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The role of auxiliary oxidants in the maintenance of a balanced redox poise for photosynthesis in bacteria

A.G. McEwan *, N.P.J. Cotton, S.J. Ferguson ** and J.B. Jackson ***

Department of Biochemistry, University of Birmingham, P.O. Box 363, Birmingham, B15 2TT (U.K.)

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Carotenoid absorbance changes were used to monitor the development of membrane potential in intact cell suspensions of *Rhodopseudomonas capsulata* strain N22. Low concentrations of phenazine methosulphate almost completely inhibited the generation of membrane potential in the light by anaerobic cells. The light-dependent reactions were restored by addition of either trimethylamine *N*-oxide, dimethylsulphoxide, nitrous oxide, or oxygen. In *Rhodopseudomonas capsulata* strain N22 DNAR$^+$ addition of nitrate was also effective. The inhibition by phenazine methosulphate and restoration by auxiliary oxidant were observed in the presence of sufficient rotenone to block NADH dehydrogenase or with low concentrations of uncoupling agent to dissipate the membrane potential under dark, anaerobic conditions. It is suggested that in intact cells of these organisms there are mechanisms which operate to maintain the electron-transport chain at an optimal redox poise for efficient photosynthesis. Phenazine methosulphate perturbs the optimal redox poise by hastening equilibrium of the photosynthetic electron-transport chain with low-potential couples in the cell. The addition of auxiliary oxidants restores the optimal redox poise. This suggests a role in photosynthesis for the pathways of respiratory electron flow to nitrate, nitrous oxide, trimethylamine *N*-oxide/dimethylsulphoxide and oxygen.

Introduction

The photosynthetic electron-transport pathway in species of the Rhodospirillaceae, including *Rhodopseudomonas sphaeroides* and *Rhodopseudomonas capsulata,* is cyclic [1]. The absorption of light leads to the oxidation of P-870 and the reduction of bound ubiquinone in the photosynthetic reaction centre. Subsequent electron transport in the dark through the ubiquinone pool, the cytochrome b/c_1 complex and cytochrome c_2 completes the cycle. The electron-transport reactions lead to the formation of a proton electrochemical gradient (Δp) across the cytoplasmic membrane of the cell and to ATP synthesis. Light-driven cyclic electron-transport proceeds within a range of ambient redox potential that is defined by the standard potentials of the P-870 and the primary bound quinone [2]. When the ambient redox potential was poised with mediators in experiments with isolated membranes (chromatophores), rapid electron transport and consequently the maximum development of Δp and high rates of ATP synthesis were observed only within narrow limits, close to 100 mV [3–7].

* Present address: Department of Microbiology, 131 Burrill Hall, 407 South Goodwin Avenue, Urbana, IL 61801, U.S.A.

** Present address: Department of Biochemistry, University of Oxford, South Parks Road, Oxford, U.K.

*** To whom correspondence should be sent.

Abbreviations: TMAO, trimethylamine *N*-oxide; DMSO, dimethylsulphoxide; FCCP, carbonyl cyanide *p*-trifluoromethoxyphenylhydrazone; PMS, phenazine methosulphate.

It is widely held that in intact cells of these organisms the cyclic photosynthetic electron-transport chain is linked to low-potential donors such as NADH via the ubiquinone pool and high-potential acceptors such as oxygen via cytochrome c_2 [8,9]. Consequently, the photosynthetic chain of these cells might be vulnerable to overreduction or overoxidation and cease to function efficiently. Since these bacteria can grow phototrophically at a range of oxygen tensions and on a variety of carbon substrates, control mechanisms must exist in vivo to adjust the redox potential poise of the cycle to an appropriate value.

The respiratory pathway of *Rps. capsulata* is branched [10]. NADH and succinate are oxidised via a pathway of electron flow which terminates in either cytochrome c_2 oxidase or in 'alternative oxidase'. Membranes prepared from a mutant strain (M6) of *Rps. capsulata* which was deficient in the alternative oxidase (cytochrome *b*-260) were shown in some circumstances to be unable to maintain a proton-motive force during short periods of illumination [11]. In the related species *Rhodospirillum rubrum* a mutant, F11, was isolated which would not grow phototropically and which would catalyse photophosphorylation only in the presence of oxygen [12,13]. This mutant which is deficient in rhodoquinone [14] has lost 'photo-oxidase' activity, believed to be associated with an alternative oxidase [15]. These observations have suggested that the alternative oxidase may function as a pathway for the removal of excess reducing equivalents from the photosynthetic electron-transport chain [11,13]. The alternative oxidase probably branches from the photosynthetic chain at the level of the ubiquinone pool [16]. We have shown recently that a number of other terminal oxido-reductases, which we term the auxiliary terminal oxido-reductases, and which include nitrate reductase, trimethylamine *N*-oxide reductase and nitrous oxide reductase, also branch at the level of ubiquinone [17–19]. We proposed that these auxiliary oxido-reductases, which are located in the periplasmic space, may also serve as redox-balancing systems for photosynthetic electron flow [19–21].

In this report we show how photosynthetic electron transport in intact cells of *Rps. capsulata* depends critically upon an established disequilibrium between the cellular redox couples, and how the auxiliary oxido-reductases might be involved in the maintenance of an optimal redox poise for photosynthesis.

Methods

Rps. capsulata strain N22, a green mutant of strain St. Louis (provided by Dr. O.T.G. Jones, University of Bristol) was grown phototrophically at 30°C in RCV medium (for a definition, see Ref. 22) in completely filled screw-capped bottles approx. 30 cm from two banks of 100 watt tungsten lamps. *Rps. capsulata* strain N22 DNAR$^+$, a nitrate-respiring mutant of strain N22 [17], was grown under similar conditions, but in RCV medium in which the ammonium N-source was replaced by 15 mM $NaNO_3$. The cells were harvested in late exponential phase by centrifugation, washed and resuspended in 10 mM sodium phosphate (pH 7.0) and stored on ice for use within 6 h. Bacteriochlorophyll was determined by extraction into acetone : methanol [23].

Experiments were performed at 30°C in fresh RCV medium with cells resuspended to give a final bacteriochlorophyll concentration of 20 μM. Anaerobiosis was maintained by flushing the spectrophotometer cuvette with argon (less than 3 ppm O_2) by way of butyl rubber and metal tubing. Carotenoid absorbance changes were measured at 503–487 nm in a chopped, dual wavelength spectrophotometer (Perkin Elmer 356) as described previously [24]. Photosynthetic illumination, at 90° to the measuring beam, was provided with a 150 watt quartz-halogen lamp passed through Wratten 88A gelatin filter. At the conclusion of each experiment the addition of a high concentration of FCCP was used to establish the base line carotenoid absorbance which was equivalent to zero membrane potential.

Results and Discussion

Suppression of photochemistry with redox mediator

The midpoint potential of the primary electron-accepting ubiquinone (UQ_A) in the photosynthetic reaction centre is in the region of -20 mV at pH 7.0 [2]. The midpoint potential of $NAD^+/NADH$ is -320 mV at pH 7.0. From the

data of Schon [25], the ratio of $NAD^+/NADH$ in the cytoplasm of photosynthetic bacteria varies considerably with the metabolic state, but the redox potential of the couple is usually well below −250 mV. Therefore, if the pyridine nucleotides were permitted to come into simple redox equilibrium with UQ_A, then the latter would be extensively reduced and photochemistry would be restricted. The two couples are linked by way of the NADH dehydrogenase and the ubiquinone pool [8] and the quantity of pyridine nucleotide in the cytoplasm probably exceeds the quantity of reaction centre in the cytoplasmic membrane by an order of magnitude. According to this reasoning the equilibrium reduction of UQ_A by NADH would interfere with the photosynthetic reactions of the bacteria.

Nevertheless, washed cell suspensions of *Rps. capsulata* maintained even for several hours under dark anaerobic conditions are prepared for rapid photosynthesis immediately upon illuminating, as though their electron-transport chains had been held homeostatically at the optimal value for efficient, electrogenic electron transport [26]. In Figs. 1 and 2 the carotenoid band shift was used to monitor the membrane potential ($\Delta\psi$) developed during photosynthetic electron flow in intact cells. The suspension had been anaerobic for 10 min before recording commenced. Each experiment was begun with a single, short period of illumination. This gave rise to an absorbance change in the endogenous carotenoid spectrum which is characteristic of the formation of a cytoplasmic membrane potential. Separate experiments (not shown) revealed that repeated, short periods of illumination at intervals continued to yield similar absorbance changes for over an hour. The addition of 10 μM PMS led to a drop in the base line absorbance change under dark anaerobic conditions for reasons that will be discussed below. The important observation is that the PMS almost completely blocked the subsequent light-induced carotenoid absorbance changes. The effective concentration of PMS varied with the preparation of bacterial cells. Generally between 1 and 10 μM was sufficient. In the absence of further additions, the absorbance changes did not recover if the sample was kept in the dark for more than 1 h or if it was illuminated for prolonged periods.

In experiments with membrane vesicles, including chromatophores from photosynthetic bacteria, PMS is commonly used as a mediator for hastening redox equilibrium between membrane-bound components [27]. In the micromolar range, it has no known inhibitory effects on electron transport in chromatophores. We interpret its effect on anaerobic intact cells simply by its behaviour as a mediator – it permits equilibration between low-potential couples in the bacterial cytoplasm, such as $NAD^+/NADH$, and the membrane-bound carriers of the photosynthetic electron-transport chain. The resulting overreduction of the photosynthetic carriers which is normally avoided by unknown mechanisms, blocks light-driven cyclic electron flow and the formation of $\Delta\psi$.

A similar effect to that found with PMS was also observed with equivalent concentrations of phenazine ethosulphate, but not with up to 100 μM tetramethylphenylenediamine or with 30 μM diaminodurene (data not shown). Although the latter two mediators can interact with electron-transport components of intact cells of *Rps. capsulata* (unpublished observations), their standard redox potentials are considerably higher than the phenazine derivatives, and this may prevent them from catalysing rapid redox equilibration of low-potential couples of the bacteria.

Restoration of light-driven electron flow with auxiliary oxidants

We have recently discovered three auxiliary terminal oxidoreductases in the periplasmic fraction of cells from *Rps. capsulata*: nitrate reductase [20], trimethylamine *N*-oxide (TMAO) reductase which also reduces dimethylsulphoxide (DMSO) [21], and nitrous oxide reductase [19]. Electron transport to these components proceeds through NADH dehydrogenase and probably through the ubiquinone pool via largely uncharacterised redox components, though cytochrome *c′* may participate in electron flow to TMAO and N_2O (see Ref. 19 and see also unpublished observations). The antimycin insensitivity of all three pathways suggests that the cytochrome b/c_1 complex is not involved [17–19]. The effect of NO_3^-, TMAO, DMSO and N_2O on cells treated with low concentrations of PMS is shown in figs. 1 and 2. The experiments with TMAO, DMSO and N_2O were

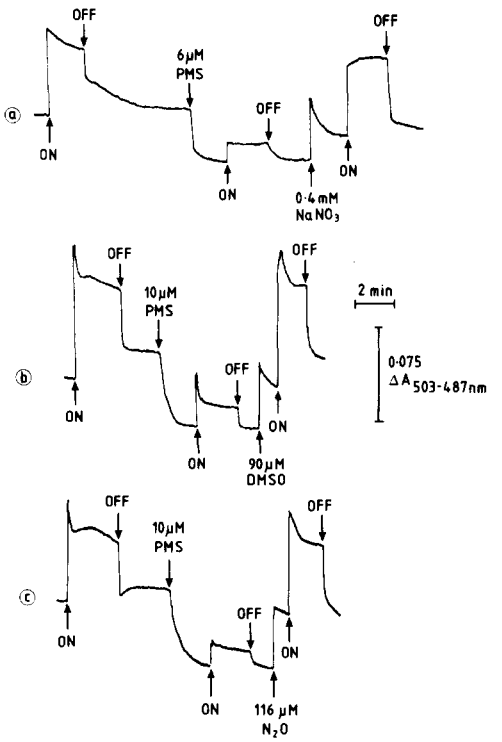

Fig. 1. Abolition of light-induced carotenoid absorbance changes by PMS and restoration with nitrate, dimethylsulphoxide and nitrous oxide. Harvested and washed, intact cells of *Rps. capsulata* (from strain N22 DNAR$^+$ in trace (a) and from strain N22 in traces (b) and (b)) were suspended anaerobically in fresh RCV medium (lacking nitrogen source in (a) and in the complete medium in (b) and (c)) to give 20 μM bacteriochlorophyll. The actinic light was switched on and off and the additions of reagents are as indicate dby the arrows (final concentrations of reagents are given).

performed with *Rps. capsulata* strain N22 and those with NO_3^- were performed with the nitrate-respiring mutant *Rps. capsulata* N22 DNAR$^+$. It is emphasised that the bacteria used for the experiments with TMAO, DMSO and N_2O were harvested from cultures grown phototrophically in the absence of these oxidants. The addition of oxidant to suspensions of bacteria in the dark (Figs. 1 and 2) led to a carotenoid absorbance change, indicating the formation of a cytoplasmic membrane potential. This confirms earlier observations that electron transport in the dark through the terminal auxiliary oxido-reductases is linked to proton translocation [17–19]. Figs. 1 and 2 show that the presence of the oxidant also resulted in the restoration of the PMS-inhibited, light-induced absorbance change. This suggests that the auxiliary oxidants are able to raise the redox potential of the over-reduced carriers to a value which is once again suitable for photosynthetic electron transport.

The addition of oxygen had a similar effect to the auxiliary oxidants (data not shown) and this may indicate that the alternative oxidase has a similar role to the NO_3^-, TMAO/DMSO and N_2O reductases. However in this case restoration of the light-induced formation of membrane potential might also be the result of a non-enzymic oxidation of reduced PMS by molecular oxygen. In separate experiments we showed that NO_3^-, TMAO and N_2O cannot directly oxidise reduced PMS. Two approaches were used. (1) 20 μM PMS in 10 mM sodium phosphate buffer (pH 7.0) under an atmosphere of argon was reduced by the addition of 100 μM NADH. This resulted in the loss of an absorbance band at 386 nm. The subsequent addition of 500 μM TMAO or 500 μM $NaNO_3$ did not lead to the restoration of the absorbance peak of oxidised PMS, nor did it produce any oxidation of the NADH measured at 340 nm. The addition of oxygen, however, led to PMS and NADH oxidation presumably in the sequence NADH → PMS → O_2 (data not shown). (2) In RCV medium there was no detectable reduction of N_2O measured with an electrode [28] in the presence of 1 mM NADH and 10 μM PMS (data not shown).

The involvement of the auxiliary reductases to rebalance the disturbed redox potential of the photosynthetic electron-transport chain was demonstrated in other ways. Firstly, it was shown that in the parent strain *Rps. capsulata* N22, which lacks the dissimilatory NO_3^--reducing pathway (NRII, [20]), the addition of NO_3^- was unable to restore the light-induced absorbance changes after treatment with PMS. Secondly, the presence of 780 μM acetylene, a specific inhibitor of N_2O reductase in this organism [19], prevented N_2O-dependent restoration of the PMS-inhibited, light-induced absorbance change. The acetylene also blocked the generation of membrane potential upon N_2O addition in the dark, but it had no effect on the formation of the light-induced mem-

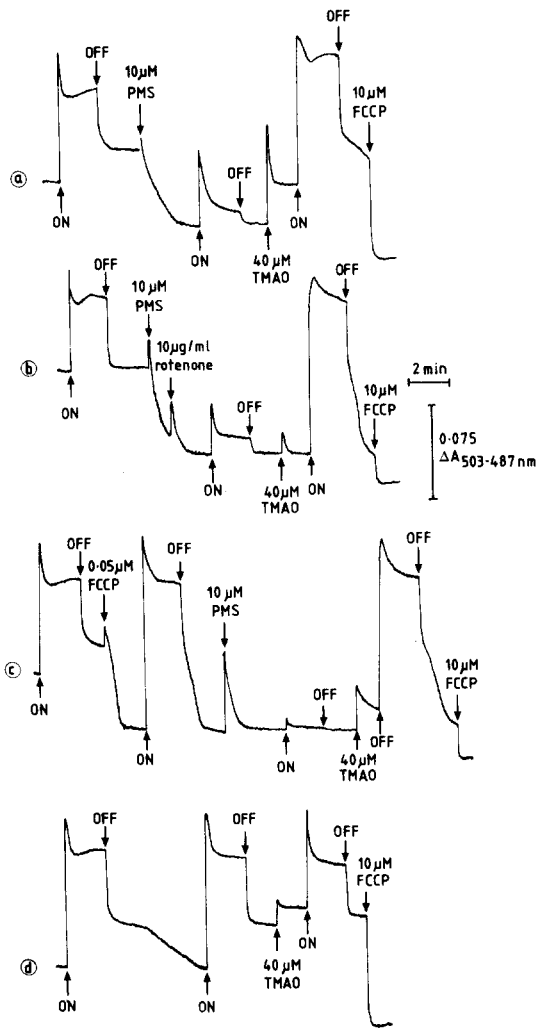

Fig. 2. Abolition of light-induced carotenoid absorbance changes by PMS and restoration by trimethylamine *N*-oxide. The effect of rotenone and low concentrations of FCCP. Conditions as in Fig. 1, using *Rps. capsulata* strain N22.

brane potential in the absence of PMS or N_2O (data not shown).

The effect of rotenone is shown in Fig. 2. This inhibitor of the membrane-bound NADH dehydrogenase prevented the formation of membrane potential on addition of TMAO in the dark, consistent with the pathway proposed for electron transport from NADH to the auxiliary oxidant [8]. The presence of rotenone did not, however, prevent the TMAO from restoring the PMS-inhibited light-induced absorbance change. In fact the restoration with TMAO appeared more striking, because in this instance the elimination of membrane potential arising from the dark pathway, NADH → TMAO, reduced the basal level from which the light-induced absorbance change began. The oxidising effect of TMAO on the components of the photosynthetic electron-transport chain is not expected to be blocked by rotenone, since the block is on the low-potential side of the ubiquinone pool.

Fig. 2d shows a control experiment in the absence of PMS. TMAO added in the dark shortly after the second illumination gave rise to a membrane potential. During a subsequent illumination period the membrane potential rose to a value commensurate with that achieved during the earlier periods. This shows that the auxiliary oxidant does not affect the proton-translocating activity of the photosynthetic electron-transport chain, unless the redox potential is disturbed, for example after treatment with PMS.

The influence on redox poising of the membrane potential which exists in dark, anaerobic suspensions of the photosynthetic bacteria

Even in dark, anaerobic suspensions of *Rps. capsulata* and related species, there exists a significant membrane potential. The origin of this potential is not clear although three possibilities can be considered: (1) a low, respiratory proton-translocating activity due to small quantities of oxygen contaminating the argon gas stream [24]; (2) a diffusion potential due to the efflux of cations from the cytoplasmic compartment of the cells; (3) H^+-efflux accompanying ATP hydrolysis by the F_0F_1-ATP synthase [29]. It is important to appreciate that because of the non-ohmic conductance properties of the bacterial membrane extremely small ion-transfer rates, by whatever mechanism, would be sufficient to maintain the dark, anaerobic membrane potential. Hence, this membrane potential can be largely eliminated by very low concentrations of uncoupling agent [30]. In unpublished experiments (Jackson, J.B. and Cotton, N.P.J.) we have been unable to provide positive evidence in favour of the first and third possibilities because the concentrations of agents required to inhibit either respiration or ATP hydrolysis (rotenone and dicyclohexylcarbodiimide, respec-

tively) give rise to a slight uncoupling activity.

The importance of the maintenance of a small $\Delta\psi$ in dark anaerobic conditions in the present work is that in principle it provides an alternative explanation for the inhibition by PMS of the photochemistry of the bacteria and the restoration by auxiliary oxidants. The fall in the level of the dark, anaerobic membrane potential upon addition of PMS (Figs. 1 and 2) could be explained a weak uncoupling effect of the mediator. Since there is an energy-coupling site between NADH and the ubiquinone pool [31], the existence of a cytoplasmic membrane potential would serve partly to prevent reduction of the quinone by the pyridine nucleotide. When the potential is dissipated, then redox equilibrium would shift in favour of quinone reduction, with adverse effect on subsequent photochemistry. The experiment shown in Fig. 2c argues against this possibility. Following the preliminary illumination period, the residual, dark, anaerobic membrane potential was dissipated with 0.05 μM FCCP. Despite this, the membrane potential reached during the next illumination period, an index of light-driven, coupled electron transport, was not significantly decreased. After the FCCP treatment, the PMS addition did not further lower the membrane potential (Fig. 2c), but it did lead to a loss of the light-induced change in membrane potential. As in the absence of uncoupler the addition of TMAO restored the light-induced absorbance changes. Higher concentrations of FCCP than were used in Fig. 2c led to complete elimination of the light-induced and TMAO-induced carotenoid absorbance changes. The conclusion from these experiments is that dissipation of the membrane potential in dark anaerobic bacterial suspensions with uncoupling agent does not lead to redox equilibration between NADH and ubiquinone and consequent abolition of photochemistry. Thus the inhibition of photochemistry by PMS can be attributed to its electron transferring properties and not any putative minor action as an uncoupler.

General discussion

It is already established that DMSO or TMAO [18], N_2O [19] and NO_3^- [17] can serve as terminal acceptors for proton-translocating electron-transport chains in *Rps. capsulata* and the energy conserved during anaerobic respiration to these acceptors is sufficient in some cases [19,32] to support growth in the dark. This may not be the only function of the auxiliary oxido-reductases for DMSO, TMAO, N_2O and NO_3^-. The fact that the TMAO- and N_2O reductase activities in strain N22 and NO_3 reductase in the spontaneous mutant N22 DNAR$^+$ are present constitutively in cells grown phototrophically in the absence of added auxiliary oxidant is impressive and suggests that in some circumstances these proteins may have a function in photosynthesis. The experiments described above suggest a plausible role for the auxiliary oxido-reductases in maintaining a balanced redox potential for efficient photosynthetic cyclic electron transport.

The concept of a balanced or poised redox potential arose many years ago with the finding in chromatophore membranes that, because the photosynthetic electron-transport chain is cyclic, components of the cycle can become overreduced or overoxidised by added redox coupled [33,34]. Attempts to put these experiments on a quantitative footing showed that the rate of photophosphorylation is dependent on the equilibrium value of the ambient redox potential adjusted with mediator dyes before illuminating [3–7]. The range of redox potential which gave maximum rates of ATP synthesis compared favourably with the range which promoted maximum rates of electron transport and membrane-potential generation after short light flashes [7]. Experiments with very low concentrations of dyes [7] to minimise their electron-donating and -accepting properties support the concept of an optimal redox poise for coupled photosynthetic electron transport.

The technique used in the present work for disturbing the intrinsic redox-balancing mechanism of anaerobic intact cells with PMS is imprecise. It is remarkable, however, that following the addition of auxiliary oxidant the light-generated membrane potential reached values which were not greatly different from those achieved in the absence of PMS. Also when oxidants were added in the absence of PMS or in the presence of rotenone to inhibit the reduction of ubiquinone by NADH, the membrane potential developed during illumination was remarkably constant. To some

extent this could be explained by the regulating effect of the membrane ionic conductance on the value of the membrane potential [35]. However, it may also indicate that the redox potential of the photosynthetic electron carriers is controlled by regulation of the rate of loss of surplus-reducing equivalents to the auxiliary oxidants. It is not known how the optimal poise is sensed and recognised, or how the electron-transport rate to the auxiliary oxidants is adjusted. One factor is the thermodynamic constraint imposed by the proton-motive force across the cytoplasmic membrane: the proton-motive force generated by the powerful proton-translocating activity of the photosynthetic electron-transport chain is sufficiently large to exert a back-pressure on the electron-transport reactions to oxygen [36,37], NO_3^- [38] and N_2O [19].

In phototrophic cultures growing in the absence of auxiliary oxidant other mechanisms must contribute to the maintenance of an optimal redox poise for photosynthetic electron transport. As mentioned above the presence of a large membrane potential across the coupling site of the NADH dehydrogenase could in principle be an important factor in preventing overreduction of the photosynthetic carriers by the cytoplasmic pyridine nucleotide pool. For example, a proton-motive force of 0.20 volt would maintain the equilibrium potentials of the NAD/NADH and the pool UQ/UQH_2 couples 0.2 volt or 0.4 volt apart, respectively, for 2 H^+ or 4 H^+ translocated per two electrons transferred across the NADH dehydrogenase coupling site. Metabolites of suitable oxidising power might also serve as electron sinks for the disposal of excess-reducing equivalents. The reduction of fumarate to succinate would be a prime candidate because of its close coupling to the redox state of the quinone pool. In *Rps. capsulata* the iron-sulphur centre associated with succinate dehydrogenase is in redox equilibrium with the quinone pool [39], and hence may fulfil a redox-poising role in this organism. In such instances, excess-reduced metabolite (e.g., succinate) might be excreted from the cell. This reasoning could account for the constitutive nature of the auxiliary oxido-reductases: in their natural environment it would be more efficient for the bacteria to consume auxiliary oxidant than to waste intermediary metabolites in the necessary process of attaining an optimal redox poise for rapid rates of photosynthetic electron flow.

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References

1 Cramer, W.A. and Crofts, A.R. (1982) in Photosynthesis, Vol. 1, Energy Conversion by Plants and Bacteria (Govindjee, ed.), pp. 387–467, Academic Press, New York
2 Prince, R.C. and Dutton, P.L. (1978) in The Photosynthetic Bacteria (Clayton, R.K. and Sistrom, W.R., eds.), pp. 439–453, Plenum Press, New York
3 Cusanovich, M.A., Kamen, M.D. (1968) Biochim. Biophys. Acta 153, 418–426
4 Culbert-Runquist, J.A., Hadsell, R.M. and Loach, P.A. (1973) Biochemistry 12, 3508–3514
5 Baccarini-Melandri, A., Melandri, B.A. and Hauska, G. (1979) J. Bioenerg. Biomembr. 11, 1–16
6 Baccarini-Melandri, A., Bagellini, N., Melandri, B.A., Hurt, E. and Hauska, G. (1980) J. Bioenerg. Biomembr. 12, 95–110
7 Van den Berg, W.H., Bonner, W.D. and Dutton, P.L. (1983) Arch. Biochem. Biophys. 222, 299–309
8 Zannoni, D., Jasper, P. and Marrs, B. (1978) Arch. Biochem. Biophys. 191, 625–631
9 Baccarini-Melandri, A., Jones, O.T.G. and Hauska, G. (1978) FEBS Lett. 86, 151–154
10 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, U.S.A.
11 Zannoni, D. and Marrs, B. (1981) Biochim. Biophys. Acta 637, 96–106
12 Del Valle-Tascon, S., Giménez-Gallego, G. and Ramírez, J.M. (1975) Biochem. Biophys. Res. Commun. 66, 514–519
13 Giménez-Gallego, G., Del Valle-Tascon, S., Ramírez, J.M. (1976) Arch. Microbiol. 109, 119–125
14 Ramírez-Ponce, M.P., Giménez-Gallego, G. and Ramírez, J.M. (1980) FEBS Lett. 114, 319–322
15 Del Valle-Tascon, S., Giménez-Gallego, G. and Ramírez, J.M. (1977) Biochim. Biophys. Acta 459, 76–87
16 Zannoni, D. (1982) Biochim. Biophys. Acta 680, 1–7
17 McEwan, A.G., George, C.L., Ferguson, S.J. and Jackson, J.B. (1982) FEBS Lett. 150, 277–280
18 McEwan, A.G., Ferguson, S.J. and Jackson, J.B. (1983) Arch. Microbiol. 136, 300–305
19 McEwan, A.G., Greenfield, A.J., Wetzstein, H.G., Jackson, J.B. and Ferguson, S.J. (1985) J. Bacteriol. Vol. 169, in the press
20 McEwan, A.G., Jackson, J.B. and Ferguson, S.J. (1984) Arch. Microbiol. 137, 344–349

21 McEwan, A.G., Wetzstein, H.G., Ferguson, S.J. and Jackson, J.B. (1985) Biochim. Biophys. Acta 806, 410–417
22 Weaver, P.F., Wall, J.D. and Gest, H. (1975) Arch. Microbiol. 105, 207–216
23 Clayton, R.K. (1963) Biochim. Biophys. Acta 75, 312–323
24 Clark, A.J. and Jackson, J.B. (1981) Biochem. J. 200, 389–397
25 Schon, G. (1971) Arch. Mikrobiol. 79, 147–163
26 Cotton, N.P.J. and Jackson, J.B. (1982) Biochim. Biophys. Acta 679, 138–145
27 Dutton, P.L. (1971) Biochim. Biophys. Acta 226, 63–80
28 Alefounder, P.R. and Ferguson, S.J. (1982) Biochem. Biophys. Res. Commun. 104, 1149–1155
29 Fenoll, C., Gómez-Amores, S. and Ramírez, J.M. (1985) Biochim. Biophys. Acta 806, 168–174
30 Taylor, M.A. and Jackson, J.B. (1986) Biochim. Biophys. Acta 810, 209–223
31 Klemme, J.H. (1969) Z. Naturforsch. 24b, 67–76
32 Schulz, J.E. and Weaver, P. (1982) J. Bacteriol. 149, 181–190
33 Bose, S.K. and Gest, H. (1963) Proc. Natl. Acad. Sci. USA 49, 337–345
34 Horio, T. and Kamen, M.D. (1962) Biochemistry 1, 144–153
35 Clark, A.J., Cotton, N.P.J. and Jackson, J.B. (1983) Eur. J. Biochem. 130, 575–580
36 McCarthy, J.E. and Ferguson, S.J. (1982) Biochem. Biophys. Res. Commun. 107, 1406–1411
37 Cotton, N.P.J., Clark, A.J. and Jackson, J.B. (1983) Eur. J. Biochem. 130, 581–587
38 McEwan, A.G., Cotton, N.P.J., Ferguson, S.J. and Jackson, J.B. (1984) in Advances in Photosynthesis Research (Sybesma, C., ed.), Vol. II, pp. 449–452, Martinus Nijhoff/Dr. W. Junk Publishers, Dordrecht, The Netherlands
39 Zannoni, D. and Ingledew, W.J. (1983) Arch. Microbiol. 135, 176–181