# On the Orientation of Photosystem II Inhibitors in the $Q_B$-Binding Niche: Acridones, Xanthones and Quinones

Walter Oettmeier, Klaus Masson, Ralf Kloos, and Ellen Reil

Lehrstuhl Biochemie der Pflanzen, Ruhr-Universität,
Postfach 102148, D-W-4630 Bochum 1, Bundesrepublik Deutschland

Z. Naturforsch. **48c**, 146–151 (1993); received December 3, 1992

*Chlamydomonas reinhardtii*, Mutants, D1 Protein, Antibody, Trypsination

The orientation of acridones, xanthones, 1,4-benzo- and naphthoquinones within the photosystem II $Q_B$ herbicide-binding niche was studied by means of mild trypsination and by estimation of $pI_{50}$-values in *Chlamydomonas reinhardtii* D1 mutants ($Val_{219}$ > Ile, $Ala_{251}$ > Val, $Phe_{255}$ > Tyr, $Ser_{264}$ > Ala, $Asn_{266}$ > Thr, and $Leu_{275}$ > Phe). As judged from the R/S-values (ratios of $I_{50}$-values resistant *versus* susceptible type) close to 1 in all mutants, the acridones and xanthones do not have strong interactions with the parent amino acids. Contrary, the quinones exhibit extreme low R/S-values down to 0.003 (for 2,5-dibromo-3-methyl-6-isopropyl-1,4-benzoquinone; DBMIB) in the $Ser_{264}$ mutant. This extreme negative cross resistance or supersensitivity indicates that the quinones do not form a hydrogen bond to the serine hydroxyl group.

## Introduction

The photosystem II reaction center proteins D1 and D2 carry the pigments which are necessary for the primary charge separation. At the secondary quinone acceptor site $Q_B$ photosystem II inhibitors and herbicides can compete with the native plastoquinone for binding which eventually leads to a blockage of photosynthetic electron flow. The $Q_B$-binding site is associated with the D1 protein and, hence, the D1 protein has been formerly called the "herbicide-binding protein" [1–3]. The herbicide-binding protein has been originally identified by the photoaffinity labeling technique. Pfister *et al.* [4] demonstrated that the azido-analogue of the *s*-triazine herbicide atrazine binds to a protein of the approx. molecular weight of 32 kDa. Furthermore, these authors also reported that azido-atrazine did not bind to thylakoids isolated from the atrazine-resistant weed *Amaranthus hybridus*. The reason for this altered binding behaviour was found to be a mutation of $Ser_{264}$ > Gly in the D1 protein [5]. In this way, $Ser_{264}$ was identified as the first amino acid in the D1 protein which participates in herbicide binding. Subsequently, by chemically induced mutagenesis and site-directed mutagenesis in algae a variety of single and multiple D1 mutants have been generated (for review, see [6]). They allowed the mapping of the herbicide-binding region of the D1 protein, which ranges from $Phe_{211}$ to $Leu_{275}$.

The above mentioned D1 mutants exhibit resistance against certain classes of herbicides, like *s*-triazines, triazinones or ureas. However, they show an increased sensitivity against other types of herbicides. This supersensitivity or negative cross resistance has been demonstrated first for the D1 $Ser_{264}$ mutant.

In this mutant some phenolic herbicides like dinoseb, dinoterb or ioxynil exhibit $pI_{50}$-values which are about one order of magnitude higher than in the wild type; *i.e.* the R/S-value (ratio of $I_{50}$-value resistant *versus* susceptible) is <1 [7, 8]. This has led Trebst [2] to a grouping of the "classical" herbicides like triazines, triazinones or ureas into the "$Ser_{264}$-family" and the phenolic herbicides into the "$His_{215}$-family", according to their preferential binding orientation within the $Q_B$-niche.

Negative cross resistance has also been found for certain triazinones in the $Ser_{264}$ > Ala, $Ala_{251}$ > Val, $Phe_{255}$ > Tyr, and $Leu_{275}$ > Phe *Chlamydomonas reinhardtii* mutants [9, 10], for ketonitriles in the same mutants [11] and in the $Phe_{255}$ > Tyr mutant for some ureas [11]. Negative cross resistance indicates that the parent amino acid in the

*Abbreviations:* DCIP, dichlorophenolindophenol; DBMIB, 2,5-dibromo-3-methyl-6-isopropyl-1,4-benzoquinone; SDS-PAGE, sodium dodecylsulfate-polyacrylamide gel electrophoresis.

Reprint requests to Prof. Dr. W. Oettmeier.

Verlag der Zeitschrift für Naturforschung,
D-W-7400 Tübingen
0939–5075/93/0300–0146 $ 01.30/0

wild type strain does not interact with the inhibitor and does not participate in binding. Contrary, resistance points out that the corresponding amino acid is involved in inhibitor binding and that the decreased inhibitory activity results from a loss of a strong bond, presumably a hydrogen bond. R/S-values derived from different mutants can serve, therefore, as a tool to probe the orientation of a certain compound within the $Q_B$-binding niche.

We wish to report here on the orientation of acridones, xanthones and quinones in the photosystem II $Q_B$-binding niche. Acridones have been found most recently to be efficient inhibitors of electron transport through photosystem II [12] and the mitochondrial respiratory chain [13]. They exhibit negative cross resistance in the $Phe_{255}$ mutant only and, thus, are distinct from either the triazine type or phenolic herbicides. Xanthones have not been described as photosystem II inhibitors so far. 1,4-Benzo- and naphthoquinones show negative cross resistance in the $Ser_{264}$ mutant. This supersensitivity, as expressed by the R/S ratio, can be as low as 0.003 for DBMIB, which means a 300-fold increase in inhibitory activity. To our knowledge, this is the lowest R/S ratio reported so far.

## Materials and Methods

The synthesis of the acridones is described in [13], of the xanthones in [14, 15], of the 1,4-benzoquinones in [16] and of the 1,4-naphthoquinones in [17].

Mutants of *Chlamydomonas reinhardtii* (MZ1, MZ2, and MZ4) were obtained and grown as described recently [11]. Strains Ar207 and Dr2 were originally isolated by Galloway and Mets [18] and were kindly provided by Dr. J. D. Rochaix, University of Geneva, Switzerland. Strain NNP1 ($Asn_{266}$ > Thr) was a generous gift by Dr. E. Przibilla [19].

Thylakoids from spinach and *Chlamydomonas reinhardtii* wild type and mutant lines were isolated and assayed for photosystem II activity (DCIP reduction with water as electron donor) in the presence of inhibitors according to previously established protocols [11, 20]. Trypsination of spinach thylakoids was performed as described in [21]. The antibody against the spinach D1 protein was kindly supplied by Dr. U. Johanningmeier [22]. Immunoblots were performed as previously reported [23].

## Results and Discussion

### *Acridones*

We have recently reported on the inhibitory activity of a few acridones on photosystem II electron transport in thylakoids from spinach [12]. $pI_{50}$-values of these acridones together with some of newly synthesized ones in the *Chlamydomonas* system are listed in Table I. It is evident that acridones substituted by four strongly electron withdrawing substituents, like nitro or halogen, are efficient inhibitors. The best ones are bromo-trinitro-acridones, where the 4-isomer ($pI_{50}$-value 7.28) is more active than the 2-isomer ($pI_{50}$-value 6.52).

4-Hydroxy-acridone displaces [$^{14}$C]atrazine and [$^{14}$C]ioxynil competitively from the thylakoid

Table I. $pI_{50}$-Values of various acridones in photosystem II electron transport from susceptible and R/S-values of six herbicide-resistant strains of *Chlamydomonas reinhardtii*.

| No. | -acridone | $pI_{50}$-Value Wild type | R/S Dr2 $Val_{219}$ | MZ2 $Ala_{251}$ | Ar207+ $Phe_{255}$ | MZ1 $Ser_{264}$ | NNP1 $Asn_{266}$ | MZ4 $Leu_{275}$ |
|---|---|---|---|---|---|---|---|---|
| **1** | 2,4,7-Trinitro- | 5.10 | 0.16 | 0.31 | 0.04 | 0.31 | 0.20 | 0.66 |
| **2** | 4-Hydroxy- | 5.33 | 0.06 | 1.36 | 0.16 | 4.55 | 0.91 | 1.00 |
| **3** | 2-*tert.*-Butyl- | 5.61 | 0.22 | 0.80 | 0.10 | 5.60 | 0.88 | 2.00 |
| **4** | 2-*sec.*-Butyl- | 5.70 | 0.18 | 0.50 | 0.18 | 1.90 | 0.53 | 1.00 |
| **5** | 2-Fluoro-4-methyl-5,7-dinitro- | 6.00 | 0.48 | 1.00 | 0.38 | 2.70 | 0.95 | 5.44 |
| **6** | 2,4,5-Trinitro- | 6.07 | 0.94 | 1.12 | 1.12 | 2.55 | 0.83 | 1.00 |
| **7** | 2,4,5,7-Tetranitro- | 6.26 | 1.09 | 0.67 | 0.87 | 0.84 | 1.15 | 0.53 |
| **8** | 1,4-Dichloro-5,7-dinitro- | 6.30 | 0.80 | 0.70 | 0.34 | 1.20 | 0.66 | 2.10 |
| **9** | 2,4-Dichloro-5,7-dinitro- | 6.36 | 0.57 | 1.09 | 1.14 | 1.00 | 0.71 | 1.59 |
| **10** | 2-Bromo-4,5,7-trinitro- | 6.52 | 1.57 | 1.17 | 1.27 | 2.67 | 1.56 | 0.83 |
| **11** | 4-Bromo-2,5,7-trinitro- | 7.28 | 0.72 | 1.54 | 0.85 | 1.27 | 0.73 | 21.8 |

membrane, which indicates that acridones are $Q_B$ site inhibitors [12]. However, this does not allow for discrimination of the acridones as "classical" or phenolic type herbicides.

In this respect, more information can be gained by trypsination experiments. It has been demonstrated that ureas or *s*-triazines can inhibit photosystem II electron transport to a much lesser extent in trypsinated thylakoids as compared to an untreated control [21]. This is shown in Fig. 1 for atrazine and diuron, where the residual electron transport rate after trypsin treatment is much higher as compared to an untreated control. Contrary, inhibition of photosystem II electron transport is not affected after trypsination in the presence of 2,4-dichloro-5,7-dinitro-acridone (No. 9, Table I) (Fig. 1). Therefore, the acridones do not behave like "classical" herbicides. This notion is further corroborated by a direct analysis of the D 1 content of thylakoids after trypsination in the presence and absence of acridones. Fig. 2, lane 1 shows an immunoblot of untreated thylakoid membranes with a spinach D 1 antibody [22]. The content of the D 1 protein is drastically diminished after 10 min of trypsination. 2 μM 1,4-dichloro-5,7-dinitro- (Table I, No. 8), 2,4-dichloro-5,7-dinitro- (Table I, No. 9) and 2-fluoro-4-methyl-5,7-dinitro-acridone (Table I, No. 5) do not protect the D 1 protein during trypsination (Fig. 2, lanes 3, 4, and 5, respectively) because the amount of the D 1 protein is similar to that of lane 2. However, 2 μM diuron exerts a protective effect (Fig. 2, lane 6) [21]. Note the 9 kDa trypsination fragment of the D 1 protein, detectable by the antibody, in lanes 2 to 5.

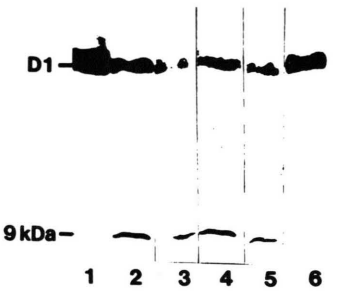


Fig. 2. Immunoblot of thylakoids with an antibody directed against the photosystem II D 1 protein. Thylakoid proteins were separated on a SDS-PAGE (10–15%). Lane 1: control without trypsin; lane 2: incubated with 200 μg trypsin for 10 min; lanes 3–6: incubated with 200 μg trypsin for 10 min in the presence of the following compounds: lane 3: 2 μM 1,4-dichloro-5,7-dinitro-; lane 4: 2 μM 2,4-dichloro-5,7-dinitro-; lane 5: 2 μM 2-fluoro-4-methyl-5,7-dinitro-acridone; lane 6: 2 μM diuron.

A more detailed insight into the orientation of herbicides can be gained by studies with herbicide-resistant mutants. This is demonstrated for acridones in Table I and Fig. 3. In the Dr2 mutant ($Val_{219}$ > Ile) the R/S-value for acridones are about unity or below. This indicates that the acridones do not interact with $Val_{219}$, because the increase in bulk from isopropyl to *sec.* butyl does generally not diminish binding. The same is true

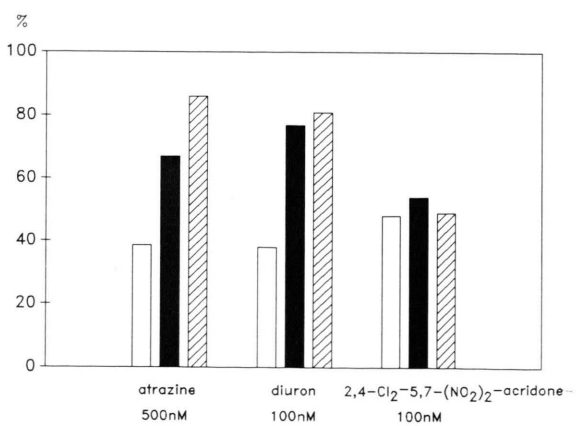


Fig. 1. Photosystem II electron transport activity after addition of various inhibitors in control (white), in 5 min (black) and in 20 min (hatched) trypsin-treated spinach thylakoids.

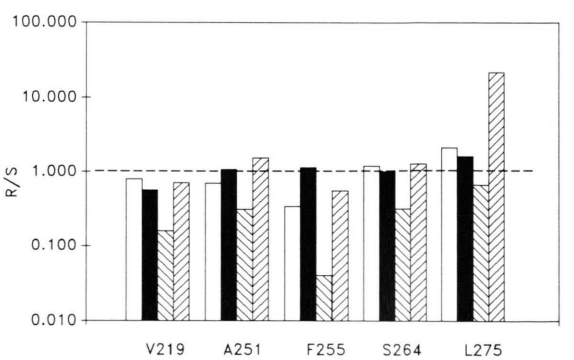


Fig. 3. R/S-Values of 1,4-dichloro-5,7-dinitro- (white), 2,4-dichloro-5,7-dinitro- (black), 2,4,7-trinitro- (left diagonal) and 4-bromo-2,5,7-nitro-acridone (right diagonal) in various *Chlamydomonas reinhardtii* mutants.

for the MZ2 mutant ($Ala_{251}$ > Val), where the increase in bulk is even greater. Similarily, the acridones in the Ar207 + mutant ($Phe_{255}$ > Tyr) show R/S-values which are around 1 or below. Therefore, the increase in bulk caused by the additional hydroxyl group does not lead to a decreased binding. Contrary, an additional hydrogen bond may be formed between the acridone and the Tyr hydroxyl group which facilitates binding. For most acridones resistance can be observed in the MZ1 mutant ($Ser_{264}$ > Ala). However, this resistance is much less pronounced as observed for *s*-triazines or triazinones, where it amounts to several orders of magnitude [8]. Consequently, the hydrogen bond to the serine hydroxyl group does not play a crucial role for the binding of acridones. Replacement of $Leu_{275}$ > Phe in the MZ4 mutant also leads to a slight resistance, but also to supersensitivity in some cases. For all acridones tested so far, negative cross resistance is observed in the NNP1 mutant ($Asn_{266}$ > Thr). In this case, an additional hydrogen group may be formed between the threonine hydroxyl group and the acridone, which stabilizes the binding. In conclusion, no really dramatic effects on acridone binding are observed in the various mutants. Furthermore, acridones in respect to their properties in the mutants cannot be regarded as a homogeneous group, because the R/S-values depend on the substitution pattern.

*Xanthones*

Xanthones (xanthen-9-ones) have not been described as photosystem II inhibitors so far. They can be visualized as acridones, where the imino group in the central ring is replaced by an oxygen bridge. Like the acridones, xanthones are efficient inhibitors if they are substituted by strongly electron withdrawing substituents (Table II). 2,7-Dibromo-4,5-dinitro-xanthone (No. 8, Table II) is the most potent xanthone inhibitor ($pI_{50}$-value 6.31). In the R/S-pattern of xanthones (Table II) no general tendency can be recognized. In some mutant strains they exhibit slight resistance but also some negative cross resistance. In most cases, the R/S-values are close to unity. It can be concluded that the xanthones like the acridones do not have strong interactions with any of the amino acids which are replaced in the mutants.

*Quinones*

Like acridones and xanthones, benzo- and naphthoquinones do not exhibit a homogeneous pattern in respect to their R/S-values (Tables III and IV, Fig. 4). However, one striking feature is immediately evident. In the MZ1 mutant ($Ser_{264}$ > Ala) all quinones tested so far show extreme negative cross resistance, which reaches up to almost three orders of magnitude, for example DBMIB (No. 3, Table III). DBMIB is an inhibitor of the cytochrome $b_6/f$-complex [24] and with a $pI_{50}$-value of 4.70 (Table III) is only a weak photosystem II inhibitor. In the $Ser_{264}$ > Ala mutant its $pI_{50}$-value is raised to 7.18 and it becomes a powerful photosystem II inhibitor in this mutant. Negative cross resistance in the $Ser_{264}$ mutant has so far been reported for phenolic herbicides and some cyanoacrylates and triazinones [8, 9, 11]. For *s*-triazines and some triazinones the hydrogen bond between serine hydroxyl and a nitrogen within the *s*-triazine or triazinone moiety plays a crucial role. If

Table II. $pI_{50}$-Values of various xanthones in photosystem II electron transport from susceptible and R/S-values of six herbicide-resistant strains of *Chlamydomonas reinhardtii*.

| No. | -xanthone | $pI_{50}$-Value | R/S | | | | |
|---|---|---|---|---|---|---|---|
| | | Wild type | Dr2 $Val_{219}$ | MZ2 $Ala_{251}$ | Ar207+ $Phe_{255}$ | MZ1 $Ser_{264}$ | NNP1 $Asn_{266}$ | MZ4 $Leu_{275}$ |
| **1** | 2-*tert.*-Butyl- | 4.79 | 0.14 | 3.16 | 0.31 | 0.41 | 0.46 | 1.75 |
| **2** | 2-*sec.*-Butyl- | 4.82 | 0.23 | 5.03 | 0.34 | 2.53 | 0.46 | 3.71 |
| **3** | 2,4-Dichloro-1,7-dinitro- | 5.59 | 1.66 | 0.54 | 0.93 | 2.25 | 0.73 | 1.39 |
| **4** | 2,4,7-Trinitro- | 5.83 | 1.23 | 0.65 | 1.09 | 1.62 | 1.28 | 0.62 |
| **5** | 2-Chloro-4,7-dinitro- | 5.88 | 1.61 | 1.34 | 2.86 | 2.24 | 3.63 | 0.91 |
| **6** | 2-Chloro-5,7-dinitro- | 6.03 | 1.29 | 1.12 | 2.52 | 1.61 | 2.67 | 1.32 |
| **7** | 2,7-Dichloro-4,5-dinitro- | 6.11 | 2.35 | 1.27 | 2.82 | 3.37 | 4.03 | 0.95 |
| **8** | 2,7-Dibromo-4,5-dinitro- | 6.31 | 2.30 | 1.19 | 2.33 | 5.24 | 6.46 | 1.42 |

Table III. $pI_{50}$-Values of various 1,4-benzoquinones in photosystem II electron transport from susceptible and R/S-values of six herbicide-resistant strains of *Chlamydomonas reinhardtii*.

| No. | 1,4-benzoquinone | $pI_{50}$-Value Wild type | R/S Dr2 $Val_{219}$ | MZ2 $Ala_{251}$ | Ar207+ $Phe_{255}$ | MZ1 $Ser_{264}$ | NNP1 $Asn_{266}$ | MZ4 $Leu_{275}$ |
|---|---|---|---|---|---|---|---|---|
| **1** | Tribromomethyl- | 4.44 | 0.92 | 0.89 | 0.69 | 0.014 | * | 0.28 |
| **2** | 2,5-Dichloro-5-*tert.*-butyl- | 4.49 | 0.44 | 0.53 | 0.59 | 0.028 | 0.53 | 0.53 |
| **3** | 2,5-Dibromo-3-methyl-6-isopropyl- | 4.70 | 0.70 | 0.75 | 0.75 | 0.003 | 0.24 | 0.65 |
| **4** | Tetrachloro- | 4.74 | 1.78 | 0.94 | 0.18 | 0.039 | 0.08 | 0.24 |
| **5** | 2,5-Dibromo-3,6-diisopropyloxy- | 4.77 | 1.18 | 0.71 | 0.82 | 0.015 | 1.37 | 0.41 |
| **6** | 2,3-Dibromo-5-*tert.*-butyl- | 5.53 | 1.79 | 11.07 | 1.64 | 0.100 | 4.00 | 3.93 |
| **7** | Tetrabromo- | 5.92 | 1.60 | 3.13 | 1.07 | 0.047 | 0.78 | 0.25 |
| **8** | Tetraiodo- | 6.76 | 1.49 | 0.74 | 0.34 | 0.049 | 1.18 | 0.48 |
| **9** | 2,3-Diiodo-5-*tert.*-butyl- | 6.85 | 3.07 | 17.86 | 1.64 | 0.464 | 3.76 | 35.7 |

* Not determinable.

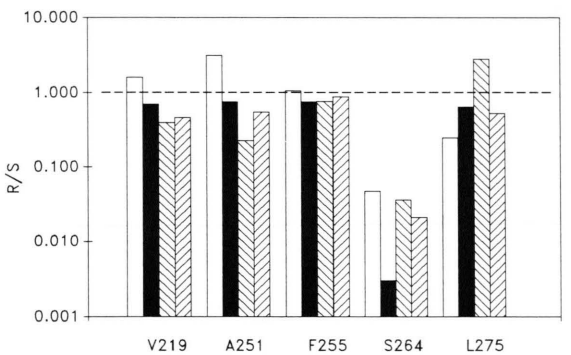


Fig. 4. R/S-Values of tetrabromo-1,4-benzoquinone (white), DBMIB (black), 2,3-dibromo-1,4-naphthoquinone (left diagonal) and 2,3-diiodo-1,4-naphthoquinone (right diagonal) in various *Chlamydomonas reinhardtii* mutants.

this hydrogen bond is lost in the $Ser_{264}$ > Ala mutant, the result is a complete loss of specific binding and a R/S-value which may be as high as more than three orders of magnitude [8]. Consequently, quinone inhibitors though resembling in their structure the native plastoquinone $Q_B$ occupant do not form a hydrogen bond to $Ser_{264}$. In this respect, quinones can be grouped into the "$His_{215}$-family". As already stressed, quinones in their R/S-pattern cannot be considered as a homogeneous group. This is clearly evident by comparison of DBMIB (Table III, No. 3) and 2,3-dibromo-5-*tert.*-butyl-1,4-benzoquinone (Table III, No. 6). DBMIB exhibits negative cross resistance in all mutants, whereas 2,3-dibromo-5-*tert.*-butyl-1,4-benzoquinone shows resistance, except for the MZ1 mu-

Table IV. $pI_{50}$-Values of various 1,4-naphthoquinones in photosystem II electron transport from susceptible and R/S-values of six herbicide-resistant strains of *Chlamydomonas reinhardtii*.

| No. | 1,4-naphthoquinone | $pI_{50}$-Value Wild type | R/S Dr2 $Val_{219}$ | MZ2 $Ala_{251}$ | Ar207+ $Phe_{255}$ | MZ1 $Ser_{264}$ | NNP1 $Asn_{266}$ | MZ4 $Leu_{275}$ |
|---|---|---|---|---|---|---|---|---|
| **1** | 2-Bromo- | 4.24 | 0.18 | 0.74 | 0.36 | 0.086 | 0.47 | 0.72 |
| **2** | 2-Chloro-3-methyl- | 5.00 | 0.26 | 1.20 | 0.83 | 0.070 | 0.35 | 1.10 |
| **3** | 2-Chloromethyl-3-chloro- | 5.00 | 0.61 | 0.42 | 0.74 | 0.080 | 1.07 | 1.96 |
| **4** | 2,3-Dichloro- | 5.33 | 0.55 | 0.36 | 0.74 | 0.034 | 1.00 | 1.65 |
| **5** | 2-Bromo-3-methyl- | 5.38 | 0.61 | 1.55 | 0.74 | 0.214 | 0.39 | 0.60 |
| **6** | 2,3-Dibromo- | 5.76 | 0.40 | 0.23 | 0.77 | 0.036 | 0.95 | 2.80 |
| **7** | 2-Bromo-3-*n*-heptyl- | 5.80 | 0.81 | 0.88 | 0.61 | 0.250 | 1.25 | 1.06 |
| **8** | 2-Bromo-3-*i*-propyl- | 5.89 | 1.59 | 1.31 | 1.50 | 0.308 | 1.30 | 1.62 |
| **9** | 2-Bromo-3-benzyl- | 6.00 | 1.23 | 0.80 | 0.80 | 0.650 | 1.20 | 1.20 |
| **10** | 2,3-diiodo- | 6.10 | 0.46 | 0.55 | 0.88 | 0.081 | 0.48 | 0.63 |

tant. It is noteworthy that both benzoquinones with a *tert.*-butyl group in the 5-position (No. 6 and 9, Table III) exhibit strong resistance in the MZ2 mutant. Obviously, the change from $Ala_{251}$ to Val does not allow for accommodation of the bulky *tert.*-butyl group and leads to a decreased binding.

As already stressed, all naphthoquinones display supersensitivity in the $Ser_{264}$ mutant. However, in all other mutants the individual naphthoquinones exhibit either resistance or supersensitivity, though the R/S-values are always close to unity. It will be beyond the scope of this paper to analyze all compounds in detail. Obviously, steric interactions will be responsible in most cases and molecular modelling studies will lead to a clearer picture in the future.

*Acknowledgements*

This work was supported by Deutsche Forschungsgemeinschaft.

[1] A. Trebst, Z. Naturforsch. **41c,** 240–245 (1986).
[2] A. Trebst, Z. Naturforsch. **42c,** 742–750 (1987).
[3] K. G. Tietjen, J. F. Kluth, R. Andree, M. Haug, M. Linding, K. H. Müller, H. J. Wroblowsky, and A. Trebst, Pest. Sci. **31,** 65–72 (1990).
[4] K. Pfister, K. E. Steinback, G. Gardner, and C. J. Arntzen, Proc. Natl. Acad. Sci. U.S.A. **78,** 981–985 (1981).
[5] J. Hirschberg and L. Macintosh, Science **222,** 1346–1349 (1983).
[6] W. Oettmeier, in: Topics in Photosynthesis (J. Barber, ed.), **Vol. 11,** pp. 349–408, Elsevier, Amsterdam, London, New York, Tokyo 1992.
[7] K. Pfister and C. J. Arntzen, Z. Naturforsch. **34c,** 996–1009 (1979).
[8] W. Oettmeier, K. Masson, C. Fedtke, J. Konze, and R. R. Schmidt, Pestic. Biochem. Physiol. **18,** 357–367 (1982).
[9] W. Oettmeier, U. Hilp, W. Draber, C. Fedtke, and R. R. Schmidt, Pestic. Sci. **33,** 399–409 (1991).
[10] A. Trebst, J. Kluth, K. Tietjen, and W. Draber, in: Pesticide Chemistry (H. Frehse, ed.), pp. 111–120, VCH Verlagsgesellschaft, Weinheim 1991.
[11] G. F. Wildner, U. Heisterkamp, and A. Trebst, Z. Naturforsch. **45c,** 1142–1150 (1990).
[12] W. Oettmeier, K. Masson, R. Kloos, and D. Godde, in: Research in Photosynthesis (N. Murata, ed.), **Vol. 3,** pp. 563–566, Kluwer Academic Publishers, Dordrecht 1992.
[13] W. Oettmeier, K. Masson, and M. Soll, Biochim. Biophys. Acta **1099,** 262–266 (1992).
[14] A. A. Goldberg and H. A. Walker, J. Chem. Soc. **1953,** 1348–1357.
[15] N. Dhar, J. Chem. Soc. **117,** 1053–1070 (1920).
[16] W. Oettmeier, S. Reimer, and K. Link, Z. Naturforsch. **33c,** 695–703 (1978).
[17] W. Oettmeier, C. Dierig, and K. Masson, Quant. Struct.-Act. Relat. **5,** 50–54 (1986).
[18] R. E. Galloway and L. J. Mets, Plant Physiol. **74,** 469–474 (1984).
[19] E. Przibilla and R. Yamamoto, in: Regulation of Chloroplast Biogenesis (J. H. Argyroudi-Akoyunoglou, ed.), pp. 561–565, Plenum Press, New York 1992.
[20] W. Oettmeier, D. Godde, B. Kunze, and G. Höfle, Biochim. Biophys. Acta **807,** 216–219 (1985).
[21] A. Trebst, B. Depka, B. Kraft, and U. Johanningmeier, Photosynth. Res. **18,** 163–177 (1988).
[22] U. Johanningmeier, Z. Naturforsch. **42c,** 755–757 (1987).
[23] R. Geiger, R. J. Berzborn, B. Depka, W. Oettmeier, and A. Trebst, Z. Naturforsch. **42c,** 491–498 (1987).
[24] A. Trebst, E. Harth, and W. Draber, Z. Naturforsch. **25b,** 1157–1159 (1970).