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ATP SYNTHESIS COUPLED TO LIGHT-DEPENDENT NON-CYCLIC ELECTRON FLOW IN CHROMATOPHORES OF *RHODOPSEUDOMONAS CAPSULATA*

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Light-dependent non-cyclic electron transport in membranes from aerobic dark-grown cells of *Rhodopseudomonas capsulata*, i.e., light-induced oxygen uptake, is clearly linked to energy transduction. It is proposed that the open chain performing the electron transfer from exogenous donors such as 2,6-dichlorophenolindophenol (DCIP) or *N,N,N',N'*-tetramethyl-*p*-phenylenediamine (TMPD) to oxygen includes cytochrome c_2, reaction centre bacteriochlorophyll, ubiquinone molecules and the alternate oxidase (cytochrome *b*-260). In contrast, cytochrome *b*-60 previously suggested to act at the branching point of the dual electron transport system of *Rps. capsulata* (Zannoni, D., Melandri, B.A. and Baccarini-Melandri, A. (1976) Biochim. Biophys. Acta 449, 386–400) does not appear to be essential for aerobic photophosphorylation. This conclusion is derived mainly from experiments with *Rps. capsulata* R126 chromatophores deficient in cytochrome *b*-60 photoreduction and electron transfer through the cytochrome *b-c* segment of the chain. Indeed, these mutant membranes present normal rates of light-induced oxygen consumption. Moreover, this activity is coupled to $\Delta\mu_{H^+}$ formation which in turn drives ATP synthesis.

Introduction

The presence of light-driven oxygen uptake in chromatophores from facultative photosynthetic bacteria has been widely documented [1–6]. This phenomenon, which is generally linked to photooxidation of exogenous cytochrome *c* or reduced artificial electron carriers, is enhanced by blocking the cytochrome *b-c* region of the photocyclic electron flow with antimycin A [5,6]. This latter result suggests that under aerobic, illuminated conditions, two electron transport pathways are operating in membrane fragments from facultative phototrophs: a cyclic and a noncyclic electron flow. They branch presumably at the ubiquinone-cytochrome *b* level and the noncyclic electron flow leads to oxygen reduction through a redox pathway different from that which contains the cytochrome *c* oxidase system [5,6].

Very recently, it has been demonstrated that light-dependent oxidase activity in chromatophores from *Rhodopseudomonas capsulata* cells requires cytochrome *b*-260, previously described as a functional respiratory oxidase present in this organism [7,6]. This finding indicates that electrons can be transported from the photosynthetic to the respiratory system and therefore these systems should be located on the same membrane fragments. Indeed, several lines of evidence tend to show that many of the redox carriers forming the bacterial ubiquinone-cytochrome *b-c* oxidoreductase complex are clearly involved in both oxidative- and light-driven electron flow [8–11]. In this context it is believed that in addition to a

Abbreviations: $[BChl]_2$, reaction centre bacteriochlorophyll; DCIP, 2,6-dichlorophenolindophenol; TMPD, *N,N,N',N'*-tetramethyl-*p*-phenylenediamine; Q, ubiquinone.

large thermodynamically homogeneous pool of ubiquinone-10 (Q-10) [12], two molecules of ubiquinone (Q_I and Q_{II}) act as primary and secondary acceptors from the reaction centre bacteriochlorophyll, $[BChl]_2$ [13,14], and a third molecule (Q_z) operates in the cytochrome *b-c* segment [15]. Moreover, it has been proposed that ubiquinone and cytochrome *b*-60, which are involved at the branching point of the dual electron transport chain of *Rhodopseudomonas capsulata* [16], are also essential to the overall process of photophosphorylation (reviewed in Ref. 11). In contrast, details of the actual nature and role of those redox carriers which are thought to participate in light-dependent noncyclic electron flow have been poorly considered.

In this report I present experimental evidence of ATP formation coupled to light-driven oxygen reduction, i.e., to noncyclic electron flow, in membranes from semiaerobically grown cells of *Rps. capsulata.*

The involvement of cytochrome *b*-60 in light-dependent noncyclic electron flow has been examined by using a photosynthetic mutant deficient in both cytochrome *b* photoreduction and cyclic photophosphorylation.

Materials and Methods

Strain Y11 is a normally pigmented, nonphotosynthetic mutant of *Rhodopseudomonas capsulata* that was isolated by La Monica and Marrs [17]. A green derivative of Y11, designed R126, was chosen for study because it accumulates a mixture of neurosporene, hydroxyneurosporene and methoxyneurosporene in place of wild-type carotenoids which present a strong absorption in the cytochrome region of the spectrum. An isogenic control strain (MR126) that regained photosynthetic competence was also isolated by Marrs and collaborators (Marrs, generous gift).

Semiaerobic cultures of MR126 and R126 cells were grown in the dark and membranes prepared as described in Ref. 6.

Light-induced oxygen uptake was measured polarographically at 30°C using a Yellow Springs Model YS1 53 oxygen meter with a jacketed Gilson Oxygraph reaction chamber (1.6 ml final volume); the illumination of the reaction chamber was provided by a 300 W lamp with a round flask filled with water as a heat filter and focusing device between the lamp and the electrode. All measurements of light-induced oxygen uptake were performed in infrared light using a Corning coloured glass filter (No. 7-69) and corrected for dark uptake for O_2.

Oxidative and light-driven phosphorylations were carried out following essentially the method described by Baccarini-Melandri et al. [18].

Oxidation of both NADH and ascorbate-DCIP was measured polarographically. Cytochrome *c* reduction was followed spectrophotometrically at 551 nm ($\epsilon = 19\ nM^{-1} \cdot cm^{-1}$) [19]. The concentrations of the various acceptors and donors used were: equine cytochrome *c*, 30 μM; DCIP, 100 μM; TMPD, 100 μM; ascorbate, 3 mM; NADH, 2 mM.

Measurements of the quenching of the atebrine fluorescence were done as described in Ref. 20. The medium for the assay comprised 100 mM Na^+–glycylglycine buffer (pH 8.25), 50 mM KCl, 5 mM $MgCl_2$, 3 μM atebrine, 4 μg/ml valinomycin, 5 μM antimycin A, 50 μM KCN and chromatophores corresponding to 39 μg BChl/ml (0.78 mg protein/ml).

Protein content was assayed by the method of Lowry et al. [21] and bacteriochlorophyll was measured by extraction with acetone/methanol (7:2) using an extinction coefficient of 75 $mM^{-1} \cdot cm^{-1}$ [22].

Results

Cyclic photophosphorylation in chromatophores of *Rhodospirillum rubrum* [23,24] and *Rps. capsulata* [25] is inhibited by antimycin A. In the former organism artificial electron carriers such as 2,6-dichlorophenolindophenol (DCIP) and *N,N,N',N'*-tetramethyl-*p*-phenylenediamine (TMPD) have been demonstrated to catalyze high rates of antimycin A-insensitive photophosphorylation [24–27].

In contrast, the antimycin A-block of the photocyclic electron transfer in chromatophores of *Rps. capsulata* can also be overcome by artificial redox dyes, but only under special redox conditions, i.e., by maintaining the ambient redox potential around +200 mV [28]. Indeed, under

aerobic, illuminated conditions, antimycin A markedly enhances light-induced oxygen consumption by inhibiting cyclic electron flow [5,6]. On the basis of these considerations it is of interest to determine whether *Rps. capsulata* chromatophores, which carry out cyclic photophosphorylation in the presence of reduced TMPD or DCIP, may also catalyze noncyclic photophosphorylation linked to light-driven oxygen reduction (see Table I). As expected, the photophosphorylation activity which is measured in the presence of 5 mM KCN to minimize light-induced oxygen reduction is completely suppressed by 5 μM antimycin A. However, if the cyanide concentration is reduced to 50 μM in order to inhibit specifically the cytochrome *c* oxidase [20] under antimycin A-inhibited conditions, a noncyclic electron flow operates and the resulting oxygen consumption is clearly linked to ATP formation (P/O ratios of about 0.17 and 0.22 with DCIP and TMPD, respectively).

To examine the specific role of cytochrome *b*-60 in noncyclic photophosphorylation, the photosynthetic mutant *Rps. capsulata* R126, lacking both photoinduced cytochrome *b* reduction and cyclic photophosphorylation, has been analyzed for its ability to perform ATP synthesis coupled to light-driven oxygen reduction.

As previously reported, the strain R126 can only grow under aerobic dark conditions due to the absence of electron flow through the cytochrome *b-c* segment of the chain [17]. Typical respiratory activities in membranes from R126 and the isogenic control strain MR126 are presented in Table II. From this it is evident that the NADH respiration in R126 chromatophores reflects markedly the negligible rate of exogenous horse-heart cytochrome *c* reduction (2% of the control strain MR126). The presence of a normal cytochrome *c* oxidase, i.e., either ascorbate-DCIP or -TMPD oxidase, in these mutant membranes strongly supports this assertion.

When membranes from R126 cells were tested for light-induced reactions, it was found that a fast oxidation of cytochrome *c* occurred upon flash excitation but rereduction did not occur [29]. Further analysis also revealed that cytochrome *b* was not photoreduced following several flashes of light [29]. Moreover, it has recently been noted that R126 membranes appear to behave as MR126 membranes in the presence of the inhibitor 5-*N*-undecyl-6-hydroxy-4,7-dioxobenzothiazole [30]. This compound, which is thought to block the interaction between the Rieske centre and cytochrome c_2, seems to inhibit also the reoxidation of

TABLE I

LIGHT-DEPENDENT ENERGY TRANSDUCTION COUPLED TO CYCLIC AND NONCYCLIC ELECTRON FLOW IN MEMBRANES FROM SEMIAEROBICALLY DARK-GROWN CELLS OF *Rps. CAPSULATA* MR126

Measurements were performed as indicated under Materials and Methods. The concentrations of the various electron donors used were: ascorbate (Asc), 2 mM; TMPD, 100 μM; DCIP, 100 μM. Antim. A, antimycin A.

Light-induced electron transport pathways	Electron donors	Inhibitors added	ATP [a] formed	Oxygen [b] uptake
Cyclic electron flow	Asc-DCIP	5 mM KCN	11.6	0.43
	Asc-TMPD	5 mM KCN	12.8	0.47
	Asc-DCIP	5 mM KCN; 5 μM antim. A	0.32	0.50
	Asc-TMPD	5 mM KCN; 5 μM antim. A	0.36	0.60
Noncyclic electron flow	Asc-DCIP	50 μM KCN; 5 μM antim. A	1.15	6.70
	Asc-TMPD	50 μM KCN; 5 μM antim. A	1.52	6.80

[a] Activities as μmol ATP/h per mg protein.
[b] Activities as μequiv./h per mg protein.

TABLE II

RESPIRATORY ACTIVITIES IN MEMBRANE VESICLES FROM SEMIAEROBICALLY GROWN CELLS OF *Rps. CAPSULATA* MR126 AND R126

Respiratory activities (R.A.) are expressed as μequiv. electron acceptor reduced/h per mg protein. In both MR126 and R126 membrane fragments the ratio mg protein/mg BChl was about 20. Experimental conditions were as indicated under Materials and Methods. Antim. A, antimycin A.

Substrates (inhibitors)	Electron acceptor	MR126		R126	
		R.A.	Inhibition (%)	R.A.	Inhibition (%)
NADH	O_2	20.1	0	12.50	0
NADH (50 μM KCN)	O_2	8.6	57	10.20	18
NADH (5 mM KCN)	O_2	1.2	94	0.30	98
NADH (5 μM antim. A)	O_2	10.0	50	11.80	5
NADH	cyt. *c* [a]	4.5	0	0.10	0
NADH (5 μM antim. A)	cyt. *c* [a]	0.9	80	0.05	50
Ascorbate-DCIP	O_2	35.0	0	18.0	0
Ascorbate-TMPD	O_2	44.0	0	24.0	0
Ascorbate-DCIP (50 μM KCN)	O_2	2.0	94	1.0	95
Ascorbate-TMPD (50 μM KCN)	O_2	2.0	96	1.2	95

[a] Activities measured in the presence of 5 mM KCN.

the photoreduced secondary ubiquinone (Q_{II}) by cytochrome *b* [31]. Thus it is reasonable to conclude that the lesion present in R126 membrane vesicles has produced a multiple effect on both cytochrome *b* reduction by Q_{II} and cytochrome *c* rereduction through the Rieske iron-sulphur centre.

Table III summarises the data obtained using R126 chromatophores during light-induced oxygen uptake experiments. It is quite evident that the lack of cytochrome *b* photoreduction does not affect at all the rate of light-dependent oxygen consumption (see also Table I for comparison). Furthermore, this activity seems to be linked to ATP formation by a reaction mechanism which is almost completely suppressed by 5 mM *o*-phenanthroline. This inhibitor is known to block electron transfer from Q_I to Q_{II} [14]. Therefore this process appears to be an essential step for light-driven oxygen consumption.

To test whether light-induced noncyclic electron flow in R126 chromatophores is really coupled to the formation of an electrochemical proton gradient, the light-dependent quenching of the fluorescence of atebrine, which should indicate a transmembrane ΔpH [32,33], has been examined.

The traces in Fig. 1 show that in the presence of ascorbate-TMPD or ascorbate-DCIP, a consistent decrease of the atebrine fluorescence induced by light takes place only under aerobic conditions. Indeed, if the anaerobiosis is reached by means of substrate- or light-driven oxygen consumption, no transmembrane $\Delta\mu_{H^+}$ is formed. The same effect was obtained by adding either 5 mM KCN or the protonophore nigericin (2 μg/ml) under aerobic

TABLE III

LIGHT-DRIVEN ATP SYNTHESIS IN CHROMATOPHORES FROM SEMIAEROBICALLY DARK-GROWN CELLS OF *Rps. CAPSULATA* R126

Measurements were performed as indicated under Materials and Methods. The electron donors used were: ascorbate, 2 mM and TMPD, 100 μM.

Inhibitors added	ATP [a] formed	Oxygen [b] uptake
5 mM KCN	0.0	0.57
50 μM KCN	1.2	6.30
5 mM *o*-phenanthroline; 50 μM KCN	0.1	0.90

[a] Activities as μmol ATP/h per mg protein.
[b] Activities as μequiv./h per mg protein.

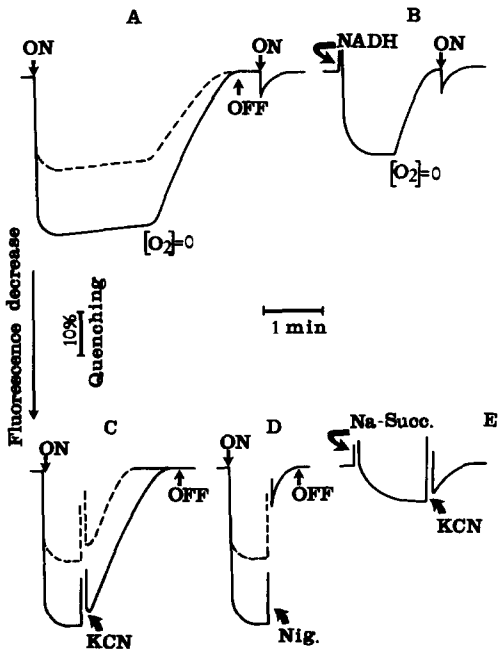

Fig. 1. Formation of an electrochemical potential of protons coupled to either light- or substrate-driven oxygen consumption in *Rps. capsulata* R126 membrane fragments as monitored with atebrine fluorescence. Traces in A, C and D represent the light-dependent quenching of fluorescence of atebrine with 2 mM ascorbate, 50 μM TMPD (————) or 50 μM DCIP (- - - - -) as electron donors. In B, ascorbate-TMPD was added before the respiratory substrate. Additions: 2 mM NADH, 3 mM sodium succinate (Na-succ.), 5 mM KCN, 2 μg/ml nigericin (Nig.). Reaction conditions were as described under Materials and Methods.

TABLE IV

NADH- AND SUCCINATE-DEPENDENT OXIDATIVE PHOSPHORYLATION BY CHROMATOPHORES FROM SEMIAEROBIC DARK-GROWN CELLS OF *Rps. CAPSULATA* R126

Measurements were performed as indicated under Materials and Methods.

Respiratory activities	Oxygen reduced [a]	ATP formed [b]	P/2e
NADH-oxidase	16.0	3.8	0.24
Succinate-oxidase	6.3	1.1	0.17

[a] Activities as μequiv./h per mg protein. The ratio: mg protein per mg BChl as in Table II.

[b] Activities as μmol/h per mg protein.

conditions. In this context the finding that succinate-dependent respiration in R126 membrane fragments is linked to both ATP and ΔpH formation (Table IV and Fig. 1, trace E) indicates that energy transduction through the ubiquinone-cytochrome *b*-260 branch of the chain is not driven only by light reactions.

Note that in all the experiments of Fig. 1, 5 μM antimycin A was present in order to prevent any residual electron transport through the cytochrome *b-c* segment.

Discussion and Conclusions

This work supports the concept that light-induced electron transport through the ubiquinone-cytochrome *b*-260-containing pathway of *Rps. capsulata* is coupled to energy transduction.

It might be suggested that light-induced oxygen uptake is not physiologically linked to ATP formation but that DCIP, a well known proton translocator, would be involved in producing artificial energy conserving sites, as proposed earlier for chloroplasts [34,35]. However this is unlikely, since TMPD, an electron carrier, is even more active than DCIP in maintaining the formation of an electrochemical proton gradient and ATP synthesis (Fig. 1 and Table I). In addition, it was previously demonstrated that light-driven oxygen reduction in *Rps. capsulata* is markedly enhanced by the addition of antimycin A. Any artificial photocyclic electron flow catalyzed by TMPD or DCIP can therefore be reasonably ruled out.

It is noteworthy that ATP formation brought about by the open chain involved in light-dependent oxygen uptake stands in contrast to the results of Hochman and Carmeli [3], who were unable to obtain any aerobic photophosphorylation in 'heavy chromatophores' from *Rps. capsulata.* Although the membrane vesicles used in the present report and 'heavy chromatophores' are opposite in their polarity, their different capacity for supporting ATP synthesis cannot be explained simply in terms of chemiosmotic coupling. One possible explanation for this discrepancy may be based on the fact that the photophosphorylation activity reported in heavy chromatophores is 4-times lower than the equivalent activity measured in regular chromatophores (65 and 232 μmol

ATP/h per mg BChl, respectively) [3]. Since it is well known that the P/O ratios in bacterial systems are invariably low even in well coupled membrane preparations, it is possible that the lack of aerobic photophosphorylation in heavy chromatophores is due to their partial uncoupling.

An important feature mainly derived from the experiments with R126 chromatophores is that cytochrome *b*-60 does not seem to operate in noncyclic photophosphorylation.

Previous results indicated that a large amount of the *b*-type complement contained in aerobic membranes from *Rps. capsulata* cells may be reoxidized during steady-state respiration via the cytochrome *b*-60 branch of the chain [16]. Since cytochrome *b*-60 represents the major *b*-type component (about 40% of the total signal at 561–570 nm) [16] it could be expected a priori to be an electron carrier also for light-induced oxygen reduction. However, only the ubiquinone-10 pool, and not cytochrome *b*-60, appears to play an essential role in light-dependent noncyclic electron transfer of *Rps. capsulata.* In fact, preliminary experiments on chromatophores from the related bacterium *Rhodopseudomonas sphaeroides* show that the extraction of more than 80% of the total Q-10 complement (possibly the whole of the homogeneous pool) produces a marked inhibition of the light-induced oxygen reduction present in this organism (Zannoni et al., unpublished data). Very similar data have also been reported by Remennikov and Samuilov [36] in *R. rubrum* chromatophores depleted of loosely bound quinones. Consequently, it seems that the minimal redox pathway supporting noncyclic photophosphorylation in *Rps. capsulata* includes the primary and secondary ubiquinone acceptors from the reaction centre bacteriochlorophyll, the ubiquinone-10 pool and the alternate oxidase (cytochrome *b*-260). The ATP formation linked to succinate respiration in R126 mutant membranes (Table IV and Fig. 1, trace E) confirms this conclusion and would also suggest restriction of the phosphorylating pathway to a segment formed only by the Q-10 pool and cytochrome *b*-260. Succinate oxidation does not appear to be affected by 5 mM *o*-phenanthroline (data not shown).

An apparent discrepancy is related to the phosphorylation efficiencies shown by NADH respiration and light-dependent noncyclic electron flow from reduced TMPD. Although they present very similar P/O ratios (0.24 and 0.22, respectively), the electrochemical potential of protons linked to NADH oxidation is lower than the ΔpH produced by light-driven oxygen consumption, as monitored by atebrine fluorescence (Fig. 1, continuous traces A and B). This observation disagrees with an essential feature of the chemiosmotic hypothesis which would require a direct control by ΔpH, $\Delta\psi$ and by the concentrations of substrates on the rate of phosphorylation. A possible explanation of the present results should involve the possibility of parallel mechanisms coupling electron flow, proton gradient and ATP synthesis as proposed earlier by Padan and Rottemberg [37] for oxidative phosphorylation in rat liver mitochondria and more recently by Baccarini-Melandri et al. [38] for photophosphorylation in non-sulphur purple bacteria. Indeed, Tables III and IV clearly indicate that NADH respiration through the cytochrome *b*-260-containing pathway proceeds at a rate nearly 3-times higher than the equivalent activity induced by light using either TMPD or DCIP as electron donors. Therefore a direct control of the electron transport on the rate of ATP synthesis would reconcile the above reported anomaly between the P/O ratios and the extent of the proton gradients observed during substrate- and light-dependent O_2 consumption in *Rps. capsulata* chromatophores.

At the present experimental stage the mechanism of $\Delta\mu_{H^+}$ formation during aerobic photophosphorylation is unclear. Nevertheless, it might be proposed that the Q-10 pool operates as electron and proton transfer between the outer and the inner face of the membrane chromatophores. In this case the alternate oxidase, which should be localized on the inner face of the membrane vesicles, would act as an electron sink providing oxidizing equivalents for the photoreduced ubiquinone-10 pool. Indeed, it has been recently shown that a large excess of Q-10 is required for photophosphorylation in continuous light [39] and it has, moreover, been suggested that some bacterial alternate oxidases are located on the periplasmic membrane phase, i.e., on the inner face of membrane vesicles (reviewed in Ref. 8).

The electrogenic proton transport coupled to either light- or respiration-induced electron trans-

fer through the Q-10-cytochrome *b*-260 segment of the chain in *Rps. capsulata* is now under active investigation.

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References

1 Vernon, P.L. and Kamen, M.D. (1953) Arch. Biochem. Biophys. 44, 298–311
2 Govindjee, R., Smith, W.R., Jr. and Govindjee, J.M. (1974) Photochem. Photobiol. 20, 191–199
3 Hochman, A. and Carmeli, C. (1977) Arch. Biochem. Biophys. 179, 349–359
4 Taniguchi, S. and Kamen, M.D. (1965) Biochim. Biophys. Acta 96, 359–429
5 Hochman, A., Ben-Hayim, G. and Carmeli, C. (1977) Arch. Biochem. Biophys. 184, 416–422
6 Zannoni, D., Jasper, P. and Marrs, B. (1978) Arch. Biochem. Biophys. 191, 625–631
7 Zannoni, D., Melandri, B.A. and Baccarini-Melandri, A. (1976) Biochim. Biophys. Acta 449, 386–400
8 Zannoni, D. and Baccarini-Melandri, A. (1980) in Diversity of Bacterial Respiratory Systems (Knowles, D., ed.), vol. 2, pp. 183–202, CRC Press, Cleveland, OH
9 Baccarini-Melandri, A., Jones, O.T.G. and Hauska, G. (1978) FEBS Lett. 86, 151–154
10 Zannoni, D., Prince, R.C., Dutton, P.L. and Marrs, B. (1980) FEBS Lett. 113, 289–293
11 Baccarini-Melandri, A. and Zannoni, D. (1978) J. Bioenerg. Biomembranes 10, 109–139
12 Takamiya, K. and Dutton, P.L. (1979) Biochim. Biophys. Acta 546, 1–16
13 Wraight, C.A. (1977) Biochim. Biophys. Acta 459, 525–531
14 Vermeglio, A. and Clayton, R.K. (1977) Biochim. Biophys. Acta 461, 159–165
15 Takamiya, K., Prince, R.C. and Dutton, P.L. (1979) J. Biol. Chem. 254, 11307–11311
16 Zannoni, D., Melandri, B.A. and Baccarini-Melandri, A. (1976) Biochim. Biophys. Acta 423, 413–430
17 La Monica, R.F. and Marrs, B. (1976) Abstract from the Primary Electron Transport and Energy Transduction in Photosynthetic Bacteria, Brussels Belgium, pp. ThB3
18 Baccarini-Melandri, A., Gest, H. and San Pietro, A. (1970) J. Biol. Chem. 245, 1224–1226
19 Chance, B. (1957) Methods Enzymol. 4, 273–329
20 Baccarini-Melandri, A., Zannoni, D. and Melandri, B.A. (1973) Biochim. Biophys. Acta 314, 298–311
21 Lowry, O.H., Rosebrough, M.J., Farr, A.L. and Randall, R.J. (1951) J. Biol. Chem. 193, 265–275
22 Clayton, R.K. (1963) Biochim. Biophys. Acta 17, 10–22
23 Smith, L. and Baltscheffsky, M. (1959) J. Biol. Chem. 234, 1575–1579
24 Geller, D.M. and Lipmann, F. (1960) J. Biol. Chem. 235, 2478–2484
25 Klemme, M.D. and Schlegel, H.G. (1968) Archiv. Mikrobiol. 63, 154–169
26 Arwidsson, B., Baltscheffsky, M. and Baltscheffsky, H. (1962) Biochim. Biophys. Acta 65, 429–433
27 Gromet-Elhanan, Z. and Gest, H. (1978) Archiv. Mikrobiol. 116, 29–34
28 Baccarini-Melandri, A., Melandri, B.A. and Hauska, G. (1979) J. Bioenerg. Biomembranes 11, 1–16
29 Marrs, B.L. (1978) in Current Topics in Bioenergetics (Sanadi, D.R. and Vernon, P., eds.), vol. 8, pp. 261–294, Academic Press, New York
30 Bowyer, J.R. and Crofts, A.R. (1981) Biochim. Biophys. Acta 636, 218–233
31 Bowyer, J.R. and Crofts, A.R. (1978) in Frontiers of Biological Energetics (Dutton, P.L., Leigh, J.S. and Scarpa, A., eds.), vol. 1, pp. 326–333, Academic Press, New York
32 Kraayenhof, R. (1970) FEBS Lett. 6, 161–165
33 Shuldiner, S., Rottemberg, H. and Avron, M. (1972) Eur. J. Biochem. 25, 64–70
34 Trebst, A. (1974) Annu. Rev. Plant Physiol. 25, 423–458
35 Hauska, G., Reimer, S. and Trebst, G. (1974) Biochim. Biophys. Acta 357, 1–13
36 Remennikov, V.G. and Samuilov, V.D. (1979) Biochim. Biophys. Acta 546, 220–235
37 Padan, E. and Rottemberg, H. (1973) Eur. J. Biochem. 40, 431–437
38 Baccarini-Melandri, A., Casadio, R. and Melandri, B.A. (1977) Eur. J. Biochem. 78, 389–402
39 Baccarini-Melandri, A., Gabellini, N., Melandri, B.A., Hurt, E. and Hauska, G. (1980) J. Bioenerg. Biomembranes 12, 95–110