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# Redox control of gene expression and the function of chloroplast genomes – an hypothesis

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**Abstract**

Two-component regulatory systems that respond to changes in redox potential have recently been discovered in bacteria. 'Redox sensors' are defined as electron carriers which initiate control of gene expression upon oxidation or reduction. 'Redox response regulators' are defined as DNA-binding proteins which modify gene expression as a result of the action of redox sensors. Redox sensors and redox response regulators may comprise a mechanism for feedback control of redox potential in photosynthetic electron transport chains, thereby protecting plants, algae and photosynthetic bacteria from damage caused by electrochemistry operating on inappropriate electron donors and acceptors. Chloroplast redox sensors and redox response regulators, themselves encoded in the nucleus, may place chloroplast gene expression under redox regulatory control. This may account for the persistence, in evolution, of chloroplast genomes, and for the constancy of the sub-set of chloroplast proteins encoded and synthesised in situ. These and other predictions are discussed.

## Introduction

Bacterial two-component regulatory systems control gene expression by means of environmental sensors and response regulators (Ronson et al. 1987, Stock et al. 1989). The sensor is usually a membrane protein and becomes phosphorylated in response to the environmental signal. Its substrate, the response regulator, is a DNA-binding protein which initiates transcription of a specific gene or genes by interacting, in its phosphorylated form, with an RNA polymerase. Sensors undergo histidine phosphorylation at a conserved site, and response regulators are phosphorylated on aspartate by a phosphotransferse or kinase (Stock et al. 1989).

The Arc (aerobic respirtory control) system of *Escherichia coli* is a two-component regulatory system which responds to redox potential. Mutations in two genes, *arc*A and *arc*B, increase the anaerobic expresion of a number of genes normally repressed by anoxyia (Iuchi et al. 1989, Guest et al. 1990). ArcB is a protein kinase which contains two membrane-spanning helices flanking a cytoplasmically-exposed loop, the latter containing a histidine which is the site of autophosphorylation. Autophosphorylation occurs in response to decreased redox potential, rather than in response to decreased oxygen concentration per se. (Iuchi et al. 1990a, Iuchi et al. 1990b). ArcA is soluble protein which is phosphorylated by ArcB (Iuchi and Lin 1988) and which contains the helix-turn-helix motif of DNA-binding proteins. I suggest that the terms 'redox sensor' and 'redox response regulator' are appropriate descriptions of ArcB and ArcA, respectively.

In the purple non-sulphur bacterium *Rhodo-*

*bacter capsulatus*, the opposing effects of light and oxygen on synthesis and assembly of photosynthetic reaction centre and light-harvesting complexes are inhibited by mutations in a single regulatory DNA sequence in the promoter region of the *puf* operon (Klug et al. 1991). The regulatory gene product has been termed PPBP, for *puf* promoter binding protein (Taremi and Marrs 1990), and the gene has been termed *reg*A (Sganga and Bauer 1992). Like ArcA, RegA shows the helix-turn-helix motif of DNA-binding proteins and sequence similarities with other response regulators. Thus, RegA is likely to be a redox response regulator in the sense defined here.

The *E. coli* FNR protein (regulating fumarate and nitrate reductases) is a helix-turn-helix DNA-binding protein with an amino-terminal segment containing cysteines that ligate a redox-active iron atom (Guest et al. 1990, Unden et al. 1990). Reduction of the iron from $Fe^{III}$ to $Fe^{II}$ is thought to cause a protein structural change which regulates transcription. From effects of poising the growth medium at different potentials on expression of a reporter gene for *frd* (encoding fumarate reductase), the standard mid-point potential ($Em_7$) of the FNR response has been estimated at +400 mV (Unden et al. 1990).

The oxygen sensor controlling expression of nitrogen-fixation genes in *Rhizobium meliloti* is a membrane haemoprotein with protein kinase and phosphotransferase activity, termed FixL (Gilles-Gonzalez et al. 1991). FixL catalyzes its own phosphorylation, and is a protein kinase for FixJ, the oxygen response regulator. Thus, certain redox sensors may also be haemoproteins, haem redox sensors in general standing in relation to haem oxygen sensors as cytochromes do to haemoglobins and myoglobins.

I suggest that FNR-type redox control of gene expression could be termed high-potential redox

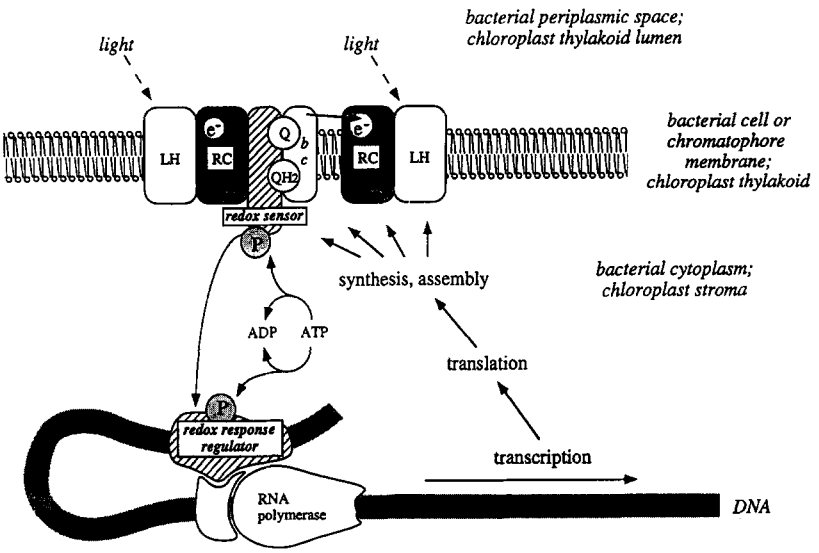


*Fig. 1.* Two-component redox regulation of transcription in photosynthetic systems. Redox sensors are redox-active components of complexes of photosynthetic electron transport chains. Redox sensors may respond to altered redox potential by autophosphorylation, which in turn causes phosphorylation of redox response regulators. Redox response regulators are sequence-specific DNA-binding proteins whose phosphorylation is required for binding of RNA polymerase and initiation of transcription at promoters of genes whose expression is thereby made subject to control by redox potential. Redox sensors and redox response regulators are shown as diagonally cross-hatched shapes. The photosynthetic membrane is a diagrammatic composite, including a cytochrome *b*/*c* complex, and photosynthetic light-harvesting (LH) and reaction centre (RC) complexes. The redox sensor shown interacting with its response regulator is a medium potential redox sensor, associated with the cytochrome *b*/*c* complex. Redox sensors and response regulators are shown as separate components, but they may be combined in a single polypeptide. Some two-component systems have sensor and response regulator domains in a single polypeptide, for example VirA/VirG of *Agrobacterium* (Stock et al. 1989). Single redox sensor-response regulator polypeptides may therefore provide points of attachment of DNA to photosynthetic membranes.

control, and the sensors termed Fe-redox sensors. Two-component redox control may operate at lower redox potentials, according to the prosthetic groups concerned. Haem, quinone, flavin, and iron–sulphur centres might each serve as the redox-active groups of different redox sensors.

Figure 1 shows a general scheme for the operation of redox sensors and redox regulators in photosynthetic systems.

## Redox regulation and chloroplast genomes

Chloroplast genomes encode a small but remarkably constant subset of chloroplast proteins. The endosymbiotic origin of chloroplasts (Whatley et al. 1979, Cavalier-Smith 1981, Margulis 1981, Cavalier-Smith 1987a,b) requires that the majority of symbiont genes have been transferred to the nucleus of the host. What do proteins encoded by chloroplast DNA have in common that confers an evolutionary advantage to location of their genes in situ?

Here I suggest a possible solution to this problem. It is assumed that any gene can be copied between chloroplast and nucleus, or between an endosymbiont and the nucleus of its host. After the addition of the appropriate presequences to nuclear copies of endosymbiont genes, intra-cellular selection may arise for each gene concerned. In the majority of cases, expression of the nuclear copy is sufficient for synthesis of the corresponding protein, and the chloroplast copy becomes redundant and is lost. In a minority of cases, however, some distinctive requirement cannot be satisfied by the nuclear-encoded copy, even granted a targeting, import and processing machinery that gave a mature protein identical to that synthesised in the organelle. I propose control by redox sensors and redox response regulators as the distinctive requirement favouring survival of the chloroplast-encoded copy, as depicted in Fig. 2. If this is correct, then the distinctive common feature of chloroplast-encoded proteins is that they directly determine redox poise, and function safely together only within ranges of redox potential that are liable to be exceeded by environmental change. The complete nucleotide sequences of some chloroplast genomes are known (Ohyama et al. 1986, Shinozaki et al. 1986, Hiratsuka et al. 1989), and it is therefore possible to test this prediction. Major chloroplast-encoded proteins are listed in Table 1.

## Redox roles of chloroplast-encoded proteins

Chloroplast gene products (Ohyama et al. 1986, Shinozaki et al. 1986, Hiratsuka et al. 1989) include proteins whose functions directly link the physical environment to electron transport and redox poise (Table 1). In particular, photosynthetic electron transport chains contain chloroplast-encoded proteins at their extremes of Em (Table 1). Chloroplast-encoded proteins include all those located at the bioenergetic or assimilatory interfaces of photosynthetic cells. In contrast, nuclear-encoded chloroplast components do not take such key roles, even where they function in light-harvesting or electron transport.

The requirement for a core of components subject to redox control in situ may disappear when chloroplasts cease to perform their primary bioenergetic role of photosynthesis, as in parasitic plants (Morden et al. 1991).

The reaction centre core proteins of both Photosystem I and Photosystem II are chloroplast-encoded. Photosystem II is highly susceptible to a light-induced damage – photoinhibition – which occurs when electron transport is restricted on either acceptor or donor sides, or both (Prásil et al. 1992). Breakdown of the D1 protein of the Photosystem II reaction centre occurs during photoinhibition (Prásil et al. 1992) The *psb*A genes encoding D1 (Erickson and Rochaix 1992) are therefore obvious candidates for redox control of gene expression. The loss of chloroplast *psb*A genes to the nucleus would tend release their expression from control by transient changes in light intensity and redox potential.

The chloroplast-encoded reaction centre polypeptides of PS I (Scheller and Møller 1990) may be subject to low-potential redox control, perhaps by Fe–S sensors and specific response regulators operating at the promoter region of *psa*A and *psa*B genes. However, both Photosystem I and chloroplast-encoded components of

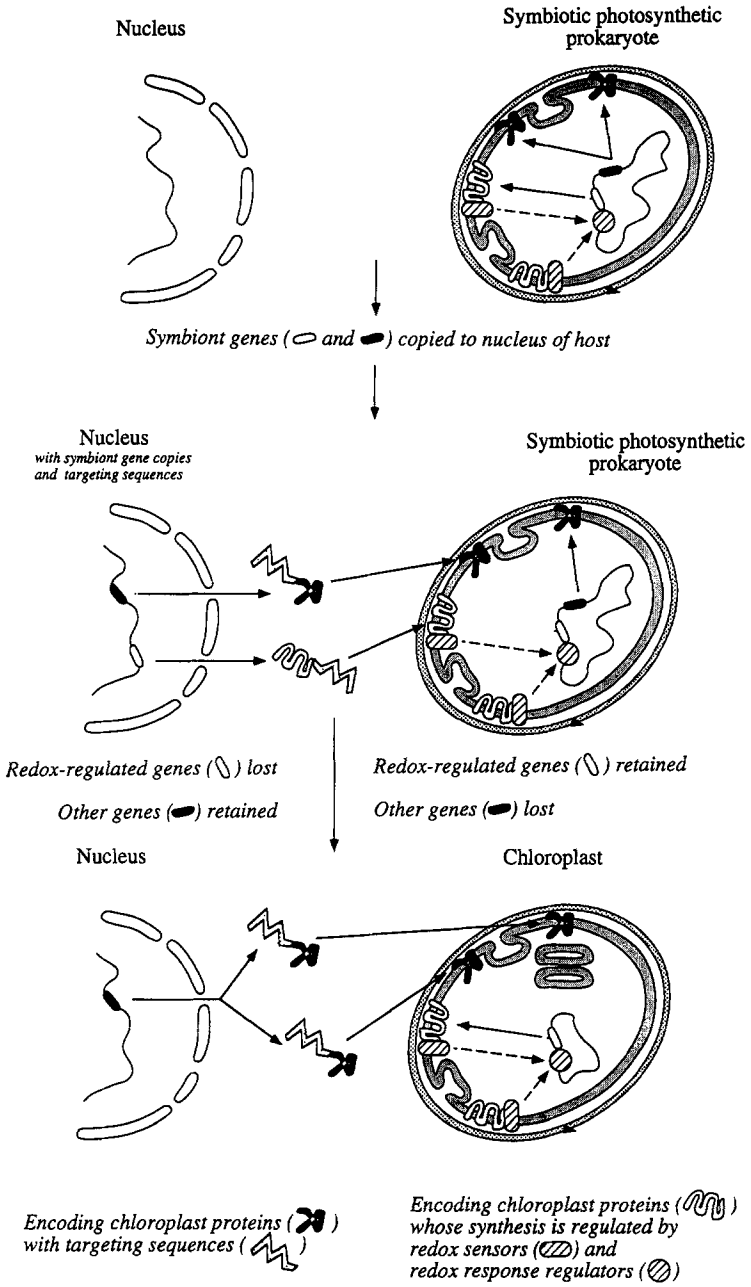


*Fig. 2.* The proposed sequence of events leading to distribution of copies of the symbiont's genes between the nucleus of the host cell and the chloroplast to which the symbiont gives rise (upper diagram).

Any and all genes in the symbiont genome are copied to the nucleus of the host cell, and there sequences are added which enable the protein to return, as a precursor, to the symbiont. Hence, any symbiont protein may be synthesised both in situ and, in precursor form, in the cytoplasm of the host (middle diagram).

For the majority of symbiont-encoded proteins, nuclear encoding, cytoplasmic synthesis and import and processing of precursors together provide an effective alternative to synthesis in situ, and the symbiont copies of these genes are lost. However, transcription of a minority of symbiont genes must be closely coupled to redox potential at specific sites in the photosynthetic membrane. For these genes, the nuclear copy continues to be expressed in inappropriate environments, and the nuclear gene product continues to be synthesised even when its function is damaging to the cell. The symbiont gene copy remains under redox control, and therefore has a selective advantage. The nuclear gene copy is therefore lost, and a stable division of genetic responsibility is reached in which the chloroplast retains a genome encoding those of its proteins whose synthesis must respond rapidly to alteration in the physical environment of the cell (lower diagram).

*Table 1.* Major chloroplast genes and gene products (Ohyama et al. 1986, Shinozaki et al. 1986, Hiratsuka et al. 1989) and their functions. $Em_7$ values are taken from Cramer and Knaff (1990)

| Gene | Protein | Standard redox potential, $Em_7$ (mV) | Substrate |
|---|---|---|---|
| *Photosystem II* | | | |
| *psb*A | D1 of PS II reaction centre | +1170 ($P^+/P$)<br>−800 ($P/P^*$) | Light |
| *psb*D | D2 of PS II reaction centre | +1170 ($P^+/P$)<br>−800 ($P/P^*$) | Light |
| *psb*B, *psb*C | CP47 and CP43 of PS II | - | Light |
| *Energy coupling* | | | |
| *pet*A | Cytochrome *f* | +350 | |
| *pet*B | Cytochrome $b_6$ | −50 | |
| *atp*A, B, E | ATP synthase $CF_1\alpha,\beta,\varepsilon$ | | |
| *atp*F, H, I | ATP synthase $CF_0$I, III, IV | | |
| *Photosystem I* | | | |
| *psa*A | A1 of PS I reaction centre | −1250 ($P/P^*$)<br>+500 ($P^+/P$) | Light |
| *psa*B | A2 of PS I reaction centre | −1250 ($P/P^*$)<br>+500 ($P^+/P$) | Light |
| *Respiration: cyclic electron transport* | | | |
| *ndh*A–F | NADH dehydrogenase subunits 1–5 | −324 ($NADH/NADP^+$) | |
| *$CO_2$ fixation* | | | |
| *rbc*L | Rubisco large subunit | - | Carbon dioxide |

the intermediary electron transport chain such as cyt *f* and cyt $b_6$ are connected in series with Photosystem II, and they therefore play a role in determining the redox potential of Photosystem II, as may the $CF_1$–$CF_0$ coupling ATP synthase, indirectly through photosynthetic control.

Chloroplast genomes also encode NADH dehydrogenases (Ohyama et al. 1986, Shinozaki et al. 1986, Matsubayashi et al. 1987, Hiratsuka et al. 1989). *ndh* genes are expresed in mature chloroplasts (Matsubayashi et al. 1987), where they may function in chlororespiration or as links between the acceptor side of Photosystem I and the cyt $b_6/f$ complex in cyclic photophosphorylation, as demonstrated recently in a cyanobacterium (Mi et al. 1992). The importance of redox poise for cyclic photophosphorylation can be demonstrated by addition of catalytic concentrations of oxidant or reductant to overcome inhibition by overreduction or overoxidation in chloroplasts (Whatley 1963, Grant and Whatley 1967, Arnon and Chain 1975, Ziem-Hanck and Heber 1980, Allen 1983) and photosynthetic bacteria (McEwan et al. 1985). Redox control of expression of chloroplast *ndh* genes may therefore be required for maintaining photophosphorylation. In *E. coli*, transcription of *ndh*

genes is under the control of the high-potential redox regulator, FNR (Spiro et al. 1989).

The chloroplast-encoded Rubisco large subunit does not itself participate in electron transfer. However, the primary carboxylation step of $CO_2$ assimilation may, under a variety of circumstances, become the limiting factor for consumption of electrons from the photosynthetic chain by triose phosphate dehydrogenase (Walker et al. 1986). The activity of the Rubisco large subunit may therefore have immediate effects on the redox potential of components of the photosynthetic electron transport chain, including, for example, D1.

The hypothesis that a requirement for redox regulation determines the location of chloroplast genes does not obviously apply to the 'energy coupling' genes listed in Table 1, nor to the chloroplast-encoded components of the chloroplast genetic system itself. For example, it is difficult to see how redox regulation might account for the chloroplast location of cytochrome *f* and $b_6$ genes but the nuclear location of Rieske iron–sulphur protein and plastocyanin genes. If the hypothesis applies equally to these components, it suggests that cytochromes could have a uniquely deleterious effect when compared with other electron carriers operating at intermediate potentials, but there seems to be no evidence for or against this proposal. Alternatively, other factors may play a role in determining the location of these genes. Although these examples are neutral for the hypothesis, there seems to be no exception to the chloroplast location of genes for proteins operating at the extremes of redox potential. The nuclear location of ferredoxin and water-oxidation polypeptides fits with the hypothesis, since these proteins are involved in secondary electron transfer with chloroplast-encoded reaction-centre components at much lower and higher potentials.

## Chloroplast redox sensors and response regulators may themselves be encoded in the nucleus

Genes encoding chloroplast redox sensors and redox response regulators need not themselves be organelle-encoded, since there is no obvious obstacle to their expression in the nucleus for cytosolic translation and uptake, in precursor form, by the organelle. It is possible that a nuclear location for chloroplast redox sensors and response regulators is advantageous, allowing them to be made subject to additional control (however, see Cavalier-Smith 1987b).

## Predictions

In photosynthetic prokaryotes, further redox sensors may be found whose redox properties and prosthetic groups permit their operation at specific points in photosynthetic electron transport chains. Potentiometric redox titration of expression of reporter genes (Unden et al. 1990) may reveal structural genes under control of redox sensors at different potentials. Redox control of translation or of messenger processing and stability may also be important.

Cyanobacteria exhibit redox control of phosphorylation of a number of polypeptides (Harrison et al. 1991), and these are candidates for redox sensors. Redox control of protein phosphorylation is also a property of a range of polypeptides in purple non-sulphur bacteria (Holmes and Allen 1988).

Redox control of response at the post-translational level is well established for chloroplasts (Allen 1992). The best-characterised substrate is the nuclear-encoded light-harvesting complex, LHC II. Phosphorylation of LHC II is regulated by a protein kinase whose activity has an $Em_7$ of approximately +80 mV, becoming activated as plastoquinone is reduced (Allen et al. 1981). A number of other chloroplast polypeptides are phosphorylated by a redox-controlled kinase or kinases (Allen 1992). The redox-sensitive LHC II kinase is a nuclear-encoded protein of 64 kDa in the mature form (Gal et al. 1990, Gal et al. 1992). It is autophosphorylated in a reaction that is also under redox control. Plastoquinone-redox control of chloroplast *psa* and *psb* gene expression may maintain balanced utilization of light energy by Photosystem I and Photosystem II (Allen 1992). The LHC II kinase is therefore a candidate for a medium-potential, quinone redox sensor.

The chloroplast genome has promoter regions with similarities to those encountered in gram-negative bacteria, including cyanobacteria, indicating the possibility of control of initiation of transcription by regulator proteins of bacterial type. In particular, '-10' and '-35' sequences resembling *E. coli* regulatory sequences are found in the chloroplast genome, upstream of the presumed sites of initiation of transcription of *psb*A, *atp*B, *atp*E and *rbc*L (Table 1) (Ohyama et al. 1986, Shinozaki et al. 1986, Hiratsuka et al. 1989).

In chloroplasts and cyanobacteria, many membrane proteins are phosphorylated and redox-active, but have no obvious role in electron transport (Allen 1992). Indeed, the ideal redox sensor might have no direct role in linear electron flow, existing instead as a side-arm of the chain. The Em of a redox sensor may be different from that of some of the proteins whose synthesis it controls, as seen in FNR ($Em_7 = +400$ mV) control of transcription of *ndh* genes (Spiro et al. 1989) encoding NADH dehydrogenase ($NADH/NAD^+$ $Em_7 = -324$ mV).

The hypothesis of redox sensors and response regulators in the function and maintenance of chloroplast genomes may help to explain the distribution of genes between the nucleus and chloroplasts in plants and algae. A major prediction of this hypothesis is the existence in chloroplasts of a range of redox-active sensors and DNA-binding redox response regulators, all encoded in the nucleus.

At present there are no data by which this hypothesis can be accepted or rejected, though this is an objective of current and planned experiments. The hypothesis may also be applicable to the evolution and function of mitochondrial genomes. The aim of this paper is to make the hypothesis, as it applies to photosynthetic systems, available for critical test in a range of experimental systems.

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