Binding of Triazines and Triazinones in the Q_B-Binding Niche of Photosystem II

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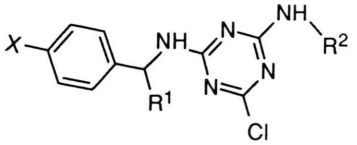
A series of 20 triazines (derivatives of 2-alkylamino-4-benzylamino-6-chloro-1,3,5-triazines) and 37 triazinones (derivatives of 3-alkyl-4-amino-6-phenyl-1,2,4-triazin-5-ones) is tested for inhibitory potency in photosynthetic electron flow through photosystem II of wild type *Chlamydomonas reinhardtii* and of five mutants with aminoacid substitutions in the D1 protein at valine 219, alanine 251, phenylalanine 255, serine 264, and leucine 275. The data are used for computer modelling of the possible location of the compounds within a three dimensional model of the Q_B-binding niche of the D1 protein.

Introduction

A large number of inhibitors, among them commercial herbicides, are known to block photosystem II by displacing plastoquinone from the Q_B-binding niche of the D1 protein in the reaction centre (for review see [1–3]). Structural models for the herbicide-binding niche have been proposed by us [2, 4] and others [5, 6]. Instrumental for modelling the binding of triazines and triazinones has been the change in potency (usually tolerance to the inhibitors) with aminoacid substitutions in the D1 protein [3, 4]. Here we describe the inhibition of mutants of *Chlamydomonas reinhardtii* by a series of triazines (structure **1**) and triazinones (structure **2**) that allows a more detailed description of their orientation in the Q_B-binding niche.



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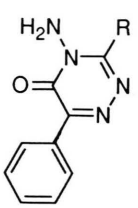

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Materials and Methods

Inhibitors were synthesized in the Bayer Crop Protection Center, Monheim, Chemical Research laboratories.

Chlamydomonas reinhardtii wild type (wt) and D1 protein mutants are described by Wildner *et al.* [7]. Inhibition was measured as inhibition of electron transfer from water to 2,6-dichlorophenolindophenol (DCPIP) in thylakoids [7].

For molecular modelling the software SYBYL from Tripos Associates Inc., St. Louis, Missouri, U.S.A., was used. The construction of the protein model has been described in [4]. The model contains only the amino acids directly surrounding the probable binding locus of the plastoquinone. These amino acids are the last part of the fourth transmembrane helix from serine 207 to serine 222, then a gap and starting again with asparagine 247, the beginning of a small helix which we call "parallel" helix, because its orientation is not transmembrane though not parallel to the membrane, and ending with alanine 277 in the first part of the fifth and last transmembrane helix. Inhibitors and protein were allowed to relax after first arbitrary fitting.

Results

The principle way to obtain precise data for the inhibitory potency of herbicides by testing in isolated thylakoid membranes is well established.

Also the testing of thylakoid systems of *Chlamydomonas reinhardtii* and mutants of it has already been used by ourselves and others several times [4, 7, 8]. The data in Table I for the tolerance of substituted triazines and in Table II of triazinones are in agreement with those for the few triazines and triazinones tested so far in literature [2, 4, 7, 9].

Discussion

Binding of triazines

In analogy to the binding of terbutryne, a triazine herbicide, to the bacterial photosynthetic reaction center [10] we have assumed binding of triazines into the D1 protein to serine 264 hydroxyl and to phenylalanine 265 amide NH [4]. This is in agreement with the D1 protein model of A. Crofts [5], but there is a difference to the recently published model of Ruffle *et al.* [6] in which the amid NH of amino acid 265 is located outwards the binding niche. Fig. 1 shows an ethoxyethyltriazine for an example of our proposition. To strengthen our view we have examined the behaviour of 37 triazines of structure **1** in five different mutants of the D1 protein (Table I). Especially we have varied the substituent R^2 which in the orientation shown is the most enclosed one within the binding niche. If R^2 contains an oxygen atom in

Table I. pI_{50} values and lg *P* values of triazines of structure **1**.
Abbreviations: (*S*) and (*R*) are stereoinformations; THF is tetrahydrofuryl, wt is wild type, A is alanine, F is phenylalanine, I is isoleucine, L is leucine, S is serine, V is valine, Y is tyrosine.

			pI_{50}						
R^2	X	R^1	wt	S264A	A251V	V219I	F255Y	L275F	lg
H	H	(*S*)CH3	6.7	4.8	6.2	6.7	5.0	6.3	2
methyl	H	(*S*)CH3	6.7	4.7	6.2	6.7	5.5	7.5	2
ethyl	H	(*S*)CH3	7.7	6.0	6.5	7.5	6.4	7.8	2
n-propyl	H	(*S*)CH3	7.4	5.3	6.0	7.0	6.2	7.6	3
n-butyl	H	(*S*)CH3	6.5	5.3	6.2	7.5	6.0	7.1	3
n-pentyl	H	(*S*)CH3	7.3	6.0	6.7	7.3	6.6	8.0	4
n-hexyl	H	(*S*)CH3	7.0	6.0	6.5	7.4	7.0	7.3	4
n-septyl	H	(*S*)CH3	8.0	7.0	7.0	7.3	7.6	7.3	5
n-octyl	H	(*S*)CH3	7.8	6.4	7.4	7.1	7.5	7.6	5
n-nonyl	H	(*S*)CH3	7.3	5.3	6.5	7.4	6.6	7.0	6
CH(CH3)–CH3	H	(*S*)CH3	7.1	5.6	6.0	6.8	5.8	7.6	3
CH(CH3)–CH2–CH3	H	(*S*)CH3	6.8	5.6	5.6	6.4	5.7	6.6	3
CH(CH3)–CH2–CH2–CH3	H	(*S*)CH3	7.0	5.8	6.0	7.4	6.3	7.3	4
CH2–CH(CH3)–CH3	H	(*S*)CH3	6.7	5.6	5.3	6.4	5.6	6.6	3
CH2–CH(CH3)–CH2–CH3	H	(*S*)CH3	7.0	5.7	5.7	6.7	6.4	6.8	4
CH2–CH2–CH(CH3)–CH3	H	(*S*)CH3	7.0	6.0	5.8	6.7	6.2	6.4	4
CH2–CH2–O–CH3	H	(*S*)CH3	6.8	4.5	5.8	6.6	5.4	7.3	2
CH2–CH2–O–CH2–CH3	H	(*S*)CH3	7.2	4.8	6.6	7.0	6.2	7.3	2
CH2–CH2–CH2–O–CH3	H	(*S*)CH3	8.3	6.2	6.6	7.7	7.0	8.4	2
CH2–CH2–CH2–O–CH2–CH3	H	(*S*)CH3	7.3	5.5	6.7	7.0	6.5	8.0	3
CH2–CH(O–CH3)–O–CH3	H	(*S*)CH3	5.6	4.2	4.6	5.7	5.4	6.0	2
(*S*)CH(CH3)-phenyl	H	(*S*)CH3	7.7	7.0	6.3	7.2	6.8	7.2	3
(*R*)CH(CH3)-phenyl	H	(*S*)CH3	4.6	4.7	5.0	4.6	4.5	4.5	3
2-(*S*)-THF	H	(*S*)CH3	6.7	4.4	5.3	6.2	5.4	7.0	2
2-(*R*)-THF	H	(*S*)CH3	5.8	4.6	4.8	5.7	5.5	6.4	2
CH2-2-(*S*)-THF	H	(*S*)CH3	6.8	4.6	5.1	6.6	6.0	7.2	2
CH2-2-(*S*)-THF	F	(*S*)CH3	7.7	5.3	5.0	7.4	5.8	8.0	2
CH2-2-(*S*)-THF	F	(*S*)CH2–CH3	8.0	6.5	7.0	8.0	6.5	8.4	3
CH2-2-(*S*)-THF	Cl	(*S*)CH2–CH3	8.0	6.3	7.3	8.5	7.2	8.4	3
CH2-2-(*S*)-THF	F	(*S*)CH2–CH2–CH3	8.1	5.5	7.0	8.0	6.4	8.3	3
CH2-2-(*S*)-THF	Cl	(*S*)CH2–CH2–CH3	8.3	6.5	7.4	8.6	7.5	8.5	4
CH2-2-(*R*)-THF	H	(*S*)CH3	5.7	4.5	5.0	6.4	5.6	6.1	2
CH2-2-(*R*)-THF	F	(*S*)CH3	6.6	5.3	5.6	6.6	5.3	6.3	2
CH2-2-(*R*)-THF	F	(*S*)CH2–CH3	7.0	5.4	5.6	7.6	5.2	7.2	3
CH2-2-(*R*)-THF	Cl	(*S*)CH2–CH3	7.5	6.1	6.3	8.2	5.8	7.8	3
CH2-2-(*R*)-THF	F	(*S*)CH2–CH2–CH3	7.1	5.4	6.2	7.6	5.7	7.7	3
CH2-2-(*R*)-THF	Cl	(*S*)CH2–CH2–CH3	7.6	6.1	6.7	7.7	6.0	7.8	4

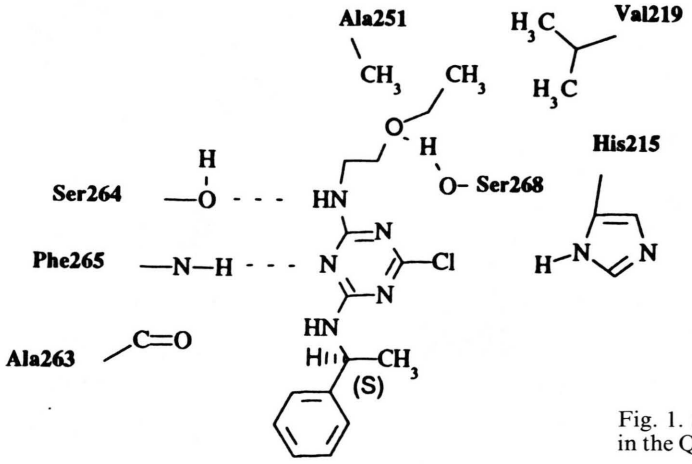

Fig. 1. Sketch of the orientation of ethoxyethyl-triazine in the Q_B-binding niche of photosystem II.

the third or fourth position, a hydrogen bond to serine 268 hydroxyl is possible as shown in Fig. 1. Fig. 2 (see Plate I on page 208) shows a molecular modelling picture of the ethoxyethyl-triazine within the D 1 protein.

The phenyl moiety of the ethoxyethyl-triazine is close to the phenyl ring of phenylalanine 265. The aromatic interaction might explain the high pI_{50} values of all our triazines. This interaction can only be optimal with the (*S*) conformation of the methylbenzyl substituent. But there is enough place to adopt an ethylbenzyl or propylbenzyl group. In agreement with our model are pI_{50} values of similar compounds in wild type reaction centres that have been published by Omokawa and Konnai [9].

The R^2 substituent is oriented into the interior of the binding niche towards alanine 251, serine 268, asparagine 247, leucine 218 and finally valine 219. Molecular modelling suggests that the contact of R^2 to the protein is not very intimate. This is confirmed by the only small effect that variation in chain length of R^2 over a large range has on the pI_{50} values (Fig. 3). The same is true for the pI_{50} values in the mutants serine 264 to alanine and alanine 251 to valine. The decrease of the pI_{50} values of all triazines in the mutant serine 264 to alanine by about two orders of magnitude is explained by loss of a hydrogen bond.

A direct comparison of the locations of the natural substrate plastoquinone and ethoxyethyl-triazine visualizes the rather peripherical position of

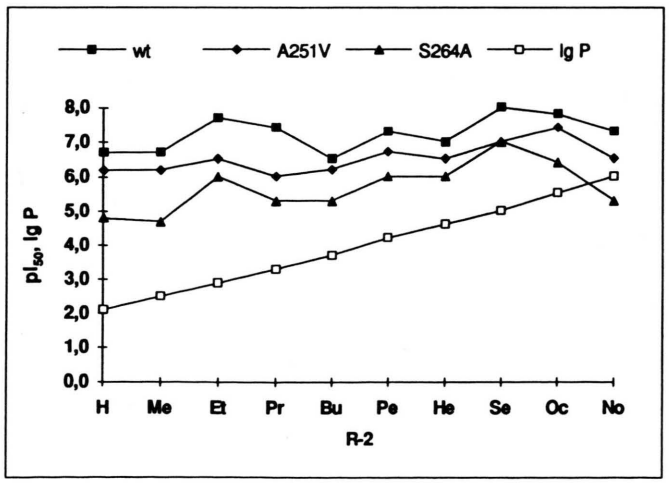

Fig. 3. pI_{50} values and lg *P* values of triazines **1** with different chain lengths in substituents R^2 in wild type (wt) and mutant (ser 264, ala, ala 251 val) thylakoids. R^1 is always (*S*)-methyl and X is hydrogen.

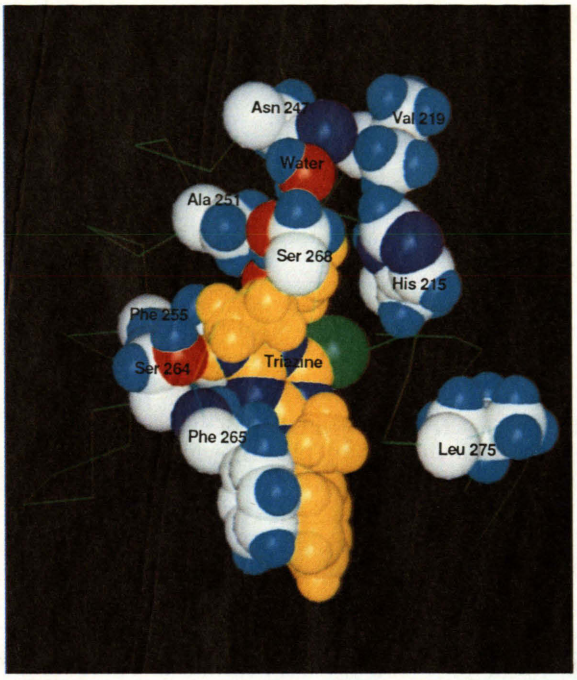

Fig. 2. A partial surface view of ethoxyethyl-triazine (*cf.* Fig. 1) in the Q_B-binding niche of photosystem II. The view is in plane of the membrane with the matrix side at the top. The carbon and hydrogen atoms of ethoxyethyl-triazine are coloured in yellow. Aminoacid side chains shown are (clockwise from left bottom): phe 265, ser 264, phe 255, ala 251, asn 247 a molecule of water and below it ser 268, val 219, his 215, and leu 275. Hydrogen bonds are as shown in Fig. 1.

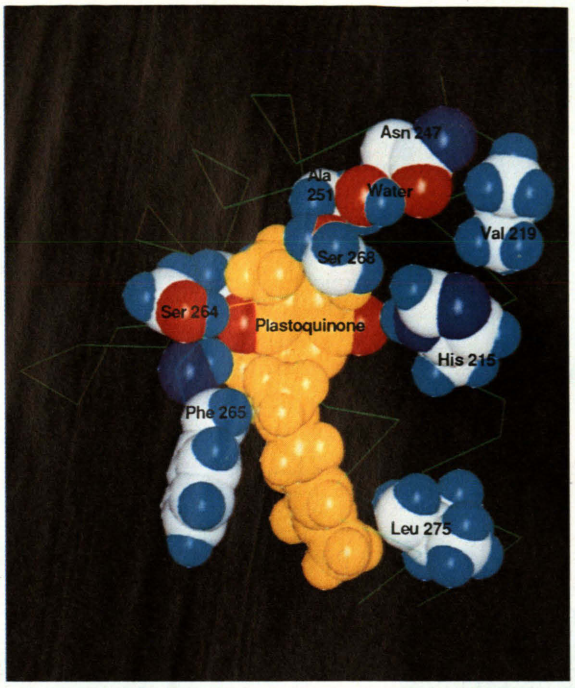

Fig. 5. A partial surface view of plastoquinone in the Q_B-binding niche of photosystem II. The carbon and hydrogen atoms of plastoquinone are coloured in yellow. View and aminoacid side chains shown are as shown in Fig. 2.

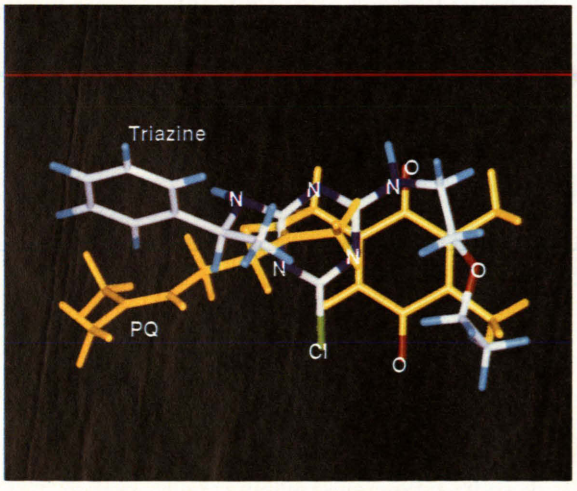

Fig. 4. Direct comparison of the locations of plastoquinone (carbon and hydrogen atoms coloured in yellow, isoprene chain truncated after two monomers) and ethoxyethyl-triazine (*cf.* Fig. 1).

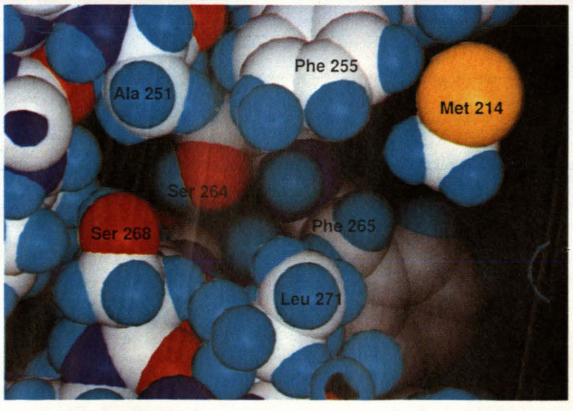

Fig. 6. An interior surface view of the Q_B-binding niche of photosystem II. The view is from his 215 towards ser 264. The plane of the quinone ring is horizontal with the isoprenoid substituent at the right bottom. The dark hydroxyl in the center is of ser 264. Right next to it is the amide NH of phe 265 with the penyl moiety at the right bottom. The bright hydroxyl left of ser 264 is from ser 268. The methyl of ala 251 is above ser 268. Phe 255 is at the top. The thiomethyl of met 214 is at the right top.

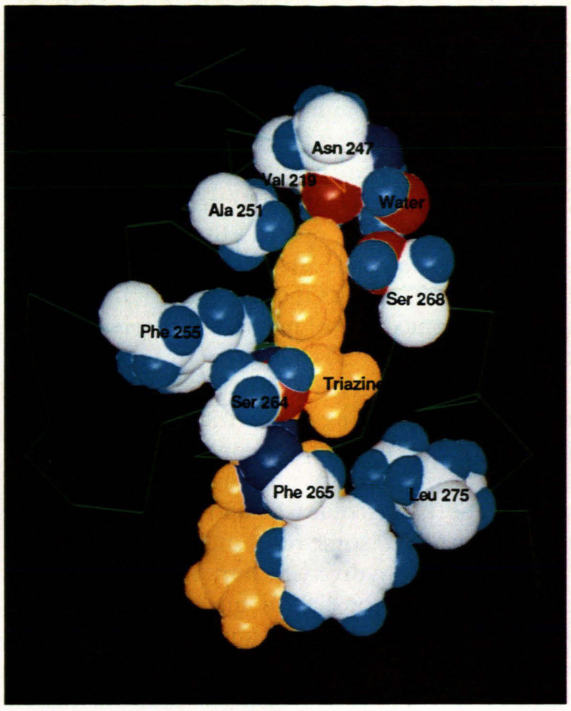

Fig. 7. A partial surface view of phenethyl-triazine (two (*S*)-phenethylamino substituents) in the Q_B-binding niche of photosystem II. The view is in plane of the membrane but perpendicular to the views of the other modelling pictures. The carbon and hydrogen atoms of phenethyl-triazine are coloured in yellow. Aminoacid side chains shown are (clockwise from the bottom): phe 265, ser 264, phe 255, ala 251, asn 247 a molecule of water, ser 268, and leu 275. The upper phenethyl moiety of the inhibitor is located between ala 251 and ser 268.

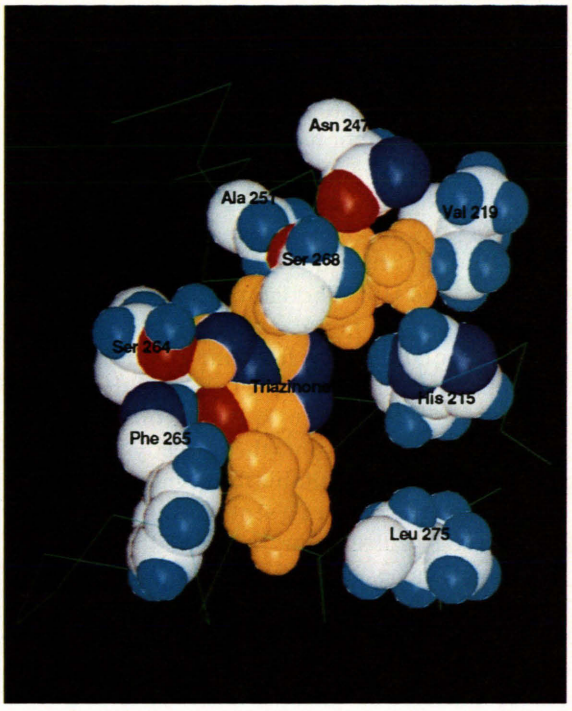

Fig. 10. A partial surface view of pentyl-triazinone (*cf.* Fig. 9) in the Q_B-binding niche of photosystem II. The carbon and hydrogen atoms of pentyl-triazinone are coloured in yellow. View and aminoacid side chains shown are as shown in Fig. 2.

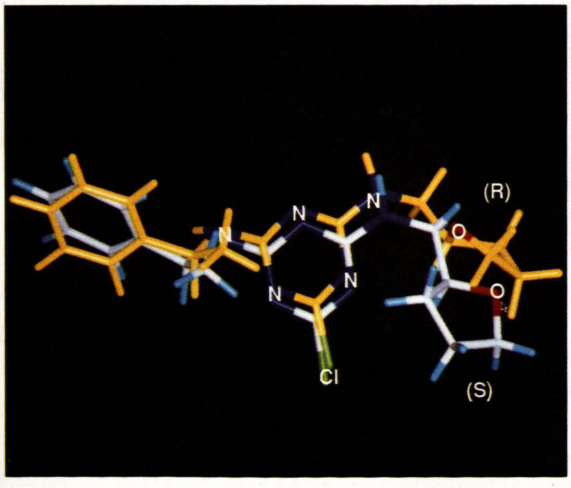

Fig. 8. Direct comparison of the locations of (*S*) tetrahydrofurfuryl-triazine (coloured by atom type, the oxygen hydrogen bound to ser 268 is marked by an arrow) and (*R*)-tetrahydrofurfuryl-triazine (carbon and hydrogen atoms coloured in yellow).

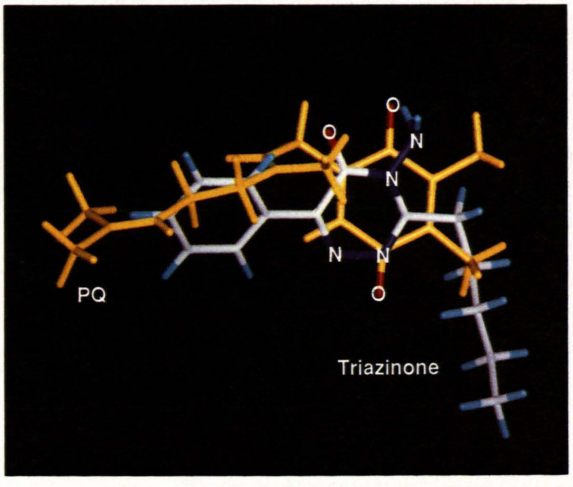

Fig. 11. Direct comparison of the locations of plastoquinone (carbon and hydrogen atoms coloured in yellow, isoprene chain truncated after two monomers) and pentyl-triazinone.

the triazines within the binding niche (Fig. 4, s. Plate I on page 208). In this context it is to consider that in our model the quinone is bound very similar but not identical to its binding in the bacterial photosynthetic reaction center (Fig. 5, s. Plate I on page 208). In the bacterial reaction center the quinone is hydrogen bound with one carbonyl group to serine 223 (corresponding to serine 264 in plants) and to the amide NH of the second following amino acid [10]. Though the amide NH of the second following amino acid in our model is in a hydrogen bond distance to serine 264 hydroxyl, it is not in a position equivalent to that in the bacterial reaction center (Fig. 6, s. Plate I on page 208). Only the amide NH of the directly following phenylalanine 265 is suitable for quinone binding.

For geometric reasons a septyl chain seems to be the longest R^2 substituent, that could fit well into the binding niche. Nevertheless there is no strong decrease in the pI_{50} values for octyl or nonyl chains. Especially the mutation valine 219 to isoleucine would be expected to limit the available space for longer R^2 groups; but it does not. The finding that R^2 without loss in affinity may also be a second (*S*)-phenethyl group leads to an explanation for the observed effects. The triazine with two (*S*)-phenethyl substituents can be fitted nicely into the protein model (Fig. 7, s. Plate II on page 209). This means that triazines with a longer alkyl substituent can bind upside-down with R^2 interchanged with the phenethyl substituent, other than the orientation shown in Fig. 1.

The oxygen in the side chain of the ethoxyethyltriazine can built up a hydrogen bond to the hydroxyl of serine 268. The analogous compound without oxygen in this position, the pentyl-triazine, has a pI_{50} value not significantly different. But there is a difference in lg *P* between the two compounds of 1.6. Since the binding niche is accessable only from the thylakoid membrane, the much lower lipophilicity of the ethoxyethyl group should expectedly lower the pI_{50} value of this compound by the same range [11, 12]. This is not the case because this effect is just compensated by the additional hydrogen bond. For the methoxypropyl-triazine the pI_{50} value is raised even by one more order of magnitude.

In molecular modelling the oxygen atom of the ethoxyethyl group may also be incorporated into a more rigid tetrahydrofurfuryl system without loss of binding. In fact the pI_{50} values of the (*S*)-tetrahydrofurfuryl-triazine are very similar to the ethoxyethyl-triazine. Only the resistance in the alanine 251 to valine mutant is significantly higher, because there is less flexibility in the ring system to accomodate into the changed protein. For the other isomer, the (*R*)-tetrahydrofurfuryl-triazine, molecular modelling predicts that there is no hydrogen bond to serine 268 possible (Fig. 8, s. Plate II on page 209). In fact the pI_{50} value of this isomer is lower by one order of magnitude, corresponding to one hydrogen bond energy.

Mutation of leucine 275 to phenylalanine does not affect the binding of the triazines very much, because there is no intimate contact to the changed atoms. The mutation of phenylalanine 255 to tyrosine in the background behind the triazine ring generally leads to some resistance for the triazines. The additional hydroxyl group of the tyrosine in our model does not directly interact with the triazines. We therefore cannot understand, why there is resistance to the triazines. A contrary phenomenon, a general supersensitivity in the tyrosin 255 mutant, can be observed for the triazinones (*cf.* Table II). It might be, that there happens an unforeseeable slight change in the overall conformation of the binding niche.

Binding of triazinones

We have assumed a binding of triazinones as shown in Fig. 9 [2, 4]. There are hydrogen bonds to serine 264 hydroxyl and to phenylalanine 265 amide NH. Fig. 10 (s. Plate II on page 209) shows a molecular modelling picture of the pentyl-triazi-

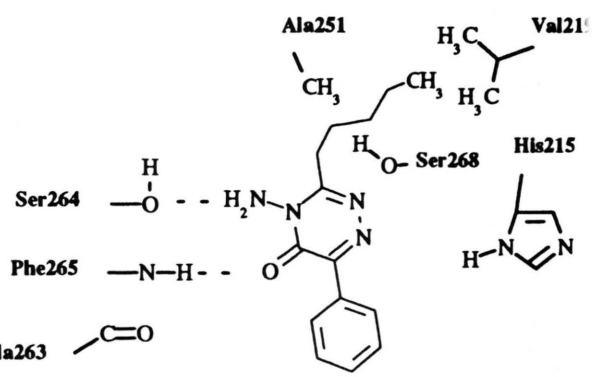

Fig. 9. Sketch of the orientation of pentyl-triazinone in the Q_B-binding niche of photosystem II.

Table II. pI_{50} values and lg *P* values of triazinones of structure **2**.
Abbreviations: as in Table I, nd is not determined.

	pI_{50}						
R	wt	S264A	A251V	V219I	F255Y	L275F	lg *P*
H	4.0	<4	<4	<4	4.3	<4	0.8
methyl	5.5	4.0	3.4	5.4	6.0	4.7	1.0
ethyl	6.4	<4	4.4	5.2	7.1	5.6	1.3
n-propyl	5.2	<4	<4	5.0	6.2	4.6	1.7
n-butyl	6.0	<4	<4	5.7	6.8	5.0	2.1
n-pentyl	6.5	<4	<4	5.7	6.8	5.2	2.5
n-hexyl	4.0	<4	<4	<4	4.4	<4	2.9
CH(CH3)–CH3	6.2	<4	<4	4.5	6.6	5.8	1.7
CH(CH3)–CH2–CH3	5.6	<4	<4	4.5	6.0	4.4	2.1
CH(CH3)–CH2–CH2–CH3	5.0	4.8	4.2	5.1	6.3	5.4	2.5
CH(CH3)–CH2–CH2–CH2–CH3	5.3	4.4	4.3	4.5	6.0	5.3	2.9
CH2–CH(CH3)–CH3	4.3	<4	<4	4.3	5.0	<4	2.0
CH2–CH(CH3)–CH2–CH3	5.2	<4	<4	5.2	5.6	4.0	2.4
CH2–CH2–CH(CH3)–CH3	6.2	<4	4.0	5.8	6.4	4.3	2.4
CH2–CH2–CH2–CH(CH3)–CH3	5.5	<4	4.2	5.4	6.3	4.6	2.8
CH(CH3)–CH(CH3)–CH2–CH3	5.0	<4	4.5	4.8	5.8	4.2	2.8
CH(CH2–CH3)–CH2–CH3	3.0	3.0	3.0	3.0	5.2	3.0	2.4
CH2–CH(CH2–CH3)–CH2–CH3	3.0	3.0	3.0	4.3	5.1	4.3	2.8
CH2–CH2–O–CH3	5.5	<4	<4	5.6	6.4	5.1	1.0
CH2–CH2–CH2–O–CH3	4	<4	nd	nd	nd	nd	1.4
cyc.-Propyl	6.7	4.1	<4	5.0	7.0	6.3	1.5
cyc.-Butyl	6.3	4.0	4.0	5.3	6.7	5.7	1.9
cyc.-Pentyl	5.2	<4	<4	5.0	6.0	5.0	2.2
cyc.-Hexyl	<4	<4	<4	<4	4.0	<4	2.5

none in the Q_B-binding niche. Though both, quinone and triazinone, bind *via* a carbonyl group, our postulated binding of the triazinones is not analogous to quinone-binding. While the quinone binds with the carbonyl to the serine, the triazinones bind with the amino substituent. Fig. 11 (s. Plate II on page 209) directly compares the locations of plastoquinone and pentyl-triazinone, visualizing the rather deep location of the triazinone within the binding niche.

Extended data on 20 triazinones of structure **2** confirm our previous view [2] (Table II) [1]. In comparison to the triazines the triazinones lie much deeper within the binding niche. The triazi-

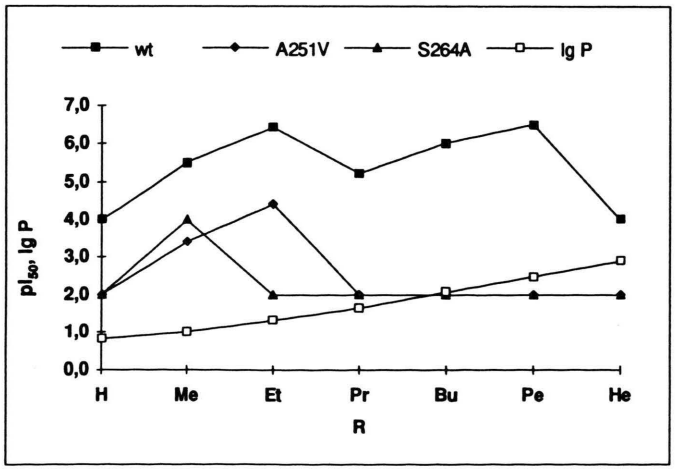

Fig. 12. pI_{50} values and lg *P* values of triazinones **2** with different chain lengths in substituents *R* in wild type (wt) and mutant (ser 264 ala, ala 251 val) thylakoids. pI_{50} values not measurable (smaller than 4) are drawn as 2.0.

nones have a close contact to alanine 251 and serine 268. In consequence not only the mutation serine 264 to alanine (loss of a hydrogen bond), but also the mutation alanine 251 to the larger valine strongly interferes with triazinone binding (Fig. 12). The pentyl-triazinone just fills the available space up to asparagine 247 which is in front of valine 219.

The lower pI_{50} value of propyl-triazinone in comparison to ethyl- or butyl-triazinone is explained, by the bottle neck between alanine 251 and serine 268 [2]. The ethyl group of ethyl-triazinone ends just before alanine 251 and can also accomodate in the valine 251 mutant. Propyl-triazinone is just within the bottle neck and cannot bind as well in the valine 251 mutant. For longer alkyl-triazinones there is some space again after the bottle neck. The 3-methylbutyl-triazinone has a branching point at the position corresponding to the bottle neck. This should prevent good binding of 3-methylbutyl-triazinone. But in fact the pI_{50} value of 3-methylbutyl-triazinone is one order of magnitude better than that of propyl-triazinone and similar to that of butyl-triazinone. Molecular modelling makes it unprobable that there is a flip in orientation for these different triazinones as we postulate for the longer alkyl-triazines. Instead we assume that in the wild type protein there is a molecule of water between the side chains of serine 268 and asparagine 247 [2]. Binding of triazinones with substituents larger than ethyl turn the serine hydroxyl out of the binding niche into the position originally occupied by the water molecule. Then there also is enough place for 3-methylbutyl-triazinone.

Comparison of the pI_{50} values of *n*-butyl and *n*-pentyl-triazinone with methoxyethyl- and methoxypropyl-triazinone demonstrates that the oxygen of methoxyethyl- but not of methoxypropyl-triazinone can reach the serine 268 hydroxyl. This can also be concluded from modelling (*cf.* Fig. 10 and 11). As with the triazines the positive effect of the new hydrogen bond of the oxygen compound on the pI_{50} value is compensated by the lower lg *P* value [11, 12].

The postulated molecule of water next to serine 268 could have a function in conducting protons into the binding niche needed for reduction of plastoquinone. It is also conceivable that there is not a molecule of water, but a molecule of carbonate instead (personal discussion with Govindjee). The carbonate could be held in place by the side chain of the arginine (or lysine) 238, involved in rapid turnover of the D 1 protein [2]. Arginine 238 is next to the azidomonuron labelled tyrosine 237 [13]. This positioning would give a link between herbicide binding, bicarbonate effects, and rapid turnover of the D 1 protein.

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