Table II. Concentration of Ethalfluralin and Trifluralin Measured with a GC–MS-NCI and a GC-ECD[a]

rate, kg/ha	days after treatment	concentration, ng/g of oven-dry soil GC–MS-NCI	GC-ECD
		Ethalfluralin	
1.0	199	46	50
1.5	199	124	129
1.0	555	12	12
1.5	555	16	23
standard error of mean for methods of analysis = 2.6			
		Trifluralin	
1.0	199	122	126
1.5	199	206	192
1.0	555	57	65
1.5	555	89	85
standard error of mean for methods of analysis = 4.8			

[a] Differences in concentration of trifluralin and ethalfluralin due to rates and sampling dates were significant and differences between GC–MS-NCI and GC-ECD were not significant ($P = 0.05$) by analysis of variance.

(Lethbridge Research Station, unpublished data). Thus, the GC–MS-NCI can be used to confirm levels of ethalfluralin and trifluralin residues that are toxic to plants.

The ability to analyze trifluralin and ethalfluralin with the GC–MS-NCI indicates that this system has potential for rapidly confirming structures and concentrations of several pesticides with a positive electron affinity in soil extracts.

ACKNOWLEDGMENT

We thank Drs. D. L. Struble and W. G. Taylor at the Agriculture Canada Research Station, Lethbridge, Alberta, for valuable advice on quantitatively measuring the herbicides with the GC–MS-NCI.

Registry No. Ethalfluralin, 55283-68-6; trifluralin, 1582-09-8.

LITERATURE CITED

Biros, F. J. *Residue Rev.* **1971**, *40*, 1.

Biros, F. J.; Cummings, J. C., Eds. "Pesticide Analytical Manual"; U.S. Department of Health, Education, and Welfare: Washington, DC, 1970; Vol. I.

Biros, F. J.; Cummings, J. C., Eds. "Pesticide Analytical Manual"; U.S. Department of Health, Education, and Welfare: Washington, DC, 1973; Vol. II (revised).

Byast, T. H.; Cotterill, E. G.; Hance, R. J. "Technical Report No. 15", 2nd ed.; Weed Research Organization: Begbroke Hill, England, 1977.

Dougherty, R. C.; Piotrowski, K. *J. Assoc. Off. Anal. Chem.* **1976**, *59*, 1023.

Hunt, D. F.; Stafford, G. C., Jr.; Crow, F. W.; Russell, J. W. *Anal. Chem.* **1976**, *48*, 2093.

Kuehl, D. W.; Whitaker, M. J.; Dougherty, R. C. *Anal. Chem.* **1980**, *52*, 935.

Moyer, J. R.; Hamman, W. M. "Research Report ECW Western Canada"; Expert Committee on Weeds: Regina, Saskatchewan, 1980; Vol. 3, p 118.

Smith, A. E. *J. Agric. Food Chem.* **1981**, *29*, 111.

Zweig, G.; Sherma, J., Eds. "CRC Handbook of Chromatography"; CRC Press: Cleveland, OH, 1972; Vol. II.

Received for review October 27, 1982. Revised manuscript received August 16, 1983. Accepted April 2, 1984.

Herbicidal Mode of Action on Chlorophyll Formation

Gerhard Sandmann,* Hildegard Reck, and Peter Böger

Oxadiazon [2-*tert*-butyl-4-(2,4-dichloro-5-isopropoxyphenyl)-Δ^2-1,3,4-oxadiazolin-5-one], MK-616 [*N*-(4-chlorophenyl)-3,4,5,6-tetrahydrophthalimide], and DTP [1,3-dimethyl-4-(2,4-dichlorobenzoyl)-5-hydroxypyrazole] exert phytotoxic activity by interfering with chlorophyll biosynthesis as a primary mode of action. When *Scenedesmus* is grown in the dark with these herbicides present, they interfere with chlorophyll biosynthesis, whereas carotene biosynthesis is unaffected. In addition, the content of plastidic cytochrome *c*-553 decreases. Peroxidative activity of oxadiazon (assayed by ethane evolution or degradation of ^{53}S-prelabeled sulfolipid) is very low at a concentration of 1 μM oxadiazon, which is the I_{50} value for inhibition of chlorophyll formation. With 10 μM oxadiazon present, peroxidative properties are evident. The fluorescence-induction kinetics of *Scenedesmus* grown with 1 μM oxadiazon present are different from those obtained with norflurazon [4-chloro-5-(methylamino)-2-[3-(trifluoromethyl)phenyl]pyridazin-3(2*H*)one] or the peroxidative herbicide oxyfluorfen [2-chloro-4-(trifluoromethyl)phenyl]3-ethoxy-4-nitrophenyl ether]. In contrast to the latter two, the signal shape is preserved with oxadiazon, while the signal height diminishes in oxadiazon-treated cells due to a decrease of the chlorophyll content.

Little information is available on the mode of action of many commercial herbicides. One of these compounds is oxadiazon, which is used for weed control in rice and soybean as well as in orchards and vineyards (Burgaud et al., 1969; Ambrosi and Desmoras, 1973). In a recent publication, evidence was presented that oxadiazon alters the pigment composition of the chloroplast (Sandmann and Böger, 1983a). So-called "bleaching" herbicides can exert their influence by either inhibiting biosynthesis of (1) chlorophyll or (2) carotenoids and (3) by causing destruction of pigments already formed (Sandmann and Böger, 1982a).

In this paper, data are presented indicating that a primary target of oxadiazon is the biosynthetic pathway of chlorophylls. In short-term experiments, peroxidative properties of this herbicide were negligible when using concentrations of 1 μM. In addition, phytotoxic activities of substituted hydroxypyrazoles (Moon et al., 1977) and

Lehrstuhl für Physiologie und Biochemie der Pflanzen, Universität Konstanz, D-7750 Konstanz, West Germany.

0021-8561/84/1432-0868$01.50/0

of a tetrahydrophthalimide derivative (Wakabayashi et al., 1979) are—at least in part—also due to inhibition of chlorophyll formation.

MATERIALS AND METHODS

Sterile liquid cultures of *Scenedesmus acutus* (strain 276-3a, Algal Culture Collection, University of Göttingen) were grown either autotrophically in the light or heterotrophically in darkness with glucose and yeast extract present, as previously described (Sandmann and Böger, 1981). Cultivation was carried out in 1-L Fernbach flasks by using carbon dioxide enriched air (3% v/v) at 22 °C on a shaker, with white fluorescent light of 45 W/m^2. Growth of the culture was determined by measuring packed cell volume (pvc) in calibrated microcentrifuge tubes of 80-μL of packed cell volume/mL of culture volume, reaching a figure of approximately 3 with the control after a 48-h growth period.

Oxygen evolution was routinely checked in a Clark-type oxygen electrode at light-saturating conditions (400 W/m^2; Böger and Schlue, 1976). All herbicidal compounds (99% pure) were added from 10 mM stock solutions in methanol or methanol–dimethyl formamide, 1:1 (v/v). The concentrations of organic solvents in the cultures were always less than 0.1% (v/v). Chlorophyll was determined after methanol extraction at 65 °C of cells according to Mackinney (1940). Extraction and partition of total colored carotenoids were improved as compared to our previous procedure. Cells were extracted with methanol for 20 min at 65 °C, containing 6% (w/v) KOH. After cooling, total carotenoids were transferred into petroleum ether (bp 60–80 °C) containing 10% (v/v) diethyl ether, and this procedure was repeated twice. Quantification was done by optical absorption. All other details of this procedure remained as noted by Sandmann and Böger (1983b).

Fluorescence induction was measured at 20 °C in a single-beam instrument with excitation light from a He–Ne laser (632.8 nm; 30 W/m^2). Emission of fluorescent light was measured above 715 nm with an RG 715 cutoff filter (Schott, Mainz). Resolution of the kinetics was down to 5 ms. Details of the setup are given by Karapetyan et al. (1983).

The peroxidative activity of oxadiazon was measured either by degradation of radioactive prelabeled ^{35}S-sulfolipid or by evolution of ethane (Sandmann and Böger, 1980a). Labeled sulfolipid was formed by growing *Scenedesmus* with 10 μCi/L [^{35}S]K_2SO_4 present in the nutrient medium before the herbicide was added (Sandmann and Böger, 1982b). Both procedures are routinely used in our laboratory for estimating the peroxidative activity of various herbicides, e.g., oxyfluorfen or paraquat (Sandmann and Böger, 1982a).

Determination of cytochrome *c*-553, preparation of cell homogenates, extraction of redox proteins, and enrichment by ammonium sulfate precipitation was according to Sandmann and Böger (1980b). Cytochrome *c*-553 was measured by difference absorption spectroscopy after chemical oxidation and reduction.

The compounds assayed in this paper are given in Table II by formulas and chemical names. Data are means of at least five independent experiments, with a tolerance of ±7%, in case the standard error is not given.

RESULTS

Application of oxadiazon at concentrations of 0.01 μM and higher to autotrophic *Scenedesmus* cells resulted in a decreased chlorophyll content (Figure 1A). An I_{50} value of 0.9 μM was determined by a modified Dixon plot (Figure 1B). The kinetics of oxadiazon action on chlorophyll, carotenoids, photosynthetic oxygen evolution, and cell

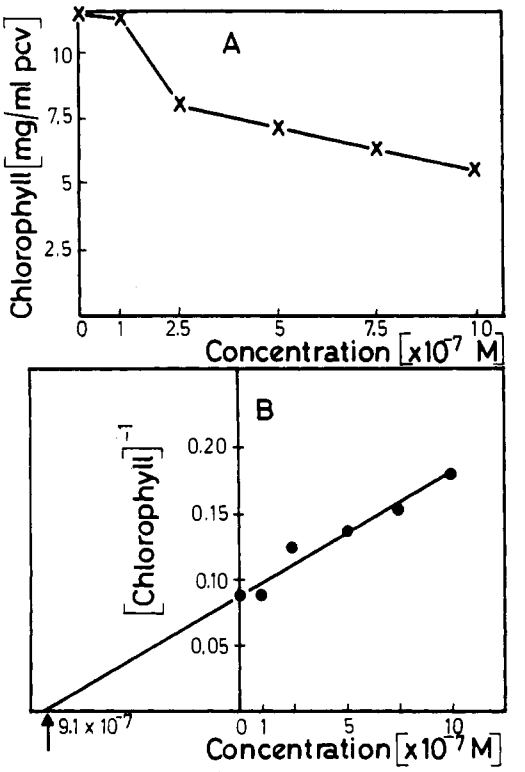


Figure 1. Formation of chlorophyll in oxadiazon-treated autotrophic cultures of *Scenedesmus* (A) and Dixon transformation of (A) for I_{50} determination (B) after 48 h.

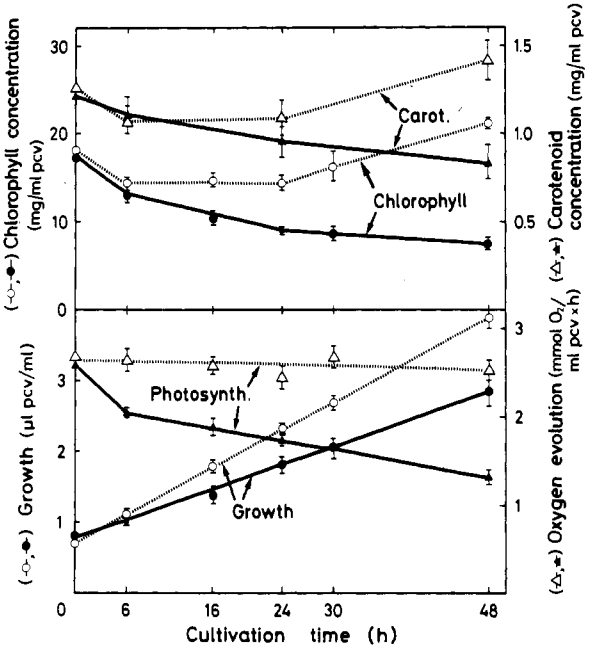


Figure 2. Pigment concentration and photosynthesis during autotrophic growth of *Scenedesmus* in control (open symbols) and oxadiazon-treated culture (1 μM; closed symbols).

growth is demonstrated in Figure 2. Both chlorophyll and carotenoid concentrations in oxadiazon-treated cells declined in parallel beyond 6 h. An incubation period of 6 h already affected photosynthetic electron transport at saturating-light conditions (400 W/m^2). However, growth under light intensities of 45 W/m^2 was not yet influenced.

A direct effect of oxadiazon on photosynthesis of *Scenedesmus* cells within 1 h, or on isolated spinach chloroplasts, was not observed with concentrations up to 10 μM. At the end of a 48-h experiment, the autotrophic culture with 1 μM oxadiazon present contained only 58%

Table I. Total Amount of Chlorophyll and Colored Carotenoids (mg/L of Suspension) in an Autotrophic *Scenedesmus* Culture Grown with Oxadiazon Present

pigments	additions	cultivation time: at start	24 h	48 h
chlorophyll	control	17.5	27.8	64.3
	+oxadiazon, 1 μM	18.6	18.6	19.4
	+oxadiazon, 10 μM	15.8	15.6	10.8
colored carotenoids	control	1.10	2.76	5.56
	+oxadiazon, 1 μM	1.04	1.12	1.76
	+oxadiazon, 10 μM	1.15	1.06	0.51

and 36% of carotenoids and chlorophyll, respectively, as compared to an untreated culture; oxygen evolution was inhibited by 50% and cell growth by 25% of the control. Higher concentrations or longer treatment with oxadiazon increased these effects.

Table I demonstrates the total amount of chlorophyll and colored carotenoids per liter of autotrophic cultures grown with 1 or 10 μM oxadiazon. In contrast to that of the control, the chlorophyll content of the culture (per volume) remained more or less the same over 48 h and carotenoids increased slightly with the 1 μM treatment. With 10 μM oxadiazon, however, the amount of both chlorophyll and carotenoids decreased below the initial values at the start.

In illuminated autotrophic cultures, inhibition of carotenoid biosynthesis affects the chlorophyll level (Sandmann and Böger, 1982a), and vice versa inhibition of chlorophyll formation might affect carotenoid content. In order to differentiate between the primary mode of action and a possible secondary, photodestructive effect on pigments of oxadiazon and the other herbicidal compounds indicated, *Scenedesmus* was cultured under heterotrophic conditions (in the dark) with glucose (Table II). Then, in contrast to autotrophic growth, oxadiazon had no substantial effect on total carotenoids, whereas the chlorophyll concentration was decreased, though to a smaller extent than under the autotrophic conditions of Figure 2.

Among the other compounds assayed, MK-616 and DTP also showed an effect on chlorophyll content. MK-616 was most effective while the activity of DTP was comparable to that of oxadiazon. None of these altered the carotenoid concentration. Levulinic acid, known as a competitive inhibitor of δ-aminolevulinic acid dehydratase, was used as a reference. Again, this compound decreased the chlorophyll level in heterotrophic cultures without affecting carotenoids. The other imidazole and pyrazole derivatives, metronidazole and SW-751, did not interfere with the chlorophyll content. In the case of W-1440, there was only a small difference as compared with the control. For comparison, the last line of Table II demonstrates the influence of norflurazon in the dark. There was some decrease of the chlorophyll level but a much stronger one of carotenoids.

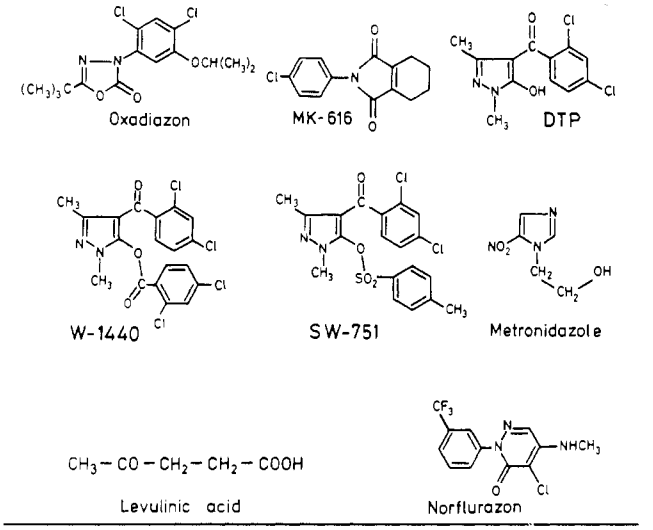
The pathway from δ-aminolevulinic acid to protoporphyrin IX is the common biosynthetic sequence to both chlorophyll and the heme moiety of cytochromes. In Table III, we assayed for the influence of oxadiazon on plastidic cytochrome *c*-553, which is formed in *Scenedesmus* cells grown in low-copper mediums (0.01 μM Cu^{2+}; Sandmann and Böger, 1980b). Indeed, the cytochrome *c*-553 concentration was decreased to about 60% by oxadiazon in autotrophic and heterotrophic cells.

The experiments of Table IV were performed to determine a possible peroxidative activity of oxadiazon that may lead to chlorophyll destruction, as is known for certain *p*-nitrodiphenyl ethers. Cells precultivated for 2 days in the presence of [^{35}S]sulfate were washed free of radioactive sulfate, divided into aliquots, and grown for another 2 days in normal medium. To one of the aliquots 1 μM oxadiazon was added. Radioactivity in (plastidic) sulfolipid per culture did not decrease during incubation with oxadiazon, indicating that under this condition no peroxidative degradation of cell components was induced. This finding was confirmed by little ethane formation (deriving from endogenous polyunsaturated fatty acids), which was in the same range as that of the control. Nevertheless, chlorophyll content was lower than that in the control. In ex-

Table II. Influence of Oxadiazon, Tetrahydrophthalimides, and Pyrazole Analogues on the Formation of Chlorophylls and Carotenoids in Heterotrophic (Dark) Cultures of *Scenedesmus* over 48 h and Formulas and Chemical Names of Compounds Used

additions	chlorophyll, mg/mL of pcv	colored carotenoids, mg/mL of pcv
control	4.8 ± 0.4	0.58 ± 0.07
oxadiazon, 1 μM	3.5 ± 0.3	0.55 ± 0.09
MK-616, 0.1 μM	3.8 ± 0.4	0.53 ± 0.10
DTP, 5 μM	3.7 ± 0.3	0.57 ± 0.07
W-1440, 5 μM	4.4 ± 0.3	0.53 ± 0.09
SW-751, 5 μM	4.6 ± 0.2	0.61 ± 0.10
metronidazole, 5 μM	4.9 ± 0.4	0.59 ± 0.08
levulinic acid, 10 mM	1.8 ± 0.3	0.52 ± 0.08
norflurazon, 1 μM	3.8 ± 0.4	0.20 ± 0.09

Formulas



Chemical Names

oxadiazon, 2-*tert*-butyl-4-(2,4-dichloro-5-isopropoxyphenyl)-Δ^2-1,3,4-oxadiazolin-5-one
MK-616, *N*-(4-chlorophenyl)-3,4,5,6-tetrahydrophthalimide
DTP, 1,3-dimethyl-4-(2,4-dichlorobenzoyl)-5-hydroxypyrazole
W-1440, 1,3-dimethyl-4-(2,4-dichlorobenzoyl)-pyrazole-5-(2,4-dichlorobenzoate)
SW-751 (Sanbird), [1,3-dimethyl-4-(2,4-dichlorobenzoyl)-pyrazol- 5-yl]-*p*-toluenesulfonate
metronidazole, 1-(hydroxyethyl)-2-nitro-5-methylimidazole
norflurazon, 4-chloro-5-(methylamino)-2-[3-(trifluoromethyl)phenyl]pyridazin-3(2*H*)one

Table III. Inhibition of Plastidic Cytochrome *c*-553 Content in Autotrophic and Heterotrophic *Scenedesmus* Grown for 48 h in the Presence of 1 μM Oxadiazon

culture	cytochrome *c*-553 content [a]: autotrophic	heterotrophic, dark
control	40.1 ± 4.6	13.9 ± 0.8
+oxadiazon	24.2 ± 1.9	9.2 ± 1.3

[a] In nmol/mL of packed cell volume.

Table IV. Degradative Properties of Oxadiazon Measured by the Level of ^{35}S-Prelabeled Sulfolipid and by Formation of Ethane

parameters	(A) cultivation for 48 h minus herbicide with [^{35}S]sulfate present	(B) after a subsequent 48-h cultivation of (A) with unlabeled sulfate ± herbicides added		
		control	1 μM oxadiazon	1 μM oxyfluorfen
radioactivity in sulfolipid of total culture, 10^3 dpm/L	203	223	239	64
total chlorophyll, mg/L	11.6	46.2	19.3 7.5[b]	7.4
cell density, μL of packed cell volume/mL of suspension	6	14	11	8.0
formation of ethane,[a] nmol (mL of packed cell volume)$^{-1}$ h^{-1}		1.0	1.2 4.6[b]	4.2 9.8[b]

[a] Determination 24 h after addition of herbicides. [b] Herbicide concentration increased to 10 μM.

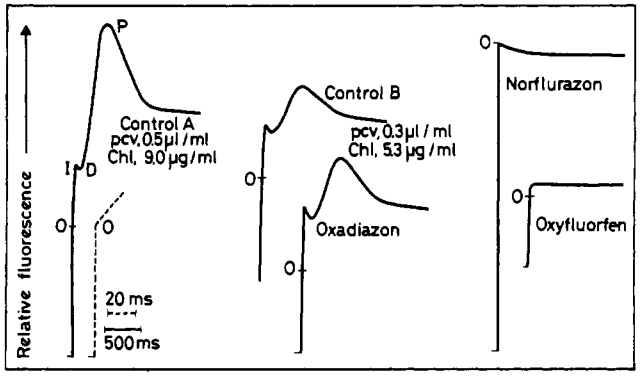


Figure 3. Fluorescence-induction signals of autotrophic *Scenedesmus* cells after growth with different herbicides present (1 μM). All samples, including control (A), were adjusted to a density of 0.5 μL of packed cell volume (pcv/mL of suspension) except for control (B), which was diluted to 5.3 μg of chlorophyll, then having the same chlorophyll concentration as the oxadiazon-treated sample (with a density of 0.5 μL of pcv/mL). The O level was determined by using shorter resolution times (dashed curve).

periments with 10 μM oxadiazon, a 4-fold higher ethane evolution was observed, accompanied by sulfolipid degradation [compare Sandmann and Böger (1982b)]. This is similar to the effect exhibited by 1 μM of the peroxidative herbicide oxyfluorfen, a *p*-nitrodiphenyl ether (last column).

Herbicides inhibiting carotene biosynthesis (e.g., norflurazon) or inducing peroxidation (like oxyfluorfen) can be recognized by their effect on the time course of chlorophyll fluorescence in the millisecond range as shown by intact *Scenedesmus* cells grown for 2 days with either oxadiazon or the two above-mentioned herbicides (Figure 3). Comparing samples of the same density (packed cell volume adjusted to 0.5 μL/mL), initial fluorescence (O) was increased by norflurazon to about the peak level (P) of the control signal (A). An additional peak in the millisecond range was not observed. In contrast, oxyfluorfen decreased initial fluorescence, but changes in the variable part were also absent. On the other hand, oxadiazon did not cause a substantial difference in the shape of the O–I–D–P fluorescence induction. The height of the signal, however, was reduced to the same degree as the chlorophyll content of the cell. Accordingly, the fluorescence-induction curves were about identical when an untreated sample (control B) was compared with the sample pregrown with oxadiazon, when both were adjusted to the same *chlorophyll* content.

DISCUSSION

Previous observations of decreased pigment content caused by oxadiazon (Sandmann and Böger, 1982a; Karapetyan et al., 1983) were investigated in detail in this study. The results shown in Figure 2 suggest that in autotrophic cells an early effect of low oxadiazon concentration is on the photosynthetic capacity, caused by lowered levels of chlorophylls and carotenoids. The subsequent inhibition of photosynthesis impairs growth.

The effect of oxadiazon on chlorophyll formation in heterotrophic (dark) cultures of *Scenedesmus* leaving the carotenoids unaffected points to the chlorophyll-biosynthesis pathway as the specific target of this compound (Table II). Under our dark conditions a possible photooxidative degradation of carotenoids can be excluded, as is seen in illuminated cultures (Figure 2), which might be a consequence of inhibited chlorophyll formation. It should be noted that a similar connection between the levels of carotenoids and chlorophylls was observed in autotrophic cells treated with inhibitors of carotene biosynthesis. Here, a parallel decrease of chlorophyll content was evident in the light (Sandmann et al., 1981).

A direct effect of oxadiazon on carotenoid content in autotrophic *Scenedesmus*, in addition to chlorophyll inhibition, was also ruled out by comparing variable fluorescence in the presence of oxadiazon against norflurazon (Figure 3). Oxadiazon-treated samples did not show the altered shape of the variable fluorescence signal observed after growth with norflurazon present, which is typical for herbicides affecting carotene biosynthesis (Böhme et al., 1981; Karapetyan et al., 1983). Furthermore, the fluorescence induction curve with oxadiazon-treated cells differed from those treated with oxyfluorfen. It should be noted that the strong oxyfluorfen-dependent decrease of initial fluorescence is more pronounced when packed cell volume (Figure 3) or milliliter culture suspension (Böhme et al., 1981) was used as a reference instead of chlorophyll (Karapetyan et al., 1983). With oxadiazon applied—at least for the experimental times as in this study—it appears that only the (chlorophyll) reference is changed and not the function of the photosystems. Furthermore, the low degradative activity of 1 μM oxadiazon (by looking at changes of ^{35}S-prelabeled sulfolipid content or evolution of ethane in Table IV) excludes a pronounced peroxidative action of oxadiazon on cell components including chlorophylls. However, at a concentration of 10 μM, the decrease of pigments per culture (Table I) and the evolution of ethane indicate that degradative processes take place in addition to inhibition of chlorophyll biosynthesis.

Formation of plastidic cytochrome *c*-553 was inhibited to a similar extent as chlorophyll formation (Table III). Consequently, it can be assumed that oxadiazon is directed against a step in the common pathway of chlorophyll and heme synthesis leading to the branching point protoporphyrin IX. However, we cannot yet exclude that the conversion of protoporphyrin IX or one of its successors is inhibited and that the accumulating intermediate may be a negative feedback inhibitor as was reported recently

for Mg-protoporphyrin IX (Chereskin and Castelfranco, 1982).

DTP and MK-616 showed the same effect on chlorophylls and carotenoids as oxadiazon in heterotrophic cultures (Table II). Structural features common to these two compounds and to oxadiazon are planar heterocyclic five-membered rings (e.g., pyrazole, pyrroline, and oxadiazoline, respectively) substituted at various positions. The imidazole ring of metronidazole has the same properties. Nevertheless, this compound was inactive. A potency of metronidazole as an inhibitor of chlorophyll biosynthesis may be impaired by unfavorable substituents. This may be true for W-1440 and SW-751, which are identical with DTP except for their substituents at position 5 but are much less effective than DTP. It is assumed that the free OH group of DTP at position 5 is necessary for proper activity.

Our evidence for the same mode of action of MK-616 and oxadiazon is not in accordance with two reports in the literature. Wakabayashi et al. (1979) proposed a herbicidal mechanism of MK-616 and other chemically related compounds as similar to that of *p*-nitrodiphenyl ethers (i.e., by peroxidative activities). However, that study did not include biochemical data. The influence of DTP on chlorophyll biosynthesis was reported by Kawakubo et al. (1979). They assumed an inhibition of protochlorophyll synthesis, a step following the formation of protoporphyrin IX, which cannot yet be ruled out.

Data on the primary enzymic interference of the herbicidal compounds mentioned should become available with cell-free systems, exhibiting at least some of the reaction steps leading to chlorophyll formation. A comparable system was successfully employed for characterization of herbicidal inhibitors of carotene biosynthesis (Clarke et al., 1982). Apparently, the type of compounds investigated in this paper represents a potential to provide for new inhibitors useful to study biosynthetic pathways of plant pigments.

ACKNOWLEDGMENT

We are indebted to Rhône-Poulenc, Vitry-sur-Seine, France, Sankyo Co., Tokyo, Japan, Celamerck, Ingelheim, West Germany, Mitsubishi Chemical Industries, Yokohama, Japan, Bayer AG, Leverkusen, West Germany, Sandoz AG, Basel, Switzerland, and Rohm and Haas, Spring House, PA, for supplying samples of pure herbicides. We are grateful to Silvia Kuhn, Konstanz, for expert technical assistance. This study was supported by the Deutsche Forschungsgemeinschaft.

Registry No. Oxadiazon, 19666-30-9; MK-616, 39985-63-2; DTP, 58010-98-3; cytochrome *c*-553, 12624-01-0; norflurazon, 27314-13-2; oxyfluorfen, 42874-03-3; SW-751, 58011-68-0; metronidazole, 443-48-1; levulinic acid, 123-76-2; W-1440, 58033-05-9.

LITERATURE CITED

Ambrosi, D.; Desmoras, J. *Proc. Eur. Weed Res. Counc. Symp. Herbic.-Soils* **1973**, 163–177.
Böger, P.; Schlue, U. *Weed Res.* **1976**, *16*, 149–154.
Böhme, H.; Kunert, K. J.; Böger P. *Weed Sci.* **1981**, *29*, 371–375.
Burgaud, L.; Deloraine, J.; Desmoras, J.; Guillot, M.; Petrinko, P.; Riotto, M. *Proc. Eur. Weed Res. Counc.* **1969**, *1*, 201–218.
Chereskin, B. M.; Castelfranco, P. A. *Plant Physiol.* **1982**, *68*, 112–116.
Clarke, I. E.; Sandmann, G.; Bramley, P. M.; Böger, P. *FEBS Lett.* **1982**, *140*, 203–206.
Karapetyan, N. V.; Strasser, R.; Böger, P. *Z. Naturforsch., C: Biosci.* **1983**, *38C*, 556–562.
Kawakubo, K.; Shindo, M.; Konotsune, T. *Plant Physiol.* **1979**, *64*, 774–779.
Mackinney, G. *J. Biol. Chem.* **1940**, *140*, 315–322.
Moon, M. W.; Bell, L. T.; Cutting, T. K.; Keyser, H. P.; Tiller, R. H.; Vostral, H. J. *J. Agric. Food Chem.* **1977**, *25*, 1039–1049.
Sandmann, G., Böger, P. *Z. Pflanzenphysiol.* **1980a**, *98*, 53–59.
Sandmann, G.; Böger, P. *Planta* **1980b**, *147*, 330–334.
Sandmann, G.; Böger, P. *Photosynth. Res.* **1981**, *2*, 281–289.
Sandmann, G.; Böger, P. In "Biochemical Responses Induced by Herbicides"; Moreland, D. E.; St. John, J. B.; Hess, F. D., Eds.; American Chemical Society: Washington, DC, 1982a; pp 111–130.
Sandmann, G.; Böger, P. *Plant Sci. Lett.* **1982b**, *24*, 347–352.
Sandmann, G.; Böger, P. In "IUPAC Pesticide Chemistry, Human Welfare and The Environment"; Miyamoto, J.; Kearney, P. C., Eds.; Pergamon Press: Oxfored, 1983a; Vol. 1, pp 321–326.
Sandmann, G.; Böger, P. *Weed Sci.* **1983b**, *31*, 338–341.
Sandmann, G.; Kunert, K. J.; Böger, P. *Pestic. Biochem. Physiol.* **1981**, *15*, 288–293.
Wakabayashi, K.; Matsuya, K.; Oha, H.; Jikihara, T. In "Advances in Pesticide Science"; Geissbühler, H., Ed.; Pergamon Press: Oxford, 1979; Part 2, pp 256–260.

Received for review December 22, 1983. Accepted March 19, 1984.