DICLOFOP-METHYL AND ITS MODE OF ACTION

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Summary. A review is given on the mode of action of diclofop-methyl, covering the present state of knowledge on uptake and translocation, pathway of degradation and biochemical sites of action in plants.

INTRODUCTION

The herbicide 2-(4-(2',4'-dichlorophenoxy)phenoxy)-methyl-propanoate (diclofop-methyl[1]) was discovered by Hoechst AG in 1971. It selectively controls a broad spectrum of grass weeds, such as wild oats *(Avena* spp.*)* wild millets *(e.g. Echinochloa* spp., *Setaria* spp.*)*, rye grass *(Lolium* spp.*)* and volunteer maize *(Zea mays)* in wheat, barley and dicotyledonous crops. It is predominantly applied post-emergence in the 2 to 4 leaf stage of the weeds, but has also some pre-emergence potential (Schumacher and Schwerdtle 1975; Breidert *et al.* 1977).

HERBICIDE SYMPTOMS

About 3 days after post-emergence application the leaves of susceptible species become chlorotic and the growth of new leaves is arrested. Usually between 7 to 10 days after spraying the chlorotic areas become necrotic, as does the growing point of the shoot. The destruction of the shoot meristematic area at the growing point is essential for complete kill of the plant. Growth of roots exposed to diclofop-methyl is strongly inhibited and necrosis starts from the meristematic zone at the root tip (Köcher and Lötzsch 1975; Walter 1977; Hoerauf and Shimabukuro 1979; Todd 1979; Hoppe 1981).

UPTAKE AND TRANSLOCATION

Uptake of diclofop-methyl after foliar application extends over a period of 4 days or more, the rate of uptake being highest on the day of treatment. Todd and Stobbe (1977) found about 70% uptake in wild oat *(Avena fatua)* and 90% in wheat *(Triticum aestivum)* 4 days after foliar application. Donald and Shimabukuro (1980), after the same period, found about 57% absorption in wild oat and 50% in wheat. Zacher and Hoppe (1981) found 79% foliar uptake in bushbean *(Phaseolus vulgaris)* and 76% in maize after 4 days. These results suggest that the selective action of diclofop-methyl cannot be explained by differential rates of uptake by tolerant and sensitive plant species. After foliar application, translocation rates of this herbicide within the treated leaf are low, both in acro- and basipetal directions. Three days after leaf application total translocation in wheat amounted to 1.7%, in wild oat to 0.9%, and in green foxtail *(Setaria viridis)* to 0.8% of the applied herbicide. The difference in susceptibility of these species to diclofop-methyl could not be explained by different translocation rates of the radioactive label (Todd 1979). In soybean *(Glycine max)* and sugarbeet *(Beta vulgaris* var. *alt.)* only 0.2% of the applied herbicide was translocated

[1] Trade name Hoegrass

from the treated leaf to the rest of the plant within one week after application (Köcher and Lötzsch 1981). Autoradiographs of leaves from grass and broadleaved species treated with radiolabelled diclofop-methyl showed that the radioactive material translocated above and below the site of application was mainly located within the conducting tissues and decreased with increasing distance from the site of application (Köcher and Lötzsch 1975). Microapplications of diclofop-methyl to different areas of the shoot (shoot base, proximal half, distal half of leaf blades) of wild oat plants showed that the nearer the site of application to the base of the shoot, the higher the concentration of herbicide at the growing point. This was correlated with increased damage to the growing point and the plant in general (Walter and Bischof 1976; Walter 1977). Thus, in agricultural practice, spraying conditions should be chosen which provide good deposition of diclofop-methyl at the base of the plant.

Diclofop-methyl was taken up by roots of plants kept in nutrient solution and was, to a limited extent, translocated to the shoot (Shimabukuro *et al.* 1979). A concentration as low as 1 µM diclofop-methyl in the nutrient solution still strongly inhibited root growth of wild oat plants (Hoerauf and Shimabukuro 1979). Placement experiments with plants growing in soil have shown that action via the soil can contribute to the overall action of diclofop-methyl applied post-emergence (West *et al.* 1980; Bieringer 1981). The leachability of this herbicide in soil is relatively low (Wu and Santelmann 1976; Mulder and Nalewaja 1979). Thus, it is obvious that the degree of soil action is very much dependent on rainfall or irrigation conditions after spray application.

DEGRADATION IN PLANTS

Hydrolysis of diclofop-methyl to 2-(4-(2',4'-dichlorophenoxy)propionic acid (diclofop) is the first degradation step in plants. Diclofop, which is also phytotoxic, is then hydroxylated to form the nonphytotoxic 2-(4-(2',4'-dichloro-5'-hydroxyphenoxy)phenoxy)propionic acid or the 3'- and 6'-isomers with respect to the position of the hydroxy group. These degradation products can conjugate with plant constituents (Gorbach *et al.* 1977; Shimabukuro *et al.* 1979). Wild oat predominantly conjugates diclofop to an ester conjugate. Provided that this reaction is reversible, the inactive ester conjugate may serve as a potential pool for the physiologically active diclofop. Wheat, in contrast to wild oat, mainly hydroxylates the 2,4-dichlorophenyl ring, which is followed by formation of phenolic conjugates. These differences in the pattern of degradation might explain the differential susceptibility of wheat and wild oat to diclofop-methyl (Donald and Shimabukuro 1980). Comparative studies of diclofop-methyl degradation in tolerant bushbean and susceptible maize did not indicate a relation between pattern of degradation and herbicide susceptibility of these two species (Zacher and Hoppe 1981).

BIOCHEMICAL EFFECTS

Photosynthesis and respiration are not primary sites of action of diclofop-methyl. The reduction of photosynthetic CO_2-fixation, which is apparent several days after foliar application of diclofop-methyl to sensitive grass species, is a secondary effect due to damage of the chloroplast membranes and an associated reduction of chlorophyll content (Köcher and Lötzsch 1975; Brezeanu *et al.* 1976; Chow and LaBerge 1978). When applied to the foliage of sensitive grasses, diclofop-methyl severely reduced assimilate transport to the roots and caused accumulation of soluble assimilates in the shoot (Chow and LaBerge 1978). Hoppe (1981) showed that within one hour after foliar application of the compound to maize assimilate transport to the roots was reduced by 52%, whereas

CO_2-fixation was almost as high as in the controls. It is not yet clear by which mechanism this rapid effect of diclofop-methyl occurs. Another rapid effect occurring a few hours after foliar application of diclofop-methyl to maize leaves is a marked inhibition of the incorporation of ^{14}C-labeled acetate into leaf lipids (Hoppe 1981). In similar experiments with leaf discs in buffer containing diclofop-methyl it was shown that 5×10^{-6} M diclofop-methyl reduced the ^{14}C-acetate incorporation into lipids by more than 70% in maize. In leaf discs of bushbean, a species highly tolerant to diclofop-methyl, even 5×10^{-5} M of the herbicide did not interfere with ^{14}C-acetate incorporation into lipids (Zacher and Hoppe 1981). Since uptake and degradation pattern of diclofop-methyl in these two species could not explain the differential herbicide sensitivity, it appears that differences exist between the two species at the biochemical site(s) of action of this herbicide.

Using maize root tips as a model system to study biochemical effects of diclofop-methyl on meristematic tissue, Hoppe (1980, 1981) has shown that this herbicide had little or no effect on incorporation of radioactive precursors into nucleic acids and proteins. Incorporation of ^{14}C-acetate into lipids of the root tips was inhibited by 5×10^{-6} M diclofop-methyl. In a concentration series, inhibition of lipid synthesis in root tips was correlated to inhibition of seedling root growth. The lipid fractions inhibited by diclofop-methyl are essential for structure and function of plant membrances (Hoppe 1981). Shimabukuro *et al.* (1978) found that in the oat coleoptile elongation test diclofop-methyl showed an antiauxin action in the sense that it inhibited IAA-induced coleoptile elongation. This inhibition could be overcome partially by increasing the IAA-concentration in the test medium.

At present it remains unclear whether diclofop-methyl acts by several independent mechanisms or whether the various effects observed are only different manifestations of one primary biochemical site of action.

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