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# EPR CHARACTERISTICS OF OXYGEN-EVOLVING PHOTOSYTEM II PARTICLES FROM *PHORMIDIUM LAMINOSUM*

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## Summary

The EPR characteristics of oxygen evolving particles prepared from *Phormidium laminosum* are described. These particles are enriched in Photosystem II allowing EPR investigation of signals which were previously small or masked by those from Photosystem I in other preparations. EPR signals from a Signal II species and high potential cytochrome *b*-559 appear as they are photooxidised at cryogenic temperatures by Photosystem II. The Signal II species is a donor close to the Photosystem II reaction centre and may represent part of the charge accumulation system of water oxidation. An EPR signal from an iron-sulphur centre which may represent an unidentified component of photosynthetic electron transport is also described.

The properties of the oxygen evolving particles show that the preparation is superior to chloroplasts or unfractionated algal membranes for the study of Photosystem II with a functional water oxidation system.

## Introduction

Investigation of the mechanism of oxygen evolution by plants and algae and of the associated Photosystem II reaction centre have been less successful than those of bacterial and Photosystem I reaction centres because of the lability of the oxygen evolving enzyme system and difficulties in monitoring reaction centre components. Studies have shown that the oxygen evolution system of the

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Abbreviation: DCMU, 3-(3′,4′-dichlorophenyl)-1,1-dimethylurea.

thermophilic blue-green alga (cyanobacterium) *Phormidium laminosum* is stable to detergent treatment and that an oxygen evolving Photosystem II reaction centre preparation can be made from this organism [1,2]. Some of the properties of this preparation have been described previously [2,3].

Electron paramagnetic resonance spectrometry (EPR) offers the possibility of detecting electron transport components containing transition metal complexes or organic redox components which are difficult to characterise by other techniques whilst the detection of low temperature photochemical activity can indicate the role of these components in the reaction centre. EPR studies of Photosystem II (see review in Ref. 4) have identified a number of signals arising from Photosystem II. These include radical signals in the $g$ 2.00 region attributed to the oxidised reaction centre chlorophyll P-680$^+$ and the component termed Signal II. Signal II is observed under a variety of conditions and represents a number of species with varied kinetic properties at least some of which act as donors to the Photosystem II reaction centre. We have recently shown that a Signal II component, Signal $II_{slt}$, functions as a donor to P-680 at cryogenic temperatures [5], confirming a role in the reaction centre for this species. An EPR signal near $g$ 3.0 arising from the photooxidation of high and low potential forms of cytochrome $b$-$559_{HP}$ by Photosystem II at cryogenic temperatures has also been identified in chloroplasts and subchloroplast particles [6,7].

The interpretation of Photosystem II experiments is difficult in unfractionated material because of the large and well characterised EPR signals of Photosystem I whilst the use of Photosystem II preparations devoid of oxygen evolution may give misleading results. We have now characterised the low temperature EPR properties of the oxygen evolving particle and investigated the photochemical activity of these particles at cryogenic temperatures.

## Materials and Methods

*P. laminosum* was grown as described in Ref. 1 and oxygen evolving particles were isolated as described in Ref. 2. The pellet obtained after removal of the oxygen evolving particles was enriched in Photosystem I and was used as a Photosystem I preparation. Chlorophyll $a$ concentration was determined as described in Ref. 8.

The EPR spectrometry was performed as described in Ref. 5 and details of EPR conditions are given in the legends to the figures. $g$-value scales shown in figures are approximate. Spectra were stored using a Tektronix 4051 computer which was used to obtain difference spectra and plot figures. Samples were illuminated with a 1000-W projector.

## Results

### *Overall spectral features*

Fig. 1A shows a wide scan EPR spectrum of unfractionated membrane fragments from *P. laminosum* which had been frozen in the dark 15 min after the addition of 10 mM sodium ascorbate and then illuminated at 15 K. The spectrum shows the prominent features of reduced iron-sulphur centres A and B of Photosystem I. The large radical near $g$ 2.0 mainly due to P-700$^+$, the reaction

centre chlorophyll of Photosystem I has been omitted.

Fig. 1B shows a dark spectrum of oxygen evolving particles from *P. laminosum* frozen 15 min after the addition of ascorbate. The signal at *g* 4.3 is due to high-spin $Fe^{3+}$ in a rhombic environment and has a small contribution from the helium cryostat. However most of the signal originates from the particles and may be a storage form of iron [9] although electron spin echo studies [10] have indicated that it may arise from a cytochrome in an unusual environment or conformational state. Further small high-spin $Fe^{3+}$ signals are seen near *g* 6.0 similar to those reported in subchloroplast particles [7]. The most striking signal present in Fig. 1B compared to the original membranes is near *g* 3.0 where oxidised low-spin cytochrome signals occur. The signals attributed to both native and modified potential forms of high-potential cytochrome *b*-559 (cytochrome $b\text{-}559_{HP}$) [6,7] are reduced by ascorbate indicating that the signal in Fig. 1B arises from a component with a midpoint redox potential below +100 mV. This will be discussed in a later section.

Around the *g* 2.0 region small signals due to $Mn^{2+}$ and a large signal due to $Cu^{2+}$ are the prominent features. The manganese signal is decreased in size by illumination before freezing of samples. After illumination of the sample at 15 K the spectrum changes to that shown in Fig. 1C. The presence of Photosystem I in the preparation is shown by the photoreduction of iron-sulphur centres A and B. However the decreased signal size compared to the unfractionated membranes indicates considerably decreased Photosystem I to chlorophyll ratio in the particles. Based on the ratio of photoreduced centres A and B to chlorophyll *a*, a comparison between the particles and original membranes indicates that the particles contain 15—25% of the full Photosystem I content allowing for variation between preparations. This is in good agreement with the figures presented in Ref. 3 and based on P-700 content measurements.

The *g* 3.0 peak shows an irreversible increase upon 15 K illumination which will be discussed in greater detail below.

Fig. 1D shows the spectrum of particles frozen under illumination in the presence of ascorbate. By comparison with Fig. 1C this shows a reduction in size of the *g* 3.0 signal together with the appearance of a spectrum of fully reduced centres A and B below *g* 2.0. However superimposed on these latter signals is a signal at *g* 1.92 not seen in samples frozen in the dark.

Fig. 2A shows in more detail the spectrum in the *g* 2.00 region of oxygen evolving particles frozen in the dark after reduction with ascorbate. Only signals due to $Cu^{2+}$ and $Mn^{2+}$ are seen. There is no indication of the signal at *g* 1.89 of the Rieske iron-sulphur centre, a component in the intermediary electron transport chain (reviewed in Ref. 11) between the photosystems, indicating that it has been removed during purification. After illumination at 15 K, Fig. 2B, a small amount of centres A and B are reduced together with the appearance of a $P\text{-}700^+$ radical. The difference spectrum is shown in Fig. 2C. Fig. 2D shows the *g* 2.0 region of Fig. 1D showing the spectrum of fully reduced interacting centres A and B together with the *g* 1.92 signal which is superimposed on these.

Membrane preparations of *P. laminosum* were examined previously by Cammack and coworkers [12] and the EPR properties of their Photosystem I were studied. The Photosystem I content of oxygen evolving particles displayed simi-

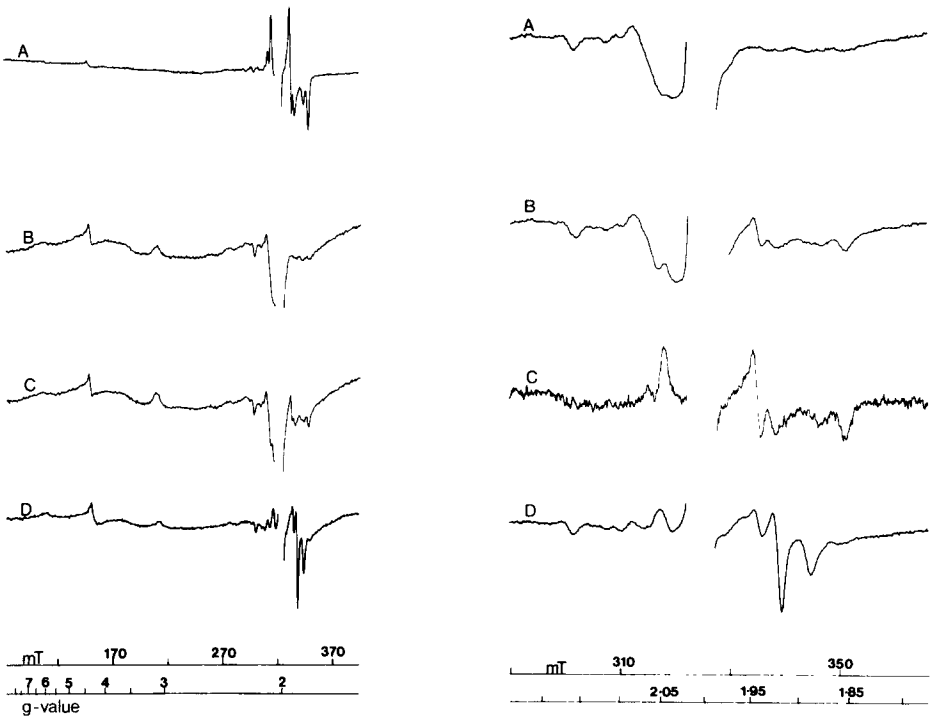


**Fig. 1. EPR spectrum of: (A) Unfractionated membrane preparation from *P. laminosum* dark adapted in the presence of 10 mM sodium ascorbate for 15 min, frozen and then illuminated at 15 K. Microwave power 10 mW; time constant, 0.1 s; scan rate 2 mT/s; modulation amplitude 1 mT; gain 1000; temperature 15 K, frequency 9.11 GHz. Chlorophyll a concentration 0.91 mg/ml pH 7.5. (B) Oxygen-evolving particles dark adapted with 10 mM sodium ascorbate and then frozen. Conditions as above except gain 2000. Chlorophyll a concentration 0.817 mg/ml. (C) As (B) after illumination at 15 K. (D) Oxygen-evolving particles frozen under illumination in the presence of 10 mM ascorbate. EPR conditions as (B) except gain 1600.**

**Fig. 2. (A) EPR spectrum of oxygen evolving particles frozen after dark adaptation in the presence of 10 mM ascorbate. Conditions as Fig. 1 except scan rate 25 mT/min. (B) After illumination at 15 K. (C) Difference spectrum ((B) — (A)) × 3. (D) Oxygen-evolving particles frozen under illumination in the presence of 10 mM ascorbate. EPR conditions as Fig. 1D.**

lar properties to those of intact membranes and to barley chloroplasts [13] with significant amounts of Centre B being photoreduced at cryogenic temperatures.

*The g 1.92 signal*

In the absence of ascorbate, freezing under illumination blocks Photosystem I as P-700$^+$ accumulates after electrons from Centres A and B are passed to oxygen. Under these conditions the spectrum of the *g* 1.92 signal was obtained without contributions from Centres A or B, Fig. 3. The same spectrum is obtained by dithionite reduction of particles which results in only a partial reduction of the sample due to the presence of the detergent lauryldimethylamine oxide which acts as an oxidant, preventing chemical reduction of Centres A and B.

A similar signal at *g* 1.92 with an approximate midpoint redox potential of —270 mV was found by Cammack and coworkers [9] in cells from *Nostoc*

*muscorum* and *Anabaena cylindrica* and membrane fragments from *P. laminosum* [12]. They suggested that this signal was due to a reduced iron-sulphur centre and that a line with a higher *g*-value should also be expected. This is shown in Fig. 3 at *g* 2.05 confirming the iron-sulphur centre characteristics of the signal. The function of the component is unclear, it is photoreduced at room temperature and it does not donate electrons to Photosystem I. Its redox potential would be compatible with a role in either cyclic electron transport or the electron acceptor side of Photosystem II. The room temperature photoreduction of the signal is blocked by the addition of either 50 $\mu$M 3-(3′,4′-dichlorophenyl)-1,1-dimethylurea (DCMU) or 2 mM 1,10-phenanthroline. However we do not regard this as conclusive evidence of a role as a direct electron acceptor from Photosystem II as the preparation is impure and the component could be part of the respiratory chain or other enzyme system and be reduced indirectly by Photosystem II turnover. The origin of the signal near *g* 1.97 is also unknown.

*Low spin cytochrome signals*

The membrane bound cytochromes of *P. laminosum* [1], cytochrome *f*, cytochrome *b*-563, cytochrome *c*-549 and both forms of cytochrome *b*-559 are present in the particles but cytochrome $b$-$559_{HP}$ and cytochrome *c*-549 are enriched several fold compared to the original membranes [3]. The spectrum in Fig. 4 shows the *g* 3.0 region spectrum of particles reduced by ascorbate and frozen in the dark. The signals in this region arise from low-spin cytochromes. The signal at *g* 3.0 is not due to cytochrome $b$-$559_{HP}$ as even modified forms of this cytochrome are reduced by ascorbate at normal pH values. Therefore the signal may be either oxidised cytochrome *c*-549, a low potential cytochrome

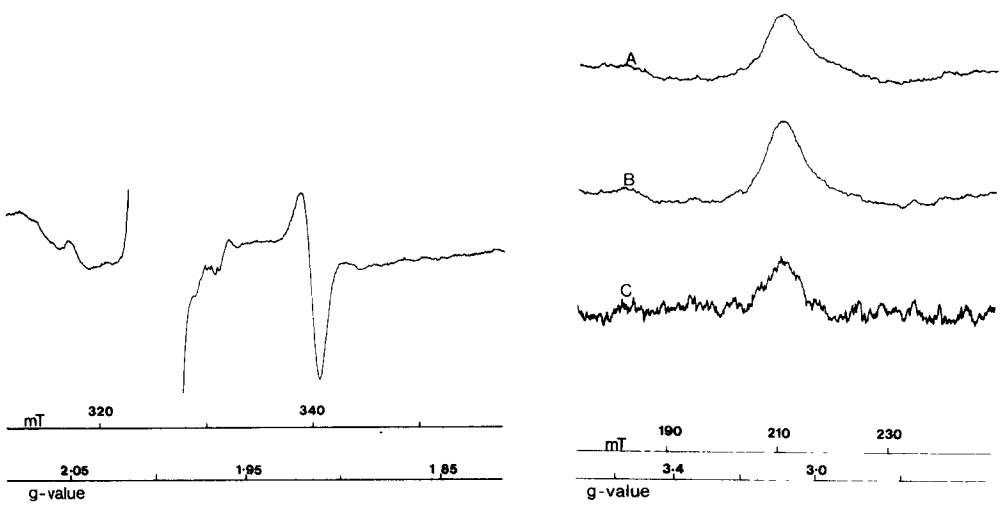


**Fig. 3. EPR spectrum of oxygen evolving particles frozen under illumination. Conditions as Fig. 1D.**

**Fig. 4. (A) EPR spectrum of oxygen evolving particles frozen after dark adaptation in the presence of 10 mM ascorbate. Conditions as Fig. 2A except time constant, 0.3 s; gain $10^4$. (B) as (A) after illumination at 15 K. (C) Difference spectrum ((B) — (A)) × 3.**

with a midpoint redox potential of −250 mV [1,3] or low potential cytochrome *b*-559 (cytochrome $b$-$559_{LP}$). The latter has an EPR signal near *g* 3.0 [6] whilst EPR characterisation of cytochrome *c*-549 has not been made. Cytochrome *b*-563 has a redox potential below that of cytochrome $b$-$559_{LP}$ [14] but exhibits properties of a high spin cytochrome with an EPR signal near *g* 6.0 [7,15]. The assignment of the *g* 3.1 signal to either cytochrome $b$-$559_{LP}$ or cytochrome *c*-549 could not be made as the presence of lauryldimethylamine oxide prevented normal redox titrations over the required potential range.

After illumination of the sample in Fig. 4A at 15 K the spectrum in Fig. 4B was obtained and the irreversible light induced signal is shown in the difference spectrum (B) − (A), Fig. 4C. Oxidation of the sample with 2,3-dichloro-5,6-dicyanoquinone prior to freezing prevented the low-temperature photooxidation and induced the fully oxidised spectrum. The chemically oxidised signal was larger than shown in the spectrum in Fig. 4 and inferred that approximately one third of the reduced cytochrome was photooxidised at cryogenic temperatures in these samples.

The low temperature light induced signal was largest in particles frozen without additions and represented over half of the chemically oxidised signal. Cytochrome *f* with an EPR signal near *g* 3.5 [6,15] was not detected.

The photooxidised signal does not decay if the sample is stored at 77 K in the dark for several days and is also absent or small in samples frozen under illumination although the total signal present is smaller than that of the corresponding sample frozen dark and illuminated at low temperature. This indicates that the photooxidation is blocked in this sample although room temperature illumination may have changed the part of the signal not involved in low temperature photochemistry.

Oxidation of the particles with benzoquinone or reduction with hydroquinone decreased but did not prevent the photoinduced signal increase at low temperature. We therefore attribute the signal in Fig. 4C to the photooxidation of cytochrome $b$-$559_{HP}$. This species is enriched in the particles [3] and its slow decay to lower potential forms may account for the smaller signal in the hydroquinone reduced sample. Slight variations in the *g*-value of cytochrome *b*-559 signals around *g* 3.0 [6,7] have suggested that in chloroplasts and subchloroplast particles the *g*-value is indicative of the particular cytochrome and that this may be used in identification. However other factors such as protonation, freezing and perhaps lauryldimethylamine oxide which may affect the haem environment could shift *g*-values. The *g*-value seen here of 3.1 is similar to that found in chloroplast and subchloroplast particles [6,7] for the low temperature photooxidisable signal attributed to cytochrome $b$-$559_{HP}$. Chemically oxidised signals attributed to cytochrome $b$-$559_{LP}$ had slightly lower *g*-values of 2.9 to 3.0. Cytochrome $b$-$559_{LP}$ present in the particles may be represented by the high field tail of the peak in Fig. 4 not present in the light induced signal.

*g 2.0 radicals*

Fig. 5A shows the spectrum of the free radical species at *g* 2.00 in dark adapted oxygen evolving particles at 16 K. The lineshape is characteristic of Signal II. When particles were frozen after dark adaption in the presence of

5 mM ferricyanide the spectrum in Fig. 5B was obtained. The radical has increased in size due to the oxidation of P-700 which blocks Photosystem I. The P-700$^+$ contribution to the spectrum is much smaller than that seen in untreated membranes allowing the outer features of Signal II to be observed. After illumination at 16 K of the ferricyanide oxidised sample the radical increases irreversibly, Fig. 5C. The signals photoinduced are shown in the difference spectrum (C) — (B), Fig. 5D. There appear to be two components, a Signal II species plus a narrower 8—10 gauss wide radical. The latter signal may be attributed to a chlorophyll radical as found in [16] and later demonstrated to be a secondary donor to P-680$^+$ at high redox potentials [17—19]. Careful subtraction of the Signal II contribution to the photoinduced signal suggests

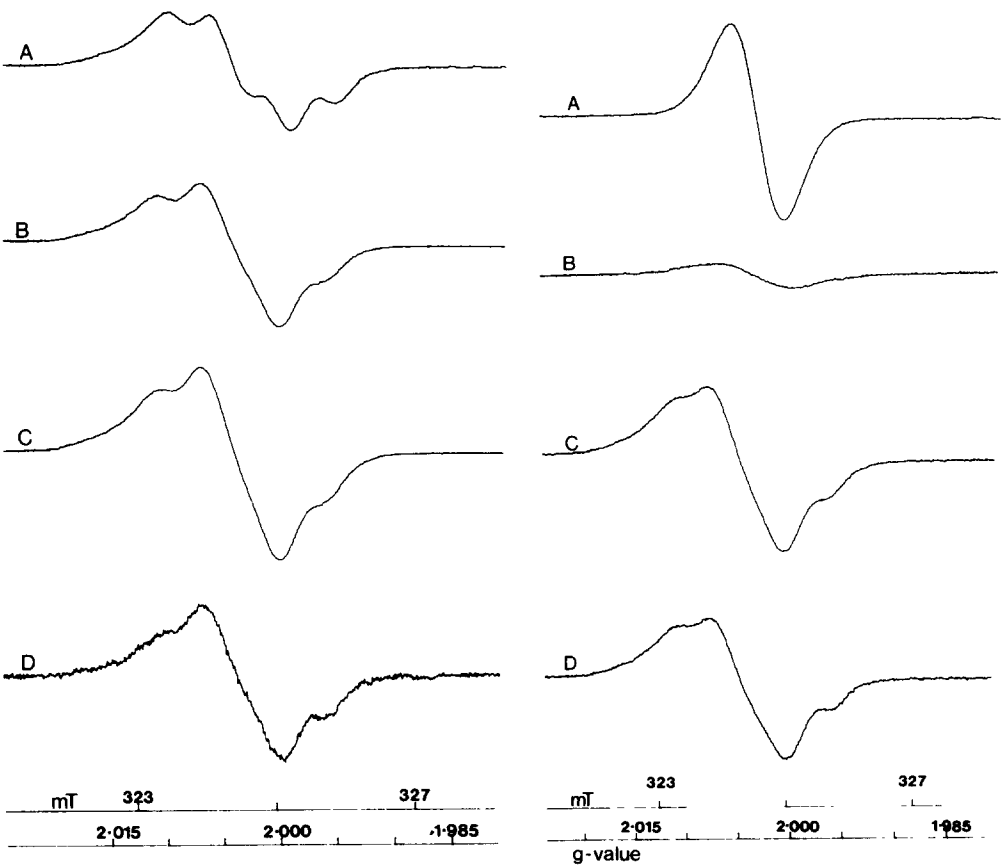


Fig. 5. (A) EPR spectrum of dark-adapted oxygen-evolving particles at 16 K. (B) Frozen after dark adaption in the presence of 5 mM ferricyanide. (C) After illumination of (B) at 16 K. (D) Difference spectrum ((C) — (B)) × 3. EPR conditions as Fig. 1 except: microwave power 100 μW; scan rate 5 mT/min; modulation amplitude 0.1 mT; temperature 16 K; gain (A) 500, (B) and (C) 250.

Fig. 6. (A) EPR spectrum of the Photosystem I enriched pellet from *P. laminosum* after illumination at 16 K. Instrument gain 250, chlorophyll *a* concentration 1.7 mg/ml. (B) Oxygen-evolving particles frozen after dark adaptation in the presence of 10 mM ascorbate. Instrument gain 500. (C) After illumination of (B) at 16 K. Instrument gain 500. (D) Difference spectrum ((C) — (B)). EPR conditions as in Fig. 5.

that the linewidth may be broader than that seen in Refs. 16—19 and contains a contribution from another component.

Fig. 6A shows the spectrum obtained by illuminating a Photosystem I enriched membrane preparation of *P. laminosum* which had been frozen after dark adaptation in the presence of ascorbate. The spectrum demonstrates the lineshape of P-700$^+$. Fig. 6B shows the spectrum of a sample of oxygen evolving particles frozen in the dark after reduction with ascorbate. The Signal II seen in Fig. 5 has been reduced. After illumination at 16 K, Fig. 6C, P-700$^+$ and a large Signal II component have been photoinduced. These are shown in the difference spectrum, Fig. 6D. The decreased Photosystem I content of the particles and therefore decreased P-700$^+$ signal size allows the observation of the Signal II increase.

When the sample shown in Fig. 6C was stored at 77 K in the dark for several days, the signals decayed almost to the original state. Illumination at 16 K then restored the signals shown in Fig. 6D but with an increased Signal II contribution. This effect has also been observed in chloroplasts from a barley mutant lacking Photosystem I [20].

Oxygen-evolving particles frozen under illumination and then stored in the dark at 77 K overnight also show the photoinduced Signal II termed Signal $II_{lt}$ shown in Ref. 5. Addition of 50 $\mu$M DCMU, to the particles prior to treatment reduced all signal sizes but did not prevent Signal $II_{lt}$ photooxidation. The reasons for this are unclear.

## Discussion

The results confirm the Photosystem II enrichment of the oxygen-evolving particles in that the low temperature light induced P-700$^+$ and iron-sulphur centres A and B are present but by comparison with unfractionated membranes are much smaller in relation to Photosystem II components such as Signal II and cytochrome $b$-559$_{HP}$. The presence of two further EPR signals, the low potential cytochrome signal near $g$ 3.0 and the iron-sulphur centre near $g$ 1.92 are probably a reflection of the impurity of the preparation. However these particles still represent a significant advance on previous oxygen evolving preparations in allowing improved observation of cytochrome $b$-559$_{HP}$ and Signal II photoreactions due to both the Photosystem II enrichment and the partial removal of signals from Photosystem I. The Photosystem I present in the particles has properties similar to those discussed in Ref. 12.

The low spin cytochrome signal near $g$ 3.0 produced by illumination at cryogenic temperatures and attributed to cytochrome $b$-559$_{HP}$ appears to reflect the oxidation of the higher potential form of this cytochrome. A recent study [14] has confirmed the presence of two distinct cytochrome $b$-559 species and species with intermediate redox potentials thought to be modified cytochrome $b$-559$_{HP}$. The photooxidised signal seen in oxygen-evolving particles at low temperature agrees with previous EPR studies using chloroplasts and subchloroplast particles [6,7] in showing that native or modified cytochrome $b$-559$_{HP}$ functions as a low temperature donor to the Photosystem II reaction centre and confirms earlier optical experiments [21—24]. The normal form of this cytochrome donor appears to be the high potential species although a species with

lower potential can function at cryogenic temperatures. The lowering of the redox potential of cytochrome $b$-$559_{HP}$ accompanying loss of oxygen evolution by a variety of treatments does not affect the photooxidation at cryogenic temperatures but may prevent the normal pathway of reduction occurring at physiological temperatures.

Signal II has been associated with Photosystem II for many years but it has only been since the discovery of fast transient components [25—27] of this signal at room temperature that a role as a donor to the Photosystem II reaction centre has been assigned. The discovery in recent years of EPR and electron spin echo components of Signal II photooxidised by illumination at cryogenic temperatures [5,28] have confirmed this role. Indeed the low temperature species probably reflect the same components as the room temperature transients. In these experiments we have been able to observe photooxidation of Signal II in dark adapted samples due to the decreased size of the P-700$^+$ radical which obscures Signal II changes in chloroplasts or unfractionated algal membranes. The decay of the light induced $g$ 2.0 radical signals on dark storage at 77 K contrasts with the behaviour of cytochrome $b$-$559_{HP}$ for which the photooxidation at low temperature is irreversible. This suggested that the species giving rise to the Signal II photooxidised at cryogenic temperatures is more closely coupled to the reaction centre than is cytochrome $b$-$559_{HP}$ and confirms our suggestion that Signal $II_{lt}$ reflects the oxidation of an immediate donor to P-680.

The species giving rise to Signal II is though to be a semiquinone or plastochromanoxyl radical or a mixture of the two [28]. It is clear from our studies and those of previous workers that several types of Signal II exist and this raises the question of where they fit into Photosystem II. One possibility is that they represent parts of the pool of quinone between the photosystems perhaps acting as a deactivating mechanism for the oxygen evolving system. A second closely related possibility is that they may represent part of the oxygen evolution system itself. They have high redox potentials [29] and are close to the reaction centre as demonstrated here. The unusual shape of Signal II components may be influenced by the manganese postulated to be involved in water splitting as previous studies have indicated a close relationship between manganese and Signal II [27,30]. The fact that Signal $II_{vf}$ changes to Signal $II_f$ [25—27] when oxygen evolution is lost and also that low temperature photooxidisable forms of Signal II occur, may reflect partial charge accumulation in the oxygen evolving complex when water oxidation is prevented.

These experiments show that the oxygen evolving particles from *P. laminosum* are sufficiently pure to allow direct identification of Photosystem II EPR signals and to identify the role of Signal $II_{lt}$ and cytochrome $b$-559 as donors to the reaction centre. Further work with this preparation will enable us to identify the role of these components in water oxidation.

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## References


1 Stewart, A.C. and Bendall, D.S. (1980) Biochem. J. 188, 351—361
2 Stewart, A.C. and Bendall, D.S. (1979) FEBS Lett. 107, 308—321
3 Stewart, A.C. and Bendall, D.S. (1981) Biochem. J., submitted
4 Hoff, A.J. (1979) Phys. Rep. 54, 75—200
5 Nugent, J.H.A. and Evans, M.C.W. (1979) FEBS Lett. 101, 101—104
6 Malkin, R. and Vänngard, T. (1980) FEBS Lett. 111, 228—231
7 Nugent, J.H.A. and Evans, M.C.W. (1980) FEBS Lett. 112, 1—4
8 Arnon, D.I., McSwain, B.D., Tsujimoto, H.Y. and Wade, K. (1974) Biochim. Biophys. Acta 357, 231—245
9 Cammack, R., Luijk, L.J., Maquire, J.J., Fry, I.Y. and Packer, L. (1979) Biochim. Biophys. Acta 548, 267—275
10 Nishi, N.N., Hoff, A.J. and Van der Waals, J.H. (1980) Biochim. Biophys. Acta 590, 74—88
11 Malkin, R. and Bearden, A.J. (1978) Biochim. Biophys. Acta 505, 147—181
12 Cammack, R., Ryan, M.D. and Stewart, A.C. (1979) FEBS Lett. 107, 422—426
13 Nugent, J.H.A., Møller, B.L. and Evans, M.C.W. (1981) Biochim. Biophys. Acta 634, 249—255
14 Rich, P.R. and Bendall, D.S. (1980) Biochim. Biophys. Acta 591, 153—161
15 Rich, P.R., Heathcote, P.H., Evans, M.C.W. and Bendall, D.S. (1980) FEBS Lett. 116, 51—56
16 Malkin, R. and Bearden, A.J. (1973) Proc. Natl. Acad. Sci. U.S.A. 70, 294—297
17 Lozier, R.H. and Butler, W.L. (1974) Biochim. Biophys. Acta 333, 465—480
18 Malkin, R. and Bearden, A.J. (1975) Biochim. Biophys. Acta 396, 250—259
19 Visser, J.W.M., Rijyersberg, C.P. and Gast, P. (1977) Biochim. Biophys. Acta 460, 36—46
20 Nugent, J.H.A., Møller, B.L. and Evans, M.C.W. (1980) FEBS Lett. 121, 355—357
21 Knaff, D.B. and Arnon, D.I. (1969) Proc. Natl. Acad. Sci. U.S.A. 63, 963—969
22 Bendall, D.S. and Sofrová, D. (1971) Biochim. Biophys. Acta 234, 371—380
23 Erixon, K. and Butler, W.L. (1971) Biochim. Biophys. Acta 234, 381—389
24 Erixon, K. and Butler, W.L. (1971) Photochem. Photobiol. 14, 427—433
25 Babcock, G.T. and Sauer, K. (1975) Biochim. Biophys. Acta 376, 315—328
26 Babcock, G.T. and Sauer, K. (1975) Biochim. Biophys. Acta 376, 329—344
27 Warden, J.T., Blankenship, R.E. and Sauer, K. (1976) Biochim. Biophys. Acta 423, 462—476
28 Nishi, N.N., Hoff, A.H., Schmidt, J. and Van der Waals, J.H. (1978) Chem. Phys. Lett. 58, 164—170
29 Velthuys, B.R. and Visser, J.W.M. (1975) FEBS Lett. 55, 109—112
30 Weaver, E.C. and Bishop, N.I. (1963) Science 140, 1095—1097