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Physiological electron donors to the photochemical reaction center of *Rhodobacter capsulatus*

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The nature and number of physiological electron donors to the photochemical reaction center of *Rhodobacter capsulatus* have been probed by deleting the genes for cytochromes c_1 and b of the cytochrome bc_1 complex, alone or in combination with deletion of the gene for cytochrome c_2. Deletion of cytochrome c_1 renders the organism incapable of photosynthetic growth, regardless of the presence or absence of cytochrome c_2, because in the absence of the bc_1 complex there is no cyclic electron transfer, nor any alternative source of electrons to rereduce the photochemically oxidized reaction center. While cytochrome c_2 is capable of reducing the reaction center, there appears no alternative route for its rereduction other than the bc_1 complex. The deletion of cytochromes c_1 and c_2 reveals previously unrecognized membrane-bound and soluble high potential *c*-type cytochromes, with $E_{m7} = +312$ mV and $E_{m6.5} = +316$ mV, respectively. These cytochromes do not donate electrons to the reaction center, and their roles are unknown.

Introduction

The cyclic photosynthetic electron-transfer systems of purple, non-sulfur bacteria such as *Rhodobacter capsulatus* consist of a photochemical reaction center and a cytochrome bc_1 complex [1–3]. These are both membrane-bound, multisubunit complexes, and it has long been considered that electron flow between them must be mediated by small, mobile components. Freely diffusible ubiquinone has been thought to be the connector for electrons emanating from the reaction center, while cytochrome c_2 has been considered to be the carrier of electrons to the reaction center. During aerobic growth the cytochrome bc_1 complex receives electrons from dehydrogenases, and passes them to a terminal oxidase, and these interactions have also been assumed to occur via the mediation of freely diffusible ubiquinone and cytochrome c_2 [3]. These notions became open to question with the finding that cytochrome c_2 was not a mandatory participant in photosynthetic electron flow [4]. The single gene for cytochrome c_2 was identified, and a large portion specifically deleted, and yet the genetically engineered strain was still capable of photosynthetic growth at essentialy normal rates [4]. Furthermore, photosynthetic electron transfer still involved the cytochrome bc_1 complex, and appeared to involve electron transfer directly between cytochrome c_1 and the reaction center [5].

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In this paper we report on experiments wherein we have deleted the gene for cytochrome c_1 in order to test whether this is the only component capable of donating electrons to the reaction center in the absence of cytochrome c_2. We find that, while this reveals a previously unsuspected membrane-bound high potential *c*-type cytochrome, there are no other donors in this organism.

Materials and Methods

Rhodobacter capsulatus strains were grown by respiration (aerobic, less than 2% dissolved oxygen, dark) or photosynthesis (anaerobic, saturating light intensity) on PYE medium supplemented with 2 mM $MgCl_2$ and $CaCl_2$ (MPYE-medium, Ref. 6) for the genetic constructions, and on RCV medium [6] supplemented with 0.1% yeast extract and casamino acids for subsequent analysis. All strains were derived from MT1131 (*rif, crt*D121), a 'green' derivative of SB1003 (*rif*) [6], and are listed in Table I.

Strain MT-G4/S4 carries a deletion of the heme-binding region of *cyc*A, the structural gene for cytochrome c_2 [4]. Strain MT-CBC1 has a deletion of most of the *pet*B (cytochrome *b*) and all of the *pet*C (cytochrome c_1) genes of the *pet* operon, leaving the Rieske iron-sulfur protein structural gene (*pet*A) intact [7]. Strain MT-GS18 was constructed by introduction of the Δ_1 (*cyc*A)::*kan* mutation into strain MT-CBC1 via Gene Transfer Agent (GTA) mediated genetic crosses using pG4/S4 in R121 [4] as a donor, and selecting for kanamycin resistance under respiratory growth conditions.

The preparation of chromatophores using a French pressure cell (without subsequent density gradient centrifugation), optical spectroscopy, and redox potentiometry followed standard methods [5]. Electron-spin resonance spectra were obtained with a Varian E-109 spectrometer equipped with a EIP model 548A microwave frequency counter and an Oxford flowing helium cryostat.

Sodium dodecyl sulfate (SDS) polyacrylamide gel electrophoresis was done according to Laemmli [8], except that for chromatophores a 9–18% gradient gel was used. Chromatophore supernatants were dialyzed against 5 mM *N*-morpholinopropane sulfonate, 1 mM KCl (pH 7.0) 10-fold concentrated, and analyzed by a 12% SDS-polyacrylamide gel electrophoresis for their cytochrome content. Protein concentrations were determined using the Biorad protein assay and approx. 100 μg of protein was loaded into each lane. The samples were diluted with 2 × Laemmli loading buffer, supplemented with 0.1 M dithiothreitol, and heated at 70 °C for 5 min. Heme staining followed the method of Thomas et al. [9] using tetramethylbenzidine.

Results

Of the four strains considered here (Table I), only two MT1131, the 'wild-type', and MT-G4/S4, deleted of cytochrome c_2, are capable of photosynthetic growth [4,5], so all the comparative results presented in this paper are for cells grown aerobically with gentle shaking. Fig. 1 shows spectra of supernatants obtained after cells broken in the French Pressure cell were spun at 200 000 × *g* for 12 h. The spectra are ascorbate-reduced minus ferricyanide-oxidized difference spectra, normalized to an equivalent amount of cytochrome *c'*. This latter cytochrome, of no known function [10], was chosen as unlikely to vary in the four strains. Fig. 1 shows that strains MT1131 and MT-CBC1

TABLE I

RHODOBACTER CAPSULATUS STRAINS

*crt*D, a mutation in the carotenoid biosynthesis pathway, rendering the strains 'green' due to the abolition of the synthesis of spheroidene and spheroidenone, and the accumulation of neurosporenes; *cyc*A, structural gene for cytochrome c_2; *pet*B and *pet*C, structural genes for the cytochrome *b* and c_1 subunits of the bc_1 complex; *rif, kan, spe*, resistance to rifamycin, kanamycin and spectinomycin, respectively; Ps, photosynthetic growth; Cyt, cytochrome.

Strain	Genotype	Relevant phenotypes
MT1131	*crt*D, *rif*	Cyt c_1^+, Cyt c_2^+; Ps^+
MT-G4/S4	*crt*D, *rif*, Δ_1 (*cyc*A)::*kan*	Cyt c_1^+, Cyt c_2^-; Ps^+
MT-CBC1	*crt*D, *rif*, Δ_{18}(*pet*B, *pet*C)::*spe*	Cyt c_1^-, Cyt c_2^+; Ps^-
MT-GS18	*crt*D, *rif*, Δ_1(*cyc*A)::*kan* Δ_{18}(*pet*B, *pet*C)::*spe*	Cyt c_1^-, Cyt c_2^-; Ps^-

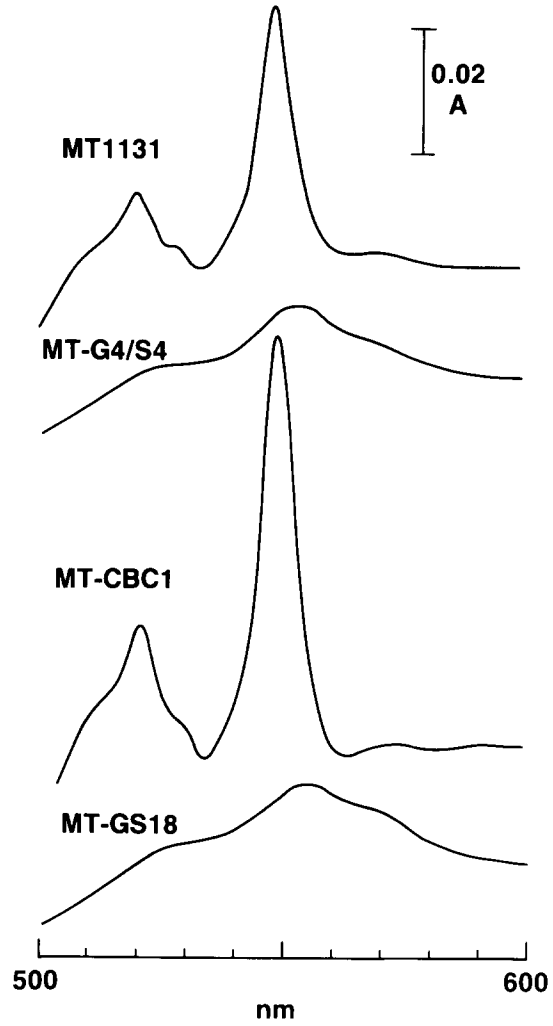

Fig. 1. Soluble cytochromes of the four strains used in this work. Supernatants obtained from the preparation of chromatophores were spun for 12 h at 200000 × *g*. These are ascorbate reduced minus ferricyanide oxidized spectra, normalized to an equivalent amount of cytochrome *c'* (Soret band dithionite-reduced minus ferricyanide-oxidized spectra (426–398 nm) = 0.23).

contain cytochrome c_2, while MT-G4/S4 (4) and MT-GS18 do not.

Fig. 2 shows spectra of cytochromes found in chromatophores of the four strains. Ascorbate, plus a trace of the redox mediator *N*-methylphenazonium methosulfate, reduces the *c*-type cytochromes, together with the high potential *b*-cytochromes of the terminal oxidase [11], while dithionite reduces all the cytochromes of the membrane. As shown in our earlier work [4,5], the deletion of cytochrome c_2 in strain MT-G4/S4 removes a third to a half of the total amount of *c*-type cytochrome, while leaving the amount of *b*-type cytochrome unaffected. The deletion of cytochromes c_1 and *b* of the *pet* operon in strain MT-CBC1 also reduces the amount of *c*-type cytochrome by about one-third, and in addition approximately halves the amount of *b*-type cytochrome. The deletion of both cytochromes c_1 and *b* of the bc_1 complex and cytochrome c_2 in strain MT-GS18 still leaves at least one apparently *c*-type cytochrome in the chromatophore membrane, together with the amount of *b*-type cytochrome seen in MT-CBC1. From these results we conclude that cytochromes c_1 and c_2 together comprise about two-thirds of the total *c*-type cytochromes in the chromatophore membrane, while the two *b*-type cytochromes of the bc_1 complex comprise about half of the *b*-type cytochromes of the membrane.

Fig. 3 shows heme-stained gels of the soluble cytochromes (panel A) and chromatophores (panel B) of the four strains used in this work. While all the strains contain cytochrome *c'* in the soluble fraction, this cytochrome is apparently absent in chromatophores (see Ref. 10, and lanes 2 and 4 in panel B). Clearly the strains MT-G4/S4 and MT-GS18 lack cytochrome c_2, while MT-CBC1 and MT-GS18 lack cytochrome c_1. The nature and identity of the two other heme-staining proteins, of approx. 26 and 33 kDa molecular weight and present in all strains, are unknown. Nonetheless, it is clear that these are not *b*-type cytochromes, since under the conditions used here such cytochromes are not detected. Another heme-staining protein of approx. 38–40 kDa molecular mass, especially visible in MT-CBC1, was also detected (lane 3, panels A and B). Whether this contributes at least partially to the additional amount of *c*-type cytochrome seen in this strain (Fig. 1), is an interesting possibility.

Fig. 4 shows an equilibrium redox titration of the apparently *c*-type cytochrome(s) in chromatophores of strain MT-GS18. The titration is consistent with a single component, $E_{m\,7} = +312$ mV.

Fig. 5 shows an equilibrium titration of the soluble, apparently *c*-type cytochrome seen in MT-G4/S4 and MT-GS18. It has a split α-band, with the major peak at 557 nm. Its equilibrium $E_{m6.5}$ is +316 mV.

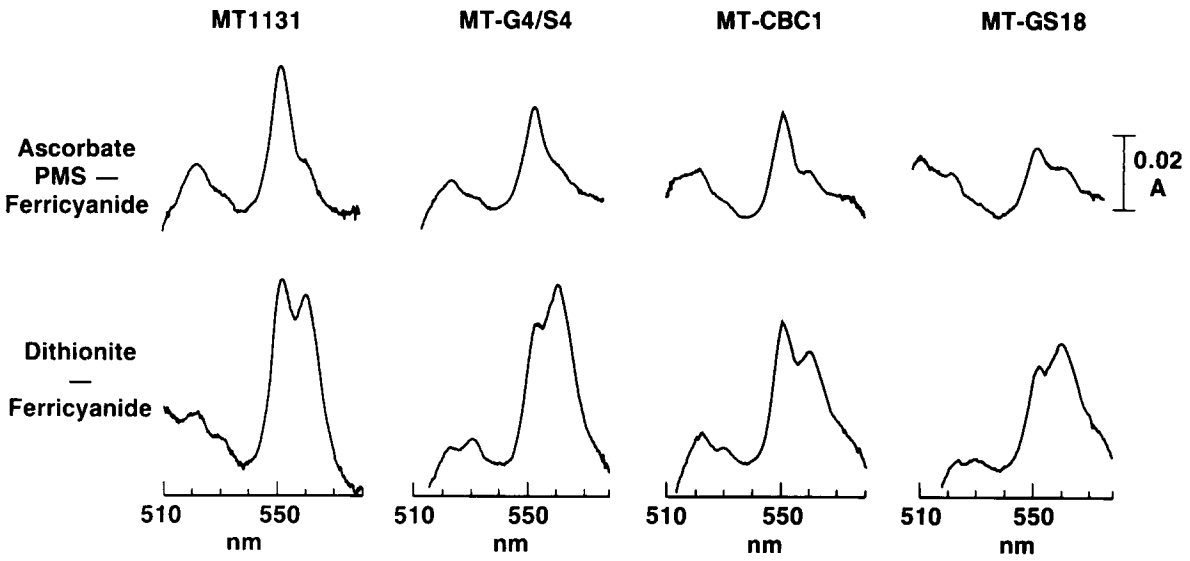

Fig. 2. Cytochromes in chromatophores of the four strains used in this work. Chromatophores (113 μM bacteriochlorophyll) in 20 mM *N*-morpholinepropanesulfonate, 100 mM KCl (pH 7.0).

Robertson, D.E. and Daldal, F. (unpublished data) have shown that deletion of any of the genes of the *pet* operon (coding for cytochromes *b* and c_1 and the Rieske iron-sulfur protein [12]) results in the absence of the entire complex from the membrane. This is in accord with our findings on

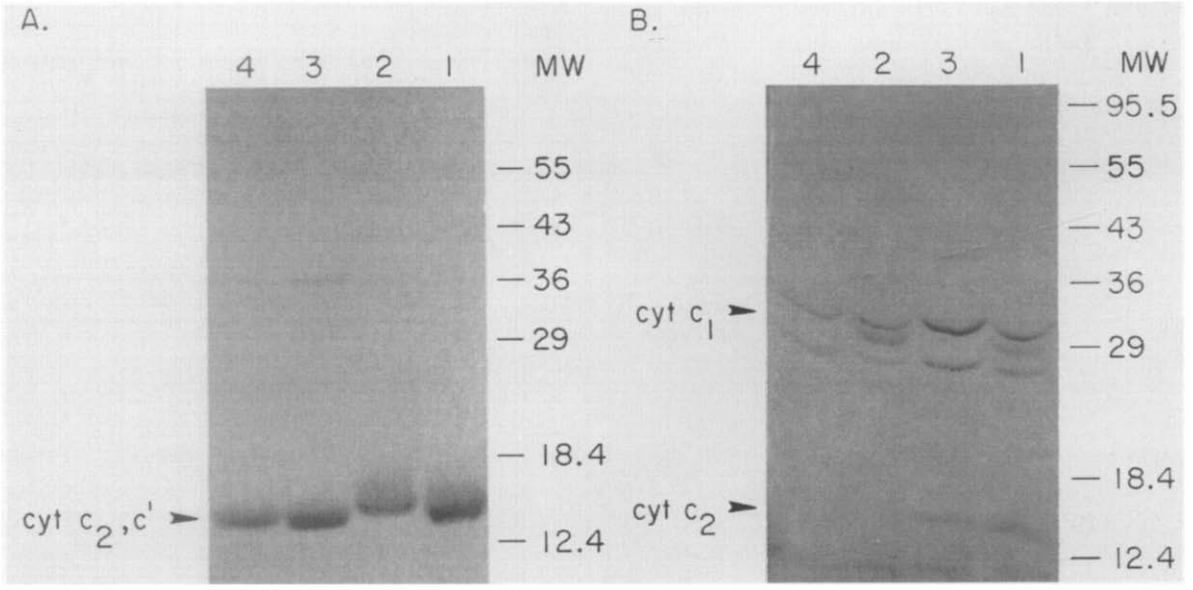

Fig. 3. Heme-stained gel of the soluble cytochromes (panel A) and of chromatophores (panel B) of the four strains used in this work. Approx. 70 μg of soluble protein or 100 μg of chromatophores, prepared as described in Materials and Methods from strains MT1131 (Cyt c_1^+, Cyt c_2^+, lane 1), MT-G4/S4 (Cyt c_1^+, Cyt c_2^-, lane 2), MT-CBC1 (Cyt c_1^-, Cyt c_2^+, lane 3) and MT-GS18 (Cyt c_1^-, Cyt c_2^-) were analyzed by SDS-polyacrylamide gel electrophoresis using either a 12% (panel A) or 9–18% gradient (panel B) separating gel. The gels were subsequently stained for heme-containing proteins using tetramethylbenzidine as in Ref. 9. Cyt c_1, c_2 and c' correspond to cytochromes c_1, c_2 and c', respectively.

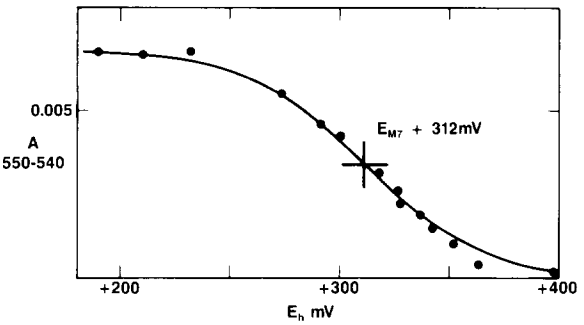

Fig. 4. Redox titration of the *c*-type cytochrome found in chromatophores of strain MT-GS18. Chromatophores were suspended in the buffer of Fig. 2, with 40 μM *N*-methylphenazoniummethosulfate, *N*-ethylphenazoniumethosulfate and 2,3,5,6-tetramethylphenylenediamine. Spectra were recorded as a function of ambient redox potential, and the amount of reduced cytochrome recorded.

strain MT113 [13], where a single mutation, which may be in the system that covalently inserts the heme into *c*-type cytochromes, results in the absence of cytochromes c_1 and c_2, the antimycin binding site thought to be near cytochrome b_H (see Ref. 1) and most of the Rieske iron-sulfur cluster. Of relevance to this work, the deletion of the genes for cytochromes *b* and c_1 in strains MT-CBC1 and MT-GS18 also results in the absence of the Rieske iron-sulfur cluster (prominent g_y at *g* 1.90) in these strains (Fig. 6, and data not shown for MT-CBC1).

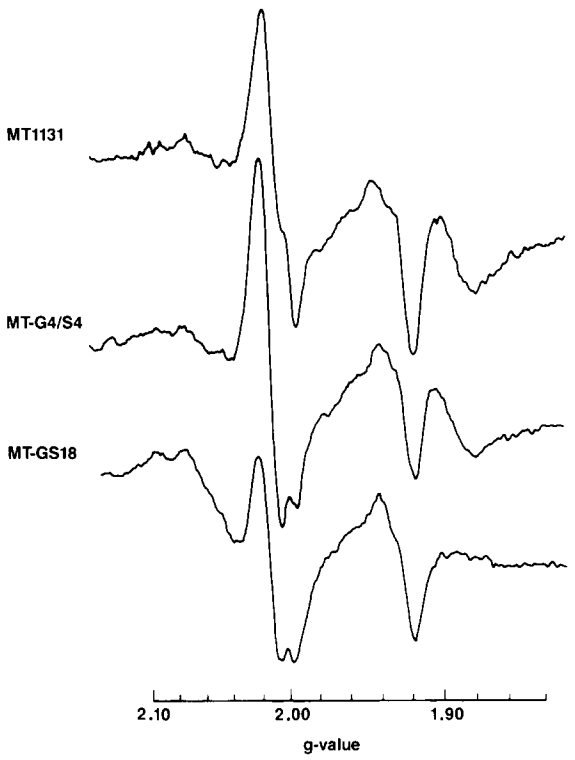

Fig. 6. The Rieske iron-sulfur cluster of three of the strains used in this work. Chromatophores (2 mM bacteriochlorophyll) were suspended in the buffer of Fig. 2, and reduced with ascorbate in the presence of 40 μM *N*-methylphenazoniummethosulfate. Spectrometer settings; 10 mW applied power, 17 K, 1.6 mT modulation, 9.234 GHz.

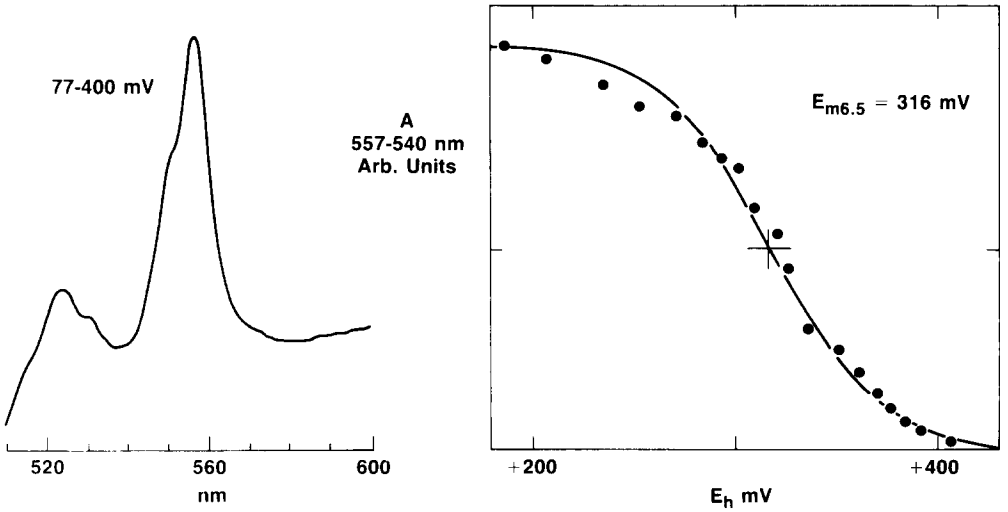

Fig. 5. Reduced minus oxidized difference spectrum, and redox titration, of the soluble *c*-type cytochrome of strain MT-G4/S4. Supernatant as described for Fig. 1 was concentrated approx. 10-fold by pressure filtration, and redox mediators were added as for Fig. 4. The spectrum reflects the difference between spectra recorded at E_h = 77 mV and E_h = 400 mV.

Fig. 7 shows the response of whole cells of the four strains under consideration to a train of four saturating single-turnover flashes of light, separated by 32 ms. The top row of traces are the responses of the reaction center bacteriochlorophyll dimer, while the second row are the responses of cytochromes c_1 and c_2. The bottom row of traces are the responses of the carotenoid bandshift, which monitors the membrane potential generated by light-driven electron flow (see Refs. 1–3). Fig. 8 shows the results of the same experiment repeated in the presence of the inhibitor 2-hydroxy-3-undecyl-1,4-naphthoquinone (UHNQ) [14] to inhibit electron transfer from the Rieske iron-sulfur cluster to cytochrome c_1, and therefore allow the observation of all *c*-type cytochrome oxidation. As discussed in our previous paper [5], cytochrome c_1 is apparently able to donate electrons directly and rapidly to the reaction center in the absence of cytochrome c_2 (MT-G4/S4), and this is revealed more clearly in the presence of UHNQ. Cytochrome c_2 is able to donate electrons to the reaction center in both the presence (MT1131, c.f. MT-G4/S4) and absence (MT-CBC1) of cytochrome c_1), but note that in the absence of the bc_1 complex (MT-CBC1) there is no reduction of the oxidixed cytochrome c_2 between flashes, even in the absence of UHNQ (Fig. 7). The absence of both the bc_1 complex and cytochrome c_2 (MT-GS18) leaves no rapid electron donor to the reaction center. Photochemistry then only occurs on the first flash, confirming that the flashes are essentially completely saturating, even in scattering samples such as the whole cells used in the experiments of Figs. 7 and 8.

Fig. 9 repeats the experiment of Figs. 7 and 8, except that it uses chromatophores instead of whole cells, and the inhibitor antimycin. In contrast to UNHQ, which has the effect of eliminating all cytochrome *b* involvement under typical conditions, antimycin prevents the oxidation of cytochrome b_H [1–3], but still allows its reduction. It also allows the Rieske iron-sulfur cluster to reduce cytochrome c_1 after the first flash. The responses of MT1131 and MT-G4/S4 to the train of flashes in Fig. 9 suggest that the absence of cytochrome c_2 does not diminish the amount of cytochrome b_H reduction after the first flash. It also conclusively demonstrates that there is no detectable involvement of *b*-type cytochromes other than those of the bc_1 complex (MT-CBC1 and MT-GS18), and no detectable alternative electron donors to the reaction center in the absence of the bc_1 complex and cytochrome c_2 (MT-GS18).

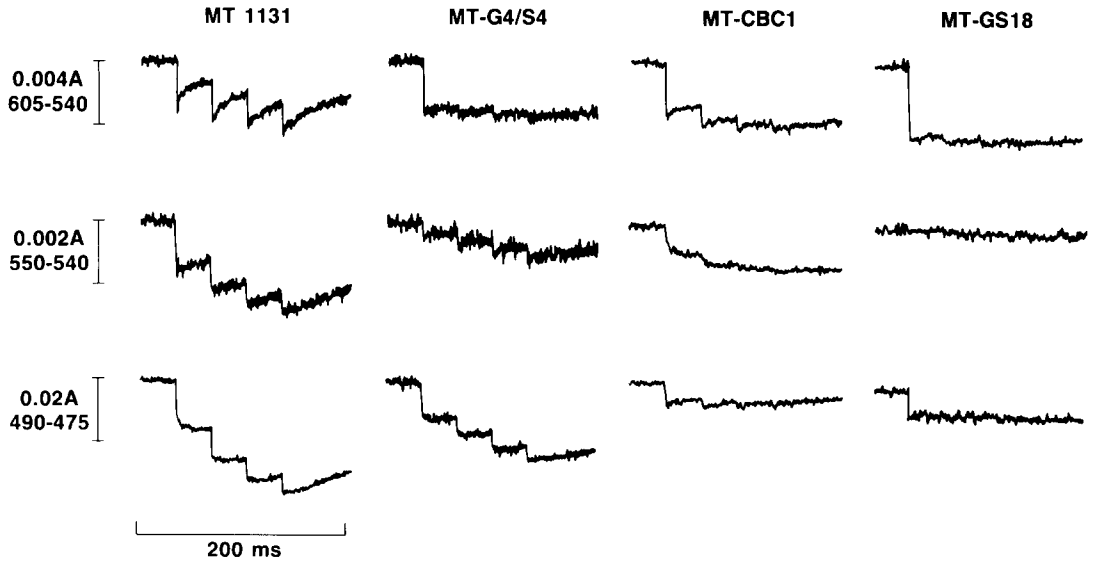

Fig. 7. Responses of the reaction center (605 − 540 nm), *c*-type cytochromes (550 − 540 nm) and the carotenoid bandshift (490 − 475 nm) of whole cells to four actinic flashes. Whole cells were suspended (20 μM bacteriochlorophyll) in 20% sucrose.

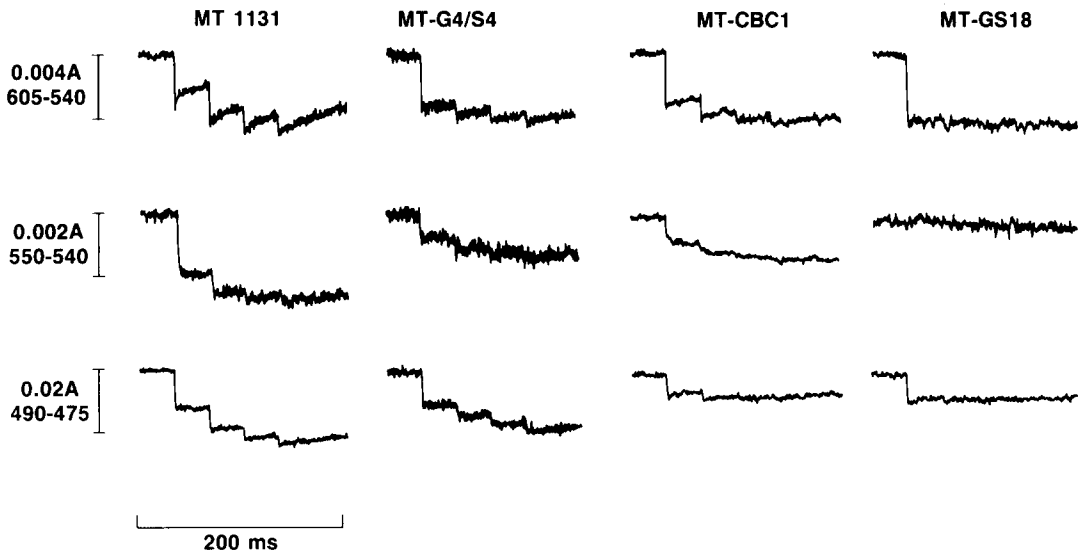

Fig. 8. Responses of the reaction center (605 − 540 nm), *c*-type cytochromes (550 − 540 nm) and the carotenoid bandshift (490 − 475 nm) of whole cells to four actinic flashes. Whole cells were suspended (20 μM bacteriochlorophyll) in 20% sucrose as in Fig. 7, with the addition of 10 μM 2-hydroxy-3-undecyl-1,4-naphthoquinone.

Fig. 10 shows the carotenoid bandshift in uninhibited chromatophores of the four strains. Note that there is almost as much bandshift in MT-G4/S4 as in MT1131, and that it is additive over many turnovers. In contrast, the amount of bandshift in the other two strains is much smaller, emphasizing the role of the cytochrome bc_1 complex in the generation of membrane potential and cyclic electron flow [1–3]. The reaction center is fully functional in generating membrane potential in all strains, but in the absence of the bc_1 complex and cytochrome c_2 (MT-GS18) there is no rereduction of the reaction center after the first turnover, and so no subsequent photochemistry (Figs. 7–9) or generation of membrane potential. Even in the presence of cytochrome c_2 (MT-

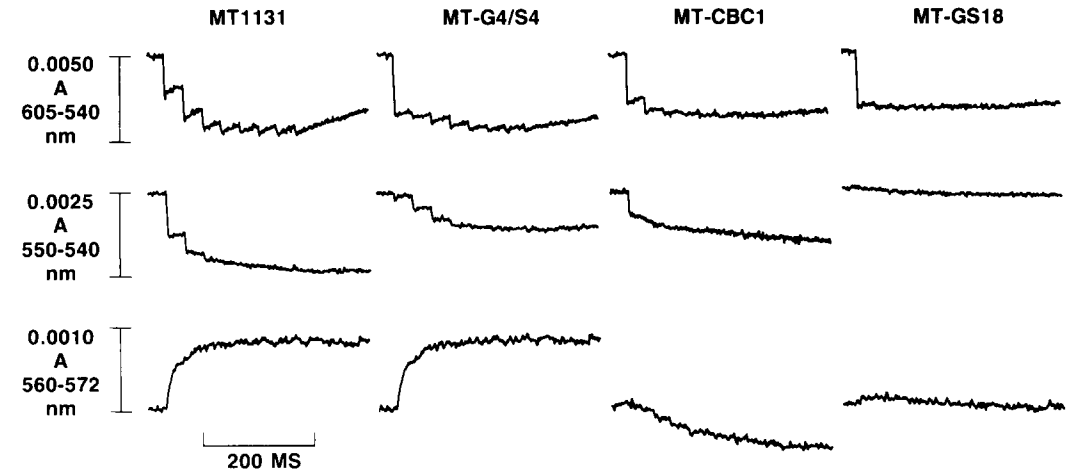

Fig. 9. Responses of the reaction center (605 − 540 nm), *c*-type (550 − 540 nm) and *b*-type (560 − 572 nm) cytochromes in chromatophores. Chromatophores (20 μM bacteriochlorophyll) were suspended in the buffer of Fig. 2 with 2 μM valinomycin and antimycin. A small amount of solid sodium ascorbate was added to bring the ambient redox potential to about 150 mV.

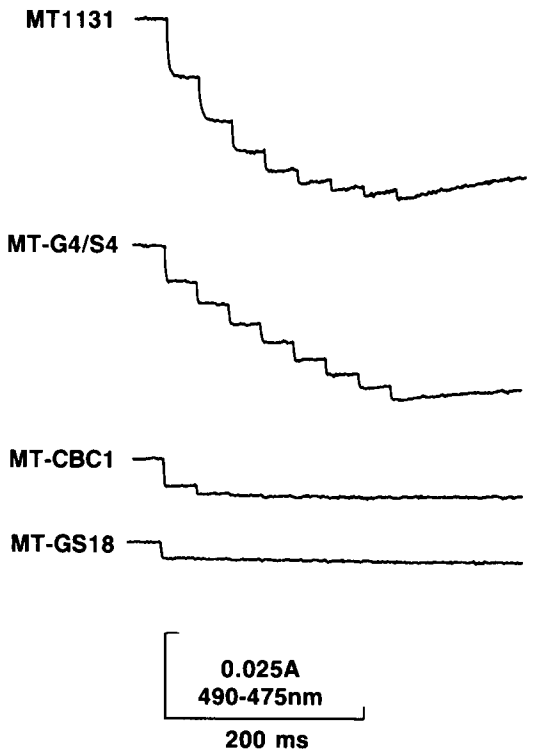

Fig. 10. The carotenoid bandshift in chromatophores of the four strains. Chromatophores were suspended as in Fig. 9, without valinomycin or antimycin.

CBC1), the absence of the bc_1 complex eliminates cyclic electron flow, and the reaction center runs out of electron donors after the second flash.

Discussion

The deletion of the gene for cytochrome c_2 in *Rb. capsulatus* revealed that cytochrome c_1 of the bc_1 complex was able to rapidly and directly donate electrons to the reaction center in this organism [5]. This indicated that cytochromes c_1 and c_2 operated, at least partially, as parallel electron donors to the reaction center. In this paper we have continued our analysis of the electron donors to the reaction center in *Rb. capsulatus* by deleting the gene for cytochrome c_1. This was done in concert with deleting the cytochrome *b* gene, and had the pleiotropic effect of deleting the entire bc_1 complex. It therefore precludes testing whether the Rieske iron-sulfur protein can act as a direct electron donor to the reaction center.

The deletion of the cytochrome bc_1 complex renders the organism photosynthetically incompetent, regardless of the presence or absence of cytochrome c_2. The data of figs. 6–9 show why this is so: in the absence of the bc_1 complex there is no alternative route to return the photo-electron from the reducing end of the reaction center back to the oxidizing end, and so the reaction center is starved of electrons, and cannot function after the first one or two turnovers. Presumably, the photo-electron gets as far as the secondary quinone of the reaction center, but can go no further. The fact that the deleted strains appear totally unable to grow photosynthetically, even at very high light intensities, suggests that the return pathway of the electron is either so slow that photochemical generation of membrane potential cannot compete with endogenous 'leaks', or that the return of the electron from reduced quinone to oxidized bacteriochlorophyll dimer in the reaction center collapses the potential generated by photochemistry.

The absence of both cytochromes c_1 and c_2 reveals the presence of additional high potential membrane-bound and soluble cytochromes (α band maximum at approx. 551 nm and 557 nm, Figs. 2 and 5) with $E_{m\,7} = +312$ mV (Fig. 4) and $E_{m\,6.5} = +316$ mV (Fig. 5), respectively. The membrane-bound cytochrome has not been recognized before, and if the amount of this cytochrome is unaffected by the levels of cytochromes c_1 and c_2, it accounts for approx. one-third of the total high-potential *c*-type cytochrome in *Rb. capsulatus* grown aerobically with low aeration (less than 2%) (Fig. 2).

Figs. 1 and 3 indicate that the strain lacking the bc_1 complex contains about 50% more cytochrome c_2, on a per cytochrome c' basis, than does the wild type. It may also contain another soluble cytochrome of approx. 39 kDa molecular weight. However, it makes no more reaction centers on a per bacteriochlorophyll basis (Fig. 9). Is the 'extra' cytochrome c_2 present in chromatophores as well? As it will be discussed elsewhere (Robertson, D.E. and Daldal, F., unpublished results) and as shown in Fig. 6, deletion of any part of the *pet* operon results in the pleiotropic loss of the entire bc_1 complex, and such a massive loss of membrane protein may well alter the amounts of other proteins in the membrane, including the newly recog-

nized high-potential cytochrome. It is also equally possible that in the absence of the bc_1 complex, the amount of cytochrome c_2 encapsulated in chromatophores [14] may be decreased, thereby increasing the amount found in soluble fractions.

The similarity of the E_{m7} of the newly detected membrane-bound cytochrome to that of c_1 (E_{m7} = 345 mV, Ref. 5) and C_2 (E_{m7} = +340 mV, Ref. 15) precluded its identification from redox titrations that included the other high-potential cytochromes. Its function is unknown. It does not seem to be the *c*-type cytochrome that co-purifies with cytochrome *c* oxidase, since the E_{m7} of that detergent-solubilized cytochrome is +234 mV [16]. The molecular weight of the newly detected cytochrome is not yet known. Although analysis of chromatophores by SDS-polyacrylamide gel electrophoresis and heme-staining (Fig. 3B) indicates that at least two proteins with covalently attached heme are present in membranes that lack cytochromes c_1 and c_2, further experiments will be required to ascribe the cytochrome of Fig. 4 to a specific band.

The soluble cytochrome detected in supernatants of the strains lacking cytochrome c_2 also appears to be a previously unrecognized cytochrome. It has a split α-band in the reduced form, with a wavelength maximum at 557 nm, and $E_{m6.5}$ = +316 mV (Fig. 5), and it might correspond to the faint band of 36–38 kDa molecular weight seen in Fig. 3A. If it has a similar extinction coefficient to other *c*-type cytochromes, it is present at approx. 15% the level of cytochrome c_2 (Fig. 1). Earlier reports of the soluble cytochromes in purple photosynthetic bacteria [17,18] have not specifically addressed *Rb. capsulatus* (see Ref. 17), but *Rhodopseudomonas palustris* contains a cytochrome with an α-band maximum at 556 nm, E_{m7} = +230 mV, and *Rb. sphaeroides* contains a *c*-554 with E_{m7} = 203 mV [17]. Whether the cytochrome reported here is related to these cytochromes remains an open question.

Acknowledgements

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