orthophosphate-dikinase [25]. It was suggested that the activity of the protein kinase could be suppressed in the presence of a high ΔpH, a suggestion confirmed in isolated thylakoids [26]. The mode of action of the ΔpH is unknown, although the stimulatory effect of antimycin A on LHC-phosphorylation, suggested the involvement of a component (ferredoxin quinone reductase) associated with the cytochrome *b–f* complex in kinase control [26], consistent with the absence of phosphorylation in cytochrome *b–f* deficient mutants [27,28]. The physiological implications of these observations are clear. They suggest that the level of phosphorylation of LHC-II (and the distribution of excitation energy) is determined by the need to provide ATP and NADPH in the appropriate stoichiometry [13,29]; a low ΔpH (indicating ATP deficiency) would allow reductive activation of the kinase whilst a high ΔpH (indicating ATP surplus) would be inhibitory. The corollary of this hypothesis is that Emerson enhancement itself is related to the metabolic requirement for ATP. Experiments with isolated spinach chloroplasts showed that enhancement was greater for CO_2 reduction than for electron transport [30,31], consistent with the relative ATP requirement.

In this paper, maize mesophyll chloroplasts are again used to: (a) establish the relationship between the ATP/NADPH stoichiometry and enhancement; (b) further examine the levels of LHC-II phosphorylation in the presence of concentrations of PGA and pyruvate likely to be encountered physiologically; and (c) to examine the relationship between ΔpH-mediated control of protein phosphorylation and of electron transport.

Materials and Methods

Intact mesophyll chloroplasts were isolated from maize (greenhouse-grown, 14 days old) by the method of Jenkins and Russ [32]. Simultaneous measurements of O_2 evolution, 9-aminoacridine fluorescence and chlorophyll fluorescence used modification of our original device as described by Oxborough and Horton [33]. Incorporation of ^{32}P into LHC-II using exogenous labelled phosphate followed the procedure described by Fernyhough et al. [24]. Illumination was either broadband red light (defined by Balzars K65 filter plus a Schott RG610 filter) or 650 nm or 707 nm light defined by Balzars interference filters plus Schott RG610 or RG715 filters, respectively. Intensity was varied using neutral density filters.

Results

Control of electron transport

The rate of oxygen evolution in intact maize mesophyll chloroplasts is totally dependent on the addition of exogenous electron acceptors. Both oxaloacetate and PGA are the consumers of NADPH in these chloroplasts. In the presence of PGA, ATP and NADPH are consumed in a 1 : 1 ratio. The rate of ATP consumption can be independently increased by addition of pyruvate which is phosphorylated to phospho*enol*-pyruvate in an ATP-dependent reaction catalysed by stromal pyruvate-orthophosphate-dikinase. Fig. 1 shows the O_2 evolution in the presence of 1.0, 2.0 and 3.0 mM PGA, and the effect of adding 1.5 mM pyruvate. With 1.0 mM PGA, addition of pyruvate resulted in a pronounced inhibition of O_2 evolution (Fig. 1A). When the PGA concentration was raised to 2 mM, pyruvate only marginally inhibited O_2 evolution (Fig. 1B). In fact, small amounts (0.75 mM) of pyruvate were found to cause a slight stimulation of O_2 evolution (data not shown). With 3 mM PGA, pyruvate was found to stimulate O_2 evolution (Fig. 1C). This transition from inhibitory to stimulatory effect of pyruvate can be explained as follows. The rate of reduction of PGA has been shown to be dependent on the mass action ratio [PGA] · [ATP] · [NADPH]/[triose P] · [ADP] · [NADP]

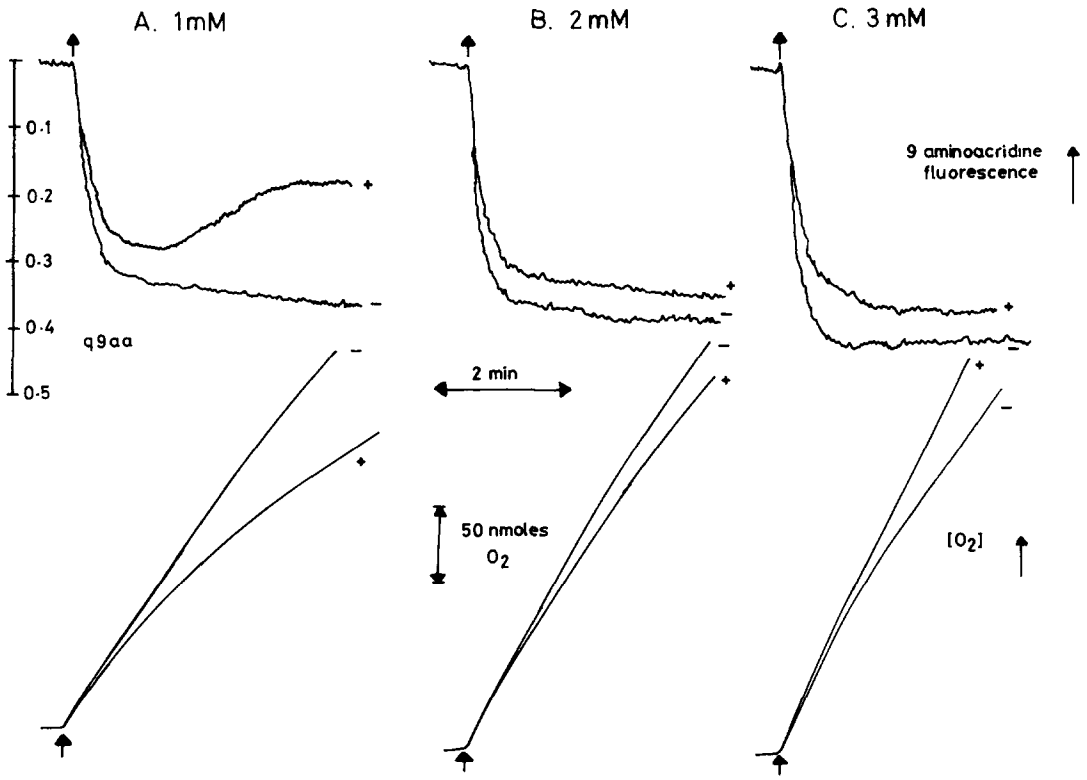

Fig. 1. Effects of pyruvate on the rate of O_2 evolution and 9-aminoacridine fluorescence in maize mesophyll chloroplasts illuminated with saturating red light (280 Wm^{-2}) in the presence of PGA at concentrations of 1 mM (A), 2 mM (B) and 3 mM (C). Control (−), 1.5 mM pyruvate (+).

[34]. At low PGA, the net rate of reaction would be expected to be more sensitive to the ATP/ADP ratio than at high concentrations. The low value for the ATP/ADP ratio known to result from pyruvate addition would be insufficient to maximally drive PGA reduction to triose phosphate. However, at high PGA, the effect of the pyruvate-induced decrease in ATP/ADP ratio would be much less. In this case, the addition of pyruvate results in stimulation. Fig. 1 shows that the increased ATP consumption brought about by pyruvate addition causes a decrease in ΔpH. This decrease in ΔpH may relieve restriction over the rate of electron transport. There is a tendency for ΔpH to build up in the presence of PGA because the stoichiometry for ATP : NADPH consumption is 1 : 1, yet for production is at least 1.33 : 1. Addition of pyruvate then stimulates electron transport since it allows extra ATP turnover. Thus there are two opposing effects of pyruvate, it lowers the ATP/ADP ratio thereby lowering the assimilatory force and tending to inhibit PGA reduction (low concentration), but at the same time the decrease in ΔpH may relieve photosynthetic control over electron transport (high concentration). At intermediate PGA concentrations these two opposing effects are seen to cancel each other out, and a negligible effect of pyruvate addition is observed.

LHC-II phosphorylation

Data obtained for the level of LHC-II phosphorylation under the contrasting conditions demonstrated in Fig. 1 are shown in Table I. The results reveal a complex control over the level of LHC-II phosphorylation. In the absence of pyruvate, the level of phosphorylation decreased from 48 to 14% upon increasing the concentration of PGA; in the presence of 1.5 mM

TABLE I

Phosphorylation of LHC-II at different concentrations of pyruvate and PGA

Phosphorylation was measured after 4 min light, subtracting the dark levels which were between 90 and 200 cpm. Each data point is obtained from a 30 min count and is a mean of four counts obtained from duplicate gel tracks of duplicate samples. Standard errors were always less than 20%. In brackets, these values are expressed as a percentage of the maximum LHC-II phosphorylation observed upon illumination with 5 mM pyruvate alone.

[Pyruvate] (mM)	LHC-II phosphorylation (cpm)			
	0.5 mM PGA	1.0 mM PGA	2.0 mM PGA	3.0 mM PGA
0	1129 (48)	710 (30)	439 (19)	340 (14)
0.75	1994 (84)	1545 (65)	1076 (45)	564 (24)
1.5	2154 (91)	1602 (68)	1287 (54)	613 (26)

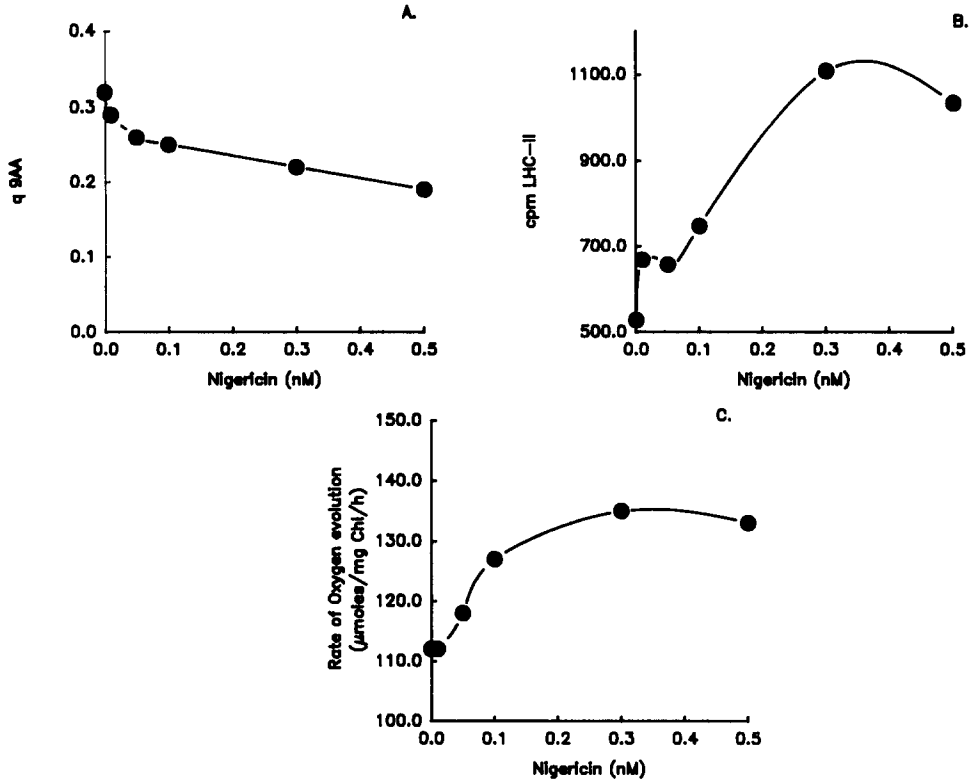

Fig. 2. Effects of nigericin on 9-aminoacridine fluorescence (q9aa (A)), LHC-II phosphorylation (B) and rate of O_2 evolution (C) in chloroplasts after 4 min illumination with red light (280 $W \cdot m^{-2}$) in the presence of 3.0 mM PGA. q9aa is obtained from the amplitude of fluorescence quenching divided by the dark level.

pyruvate, increasing the concentration of PGA caused a decrease from 91 to 26%. In the case of the change between 0.5 and 2.0 mM PGA, the decreased level of phosphorylation was accompanied by an increase in rate of O_2 evolution. Similarly, with 0.5 mM and 1.0 mM PGA, the presence of pyruvate (which inhibited O_2 evolution) substantially raised the level of phosphorylation (by factors of 1.9 and 2.3, respectively). These correlations are consistent with redox control over the activity of the LHC-II kinase, since at constant light intensity the steady state level of reduction of the electron transport chain will increase if the rate of electron transport is limited by the availability of electron acceptor. However, redox control alone cannot explain the data in Table I. For example, with 2 mM PGA, the effect of pyruvate on O_2 evolution were marginal and yet a 2.8-fold increase in phosphorylation was observed. At 3.0 mM PGA, addition of pyruvate caused a 1.9-fold increase in phosphorylation, yet O_2 evolution was stimulated. The increased phosphorylation in these cases is perhaps correlated with a decrease in thylakoid ΔpH. In fact, a synergism between high levels of reduction and low levels of energisation is suggested by the data in Table I. Near maximum O_2 evolution and maximum ΔpH (3.0 mM PGA, no pyruvate) gave 14% of the maximum level of LHC-II phosphorylation. Conversely, a low rate of electron transport and low energisation (0.5 mM PGA, 1.5 mM pyruvate) gave 91% LHC-II phosphorylation. Low electron transport and relatively high ΔpH (0.5 mM PGA, zero pyruvate) caused 48% phosphorylation whilst with high electron transport and low ΔpH (3.0 mM PGA, 1.5 mM pyruvate) 26% phosphorylation was observed.

To further probe the relationship between LHC-II phosphorylation and ΔpH, the effects of the uncoupler nigericin were examined. A progressive increase in the concentration of nigericin resulted in a decrease in ΔpH (Fig. 2A) and a 20% increase in rate of O_2 evolution (Fig. 2C). This increase in O_2 evolution caused by nigericin is consistent with the ΔpH-induced restriction on the rate of electron transport when PGA is high (see above). Over this range of nigericin concentration, phosphorylation of LHC-II increased by 107% (Fig. 2B). The complexity of the dependence of LHC-II phosphorylation on nigericin concentration can be explained by dual control by ΔpH and redox state; initially phosphorylation rises as the ΔpH falls, but when O_2 evolution is stimulated by higher concentration of nigericin (causing oxidation), increased phosphorylation is balanced by a tendency for reduced kinase activity. When O_2 evolution approaches a saturating rate, the stimulatory effect of nigericin on phosphorylation is again observed. These observations are consistent with the effects of pyruvate described above. In Fig. 3, phospho-

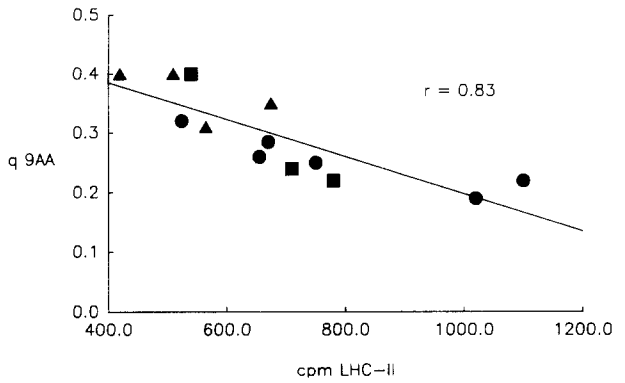

Fig. 3. Levels of LHC-II phosphorylation and quenching of 9-aminoacridine fluorescence (q9aa) after illumination of chloroplasts in the presence of different concentrations of pyruvate (▲), nigericin (●) and ammonium chloride (■). PGA concentration was 3.0 mM. Conditions as in Fig. 2 and LHC-II phosphorylation measured as described under Table I.

rylation and ΔpH are compared for a number of experiments in which ΔpH was perturbed by nigericin, pyruvate or NH_4Cl; a correlation was found between ΔpH and LHC-II phosphorylation.

There is strong evidence for the stimulation of cyclic electron transport when pyruvate is added to maize mesophyll chloroplasts [23]. This effect was interpreted as being the response of the thylakoid to an ATP deficit, cyclic photophosphorylation being responsible for increasing the supply of ATP. If LHC-II phosphorylation is also a response to an ATP deficiency, then suppression of cyclic electron transport by antimycin A might therefore be expected to stimulate phosphorylation. Indeed, Fig. 4 shows a 120% increase in phosphorylation associated with a 10% decrease in ΔpH. However, it is noticeable that part of the increase in phosphorylation occurred at an antimycin A concentration which caused no ΔpH decrease; in Fig. 4, note that addition of 0.01 μM antimycin caused a 55% increase in phosphorylation without a ΔpH decrease.

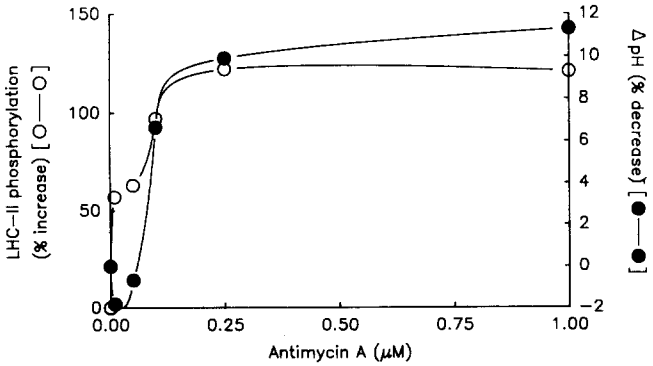

Fig. 4. Effect of antimycin A on the level of LHC-II phosphorylation (○) and quenching of 9-aminoacridine fluorescence (●) after illumination as described in Fig. 1. PGA concentration 3 mM. Data are expressed as % change of control values in the absence of antimycin.

TABLE II

Effect of antimycin A and nigericin on LHC-II phosphorylation and ΔpH

Data was obtained as described for Table I. Concentrations were 3.0 mM PGA, 5 mM pyruvate (pyr); 1 μM antimycin A (anti A); 0.1 nM nigericin (nig). Values are expressed as a percentage of a control maximum, obtained after illumination with 5 mM pyruvate alone.

Conditions	LHC-II phosphorylation	q9aa
PGA	899 (31)	0.40
PGA + pyr	1113 (38)	0.35
PGA + anti A	1613 (56)	0.41
PGA + pyr + anti A	2557 (88)	0.30
PGA + pyr + nig	1622 (56)	0.29

In Table II, results are shown of an experiment which compares the effectiveness of antimycin, pyruvate and nigericin on stimulating LHC-II phosphorylation. In this experiment, pyruvate stimulated phosphorylation by 30%, whilst lowering the q9aa from 0.40 to 0.35. In comparison, antimycin induced a 79% increase in phosphorylation without lowering ΔpH. When pyruvate and antimycin A were added together, there was a large increase in phosphorylation to 88% of the maximum level. The sensitivity of the ΔpH to antimycin in the presence of pyruvate is consistent with operation of cyclic electron transport making a significant contribution to the maintenance of the ΔpH. However, that antimycin A has an additional effect, not due to ΔpH change, is shown by the fact that a nigericin concentration giving the same ΔpH change in the presence of pyruvate was less effective in stimulating phosphorylation (56 compared to 88%).

Emerson enhancement

Under limiting PS II light, Emerson enhancement of O_2 evolution by maize chloroplasts can be observed. Addition of PS I light, which itself caused no O_2 evolution, brought about a 2.5-fold increase in the rate observed in PS II light alone (Fig. 5A). Addition of pyruvate under these conditions caused inhibition of O_2 evolution in PS II light which could, however, be restored by addition PS I light (Fig. 5B). Hence, the extent of enhancement was greater in the presence of pyruvate. The change in ΔpH and quenching of chlorophyll fluorescence by PS I light is also shown in Fig. 5. Again, in the presence of pyruvate, the proportional 'enhancement' of ΔpH and chlorophyll fluorescence quenching is much larger. Indeed, in the absence of pyruvate, the stimulation of O_2 evolution by PS I light causes a decrease in ΔpH (Fig. 5A), whereas in the presence of pyruvate, PS I light causes an increase in ΔpH (Fig. 5B). Fig. 6 shows the effect of increasing pyruvate concentration on the rate of oxygen evolution in PS II light and after supplementation with PS I light. Enhancement ratios as high as 5.5 were observed at 10

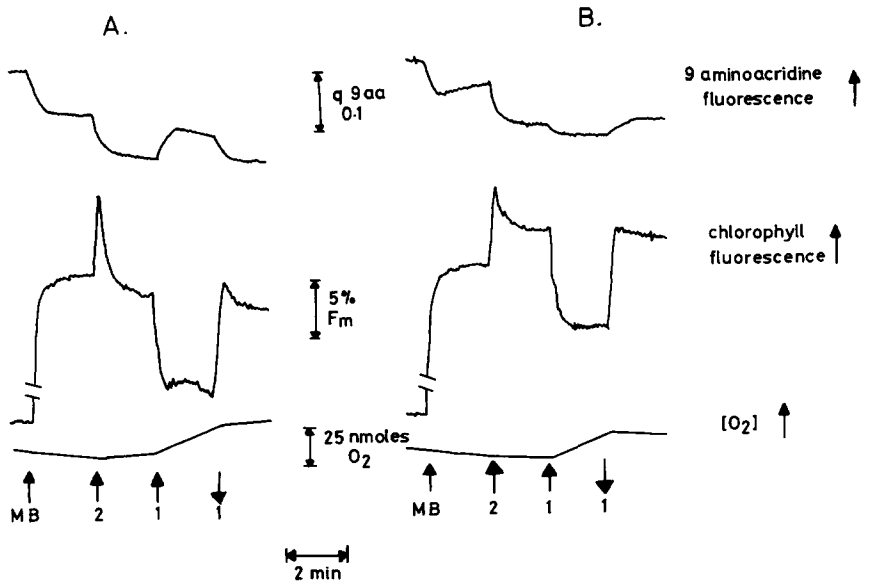

Fig. 5. Rate of O_2 evolution, 9-aminoacridine fluorescence and chlorophyll fluorescence in maize chloroplasts illuminated with 650 nm light (2) or 707 nm light (1) in the absence (A) and presence (B) of 1.5 mM pyruvate. Light on (↑), light off (↓) PGA concentration, 3.0 mM. MB indicates turning on of the fluorescence measuring beam, intensity 1 $\mu E/m^2$ per s. 650 intensity was 20 $\mu E/m^2$ per s. 707 nm light intensity was set empirically to give the maximum amount of enhancement without itself giving any O_2 evolution.

mM pyruvate. The extent of the inhibition of the rate of reduction of PGA was increased when the pyruvate concentration was raised. PS I light was found to partially offset this inhibition; for example, 5 mM pyruvate inhibited the rate of O_2 evolution in PS II light by 60%, but only by 25% in PS II and PS I light.

Discussion

Addition of pyruvate to isolated maize mesophyll chloroplasts can either stimulate or inhibit reduction of PGA depending on whether the system is under- or over-energised. This points to the importance of optimising the ATP/ADP ratio and the ΔpH so that the former is maintained at a level adequate to drive PGA reduction whilst the latter is not allowed to get large enough to restrict electron transport via 'photosynthetic control' but is still of sufficient magnitude to drive ATP synthesis. The stimulatory effect of an uncoupler and pyruvate on the rate of O_2 evolution indicates that in the presence of PGA that photosynthetic control does inhibit electron transport. The inhibitory effect of pyruvate is observed either at low PGA or in low light. In the latter case, it is suggested that the increased rate of ATP consumption can outpace the capacity of electron transport to maintain a ΔpH. The marked stimulation by PS I light therefore implicates PS I cyclic electron transport as a process responsible for making up ATP deficits. Evidence for cyclic electron flow in maize mesophyll has been obtained previously [23]. The ATP deficit induced by pyruvate results in large Emerson enhancement ratios and is again most readily explained by PS I cyclic photophosphorylation. Similar observations have been made using spinach chloroplasts reducing CO_2, PGA or nitrite [30] where the extent of enhancement was correlated with the ATP demand. The requirement for increased cyclic electron transport in the presence of pyruvate provides an explanation for previously seen high levels of LHC-II phosphorylation [24]. In the present study, we have shown that the elevated phosphorylation occurs under conditions close to those necessary for maximum rates of reduction of PGA. Thus, the change in distribution of excitation energy resulting from phosphorylation is suggested to be involved in regulating the efficiency of H^+ translocation (i.e., the ratio of H^+ translocated per electron transferred to NADP) by the thylakoid. In conditions of low ΔpH (e.g., high ATP demand, insufficient PS I

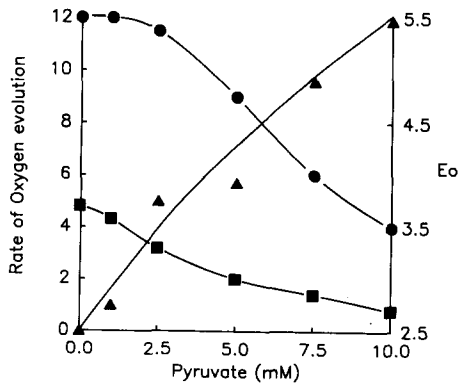

Fig. 6. Effect of pyruvate concentration of the rate of O_2 evolution in light 2 (■) and light 2+1 (●) and the enhancement ratio, E (▲). Conditions as for Fig. 5. E_0 is the rate of O_2 evolution in light 2+1 divided by the rates in light 2 and light 1 given separately. Rates of O_2 evolution are in μmol O_2/mg Chl per h.

excitation, partial uncoupling), expression of maximum protein kinase activity is allowed. Conversely, high ΔpH, indicating adequate efficiency of H^+ translocation would lead to suppression of LHC-II phosphorylation, even at high levels of redox activation.

It is well-established that the ΔpH is the signal that transmits the ATP demand to the thylakoid membrane. It is also clear that the ΔpH is 'sensed' by the thylakoid by the (presently unknown) alteration in conformation indicated by the energy-dependent quenching of chlorophyll fluorescence, *qE*. Since antimycin A can inhibit *qE* without lowering ΔpH [33], the observations in Fig. 4 and Table II can be explained; i.e., suppression of *qE* by antimycin is responsible for the elevated level of LHC-II phosphorylation [26]. The involvement of *qE* is significant, since there is now abundant evidence that thermal dissipation through this channel is an important regulatory process that suppresses PS II quantum yield as a response to excess irradiance [35–39]. Therefore, there is a switch from utilisation to dissipation once the correct ΔpH is reached. The molecular mechanism of this switch is not known, but we have recently suggested that the protonation of LHC-II/PS II complexes, that may underlie the *qE* state, diminishes their activity as protein kinase substrates [39]. Alternatively, it is now clear that the protein kinase is either controlled by the redox state of a component of, or one associated with, the cytochrome *b–f* complex [27]; electron transfer in this complex is known to respond to ΔpH and to the presence of antimycin. However, the fact that the increase in LHC-II phosphorylation can be induced by an uncoupler and by pyruvate under conditions when they stimulate linear electron transport strongly suggests the presence of another factor, in addition to the well-known redox control of the protein kinase.

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