dium depends on the system temperature and on silicate characteristics (exchange cations and layer charge). These factors also influence the catalytic hydrolysis of azinphos-methyl by the samples in an aqueous medium, under conditions of pH and temperature where the pesticide is normally stable.

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Registry No. Montmorillonite, 1318-93-0; azinphos-methyl, 86-50-0; hectorite, 12173-47-6.

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The Diphenyl Ether Herbicide Oxyfluorfen: Action of Antioxidants

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Measurement of ethane, as in index of in vivo lipid peroxidation, was done with *Scenedesmus acutus* and the diphenyl ether oxyfluorfen [2-chloro-1-(3-ethoxy-4-nitrophenoxy)-4-(trifluoromethyl)benzene] present at a concentration of 50 nM. In the light, lipid peroxidation is substantial in herbicide-treated cells, and peroxidation is directly related to pigment damage. Protection against peroxidation and pigment loss is only achieved by the antioxidants ethoxyquin (1,2-dihydro-6-ethoxy-2,2,4-trimethylquinoline) and *dl*-α-tocopherol at a concentration of 0.1 mM or by exposure to the dark. Less effective protection is found with either of the antioxidants DPPD (*N,N'*-diphenyl-1,4-phenylenediamine), BHT (2,6-di-*tert*-butyl-*p*-cresol), ascorbic acid, or the hydroxyl radical scavenger mannitol. Cells exhibiting ethane formation after a foregoing treatment with oxyfluorfen cannot be effectively protected against pigment loss by subsequent addition of ethoxyquin.

In vivo peroxidation of polyunsaturated fatty acids has been identified as one of the deleterious reactions basic to the mechanism of membrane and cellular damage (Mead, 1976). It has been reported that, in light, certain diphenyl ether herbicides seemingly damage biomembranes by peroxidation of membrane lipids (Prendeville and Warren, 1977; Vanstone and Stobbe, 1977; Kunert and Böger, 1981; Orr and Hess, 1982a,b). The herbicide-induced peroxidation was either investigated by measurement of malondialdehyde formation (Orr and Hess, 1982a), degradation of sulfolipid (Sandmann and Böger, 1983a,b), or ethane production (Kunert and Böger, 1981; Lambert et al., 1983). The noninvasive method of measuring volatile hydrocarbons released during decomposition of unsaturated fatty acid hydroperoxides provides a useful index of lipid peroxidation (Dumelin and Tappel 1977; Tappel and Dillard, 1981).

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However, little is known about the protection of algae and plants against the herbicide-induced peroxidation process. Orr and Hess (1982a) found that the antioxidant α-tocopherol inhibited membrane leakage in diphenyl ether treated cucumber cotyledons. Recently, we reported that in vivo lipid peroxidation seemed to be a function of the vitamin C concentration present in the plants after herbicide application (Kunert, 1983).

In this study, we describe some new aspects of the efficiency of antioxidants to decrease both diphenyl ether induced lipid peroxidation and pigment damage directly related to peroxidation. The sensitive measurement of ethane, a decomposition product of ω-3-unsaturated fatty acid hydroperoxides, was used as an index of in vivo lipid peroxidation. Experiments were carried out with the diphenyl ether oxyfluorfen [2-chloro-1-(3-ethoxy-4-nitrophenoxy)-4-(trifluoromethyl)benzene], and herbicidal activity was assayed with the unicellular alga *Scenedesmus acutus*. The use of microalgae has several advantages over higher plants, since absolute quantitative changes of cellular compounds can be measured when referred to a

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constant culture volume (Sandmann et al., 1979). Furthermore, the effects of herbicides and protectors against herbicidal action develop in a very short time.

MATERIALS AND METHODS

Herbicide. Oxyfluorfen was obtained from Rohm and Haas, Spring House, PA.

Antioxidants. Ethoxyquin (1,2-dihydro-6-ethoxy-2,2,4-trimethylquinoline) was purchased from ICN Pharmaceuticals, Inc., Plainview, NY; DPPD (*N,N'*-diphenyl-1,4-phenylenediamine) and BHT (2,6-di-*tert*-butyl-*p*-cresol) were from Fluka AG, Buchs, Switzerland; *dl*-α-tocopherol was from Serva, Heidelberg, West Germany; mannitol was from Sigma Chemical Co., St. Louis, MO; ascorbic acid was from Merck, Darmstadt, West Germany.

Cultivation of Algae. *S. acutus* (strain 276-3a, Algae Culture Collection, University of Göttingen, West Germany) was grown autotrophically on a shaker in Fernbach flasks (400 mL) in sterile liquid medium at 20 °C, as described by Kunert et al. (1976). Air, enriched with 5% CO_2 (v/v), was constantly supplied at a flow rate of 4.8 L/h, and the algal culture was kept under continuous light of 40 W/m^2. The initial cell density of both the control culture and the herbicide-treated culture was adjusted to 1 μL of packed cell volume/mL of cell suspension, equivalent to 4×10^6 cells/mL and 14.6 ± 1.6 μg of chlorophyll/mL of cell suspension. The herbicide and antioxidants were dissolved in methanol and added to the sterilized culture medium, with a final methanol concentration in the medium of 0.1% (v/v) at the maximum.

Analysis. The cell density was determined as packed cell volume (pcv) in graduated microcentrifuge tubes of 80-μL capacity. The chlorophyll content of cells, after methanol extraction, was determined according to the method of Kunert and Böger (1979).

For measurement of ethane, algal samples of 30 mL were taken from the Fernbach flasks and incubated in 60-mL vials sealed with rubber septa and shaken for 1 h at 20 °C under continuous light of 40 W/m^2. To provide enough CO_2, 5 mM $NaHCO_3$ was added to the samples at the beginning of the experiment. For determination of ethane, headspace gas samples of 1 mL, withdrawn from the rubber-sealed vials with a gas-tight syringe, were analyzed, after 1-h shaking, on a Perkin-Elmer F22 gas chromatograph equipped with a flame ionization detector. A stainless steel column (0.32 × 152 cm) filled with activated alumina (80–100 mesh) was used with a nitrogen carrier gas flow rate of 30 mL/min. The temperature of the column was 60 °C, and that of the injector and detector 200 °C. The relative peak area of ethane was calculated by comparison with 1 ppm of ethane as a standard (Messer Griesheim, Düsseldorf, West Germany).

Statistical Analysis. All estimates of sample variability are given in terms of the standard error (SE) of the mean. Comparisons of two means were calculated by using the Student's two-tailed t test. p values ≤ 0.05 were considered significant.

The significance of the influence of antioxidants on both ethane production and chlorophyll damage was determined by one-way analysis of variance using weighted squares of the means and Fisher's LSD test (Steel and Torrie, 1980). p Values ≤ 0.01 were considered significant.

RESULTS

Figure 1 shows the percentage of protection by antioxidants of lipid peroxidation, measured as ethane production, and of chlorophyll in oxyfluorfen-treated *Scenedesmus* cells. Maximum ethane production was found

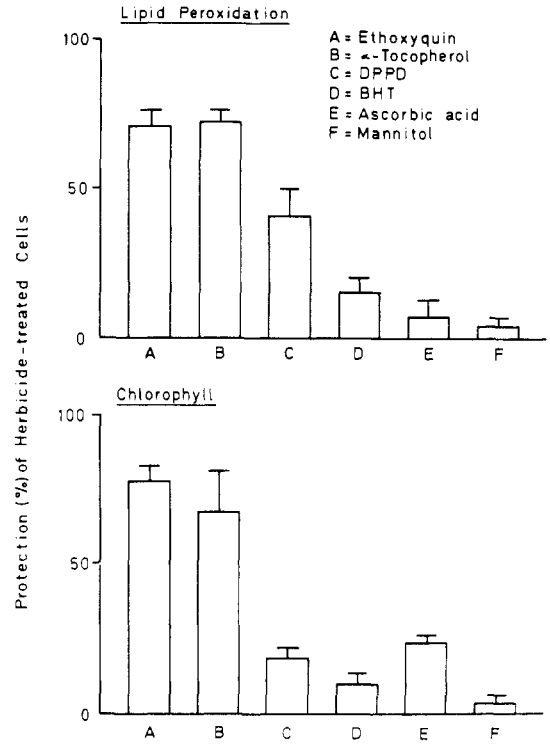

Figure 1. Percentage of protection by antioxidants against lipid peroxidation, measured as ethane production and chlorophyll damage after 6 and 24 h, respectively, of light-exposed *Scenedesmus* cells in the presence of 50 nM oxyfluorfen. At the beginning of the experiment, cells were simultaneously treated with the herbicide and 0.1 mM of either ethoxyquin (A), α-tocopherol (B), DPPD (C), or BHT (D) or 50 mM of either ascorbic acid (E) or mannitol (F). Protection against lipid peroxidation corresponds to the inhibition of 4.7 ± 0.6 pmol of ethane (μL of pcv)$^{-1}$ h^{-1} produced after 6 h by oxyfluorfen-treated cells. Chlorophyll protection of 100% corresponds to 54 ± 3.0 μg of chlorophyll/mL of cell suspension of the untreated control measured after 24 h. Data shown represent the mean ± SE of five different experiments.

after 6 h, and no higher amounts of the hydrocarbon gas were observed over 24 h. Inhibition of peroxidation and chlorophyll damage were highly significant ($p < 0.01$) in cells treated with either of the lipid-soluble antioxidants ethoxyquin or α-tocopherol. Within 6 h of herbicide treatment, both antioxidants inhibited ethane evolution by 72%. Pigment destruction was prevented by 68% and 78% over 24 h by α-tocopherol and ethoxyquin, respectively. Carotenoids and photosynthetic electron transport were also protected to the same extent (data not shown). DPPD gave a 41% protection against lipid peroxidation, which is significantly higher ($p < 0.01$) than with BHT or the water-soluble antioxidant ascorbic acid. However, DPPD inhibited chlorophyll damage by 15% only. This inhibition was comparable to that achieved by BHT or ascorbic acid. No protection against lipid peroxidation and chlorophyll damage was observed when the hydroxyl radical scavenger mannitol was added to herbicide-treated cells.

Addition of the diphenyl ether oxyfluorfen almost totally destroyed the chlorophyll of *Scenedesmus* cells within 24 h. The pigment content of herbicide-treated cells was 0.7 ± 0.2 μg of chlorophyll/mL of cell suspension after 24 h, as compared to 54 ± 3.0 μg of chlorophyll/mL of cell suspension of the untreated control (data not shown). Ethane evolution of oxyfluorfen-treated cells was 4.7 ± 0.6 pmol of ethane (μL of pcv)$^{-1}$ h^{-1} after a 6-h light-exposure time, as compared to 0.47 ± 0.2 pmol of ethane (μL of pcv)$^{-1}$ h^{-1} of the untreated control. When both ethoxyquin

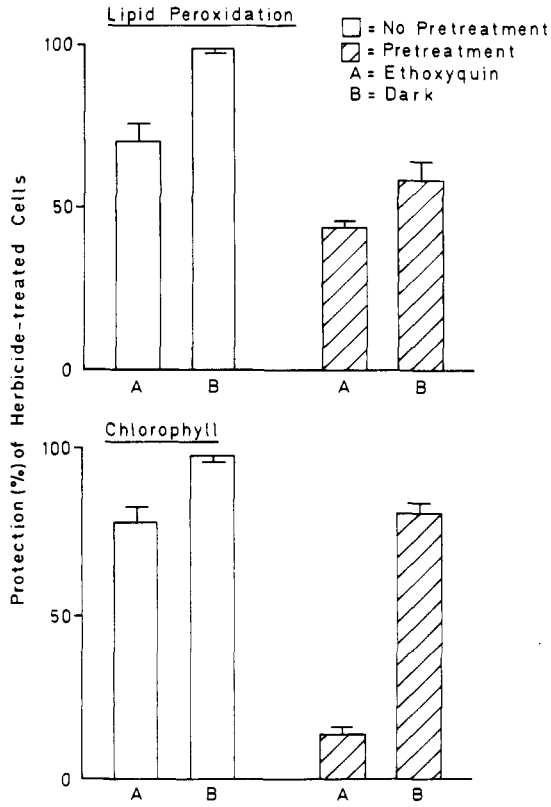

Figure 2. Percentage of protection against both lipid peroxidation (measured as light-induced ethane production) and chlorophyll damage by 0.1 mM ethoxyquin (A) or exposure to the dark (B) of *Scenedesmus* cells treated with 50 nM oxyfluorfen. Addition of the antioxidant was done either together with the herbicide at the beginning of the experiment ("no pretreatment") or after a 6-h light exposure with the herbicide present ("pretreatment"). Protection against lipid peroxidation by ethoxyquin or dark exposure of pretreated cells corresponds to the decrease of ethane production 2 h after addition of the antioxidant or dark exposure. Without antioxidants present, the cells produced 4.7 ± 0.6 pmol of ethane (μL of pcv)$^{-1}$ h^{-1} after a 6-h light-exposure time. Chlorophyll protection of 100% corresponds to either 54 ± 3.0 μg of chlorophyll/mL of cell suspension for the untreated light control or 14 ± 1.3 μg of chlorophyll/mL of cell suspension for the untreated dark control. Data shown represent the mean ± SE of five different experiments.

and the herbicide were added to an algal culture at the beginning of the experiment, pigment damage and lipid peroxidation were prevented by 71% and 78%, respectively (Figure 2). Pigment destruction and ethane evolution were completely inhibited when cells were grown in the dark, in the presence of 50 nM oxyfluorfen. No significant decrease ($p > 0.05$) of the chlorophyll content was found in cells pretreated with the herbicide in the light for 6 h, as compared to the chlorophyll content of the cells before treatment with the herbicide. However, chlorophyll protection over 24 h was only 14%, when ethoxyquin was added to cells pretreated with oxyfluorfen for 6 h. Therefore, inhibition of pigment destruction by the antioxidant was significantly lower ($p < 0.05$) in pretreated cells as compared to cells without herbicide pretreatment. After 6-h herbicide pretreatment, protection by ethoxyquin against lipid peroxidation after 2-h cultivation in Fernbach flasks, in the presence of the antioxidant, was 43%. This was significantly higher ($p < 0.05$) than protection by the antioxidant against chlorophyll damage over 24 h. Exposure to the dark of *Scenedesmus* cells, pretreated in the light for 6 h, prevented pigment degradation by 83% over 24-h dark exposure and ethane evolution by 59% after 2-h dark exposure. However, ethane production of cells was almost totally inhibited after a 24-h dark exposure.

DISCUSSION

Many studies have shown the evolution of volatile hydrocarbons to be useful indices of in vivo lipid peroxidation (Tappel and Dillard, 1981). The noninvasive method of measuring ethane seems to provide a sensitive indication of a peroxidation process (Riely et al., 1974; Kunert and Tappel, 1983).

There is strong evidence that certain bleaching herbicides, such as the diphenyl ether oxyfluorfen, are powerful initiators of in vivo lipid peroxidation in green algae (Kunert and Böger, 1981; Lambert et al., 1983). In our experiments, we have demonstrated that, in light, oxyfluorfen-treated *Scenedesmus* cells expired a significantly increased amount of ethane as compared with control cultures. Administration of antioxidants inhibited hydrocarbon gas production. The most effective nonbiological antioxidant tested, ethoxyquin, prevented ethane production to an extent comparable with that of the biological antioxidant *dl*-α-tocopherol. Various non-tocopherol antioxidants, including ethoxyquin, DPPD, or BHT and ascorbic acid, have been shown to inhibit in vivo lipid peroxidation (Tappel, 1968; Downey et al., 1978; Kunert and Tappel, 1983). However, neither the antioxidants BHT nor DPPD or ascorbic acid exhibited protection comparable to that of ethoxyquin. Recently, Orr and Hess (1982b) have also reported limited protection by both BHT and DPPD against diphenyl ether induced membrane leakage. The effect of antioxidants indicates that free radicals are involved in initiating peroxidation or/and free radicals coming up in the course of peroxidation. Apparently, free hydroxyl radicals [derived from the superoxide anion in the presence of metal ions (Elstner, 1979)] are not involved, because mannitol was not effective to protect against lipid peroxidation.

Both BHT and ascorbic acid are potent antioxidants in vivo (Logani and Davies, 1980; Kunert and Tappel, 1983). However, in addition to being an antioxidant, BHT has prooxidative activity by increasing the photooxidation of fatty acids (Logani et al., 1983). Ascorbic acid, which was used in our experiments in approximately the same concentration range as normally found in the chloroplast stroma (Gerhardt, 1964), can also act as a prooxidant, and stimulation of lipid peroxidation by ascorbate in the presence of metal ions has been well-documented (Dumelin and Tappel, 1977). Obviously, the prooxidative function of BHT and ascorbid acid became evident in our experiments with peroxidizing *Scenedesmus* cells, thus preventing effective inhibition of both lipid peroxidation and pigment destruction. In our algal system, DPPD protected cells against peroxidation but achieved only limited protection against chlorophyll damage. Diphenylamines are inhibitors of carotenoid biosynthesis in green algae, inducing formation of colorless precursors, namely, phytoene and phytofluene (Sandmann and Böger, 1983a,b). Inhibition of carotenoid formation and accumulation of precursors were also found in our experiments when DPPD was used as an antoxidant. It is generally assumed that destruction of chlorophyll in plant tissues is caused by photooxidation due to the lack of protection by colored carotenes (Kunert and Böger, 1979).

Our studies demonstrated that chlorophyll damage was a toxic consequence of the peroxidation process. Lipid peroxidation and pigment damage were totally light dependent and effectively inhibited in the light by the powerful antioxidants α-tocopherol and ethoxyquin. However, when the destruction of membranes had proceeded, ethoxyquin was no longer an efficient protector of chlorophyll

in herbicide-pretreated *Scenedesmus* cells. Chlorophyll photooxidation strongly depends on the environment, and lipids can protect chlorophyll against being photooxidized (Stillwell and Tien, 1977). Therefore, degradation of the protective lipids will cause irreversible photooxidation of unprotected pigments. Further, toxic decomposition products of lipid hydroperoxides may also be responsible for pigment degradation either by cooxidation of the pigment (Holden, 1965; Schobert and Elstner, 1980) or by inactivation of enzymes necessary for chlorophyll biosynthesis (Gardner, 1979).

Summarizing, lipid peroxidation is the dominant toxic reaction in oxyfluorfen-treated *Scenedesmus* cells. The peroxidation process is directly related to severe light-dependent consequences such as pigment damage. Cell death is finally caused by the sum of all toxic events. Among them, peroxidative membrane damage seems to be the most potent reaction. Simultaneous treatment of cells with both oxyfluorfen and antioxidants such as ethoxyquin or α-tocopherol, which are powerful protectors against lipid peroxidation and pigment destruction, strongly diminishes herbicide toxicity.

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Registry No. Oxyfluorfen, 42874-03-3; ethoxyguin, 91-53-2; *dl*-α-tocopherol, 10191-41-0; DPPD, 74-31-7; BHT, 128-37-0; ascorbic acid, 50-81-7; mannitol, 69-65-8.

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Photochemistry and Volatility of Drepamon in Water

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The aquatic environmental chemistry of the herbicide Drepamon (*S*-benzyl *N*,*N*-di-*sec*-butylthiocarbamate) was investigated in laboratory studies of photochemical reactivity and volatilization. In distilled water Drepamon photooxidized slowly to *N*-[(benzylsulfinyl)carbonyl]-*N*,*N*-di-*sec*-butylamine (Drepamon sulfoxide), an intermediate that underwent further direct photochemical conversion to benzaldehyde. Traces of hydrogen peroxide greatly accelerated the photodecomposition of Drepamon and altered the distribution of photoproducts so that phenols predominated. Peroxide-initiated free radical photooxidation included sulfur oxidation and N-dealkylation as well. Drepamon, its phenolic products, benzaldehyde, and *sec*-butylamine were analyzed by gas–liquid chromatography. The thermally unstable sulfoxide and sulfone were determined by high-pressure liquid chromatography. Drepamon volatilized from aqueous solutions more rapidly than the structurally related herbicides molinate and thiobencarb.

Drepamon or tiocarbazil (proposed common name) is an effective herbicide for the selective control of barnyardgrass (*Echinochloa* spp.) in submerged rice fields. Control of this weed increases yields, shortens the growing season, and is of major economic importance in paddy rice culture. The structurally related herbicides molinate and thiobencarb are currently used for grass control in flooded California rice fields. Previous studies in this laboratory (Soderquist et al., 1977) and others have shown that volatilization is a major route of dissipation for the thiolcarbamates under field-use conditions. Molinate and thiobencarb also are degraded photochemically in sunlight in both direct and indirect photolysis processes (Soderquist

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