The Three-Dimensional Structure of the Herbicide Binding Niche on the Reaction Center Polypeptides of Photosystem II

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The folding through the membrane of the plastoquinone and herbicide binding protein subunits of photosystem II and the topology of the binding niche for plastoquinone and herbicides is described. The model is based on the homology in amino acid sequence and folding prediction from the hydropathy analysis of the D-1 and D-2 subunits of photosystem II to the reaction center polypeptides L and M of the bacterial reaction center. It incorporates the amino acid changes in the D-1 polypeptide in herbicide tolerant plants and those indicated by chemical tagging to be involved in Q_B binding. It proposes homologous amino acids in the D-1/D-2 polypeptides to those indicated by the X-ray structure of the bacterial reaction center to be involved in Fe-, quinone- and reaction center chlorophyll-binding. The different chemical compounds known to interfere with Q_B function are grouped into two families depending on their orientation in the Q_B binding niche.

Introduction

The mode of action of inhibitors and herbicides interferring with photosynthetic electron flow at photosystem II is well known. Uncountable studies have led to the accepted view that these inhibitors block electron flow between the primary Q_A and secondary Q_B plastoquinone acceptor of photosystem II, most likely by displacing plastoquinone from the Q_B binding site [1]. Although chemically quite different, the inhibitors will displace each other from the membrane, indicating identical binding sites [2]. The herbicide or Q_B binding protein has been identified by photoaffinity labeling, particularly by azidoatrazine, as a 32 kDa polypeptide subunit of photosystem II [3]. This protein was subsequently shown to be identical to a trypsin sensitive, rapidly turning over, photogenic, chloroplast encoded polypeptide known as the D-1 protein [4]. Structure activity correlationships led already to the identification of essential atomic elements and of substituents in the chemistry of the inhibitors responsible for optimal inhibitory potency. Different inhibitors of photosystem II were grouped according to chemical specifications and inhibitory patterns into two families, an urea/triazine and a phenol family [5] each comprising many compounds. A concept of overlapping binding sites in a common binding domain on photosystem II, *i.e.* on the D-1 protein was developed [5, 6]. From these and QSAR studies the approximate dimensions of the herbicide binding niche were described [7]. New inhibitory compounds could be accommodated or were even predicted and found to be effective inhibitors *in vitro,* though not necessarily herbicides *in vivo*. Such predictions can now be done by molecular modelling of compounds into the herbicide binding niche because of a rapidly developing knowledge of the details of the molecular topology of the amino acid residues in the binding niche.

The topology of the herbicide binding niche

The gene for the rapidly turning over D-1 protein had already been localized in the chloroplast genome before its function as a plastoquinone and herbicide binding protein was known. The DNA of the gene was sequenced [8], the deduced amino acid sequence was analyzed with algorithms for membrane burried or exposed parts [9]. The large hydrophobicity of the D-1 polypeptide indicated that it is an intrinsic polypeptide extending through and out on either side of the membrane [9]. It is not just a peripheral subunit of photosystem II, as its early descriptions as a shield protein above the acceptor side of photosystem II [10] might have suggested. The first prediction of the folding of the D-1 protein in seven hydrophobic transmembrane helices [9] could not easily accom-

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modate, however, the data that successively came from the sequencing of the gene for the D-1 polypeptide in herbicide tolerant plants and algae [11–16]. The amino acid changes in the mutants seemed to be located on either side of the membrane (see for example [14]), whereas the functional studies and trypsin experiments clearly had established the Q_B and herbicide binding site to be close to the matrix side of the membrane (see [1]).

A major development for the identification of the details of the herbicide binding niche was the discovery of the D-2 polypeptide subunit of photosystem II. Although a second "diffuse" band on gels was shown early [17, 18], a role for an at first evasive D-2 polypeptide could not be proposed. The first sequencing of the gene of *Chlamydomonas* for the D-2 polypeptide [19], also located on the chloroplast genome, showed immediately the amino acid sequence homology of the D-1 and D-2 polypeptides and suggested a role as a second plastoquinone binding protein for the D-2 polypeptide. The *pspD* gene for the D-2 polypeptide has since been sequenced also in spinach [20, 21] and pea [22]. It is now established that this polypeptide D-2 occurs indeed in enriched photosystem II preparations (see [23]).

Studies on the reaction center of purple bacteria had shown the great similarities in function and composition in redox components and pigments to that of photosystem II [24, 25]. When the genes for the L and M subunits of *Rhodopseudomonas capsulata* were sequenced [26], it was recognized that they show high amino acid homologies not only among themselves, but also to the D-1 polypeptide (the D-2 polypeptide was not yet known at that time). The reaction center of *Rh. viridis* was crystallized [27] and X-ray data became available [28–30]. They provided a wealth of information on the orientation of the pigments and redox centers in the bacterial system, but also of the protein folding of the L, M and H subunits [28–30]. Functional amino acid residues like the histidines involved in bacteriochlorophyll and in Fe binding were indicated.

From the similarities of the bacterial reaction center in function and amino acid sequence homology to photosystem II it was proposed that the D-1 and D-2 polypeptides carry the reaction center of photosystem II [28, 31]. A reassessment of the hydropathy index plot considering as transmembrane spans only those sequences that are conserved in all four polypeptides, L and M and D-1 and D-2, led to a new folding model for the D-1 (Q_B binding) subunit and D-2 (called now the Q_A binding) subunit [32]. The model contradicted the assignment at that time of the photosystem II reaction center to the 47 kDa subunit [33–36]. New data now support the proposal that the D-1 and D-2 subunits carry the reaction center [23]. The model interpreted the amino acid changes in herbicide tolerant algae, as these could be now well rationalized (see Fig. 1) in the folding of the D-1 polypeptide. It points to those amino acids that are equivalent to functional amino acids identified in the X-ray structure of the *Rh. viridis* system [28–30]. For example, the phe_{255} on a parallel helix changed in the mutant AR 207 [12] is at an equivalent position to a phe in the L subunit and this in turn is equivalent to a trp in the M subunit shown in the X-ray structure to be on a parallel helix folding back into the Q_A binding niche and being located between Q_A and the pheophytin [29]. This trp is conserved in the D-2 subunit. The model based on the homology of the bacterial and plant photosystem, the X-ray structure and the mutation data allowed a detailed description of the herbicide and Q_B and Q_A binding niche [32]. Accordingly amino acids from the end of transmembrane helix IV, the beginning of transmembrane helix V, of a parallel helix between these two and a stretched sequence between the end of the parallel helix and the beginning of helix V make up the binding niche of Q_B and herbicides on the D-1 subunit (Fig. 2). Table I summarizes the amino acids that may be involved in the binding niche of Q_B and herbicides: val_{219}, ala_{251}, phe_{255}, ser_{264}, and leu_{275} are amino acids changed in mutations [10–15]; met_{214} is indicated in degradation of the azidoatrazine labeled D-1 subunit [37]; his_{215} and his_{272} are involved in Fe binding [28–30]; his_{215} and a peptide bond of an amino acid close to ser_{264} are necessary for hydrogen bridges onto the carbonyl group of a quinone [32], in homology to the X-ray structure of the bacterial system [29]. Very likely ser_{264} plays a major role in displacing the reduced Q_B from the membrane. The different allignment in lit. [31, 32] *vs* [26, 38] *vs* [39] of the D-1/D-2 amino acid sequence to that of the L and M subunits vary somewhat. Therefore the "identification" of functional amino acids in the quinone binding niche of photosystem II, if based on homology, remains hypothetical. For example, the sequence tyr_{262} to phe_{265} might be equivalent to the sequence tyr_{222} to ile_{224} in the L subunit of *Rh. viridis;* then the peptide bond involved in the quinone Q_B binding could be on

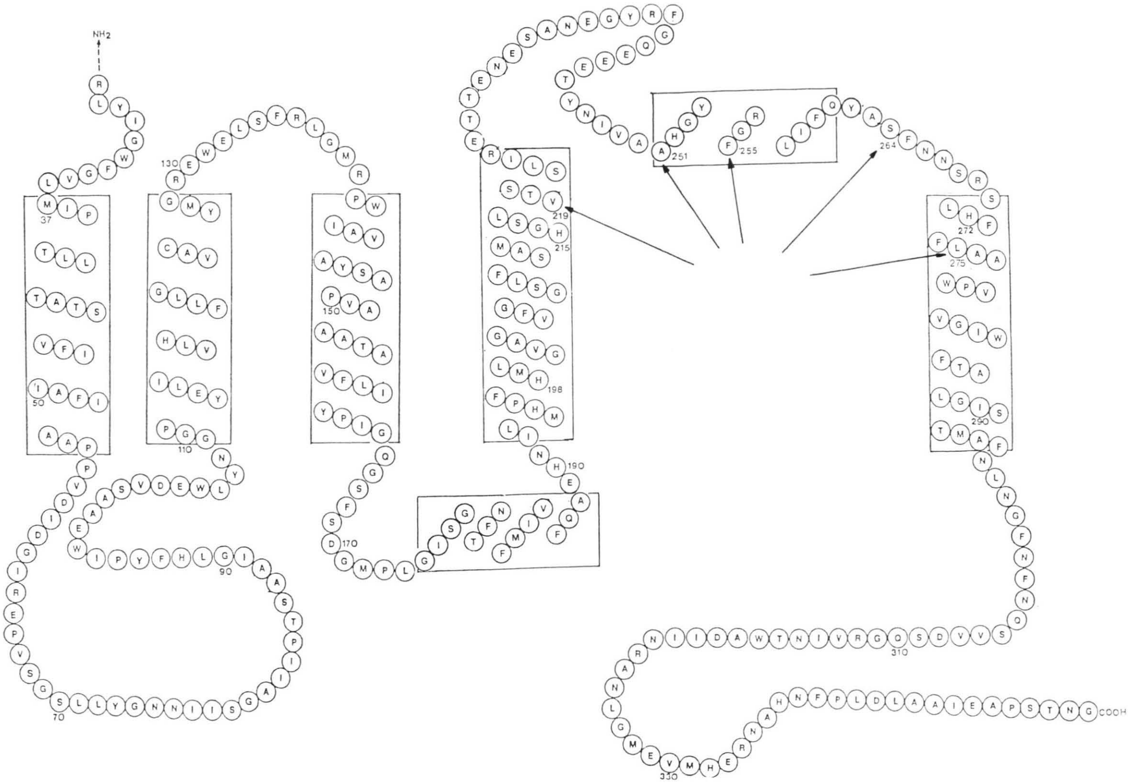

Fig. 1. The folding of the amino acid sequence of the D-1 polypeptide subunit of photosystem II. The model is to indicate five transmembrane helical spans and two parallel helices. It is based on 1. the hydropathy index plot of the amino acid sequence in homology to the D-2 polypeptide and the L and M subunit of the reaction center of purple bacteria, 2. the X-ray structure of the *Rh. viridis* reaction center, and 3. the mutations in herbicide tolerant plants. These mutations are indicated by arrows. The amino acid changes are:

$$\mathrm{Val}_{219} \rightarrow \mathrm{Ile} \qquad \mathrm{Ala}_{251} \rightarrow \mathrm{Val} \qquad \mathrm{Phe}_{255} \rightarrow \mathrm{Tyr} \qquad \mathrm{Ser}_{264} \begin{array}{l} \nearrow \mathrm{Ala} \\ \searrow \mathrm{Gly} \end{array} \qquad \mathrm{Leu}_{275} \rightarrow \mathrm{Phe}.$$

Table I. Amino acids involved in quinone binding in photosystem II. Q_A binding in *Rh. viridis* as directly seen in the X-ray structure [28–30]. Q_B binding in *Rh. viridis* according to the X-ray structure of the terbutryn derivative [30]. D-1 and D-2 subunit in spinach photosystem II according to the homology in amino acid sequences to the equivalent L and M subunit in *Rh. viridis*.

Primary quinone Q_A		Secondary quinone Q_B	
Rh. viridis M. subunit	photosystem II D-2 subunit	*Rh. viridis* L subunit	photosystem II D-2 subunit
His 217	His 215	His 190	
Ala 258	Ala 261	Tyr 222–Ser 223–Ile 224 (in terbutryn binding)	His 215 Tyr 262–Ala 263–Ser 264–Phe 265
Trp 250	Trp 254	Phe 216	Phe 255
In the neighborhood:			
Ala 215, Val 264	Ala 213, ?	Ile 229, Leu 193	?, Leu 218
Thr 220	Thr 218	Asn 213	Glu 244
		Val 220, Glu 212	?, Glu 243

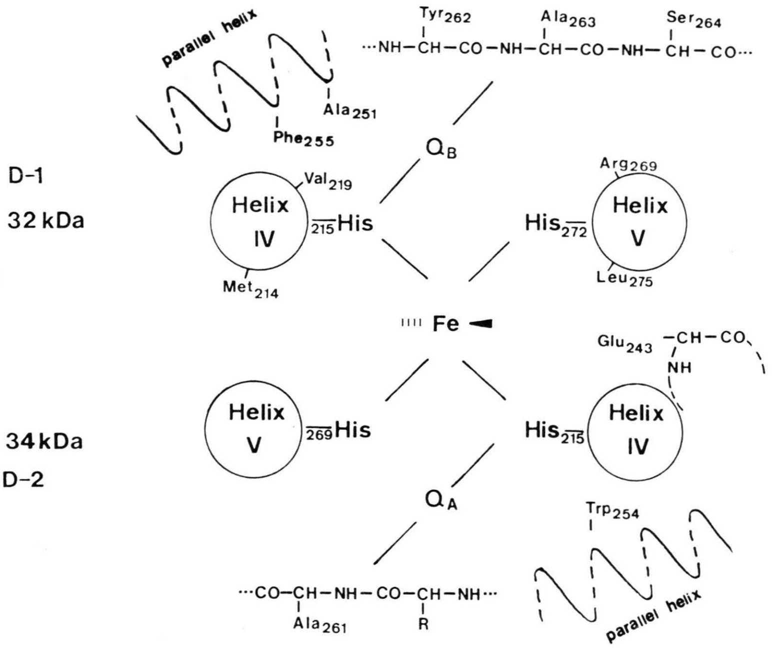

Fig. 2. The Q_B and herbicide binding niche on photosystem II. The circles are to indicated transmembrane helices, the spirales parallel helices (extending perpendicular to the page). Amino acid residues in the binding niche specified so far are those from mutations in herbicide tolerant plants, from photoaffinity labeling and from the homology of those indicated by the X-ray structure of the *Rh. viridis* reaction center.

either side of serine$_{264}$, *i.e.* either ala$_{263}$ or phe$_{265}$. According to the X-ray structure of terbutryn binding in the *Rh. viridis* crystals [30] a serine is before that peptide bond. Arg$_{269}$ might be of importance in view of the theoretical studies on overimposing of certain herbicides on a guanidine residue (*i.e.* arg) at the end of a hydrophobic helix [40].

The model of the folding of the D-1 and D-2 polypeptides, in homology to the L and M subunit, seen in the X-ray structure, predicted five transmembrane helices and three parallel helices. Exact orientations of certain amino acids can be predicted for those in helices because of the strict arrangement of amino acid residues in helices. A helical wheel (Fig. 3) of

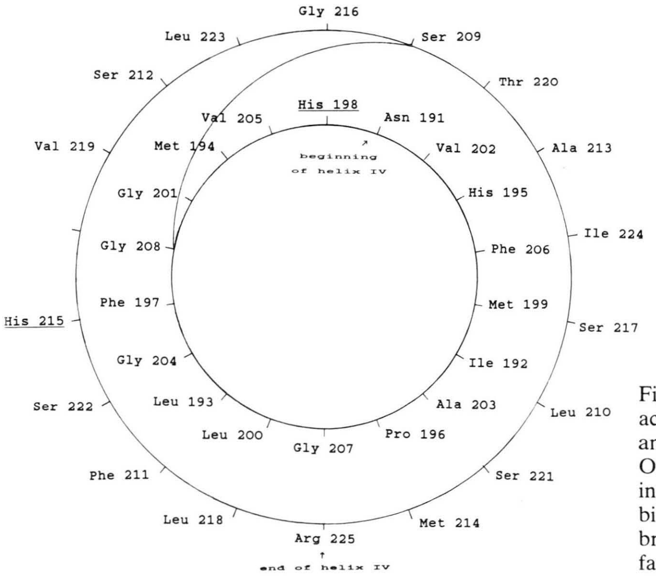

Fig. 3. Helical wheel of the, theoretically up to 33, amino acids in transmembrane helix IV of the D-1 polypeptide. In an α-helix the amino acids follow each other at a 100° angel. One circle therefore accommodates 18 amino acids. Accordingly his$_{198}$ probably involved in reaction center chlorophyll binding and his$_{215}$ in Fe binding are facing out of the membrane at about 90°. The val$_{219}$ change in triazine resistance is facing in the same direction as his$_{215}$.

the substituents of α-helix IV of either the D-1 and D-2 subunit shows that his_{198} for reaction center chlorophyll binding and his_{215} for Fe binding are at an angle of about 90°. In order to accommodate chlorophyll and Fe binding the helix has to be tilted – as directly seen in the X-ray structure [28–30]. In order to accommodate his_{272} on helix V of the D-1 (or his_{269} in the D-2) subunit in Fe binding these helices have to be tilted also. Prolines in almost the middle will bend the helices V of both D-1 and D-2 and so may the four glycines (201, 204, 207, 208), facing in the same direction, in helix IV of D-1. According to the helical wheel the angle of met_{214} and val_{219} above and below respectively to his_{215} on helix IV as well as of arg_{269} and leu_{275} above and below his_{272} on helix V are obtained. The same is true for the position of ala_{251} and phe_{255} on the parallel helix, as they are facing into the same direction, one turn of the helix orienting them besides and above the quinone. The proposed folding of the D-1 polypeptide and the Q_B binding site in Fig. 1 and 2 is extending those in print [32, 41, 42] in that it indicates also the parallel helix between transmembrane helices III and IV on the donor side (which does not contain the histidine shown to be involved in the monomeric chlorophyll binding in the bacterial system) and another between helices IV and V on the acceptor side (there is a third parallel helix after transmembrane helix V). The length of helix IV has been extended to arg_{225} equivalent to the end of helix D in *Rh. viridis.* Earlier the end at thr_{220} for the D-1 and at glu_{220} for the D-2 subunit was used because of this charged amino acid in D-2 only. The second charged position at arg_{225} is conserved in the D-1/D-2 and the L and M subunits. The theoretical length of this helix IV of the D-1 subunit in Fig. 3 from a charge on one side of the membrane (asn_{191}) to the other (arg_{225}) is probably not realized in photosystem II, as it may be too long for the membrane thickness, even when tilted. According to the model the stretched sequence from arg_{225} to the beginning of the parallel helix of ala_{251} extends into the hydrophilic environment on the matrix side with arg_{238} being among the most exposed amino acids. Indeed we showed recently with site specific antibodies that the D-2 subunit is easily split in the membrane by trypsin at arg_{234} in an easily accessible sequence of this polypeptide [43]. Similarly it can be assumed that the observed exposure is true also for the D-1 polypeptide probably split at arg_{238} [44]. The two parallel helices on the lumen side between transmembrane helices III and IV of both the D-1 and D-2 subunit run parallel to each other at right angles to the parallel helices on the matrix side. They cover the reaction center chlorophylls and most probably form the contact plain for the peripheral "Murata" 33 kDa polypeptide for a hydrophobic binding niche for the manganese in oxygen evolution. The model will not easily accommodate a planar chlorophyll for photosystem II as suggested by spin polarization data [45], but rather a perpendicular reaction center as in the purple bacteria.

The orientation of herbicides in their binding niche on photosystem II

The displacement from the membrane of the many different chemical compounds inhibiting photosystem II by each other, some competitive, some noncompetitive (see [48]), the QSAR studies [7], the overimposing (or modelling) of essential atoms in the compounds and molecular orbital calculations [46] led to the concept of overlapping specific binding sites in a common binding domain [4, 5, 47]. The two families, the urea/triazine and the phenol group, inhibiting both at the same site, are different in a number of functional aspects as well as in chemistry as summarized in [41, 46, 48]. An additional indicative difference is that the triazine tolerant mutants are still or even more sensitive to compounds of the phenol group [49–53]. We have recently described another parameter that easily distinguishes between the two groups of photosystem II herbicides [54]. In tris treated thylakoids the peripheral polypeptides of the oxygen evolution system on the donor side of the membrane are dislodged from the membrane, but also the Q_B site on the acceptor side is affected. Interesting is that the efficiency of phenoltype inhibitors in binding and inhibition is not affected by tris treatment, but that of the urea/triazines is. In a way tris treatment mimics the effect of the ser_{264} mutation. This effect on the D-1 protein on either side of the membrane is not a surprise any more, as the functional role of the D-1 protein is seen now not only on the acceptor side, but also in the reaction center chlorophyll binding. The D-1 polypeptide spans the membrane five times and provides the environment also for the donor side. A number of treatments of the membrane are now known to affect both the donor and acceptor side of photosystem II, similar to tris, like hydroxylamine [55], trypsin [56, 57], lysinase [58], mutations [16, 59] and detergents [56, 60, 61]. This explains also that certain inhibitors binding on the acceptor side also influence the donor side [62–65]. In enriched LHCP-free photosystem II preparations the D-1 polypeptide is no longer able to bind inhibitors at all [60, 61, 66], nor is it in the unstacked area of thylakoid membranes where photosystem II is not yet properly assembled [67], leave alone a purified D-1 protein.

The folding model in Fig. 1 and 2 allows now to describe the concept of overlapping binding sites in more molecular terms. As the herbicides displace Q_B, the binding characteristics of Q_B, but also of Q_A are directly relevant to herbicide binding. The quinones bind to the protein *via* two hydrogen bridges (of course, there will be other interactions less defined so far), one to his_{215} and the other to a peptide bond close to ser_{264}, as discussed above. If the herbicides and inhibitors are overimposed on plastoquinone, the substitution pattern can be rationalized: only small substituents are permitted on one side – equivalent to the methyl group(s) of plastoquinone – their size permitting up to a benzring or an alkyl sidechain that can bend away – but sufficient space for longer lipophilic substituents to the other side – equivalent to the isoprenyl sidechain of plastoquinone. The orientation in the binding niche suggests that inhibitors with a carbonyl group (like ureas, triazinones) or an equivalent group (triazines) are oriented towards the peptide bond close to

Table II. The two families of photosystem II herbicides. Typical representatives of the two families are oriented in the Q_B binding niche on the D-1 protein in two different conformations. The compounds have been described by many authors, as quoted in [41, 42] and many reviews, as are the different properties.

Compounds	Functional properties
"classical" urea/triazine type inhibitors = serine (264) family	
triazines ureas triazinones biscarbamates cyanoacrylates [73] pyrones [74, 75] chromones [76] cyclohexandiones [77] hydroxyquinoline-N-oxide benzo- and naphthoquinones [78]	predominant or even exclusive binding to the 32 kDa protein; hydrogen bridge from a NH of a peptide bond close to ser_{264} to the carbonyl or carbonyl-like function; mostly cross resistant in triazine tolerant mutants with the ser_{264} change; loss of inhibitory potency in tris-treated thylakoids; positive π-charge at a certain atom [46]
phenol-type inhibitors = histidine (215) family	
nitro-phenols azaphenanthrenes hydroxypyridines [54, 75] heterocyclic cyanoacrylates [79] (2-thiazolyliden-3-ketonitril)	binding to additional polypeptides in photosystem II besides the 32 kDa polypeptides; hydrogen bridge from his_{215} to a carbonyl or carbonyl-like function; no loss or even increased inhibitory potency in triazine tolerant mutants with the ser_{264} change; no loss of inhibitory potency in tris treated thylakoids; negative π-charge [46]; lag in inhibition that indicates an induced fit; longer residence time at the binding site [68]
not grouped yet	
cyanophenols	bind like the phenol family, but not necessarily strongly to his_{215}

serine$_{264}$ and can form a hydrogen bridge to this peptide bond. The phenol group of compounds with no substituent suitable for such a hydrogen bridge (in the case with an OH-group even unsuitable) and in case of a suitable atom for a hydrogen bridge on the other side of the molecule will be pushed away from serine$_{264}$ towards his$_{215}$. Table II summarizes properties of a "serine" and a "histidine" family with old and new compounds described recently (see [41, 42]). This proposal is consistant with the recent X-ray structure of terbutryn and phenanthroline in the Q_B binding site on *Rh. viridis* reaction center crystals [30]. Orientation of the compounds towards the his$_{215}$ will make the binding of these compounds less sensitive to disturbance of the membrane (or of the photosystem-II-complex) like tris and other treatments discussed above and also less affected by the exchange of ser$_{264}$ in the D-1 polypeptide in the mutations. The larger residence time of phenols on the membrane reflects probably also this stronger binding to the membrane *via* his$_{215}$ [68] as does the effect of inhibitors on the Fe-quinone EPR signal [69].

The "histidine" family of inhibitors shows usually a lag in inhibition in isolated membrane of about 1 to 2 minutes, *i.e.* a rather large lag [70, 71]. This might indicate an induced fit in the binding mechanism, *i.e.* the inhibitor pushes away amino acid residues in its approach to the histidine. This orientation of inhibitors towards the serine and/or the histidine might well reflect the conditions when the quinone Q_B gets reduced to the semi- and finally hydroquinone form and also has to move away from the peptide bond close to ser$_{264}$, finally to be expelled from the site altogether, if there are OH-groups oriented towards both ser$_{264}$ and his$_{215}$.

The cross resistance of the different herbicides in triazine or DCMU tolerant plants (for example [49–53] and many others [72]) suggests further subfamilies among the "serine" family. It reflects additional affinities to different amino acid residues that together contribute to the total binding energy. For example, Oettmeier *et al.* [50] showed that in an triazine resistant *Amaranthus* mutant (ser$_{264}$ change) metamitron (an aryl substituted triazinone) looses much less potency than metribuzin (an alkyl substituted triazinone). Similarly Galloway and Mets [49] observed that DCMU actually increased in inhibitory potency in the triazine tolerant AR 207 *Chlamydomonas* mutant (change of phe$_{255}$). Indeed metamitron increases also its potency in the AR 207 mutant alike DCMU (own unpublished results). Although not chemically understood, the phe/tyr change at position 255 contributes to the binding of aryl substituted inhibitors more than to alkyl substituted ones. Understanding these details will eventually clarify the distinct contributions of each amino acid residue in the binding niche to the total binding energy of the inhibitors and how this contribution varies depending on the specific substituents in the chemicals.

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