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STUDIES OF THE FUNCTION OF THE MEMBRANE-BOUND IRON-SULFUR CENTERS OF THE PHOTOSYNTHETIC BACTERIUM *CHROMATIUM VINOSUM*

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Chromatophores from the photosynthetic bacterium, *Chromatium vinosum*, have been prepared which photoreduce NAD^+ with either succinate or reduced dichlorophenolindophenol as electron donors. NAD^+ reduction is inhibited by uncouplers as well as inhibitors of cyclic photophosphorylation. These chromatophores contain several bound iron-sulfur centers which have been detected by low-temperature EPR spectroscopy. One center, having a *g* 2.01 EPR signal in the oxidized state, has $E_{m7.5}$ = +50 mV and is partially reduced by succinate in the dark. Three iron-sulfur centers having *g* 1.93 EPR signals have been resolved by redox titration, and the $E_{m7.5}$ values of these centers are −50, −175 and −250 mV, respectively. Studies of the involvement of these centers in electron transfer from donors to NAD^+ have indicated that the center with E_m = −50 mV is succinate reducible in the dark and appears to be analogous to center S-1 of succinic dehydrogenase in other systems. An additional *g* 1.93 iron-sulfur center can be photoreduced in the presence of electron donors and this reduction is inhibited by uncouplers. The possible role of the two low-potential iron-sulfur centers in relation to the dehydrogenases functioning in NAD^+ reduction is considered.

Introduction

Chromatophores from photosynthetic bacteria are known to contain multiple membrane-bound iron-sulfur centers (see Ref. 1 for a recent review). Redox potentiometry, in conjunction with EPR techniques, has led to the identification of several centers with EPR *g* values of approx. 1.94 in the reduced state that can be differentiated on the basis of differences in midpoint oxidation-reduction potentials (E_m). Another type of bound iron-sulfur center, with an EPR signal at *g* 2.01 in the oxidized form [2], has also been detected in chromatophores of purple nonsulfur bacteria.

Although the presence of these bound iron-sulfur centers has been known for several years, few studies on their function have been presented. Ingledew and Prince [2] associated several of the centers with a succinic dehydrogenase in *Rhodopseudomonas sphaeroides* by showing that three bound centers (two with *g* values at 1.94 and one with a *g* value of 2.01) could be removed from the membrane in a soluble succinic dehydrogenase. Similarly, Carithers et al. [3] isolated succinic dehydrogenase from *Rhodospirillum rubrum* chromatophores after detergent treatment and found multiple iron-sulfur centers in the solubilized enzyme. The possible role of these centers in relation to chromatophore electron-transport reactions has not been considered in detail. The purple sulfur bacterium, *Chromatium vinosum*, also is known to contain sev-

Abbreviations: BChl, bacteriochlorophyll; DCIP, dichlorophenolindophenol; FCCP, carbonyl cyanide *p*-trifluoromethoxyphenylhydrazone; TTFA, 2-thenoyltrifluoroacetone; UHDBT, 5-(*n*-undecyl)-6-hydroxy-4,7-dioxobenzothiazole; DBMIB, 2,5-dibromo-3-methyl-6-isopropyl-*p*-benzoquinone.

eral bound iron-sulfur centers [4,5]. A thermodynamic characterization of these centers has been presented, but no function has been described. In this communication, we report findings which associate several of the *C. vinosum* centers with succinic dehydrogenase and present evidence indicating a function for these centers in the reduction of NAD^+ by succinate catalyzed by these chromatophores.

Materials and Methods

C. vinosum strain D was grown in a modified Pfennig's medium [6] with succinate as the carbon source. In some studies, cells grown on malate or thiosulfate were also used. Cells from 2-l cultures were routinely used for small-scale chromatophore preparations while cells from 13-l cultures were used for the preparation of chromatophores on a larger scale, such as for EPR studies and redox titrations. In either case, optimal activity for NAD^+ reduction was found in cultures harvested 1–2 days after inoculation.

Chromatophores were prepared by resuspending approx. 4.5 g cells in a solution containing 100 mM Tris-HCl (pH 7.5) plus 100 mM KCl (this solution is hereafter referred to as buffer I) plus 20 mM sodium succinate to a final concentration of approx. 0.1 g cells per ml of solution. The cells were disrupted with a Branson model 200 sonifier at a power setting of 3. Two 45-s exposures at 50% duty resulted in a final chromatophore yield of about 1 mg BChl per g weight of cells. After sonication, the suspension was centrifuged for 30 min at 40 000 × *g* and the resulting supernatant solution centrifuged at 314 000 × *g* for 45 min. The chromatophore pellet was resuspended in buffer I plus 20 mM sodium succinate. In experiments where reduced DCIP was used as an electron donor, succinate was omitted from the final resuspension solution. In some experiments, the pellet was resuspended in 30 ml buffer I containing 30 mM sodium fumarate and repelleted by centrifugation. The chromatophores were then resuspended in buffer I. Chromatophores were used immediately, since it was found that activities decreased substantially upon storage at either –20 or 4°C.

NAD^+ photoreduction was measured at 340 nm in a Gilford spectrophotometer modified for actinic illumination. Actinic illumination ($5 \cdot 10^5$ erg/cm^2 per s) was provided by filtering white light through an 830 nm interference filter. Succinic dehydrogenase activity was measured using the phenazine methosulfate-DCIP method described by Singer [7] except that KCN was omitted from the reaction mixture. BChl was estimated by using the procedure of Clayton [8].

EPR spectra were recorded in a modified JEOL X-X-band spectrometer operating at a frequency of 9.20 Hz and using 100 kHz field modulation [9]. An Air-Products cryogenic system was used for temperature control. Redox titrations were performed as previously described [10].

UHDBT was a gift from Dr. B.L. Trumpower and DBMIB from Dr. A. Trebst. Rotenone, antimycin A and FCCP were obtained from Sigma Chemical Co. and TTFA from J.T. Baker Co.

Results

Chromatophore enzymatic activities

The chromatophores made from succinate-grown cells and prepared in the presence of succinate were able to catalyze a photoreduction of NAD^+ with either succinate or reduced DCIP as electron donors. Chromatophores from cells grown on malate rather than succinate showed considerable variability in their ability to photoreduce NAD^+, regardless of how they were prepared, and therefore were not routinely used.

As shown in Table I, NAD^+ reduction with succinate as the electron donor was inhibited by an uncoupler (FCCP), antimycin A, rotenone and two quinone analogues, DBMIB and UHDBT. Similar results were obtained using reduced DCIP as electron donor. In a series of independent experiments, DBMIB and UHDBT were found to inhibit endogenous cyclic photophosphorylation catalyzed by *C. vinosum* chromatophores. The sensitivity to an uncoupler suggests that electron flow from succinate to NAD^+ requires an input of energy. A similar mechanism was first put forward in the case of electron transport to NAD^+ in the purple nonsulfur bacteria [11–14] and differs from the uncoupler-insensitive NAD^+ reduction observed in the green sulfur bacteria [15,16].

C. vinosum chromatophores also showed succinic dehydrogenase activity with phenazine methosulfate-DCIP as an electron acceptor. The unavailability of

TABLE I

SUCCINATE-DEPENDENT NAD^+ PHOTOREDUCTION WITH *C. VINOSUM* CHROMATOPHORES – EFFECT OF INHIBITORS AND UNCOUPLERS

The reaction mixture contained 100 mM Tris-HCl buffer (pH 7.5), 100 mM KCl, 30 mM sodium succinate, 15 mM $MgCl_2$, 2 mM NAD^+, 10 mM glucose, 200 μg/ml glucose oxidase, 30 μg/ml catalase, and chromatophores at a concentration of 200 μg BChl per ml. Reactions were carried out under anaerobic conditions in 2-mm light-path cuvettes. NAD^+ photoreduction was measured as described in Materials and Methods.

	Q *	% of control activity
Complete system	7.2	100
+ 3 μM FCCP	0.7	10
+ 16 μM DBMIB	5.7	79
+ 48 μM DBMIB	1.8	25
+ 9 μM antimycin A	3.3	46
+ 18 μM antimycin A	1.0	14
+ 7.5 μM rotenone	2.2	31
+ 6 μM UHDBT	5.6	78
+ 12 μM UHDBT	2.9	40

* μmol NAD^+ reduced/mg BChl per h.

the short-chain analogues of ubiquinone prevented us from testing these compounds as electron acceptors. The specific activity of the succinic dehydrogenase in malate and succinate-grown cells (specific activity of 200–300 U *) was approx. 10-times greater than that observed in thiosulfate-grown cells (specific activity of 10 U). However (see below), this difference was not reflected in the actual amount of enzyme, based on detection of EPR components of succinic dehydrogenase, but may be related to the presence of inactivated enzyme in cells grown with different substrates.

Redox titrations of iron-sulfur centers in C. vinosum

Evans et al. [4] detected two iron-sulfur centers in *C. vinosum* chromatophores with g values in the 1.93 region ($E_{m8.0}$ = –50 and –290 mV); in a later report, Dutton and Leigh [5] reported a single g 1.93 iron-sulfur center but no midpoint potential was given. Because of these differing results, we have carried out a potentiometric titration of the g 1.93 iron-sulfur

* One unit of enzyme activity is defined as 1 μmol DCIP reduced/mg protein per h.

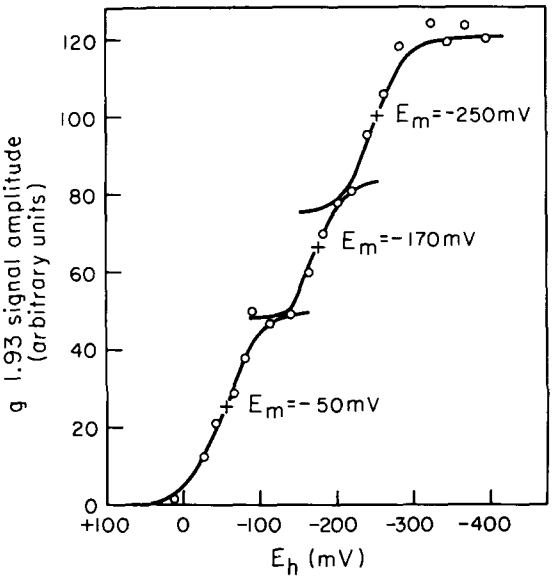

Fig. 1. Redox titration of the g 1.93 iron-sulfur centers in *C. vinosum* chromatophores. Chromatophores (2.7 mM BChl) were suspended in the standard buffer I in the presence of the following redox mediators: 80 μM methyl viologen, 80 μM benzyl viologen, 100 μM 2-hydroxy-1,4-napthoquinone, 100 μM anthraquinonedisulfonate, and 80 μM phenazine methosulfate. Redox titrations were carried out as described in Materials and Methods. EPR conditions: field setting, 3400 ± 250 G; modulation amplitude, 10 G; microwave power, 3 mW; temperature, 15 K. The line drawn through the points represents the theoretical curve for three redox components (n = 1) with the indicated midpoint potentials.

centers in our chromatophore preparation. The results of a typical titration, shown in Fig. 1, indicate the presence of three components, each showing n = 1 behavior. For further discussion, we have designated these centers as center 1 ($E_{m7.5}$ = –50 mV), center 2 ($E_{m7.5}$ = –175 mV) and center 3 ($E_{m7.5}$ = –250 mV). Lowering the redox potential below –350 mV did not result in any increase in signal intensity at g = 1.93, suggesting that no iron-sulfur centers with $E_{m7.5}$ significantly lower than –250 mV are present in *C. vinosum* chromatophores. We have also observed a g 1.89 Rieske iron-sulfur center with redox properties similar to those described by Evans et al. [4] and by Dutton and Leigh [5].

An important property of the g 1.93 centers which has been used to distinguish them in further studies is based on the temperature dependence of the reduced

EPR signals. As originally reported by Evans et al. [4], we find that the center 1 signal showed a greater intensity at 40 K than at 10 K while centers 2 and 3 were optimally observed at 10 K and broadened considerably at higher temperatures so that they were barely discernible at 40 K. Thus, EPR spectra obtained at 40 K could be used to detect only center 1 while spectra recorded at 10 K primarily gave the EPR signals from center 2 and/or center 3.

A comparison of the intensity of the g 1.93 signals in chromatophores prepared from cells grown on succinate, malate or thiosulfate showed no significant differences even though the measured succinic dehydrogenase activity of the chromatophores from thiosulfate-grown cells was only 10% of that in the other chromatophore preparations.

We have also observed a $g = 2.01$ EPR signal in our chromatophore preparations (Fig. 2A), and this signal is similar to one detected in chromatophores from *Rps. sphaeroides* and in the succinic dehydrogenase of the mammalian respiratory chain [17–20]. The g 2.01 signal has a strong temperature dependence: it is maximally observed at 10 K and above this temperature, the signal intensity decreases markedly such that it is barely detectable at 20 K. A redox titration of the chromatophore-bound g 2.01 component is shown in Fig. 3. The center shows an EPR signal in the oxidized state and has $E_{m7.5} = +50$ mV with an n value of 1.

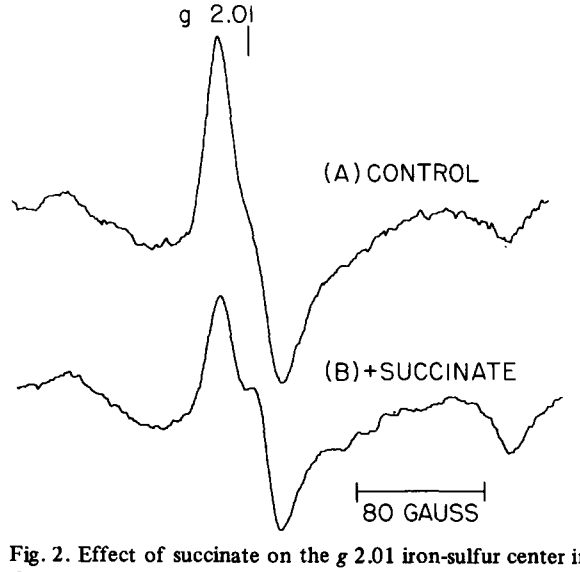

Fig. 2. Effect of succinate on the g 2.01 iron-sulfur center in *C. vinosum* chromatophores. Fumarate-washed chromatophores (2.0 mM BChl) were incubated in the dark at 4°C with 0.04 mM DCIP and excess ascorbate prior to freezing to 77 K in A. In B, 5 mM sodium succinate was also added prior to freezing. EPR conditions: field setting, 3235 ± 250 G; microwave power, 3 mW; modulation amplitude, 10 G; temperature, 10 K.

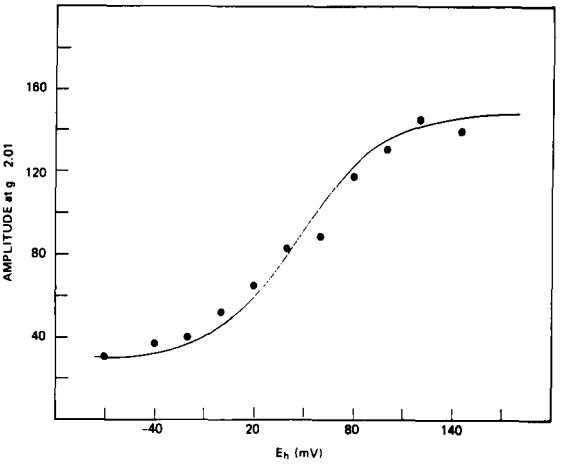

Fig. 3. Redox titration of the g 2.01 iron-sulfur center in *C. vinosum* chromatophores. Chromatophores (2.0 mM BChl) were suspended in the standard buffer I in the presence of the following redox mediators: 50 μM 2-hydroxy-1,4-napthoquinone, 50 μM duroquinone, 50 μM phenazine methosulfate, 50 μM 1,4-napthoquinone and 50 μM 2,5-dimethyl-*p*-benzoquinone. Redox titrations were carried out as described in Materials and Methods. EPR conditions were as in Fig. 2. The line drawn through the points represents a theoretical curve for a single species having $E_m = +50$ mV with an n value of unity.

Bound iron-sulfur centers under conditions of electron flow

With the identification of three g 1.93 components and the g 2.01 component, experiments were carried out to determine the function of these centers and their relationship to light-driven electron transport with various electron donors.

If the g 2.01 center in *C. vinosum* is associated with succinic dehydrogenase, as is the case in purple nonsulfur bacteria, it should respond to substrates of this enzyme. In order to study this question, the chromatophores were pretreated with fumarate (see Materials and Methods) to oxidize any centers associated with succinic dehydrogenase. It was then necessary to treat the chromatophores with reduced DCIP

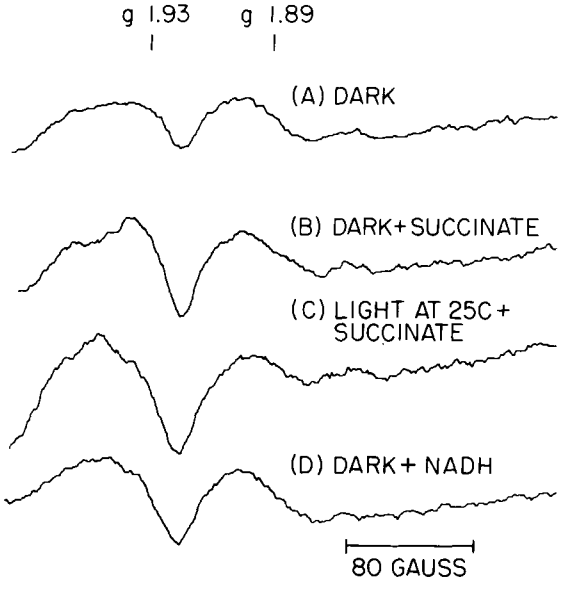

Fig. 4. Effect of succinate and NADH on the g 1.93 iron-sulfur centers (EPR at 40 K) in *C. vinosum* chromatophores. Chromatophores (2.9 mM BChl) were suspended in the standard buffer I and succinate (20 mM) or NADH (10 mM) was added where indicated. Illumination of samples was done at 25°C for 1 min and samples were frozen in liquid nitrogen in the light. EPR conditions were as in Fig. 1 except that the temperature was 40 K.

in the dark to reduce any of the high-potential iron-sulfur protein still associated with the membrane so that the EPR signal of the high-potential iron-sulfur protein would not interfere with measurements in the g 2.01 region. The g 2.01 center was oxidized in chromatophores which had been treated with reduced DCIP (E_m = +220 mV) as would be expected for an electron carrier with E_m = +50 mV. The addition of succinate in the dark (Fig. 2B) to a chromatophore sample caused a marked decrease in the g = 2.01 signal intensity. Addition of fumarate resulted in reoxidation of the g 2.01 center (data not shown).

The effect of succinate on the g 1.93 centers is shown in Fig. 4. These EPR spectra are recorded at 40 K where only center 1 is detected (see above). With no addition (Fig. 4A), a broad signal at g 1.89 (the Rieske center) is detected at this temperature and only a small signal at g 1.93 is present. After the addition of succinate in the dark, a new signal at g 1.93 is present (Fig. 3B) and because this signal is observed at 40 K, it can be associated with center 1. As was the case for the g 2.01 component, addition of fumarate resulted in oxidation of center 1. Illumination of the sample at room temperature in the presence of succinate (Fig. 4C) produces no additional change in the EPR spectrum at 40 K. The addition of NADH in the dark also results in the reduction of a g 1.93 center (Fig. 4D), and illumination in the presence of this donor causes no additional g 1.93 signal at 40 K (data not shown).

When the same samples are examined for their EPR signals at 10 K, a different pattern is observed. As shown in Fig. 5A, a large g 1.89 signal is present in the dark and virtually no g 1.93 signal is present. This situation is not altered by the addition of succinate in the dark (Fig. 5B) since center 1 shows little signal at 10 K, but after illumination in the presence of succinate a new g 1.93 signal appears at 10 K (Fig. 5C). The addition of NADH in the dark also causes the reduction of a g 1.93 center (Fig. 5D). Illumination in the presence of NADH as electron donor does not result in the reduction of any additional g 1.93 centers (data not shown).

Results with reduced DCIP as an electron donor in

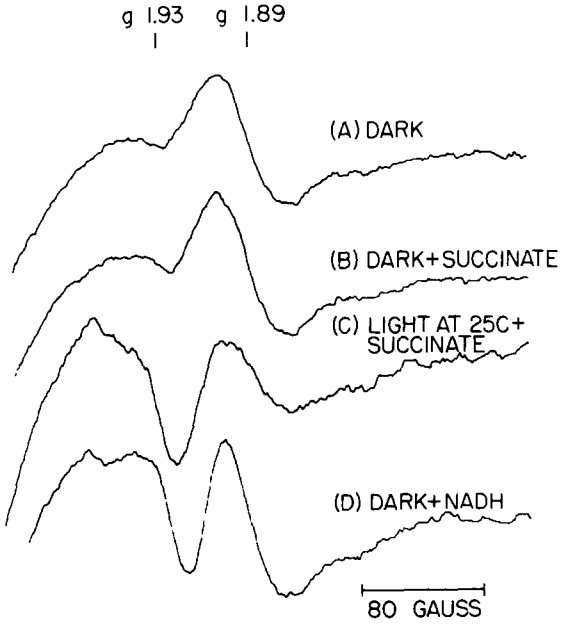

Fig. 5. Effect of succinate and NADH on g 1.93 iron-sulfur centers (EPR at 10 K) in *C. vinosum* chromatophores. Spectra of samples as prepared for Fig. 4 were recorded under appropriate EPR conditions at 10 K.

chromatophores are similar to those with succinate. At 40 K, EPR spectra show no effect of reduced DCIP on signals at g 1.93 in the dark while after illumination, a $g = 1.93$ center is present. The EPR spectrum at 40 K in the presence of reduced DCIP and light is almost identical to that observed with succinate in either the dark or light at 40 K, indicating the same center is probably reduced under both conditions. When spectra are recorded at 10 K, no g 1.93 signal is detected in the presence of reduced DCIP in the dark but after illumination, a g 1.93 center is reduced and can be detected at 10 K.

A study of the effect of inhibitors and uncouplers on the photoreduction of the g 1.93 centers with succinate as electron donor is shown in Fig. 6. When the spectra of illuminated samples are recorded at 10 K,

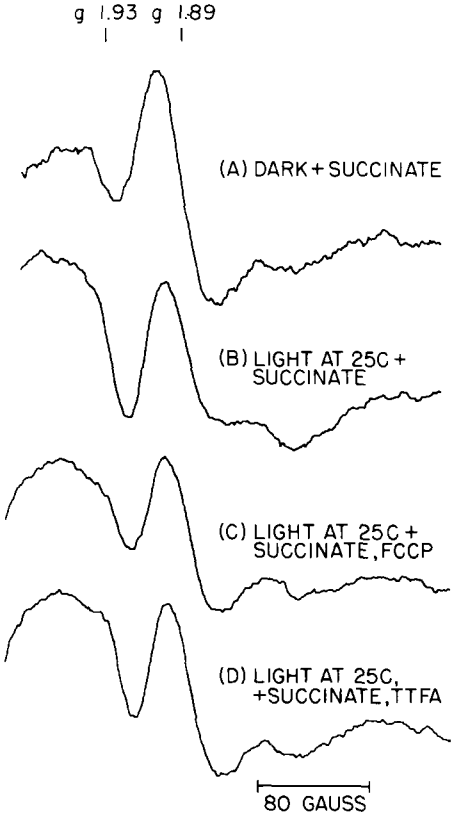

Fig. 6. Effect of FCCP and TTFA on succinate-dependent reduction of g 1.93 iron-sulfur centers in *C. vinosum* chromatophores. Conditions were as in Fig. 5 except that FCCP (0.4 mM) and TTFA (1.0 mM) were present as indicated. EPR spectra were recorded at 10 K.

FCCP almost completely inhibits the photoreduction of the g 1.93 center (Fig. 6C) while TTFA, a known inhibitor of succinic dehydrogenase activity [21,22], has a smaller effect on the photoreduction (Fig. 6D). In an additional series of experiments, the effect of rotenone, a specific inhibitor of mitochondrial NAD dehydrogenase [23,24] and an inhibitor of NAD photoreduction in our chromatophores, was also tested but results were variable in that inhibition of the photoreduction of the g 1.93 center was sometimes observed but not consistently. This variability may be due to the high concentration of this inhibitor required by the high bacteriochlorophyll concentrations used in these EPR studies. Neither FCCP nor TTFA had any significant effect on the dark reduction of the g 1.93 centers by NADH.

Although succinate as well as NADH was able to reduce at least one g 1.93 center, the addition of both substrates in the dark did not result in a fully reduced sample. The spectra at 10 K show that a maximal g 1.93 signal intensity in the presence of succinate plus NADH as compared with dithionite is not obtained. Approximately a doubling of g 1.93 intensity is observed at this temperature in the presence of dithionite. This finding indicates that dehydrogenase substrates do not reduce all the g 1.93 centers, but that at least one center, not reduced by substrates, can still be reduced by the nonphysiological reductant dithionite. Illumination of samples in the presence of succinate and NADH did not produce a g 1.93 signal as large as the dithionite-induced dark signal.

Discussion

The chromatophore preparations used in our studies of the bound iron-sulfur centers are capable of catalyzing electron transfer from either succinate or reduced DCIP to NAD^+. It was found that the preparative conditions were important in obtaining reproducibly active material. Cells from young cultures yielded the most active preparations and the inclusion of succinate in the preparative solutions was critical. In order to study the EPR changes associated with succinate reduction, it was then necessary to oxidize these components in a final step by including a fumarate wash procedure.

The aim of this study was to characterize the role of the bound iron-sulfur centers in *C. vinosum* in relation to the electron transfer activities we have measured. A series of studies was carried out in the presence of various electron donors, in both the dark and the light, in an attempt to assign functions to these multiple centers. The results of these studies and our conclusions concerning them are summarized below.

The g 2.01 center

The properties of the *g* 2.01 component in *C. vinosum* chromatophores are similar to those of a center previously seen in photosynthetic bacteria [2] and in mitochondria [18–20]. This center, known as S-3, has been associated with succinic dehydrogenase, and the similar E_m value as well as the EPR *g* value and temperature dependence suggest such a function in *C. vinosum.* The observation that the *g* 2.01 component undergoes reduction in the presence of succinate and oxidation in the presence of fumarate is consistent with this function. We have also been able to isolate a soluble succinic dehydrogenase by alkaline treatment of chromatophores [25] and the purified enzyme contains the *g* 2.01 center. It would therefore seem reasonable to assign this EPR signal to an S-3 center analogous to the one previously described.

The g 1.93 iron-sulfur centers

Center 1 ($E_{m7.5} = -50$ mV) has many properties which resemble those of center 1 in mammalian succinic dehydrogenase, and it is likely the *C. vinosum* center 1 is analogous to S-1. The E_m values of S-1 and the *C. vinosum* center are similar and the temperature dependence of the EPR signals also has a similarity in that these signals can be observed at relatively high temperatures. It has also been observed that center 1 can be reduced in the dark by succinate and can be reoxidized by fumarate. One observation of interest concerning this center is related to the finding that in the presence of reduced DCIP and light, the center undergoes photoreduction. This indicates there is some connection between the iron-sulfur centers of *C. vinosum* succinic dehydrogenase, of which center 1 (S-1) is a presumed component, and the electron carries of the light-driven electron-transport chain. Further study of this reaction is required to detail the nature of this interaction.

Of the additional two *g* 1.93 centers in the chromatophore, our conclusions must be more tentative. It is known that in mammalian succinic dehydrogenase, a low-potential *g* 1.94 center if present ($E_m = -250$ mV), but whether this center is a preparation artifact or a native component is not yet known. Albracht [26] has recently shown that this center is absent in submitochondrial particles and appears in the preparation after various treatments. We have tried to test this possibility in *C. vinosum* by preparing chromatophores by different procedures, but our preparations all contain three *g* 1.93 centers.

If we assume one of the low-potential centers in *C. vinosum* represents a center as described above (S-2), our data does not allow us to decide if it is center 2 or center 3. Our results indicate that one of these centers is photoreduced at room temperature with either succinate or reduced DCIP as the electron donor and that this reduction requires an input of energy, since it is inhibited by an uncoupler. The photoreducible center is most likely the center also reduced by NADH in the dark. It is also clear that after the addition of both succinate and NADH, an additional nonsubstrate reducible center is present which can be reduced by dithionite and these properties are suggestive of the presence of an S-2 center.

We attempted to associate one of the low-potential centers with succinic dehydrogenase by studying the iron-sulfur centers in cells grown on different substrates. Activity measurements of succinic dehydrogenase showed a 10-fold decrease in specific activity in thiosulfate-grown cells, but this decrease could not be correlated with any change in the amount of the *g* 2.01 or *g* 1.93 centers. It is likely that the enzyme is in an inactivated form under these growth conditions and that the total amount of enzyme present in the chromatophore is relatively constant and not significant altered by growth conditions.

Our results allow us to conclude that the *g* 2.01 center and two of the *g* 1.93 centers are involved in the electron-transport pathway from succinate to NAD^+. One of the low-potential centers, which is involved in the succinate pathway, is also common to the pathway from reduced DCIP to NAD^+. If we tentatively assign the third *g* 1.93 center to succinic dehydrogenase as an S-2 type center, this raises an interesting situation concerning the iron-sulfur complement of the *C. vinosum* NAD dehydrogenase system. This enzyme must necessarily contain only one

iron-sulfur center detectable by EPR and would therefore be markedly different from its mitochondrial analogue where multiple centers have been observed by redox potentiometry in conjunction with low-temperature EPR analysis [17]. We hope to be able to clarify these questions by resolution of the substrate-linked dehydrogenases from the *C. vinosum* membrane.

Acknowledgements

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