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## Interactions of Herbicides with Photosynthetic Electron Transport<sup>1</sup>

E. PATRICK FUERST and MICHAEL A. NORMAN<sup>2</sup>

**Abstract.** The two primary sites of herbicide action in photosynthetic electron transport are the inhibition of photosystem II (PS II) electron transport and diversion of electron flow through photosystem I (PS I). PS II electron transport inhibitors bind to the D1 protein of the PS II reaction center, thus blocking electron transfer to plastoquinone. Inhibition of PS II electron transport prevents the conversion of absorbed light energy into electrochemical energy and results in the production of triplet chlorophyll and singlet oxygen which induce the peroxidation of membrane lipids. PS I electron acceptors probably accept electrons from the iron-sulfur protein,  $F_a/F_b$ . The free radical form of the herbicide leads to the production of hydroxyl radicals which cause the peroxidation of lipids. Herbicide-induced lipid peroxidation destroys membrane integrity, leading to cellular disorganization and phytotoxicity.

*Additional index words:* Herbicide resistance.

### INTRODUCTION

In vitro studies have suggested that herbicides that inhibit or modify photosynthesis can be classified as: a) electron transport inhibitors, b) uncouplers, c) energy transfer inhibitors, d) inhibitory uncouplers, or e) electron acceptors (26). Carbon dioxide fixation and assimilation is not a primary site of action of herbicides. Only two biochemical mechanisms have clearly been demonstrated to be of primary importance in herbicidal action in photosynthesis: inhibition of photosystem II (PS II)<sup>3</sup> electron transport and diversion of electron transfer through photosystem I (PS I)<sup>3</sup>. The purpose of this review is to summarize the mechanisms of action of these herbicides. The components of photosynthetic electron transport, the interactions of herbicides with this process, and the secondary reactions which lead to phytotoxicity will be reviewed.

### PHOTOSYNTHETIC ELECTRON TRANSPORT

Photosynthetic electron transport occurs in chloroplast thylakoid membranes (lamellae). Thylakoid membranes are

either stacked (referred to as appressed or grana lamellae) or unstacked (referred to as stroma lamellae). Thylakoid membranes contain four membrane-spanning protein complexes: the PS II complex, the cytochrome  $b_6/f$  complex, the PS I complex, and the ATP synthase complex (Figure 1). The PS II complex is primarily but not exclusively localized in the appressed lamellae, while the PS I and ATP synthase complexes are present only in the stroma lamellae. The ATP synthase complex converts the potential energy of the proton gradient, developed during electron transport, into high-energy phosphate bond energy in the form of ATP. The cytochrome  $b_6/f$  complex is distributed approximately equally between the appressed and unappressed membranes (2). The separation of PS II and PS I complexes is thought to optimize the relative amounts of light energy transferred to each reaction center (2). Photosynthetic electron transport involves all of these complexes except for the ATP synthase complex.

The electron carriers are cofactors that are bound to chloroplast proteins. The unique protein environment in which each cofactor is bound gives that carrier its unique functional role in electron transport. Thus, the proteins comprising the various complexes do not carry the electrons directly. The only exception to this is the tyrosine amino acid residue of the D1 protein that is an electron carrier in PS II (Figures 2 and 3) (40).

**PS II electron transport.** The PS II complex includes the oxygen evolving (water-splitting) complex, a reaction center complex, and the light-harvesting chlorophyll antenna proteins (Figure 1).

Purple photosynthetic bacteria possess a photosynthetic reaction center that has many similarities with PS II of plants including the inhibition of electron transport by triazine herbicides (24). This bacterial photosynthetic reaction center was the first membrane protein to be crystallized and structurally analyzed by X-ray crystallography. The Nobel Prize in chemistry was awarded to H. Michel, J. Deisenhofer, and R. Huber in 1988 for this work (21).

A model for the PS II reaction center complex is shown in Figure 3. This model is based on homologies between PS II and the bacterial photosynthetic reaction center (6, 24, 36). The light-harvesting chlorophyll molecules associated with PS II (Figure 1) transfer excitation energy to the PS II reaction center, a chlorophyll *a* dimer known as P680. When excitation energy is transferred to the chlorophyll *a* dimer, charge separation takes place, and the excited electron is transferred to pheophytin (Figure 3). An electron derived from the splitting of water neutralizes the residual positive charge of the chlorophyll *a* dimer. From pheophytin, the electron is transferred to  $Q_A$  and then  $Q_B$ ;  $Q_A$  and  $Q_B$  are plastoquinone molecules bound in special niches of the D2 and D1 proteins, respectively.  $Q_B$  accepts two electrons from  $Q_A$ , then accepts two protons from the stroma side of the

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<sup>3</sup>Abbreviations: PS II, photosystem II; PS I, photosystem I; <sup>3</sup>chl, triplet chlorophyll; <sup>1</sup>O<sub>2</sub>, singlet oxygen; FNR, ferredoxin:NADP<sup>+</sup> oxidoreductase; PQT<sup>++</sup>, paraquat divalent cation; PQT<sup>+</sup>, paraquat monovalent cation radical (reduced paraquat); O<sub>2</sub><sup>-</sup>, superoxide; SOD, superoxide dismutase; OH<sup>·</sup>, hydroxyl radical.

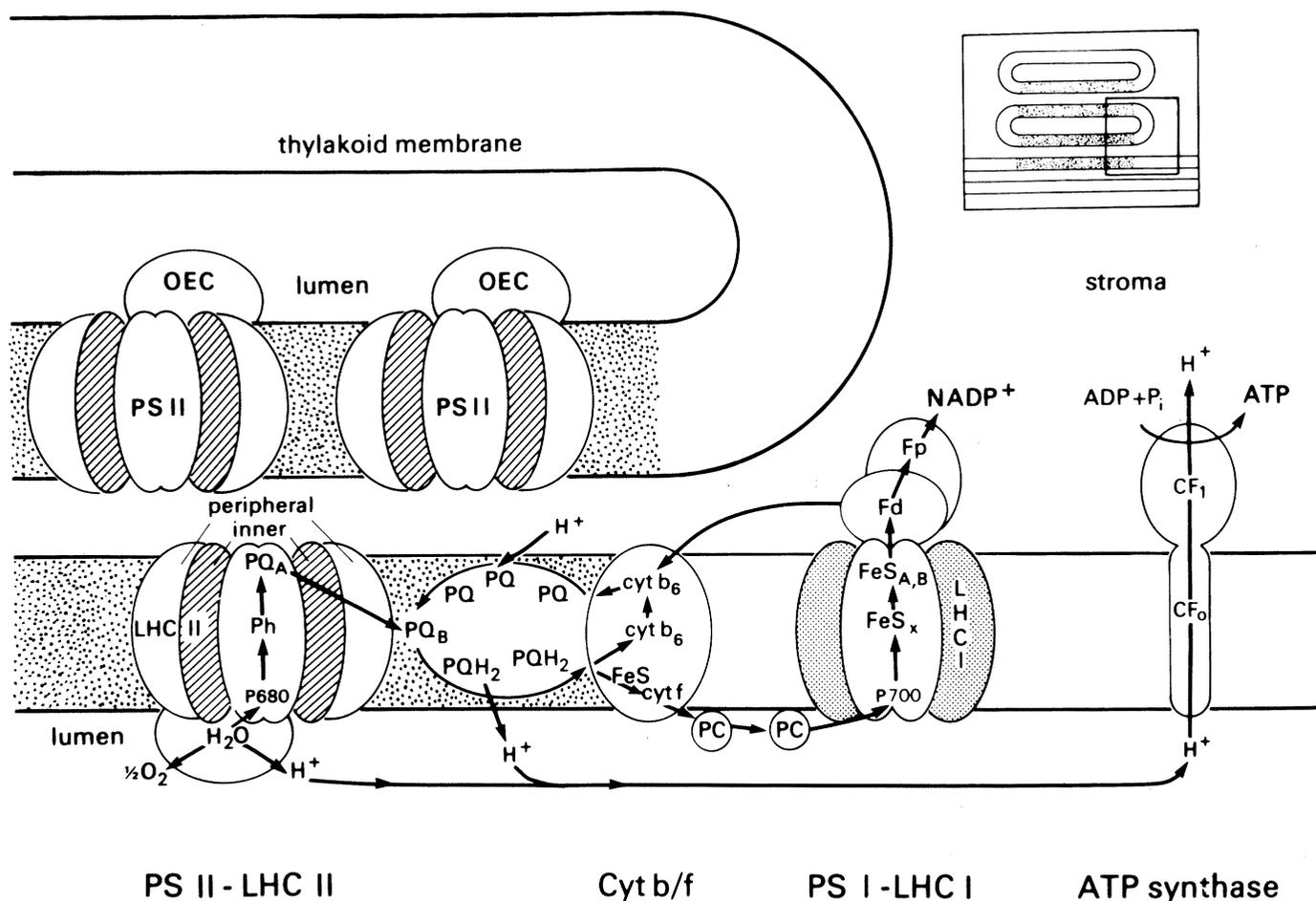


Figure 1. Organization of protein complexes in thylakoid membranes. LHC II and LHC I, light-harvesting chlorophyll proteins of PS II and PS I, respectively; OEC, oxygen-evolving complex. Reproduced from (2) with permission of publisher.

membrane, and then leaves its binding niche as plastoquinone (Figure 3). Another plastoquinone molecule then binds to the D1 protein, replacing the molecule that has left, and when bound, is called "Q<sub>B</sub>".

**Electron transport between PS II and PS I.** Plastoquinone donates its electrons to the cytochrome b<sub>6</sub>/f complex (Figure 1). The Q-cycle (not illustrated) is thought to utilize plastoquinone and the cytochrome b<sub>6</sub>/f complex to transport two protons across the membrane per electron utilized in linear electron transport from PS II to PS I (13). Plastocyanin accepts electrons from cytochrome f and shuttles the electrons along the lumen side of the thylakoid membrane to the PS I reaction center.

**PS I electron transport.** The PS I complex can be defined as the components of photosynthetic electron transport that catalyze the photoreduction of ferredoxin with plastocyanin as the electron donor (30). PS I is composed of a reaction center complex and light-harvesting chlorophyll antenna proteins which transfer absorbed light energy to the PS I reaction center, known as P700 (Figures 1 and 2). It is estimated that eight protein subunits are associated with the

PS I complex (29). Two 70 kilodalton polypeptides, designated A1 and A2 (not to be confused with the electron carriers, A<sub>0</sub> and A<sub>1</sub>), are associated with the reaction center (13).

P700 is generally considered to be a chlorophyll *a* dimer (3, 20) which undergoes a light-induced charge separation resulting in the transfer of an excited electron to A<sub>0</sub> (Figure 2). A<sub>0</sub> is generally considered to be a chlorophyll *a* monomer (3, 20). The precise nature of the next electron carrier, A<sub>1</sub>, is controversial but it may be phyloquinone (vitamin K-1) (11, 20).

The membrane-bound acceptors, F<sub>x</sub> and F<sub>a</sub>/F<sub>b</sub>, are protein-bound iron-sulfur centers. F<sub>x</sub> has one 2Fe-2S center (3) and F<sub>a</sub>/F<sub>b</sub> contains two 4Fe-4S centers (13). The sequence of electron flow through the two iron-sulfur centers of F<sub>a</sub>/F<sub>b</sub> is not clear. F<sub>a</sub>/F<sub>b</sub> is probably the intermediate from which paraquat and diquat accept electrons (27).

Ferredoxin transfers electrons from F<sub>a</sub>/F<sub>b</sub> to ferredoxin: NADP<sup>+</sup> oxidoreductase (FNR)<sup>3</sup>. FNR is restricted to the stromal surface of nonappressed lamellae (13) and catalyzes the reduction of NADP<sup>+</sup> to NADPH.

Cyclic electron transport involves the transfer of electrons from ferredoxin to cytochrome  $b_6$  (20). This transfer of electrons to cytochrome  $b_6$  drives the conversion of plastoquinone to plastoquinone and thus drives the transport of protons across the thylakoid membrane (Figures 1 and 2). This contributes to the proton gradient and ATP synthesis. Thus, the cytochrome  $b_6/f$  and PS I complexes are involved not only in linear electron transport from PS II to  $\text{NADP}^+$ , but also in cyclic electron transport (Figures 1 and 2).

#### PS II ELECTRON TRANSPORT INHIBITORS

**Mechanism of action.** PS II electron transport inhibitors, such as atrazine, bind to the  $Q_B$ -binding niche on the D1 protein. The D1 protein has also been referred to as the  $Q_B$ -binding protein and the 32 kilodalton herbicide binding protein. PS II inhibitors bind to the D1 reaction center protein and inhibit electron transport by acting as nonreducible analogs of plastoquinone (10). A model for herbicide binding is presented in Figure 4 and is based on homologies between PS II and the photosynthetic reaction center of purple photosynthetic bacteria (24, 25, 34, 36, 39).

$Q_B$  is bound to the D1 protein by two hydrogen bonds between the protein and the two carbonyl groups of plastoquinone. A hydrogen bond is formed between one carbonyl and the hydroxyl group of serine 264 (Figure 4); an amino acid adjacent to serine 264 can also hydrogen bond to the same carbonyl on  $Q_B$  (not shown) (35, 36, 39). A hydrogen bond is formed between the second carbonyl of  $Q_B$  and histidine 215 (35, 36, 39). PS II herbicides such as atrazine bind to the D1 protein due to hydrogen bonds, van der Waals forces, and hydrophobic interactions (25, 39). Hydrogen bonds between triazine herbicides and the amino acids serine 264 and phenylalanine 265 are essential for binding (Figure 4) (34, 36, 39). Phenylalanine 255 contributes to hydrophobic interactions in herbicide binding (Figure 4) (34, 39).

PS II electron transport inhibitors belong to a variety of chemical families (Table 1). Three of these families, the nitrophenols, nitriles, and pyridazinones, inhibit photosynthesis by preventing  $Q_B$  binding in vitro, but another mode of action may also be involved in their herbicidal activity, in vivo. Nitrophenols and nitriles have been shown to possess uncoupling activity (26), whereas pyridazinones inhibit lipid

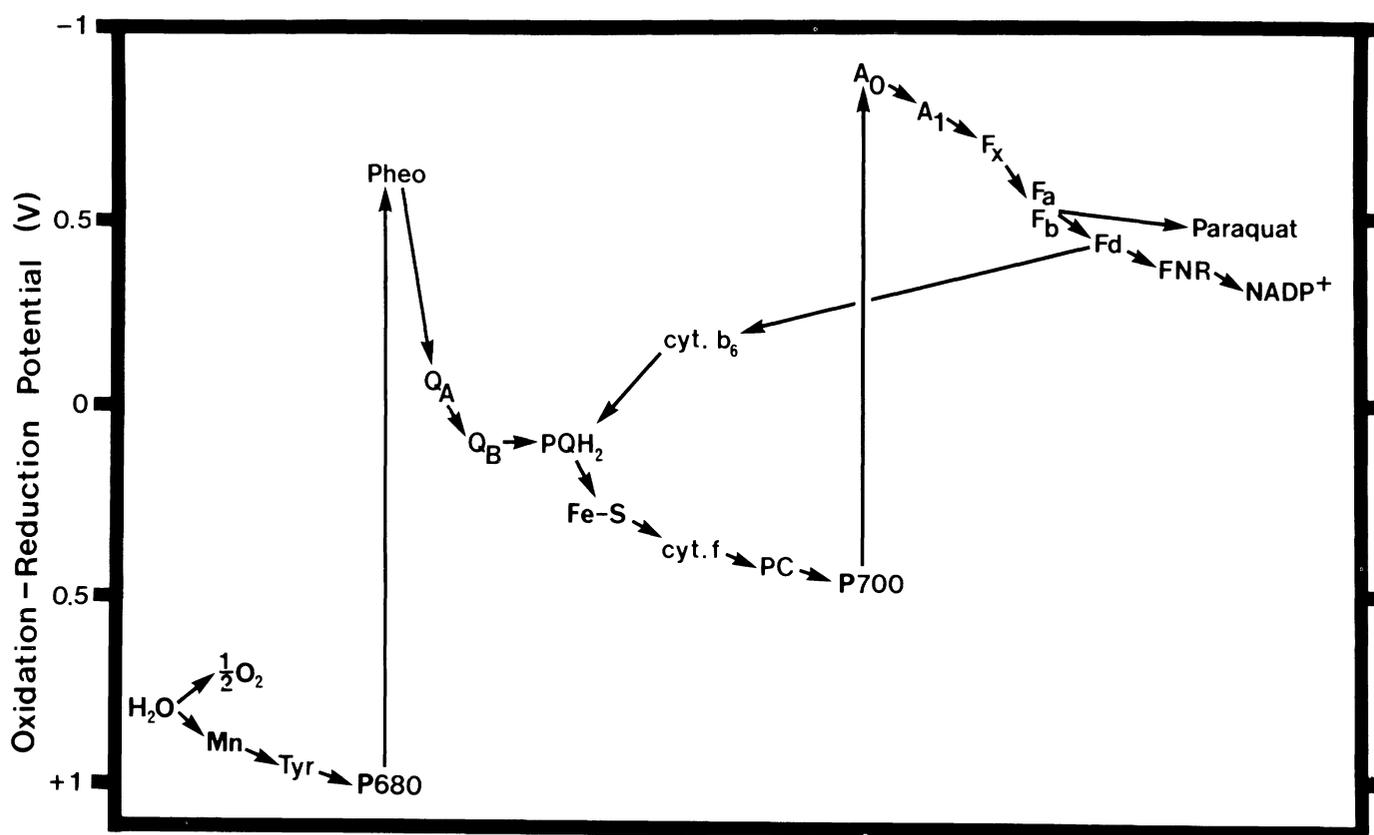


Figure 2. Photosynthetic electron transport scheme. Mn, manganese of the S-state, water-splitting enzyme; Tyr, tyrosine residue 160 of the D1 protein; P680, PS II reaction center chlorophyll *a* dimer; Pheo, pheophytin (chlorophyll *a* without magnesium);  $Q_A$ , plastoquinone bound to D2 protein;  $Q_B$ , plastoquinone bound to D1 protein;  $\text{PQH}_2$ , plastoquinone (reduced plastoquinone); Fe-S, Rieske iron-sulfur protein; cyt. f, cytochrome f; cyt.  $b_6$ , cytochrome  $b_6$ ; PC, plastocyanin; P700, PS I reaction center, probably a chlorophyll *a* dimer; Fe-S, Rieske iron-sulfur protein; cyt. f, cytochrome f; cyt.  $b_6$ , cytochrome  $b_6$ ; PC, plastocyanin; P700, PS I reaction center, probably a chlorophyll *a* dimer; A<sub>0</sub>, probably a chlorophyll *a* monomer; A<sub>1</sub>, probably phylloquinone (vitamin K-1); F<sub>x</sub>, F<sub>a</sub>/F<sub>b</sub>, membrane-bound nonheme iron-sulfur proteins; Fd, ferredoxin; FNR, ferredoxin-NADP<sup>+</sup> oxidoreductase; NADP<sup>+</sup>, nicotinamide adenine dinucleotide phosphate.

Table 1. Herbicide families discussed in text and an example of one member of each family.

Family	Example
Triazine	Atrazine <sup>1</sup>
Triazinone	Metribuzin <sup>2</sup>
Phenylurea	Diuron <sup>3</sup>
Uracil	Bromacil <sup>4</sup>
Biscarbamate	Desmedipham <sup>5</sup>
Benzothiadiazinone	Bentazon <sup>6</sup>
Nitrophenol	Dinoseb <sup>7</sup>
Nitrile	Bromoxynil <sup>8</sup>
Pyridazinone	Pyrazon <sup>9</sup>
Thiadiazole	Tebuthiuron <sup>10</sup>
Bipyridinium	Paraquat <sup>11</sup>

<sup>1</sup>6-chloro-*N*-ethyl-*N'*-(1-methylethyl)-1,3,5-triazine-2,4-diamine.

<sup>2</sup>4-amino-6-(1,1-dimethylethyl)-3-(methylthio)-1,2,4-triazin-5(4*H*)-one.

<sup>3</sup>*N'*-(3,4-dichlorophenyl)-*N,N*-dimethylurea.

<sup>4</sup>5-bromo-6-methyl-3-(1-methylpropyl)-2,4(1*H*,3*H*)pyrimidinedione.

<sup>5</sup>Ethyl [3-[[[(phenylamino)carbonyl]oxy]phenyl]carbamate.

<sup>6</sup>3-(1-methylethyl)-(1*H*)-2,1,3-benzothiadiazin-4(3*H*)-one 2,2-dioxide.

<sup>7</sup>2-(1-methylpropyl)-4,6-dinitrophenol.

<sup>8</sup>3,5-dibromo-4-hydroxybenzoxynitrile.

<sup>9</sup>5-amino-4-chloro-2-phenyl-3(2*H*)-pyridazinone.

<sup>10</sup>*N*-[5-(1,1-dimethylethyl)-1,3,4-thiadiazol-2-yl]-*N,N'*-dimethylurea.

<sup>11</sup>1,1'-dimethyl-4,4'-bipyridinium ion.

and carotenoid synthesis (8). This diverse group of herbicides binds to overlapping, but not identical, binding sites on the D1 protein (28, 39). Herbicides in the nitrophenol and nitrile families probably bind to the D1 protein due to interactions with histidine 215 rather than serine 264 (39). The reason that such a diversity of chemical families binds to the D1 protein may be due to the dual binding roles of the D1 protein; i.e., the D1 protein must bind nonreduced as well as singly reduced plastoquinone.

Treatment of plants with PS II herbicides blocks the flow of electrons through PS II, and thus also indirectly blocks the transfer of excitation energy from chlorophyll molecules to the PS II reaction center. Excited chlorophyll molecules (singlet chlorophyll) spontaneously form triplet chlorophyll (<sup>3</sup>chl) through a nonradiative energy transformation of chlorophyll, known as intersystem crossing. The <sup>3</sup>chl reacts with molecular oxygen to form singlet oxygen (<sup>1</sup>O<sub>2</sub>)<sup>3</sup> (Figure 5). Lipid peroxidation is then initiated by <sup>3</sup>chl and <sup>1</sup>O<sub>2</sub> (discussed below) (14).

**Triazine resistance.** Fifty-five weed species have evolved resistance to triazine herbicides (22). Triazine resistance in most of these weeds is due to a single mutation in the *psbA* gene that codes for the D1 protein (17). A mutation of serine 264 to glycine has been reported in all naturally occurring resistance mutations (17), including *Amaranthus hybridus* (16), *Solanum nigrum* (15), *Chenopodium album* (5), and *Phalaris paradoxa* (33). In addition, triazine resistance in canola (*Brassica napus*) originated from triazine-resistant *Brassica rapa*, and this mutation was also the same (31).

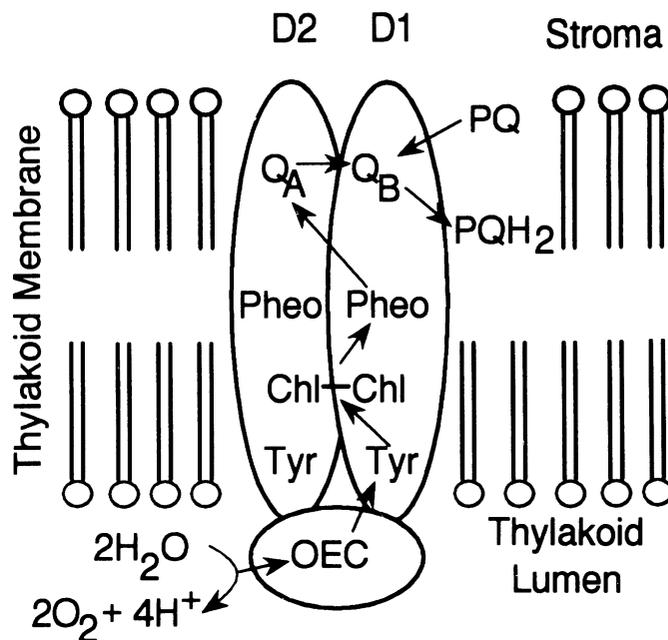


Figure 3. PS II reaction center complex. OEC, oxygen-evolving complex; Tyr, tyrosine amino acid residue 160; Chl-Chl, chlorophyll *a* dimer also known as P680; Pheo, pheophytin; Q<sub>A</sub> and Q<sub>B</sub>, bound plastoquinone molecules; PQH<sub>2</sub>, plastoquinone. Electron transport carriers bound to the D1 and D2 proteins have a twofold axis of symmetry (23, 25). The tyrosine and pheophytin of the D2 protein do not carry electrons (24, 40). The transfer of an electron from Q<sub>A</sub> to Q<sub>B</sub> is magnetically coupled by ferrous iron (not shown) (6, 24). The reaction center complex is normally associated with other proteins such as cytochrome b<sub>559</sub> and light-harvesting chlorophyll proteins *in vivo* (not shown).

At the molecular level, resistance to triazines in weeds is thought to be due to the loss of the hydrogen bond between serine 264 and the amino alkyl side chain of the triazine ring (24, 34, 36, 39) (Figure 4B). The loss of this hydrogen bond reduces the binding affinity by orders of magnitude (24, 36). The loss of the hydrogen bond from serine 264 to Q<sub>B</sub> (Figure 4A) does not prevent binding of Q<sub>B</sub> because of an alternate hydrogen bond (not shown) (24, 35, 39).

The serine to glycine mutation in the D1 protein also causes impaired electron transport (38), which occurs because over 30% of charge separation events in PS II recombine rather than being used in linear electron transport (18). This impaired electron transport reduces the yield of triazine-resistant canola (32).

Resistance to triazines in weeds results in partial resistance to other PS II herbicides, including uracils, pyridazinones, and certain phenylureas (9, 29). Most triazine-resistant weeds show a very similar spectrum of cross-resistance (9), due to similar if not identical mutations. The degree of resistance to the nontriazine PS II electron transport inhibitors is not as great as resistance to triazines, and the varying degrees of resistance are related to overlapping but not identical binding sites of these herbicides (28, 39).

The reason that the serine to glycine substitution is so common among resistant weeds may be due to the very high

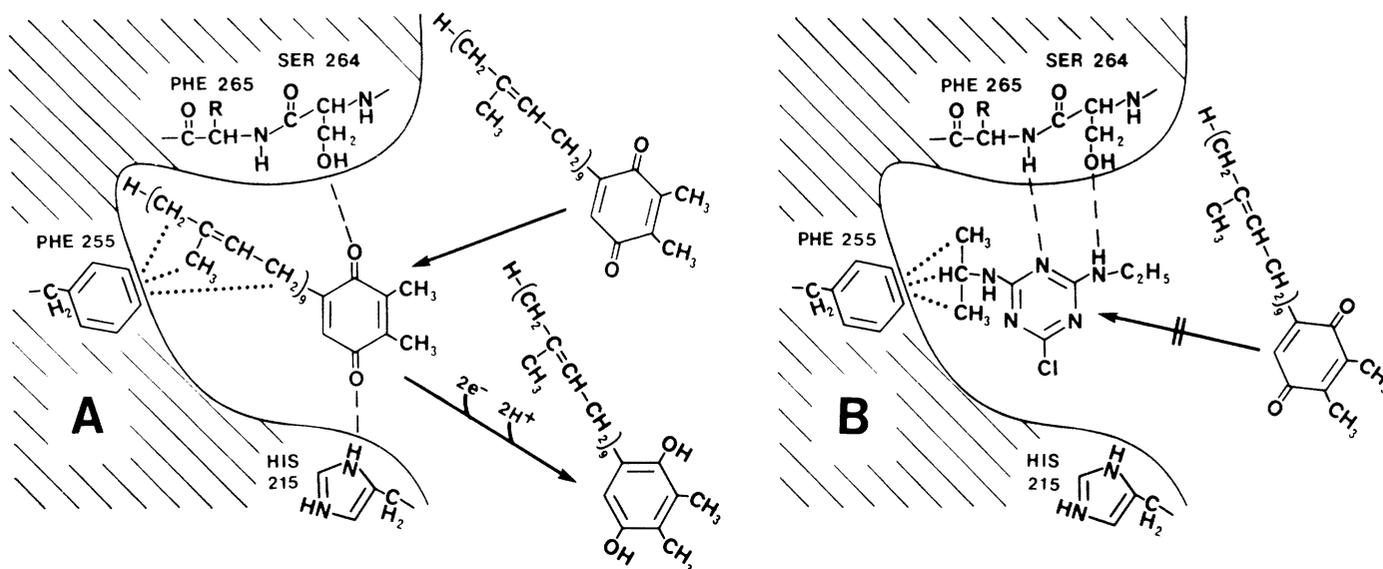


Figure 4. Schematic figure of the plastoquinone/herbicide binding pocket of the D1 protein. Dashed lines represent hydrogen bonds; dotted lines represent hydrophobic interactions. A. Plastoquinone binds to the D1 protein, accepts two electrons and two protons, and is released as plastoquinol. B. Atrazine binds to the D1 protein and prevents the binding of plastoquinone.

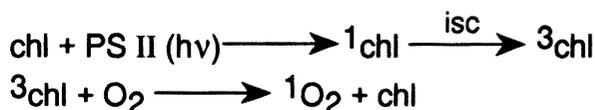
level of resistance conferred by this mutation. The resistance level to triazine herbicides due to this mutation is >1000-fold at the level of electron transport (9, 28); other mutations result in lower levels of resistance (17, 23).

Several mutations of the D1 protein have been induced artificially in algae, cyanobacteria, and higher plants (17, 23, 37, 39) which confer resistance to triazines or other PS II electron transport inhibitors. While the serine 264 to glycine mutation results in impaired electron transport, several other mutations confer resistance to PS II herbicides, but either cause no change in electron transport (23) or enhanced rates of electron transport (37). Also, the serine to glycine mutation does not confer reduced photosynthesis or plant vigor in *P. paradoxa* (17, 32). Thus, it cannot be assumed that a mutation conferring resistance to PS II herbicides necessarily confers impaired electron transport and reduced plant productivity.

#### PS I ELECTRON ACCEPTORS

The only registered herbicides that affect electron transport through PS I are paraquat and diquat, members of the bipyridinium (also called bipyridylum) herbicide family. The phytotoxic effects of these herbicides were first discovered by Brian in the 1950s (1). Although many bipyridinium compounds possess herbicidal activity, paraquat and diquat (6,7-dihydrodipyrido[1,2- $\alpha$ :2',1'-c]pyrazinedium ion) are the most active members. The quaternary bipyridinium salts with the 2,2'-(diquat), 4,4'-(paraquat), and 2,4'-configurations are phytotoxic whereas the 2,3'- and 3,3'-configurations possess little or no activity. The most active bipyridinium compounds can assume a coplanar configuration and are highly conjugated. These traits confer stability to the unpaired electron of the

#### Electron Transport Inhibitors



#### Electron Transport Acceptors

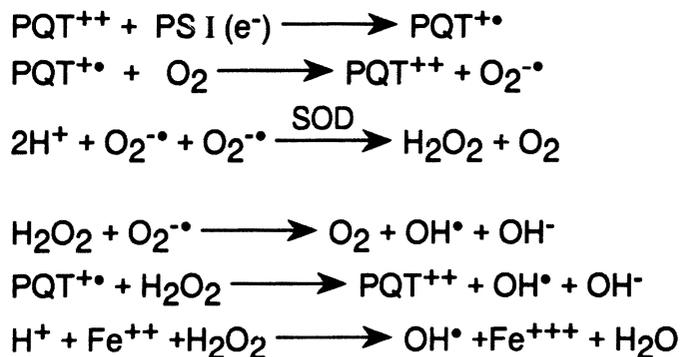
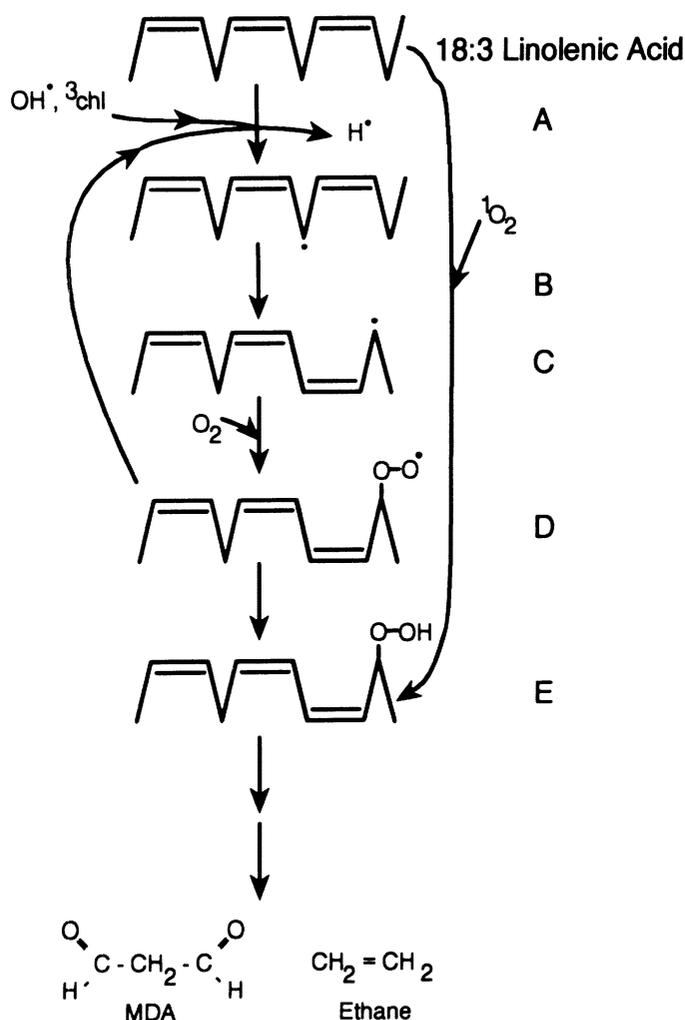


Figure 5. Reactions involved in the generation of  $^3\text{chl}$  and  $^1\text{O}_2$  by PS II electron transport inhibitors and  $\text{OH}^\bullet$  by PS I electron acceptors. Isc, intersystem crossing.



**Figure 6.** Herbicide-induced lipid peroxidation of a thylakoid membrane polyunsaturated fatty acid molecule (18:3, linolenic acid). A. Hydrogen abstraction by  $\text{OH}\cdot$ ,  $^3\text{chl}$ , or fatty acid radical, B. rearrangement, C. conjugated diene, D. lipid peroxide radical, and E. lipid hydroperoxide. MDA, malondialdehyde. [Modified from (14) by permission of the Oxford University press.]

reduced free radical form of the herbicide molecule; stability is essential for the reduction of  $\text{O}_2$  (1), discussed below.

**Mechanism of action.** The mechanism of action of paraquat and diquat is identical, but only paraquat will be discussed below. Paraquat is applied to plants as a solution of the divalent cation ( $\text{PQT}^{2+}$ )<sup>3</sup>, which is colorless. Paraquat is converted to the intensely blue monovalent cation radical ( $\text{PQT}^{+\cdot}$ )<sup>3</sup> when reduced by PS I (1). Paraquat is commonly called "methyl viologen" in the literature because of this blue color. The  $\text{F}_a/\text{F}_b$  iron-sulfur center of PS I is probably the electron donor to  $\text{PQT}^{2+}$  (27) (Figure 2). The redox potential of  $\text{PQT}^{2+}$  (-446 mV) (1) permits the acceptance of electrons from  $\text{F}_a/\text{F}_b$  (-560 mV) (27).

$\text{PQT}^{+\cdot}$  reduces  $\text{O}_2$  to superoxide ( $\text{O}_2^{\cdot-}$ )<sup>3</sup> and  $\text{PQT}^{2+}$  is regenerated in this reaction (Figure 5) (1). Thus, only

catalytic quantities of  $\text{PQT}^{2+}$  need to be associated with PS I to be phytotoxic. Superoxide dismutase ( $\text{SOD}$ )<sup>3</sup> catalyzes the conversion of paraquat-generated  $\text{O}_2^{\cdot-}$  to  $\text{H}_2\text{O}_2$  and  $\text{O}_2$  (Figure 5).  $\text{PQT}^{+\cdot}$  condenses with  $\text{H}_2\text{O}_2$  to spontaneously produce  $\text{PQT}^{2+}$ ,  $\text{OH}\cdot$ , and  $\text{OH}^-$ . The  $\text{OH}\cdot$  can also be produced by an  $\text{Fe}^{2+}$  catalyst with  $\text{H}^+$  and  $\text{H}_2\text{O}_2$  as reactants (the Fenton reaction) (Figure 5) (4).

#### LIPID PEROXIDATION AND PHYTOTOXICITY

Although the PS II electron transport inhibitors and PS I electron acceptors have extremely different primary sites of action, both types of herbicides are phytotoxic due to photooxidation of photosynthetic membranes. Plants have mechanisms of preventing membrane damage from the toxic  $^3\text{chl}$ ,  $^1\text{O}_2$ ,  $\text{H}_2\text{O}_2$ , and  $\text{O}_2^{\cdot-}$  that are normally produced to a small degree during photosynthesis (7). However, plants treated with the PS II electron transport inhibitors or PS I electron transport acceptors produce quantities of these toxic species that overwhelm native protective mechanisms, resulting in phytotoxicity (7). Protective mechanisms present in plants include the following: carotenoids protect from  $^1\text{O}_2$ ,  $^3\text{chl}$ , and lipid peroxidation;  $\alpha$ -tocopherol protects from  $\text{OH}\cdot$  and lipid peroxide radicals; ascorbate and glutathione protect from  $^1\text{O}_2$ ,  $\text{O}_2^{\cdot-}$  and  $\text{OH}\cdot$ ; SOD converts  $\text{O}_2^{\cdot-}$  to  $\text{H}_2\text{O}_2$ ; and catalase and peroxidase protect from  $\text{H}_2\text{O}_2$  (7, 12, 19). Carotenoids and  $\alpha$ -tocopherol are present in the thylakoid membranes, ascorbate and glutathione are located in the chloroplast stroma, and catalase and peroxidase are present in peroxisomes which are found in close proximity to chloroplasts.  $\text{H}_2\text{O}_2$  is also detoxified in the stroma of the chloroplast by an ascorbate/glutathione cycle driven by NADPH (14). The xanthophyll epoxide cycle may also aid in the quenching of toxic oxygen species (14).

The most abundant fatty acids of thylakoid membranes are linolenic and linoleic acids (12). These polyunsaturated fatty acids are prone to lipid peroxidation. The  $^3\text{chl}$  and  $\text{OH}\cdot$  can abstract hydrogen atoms from unsaturated fatty acids resulting in a lipid radical (Figure 6). Molecular rearrangement results in a conjugated diene configuration within the fatty acid radical that can be converted to a lipid peroxide radical with the addition of molecular oxygen. The process of lipid peroxidation is autocatalytic since the lipid peroxide radical can initiate hydrogen abstraction from adjoining lipids (Figure 6). The lipid peroxide radical can then be converted into lipid endoperoxides and hydroperoxides.  $^1\text{O}_2$  can react directly with polyunsaturated fatty acids and directly yield a lipid hydroperoxide. Lipid hydroperoxides degrade into small hydrocarbon fragments such as ethane and malondialdehyde (14) (Figure 6). The lipid peroxidation process destroys the integrity of membranes, leading to loss of cellular compartmentation and phytotoxicity (14).

The mechanism of action of photosystem II inhibitors is understood in more detail than any other site of herbicide action. Our understanding of the detailed mechanism of electron transfer to PSI electron acceptors is understood in less detail, because the precise nature and structure of PSI electron transfer components is not fully understood. Both

types of herbicides are phytotoxic as a consequence of their interactions with photosynthetic electron transport and subsequent destruction of photosynthetic membranes.

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