Chloroacetamide Mode of Action, I: Inhibition of Very Long Chain Fatty Acid Synthesis in *Scenedesmus acutus*

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Herbicidal chloroacetamides cause a very sensitive inhibition of fatty acid incorporation into an insoluble cell wall fraction of *Scenedesmus acutus*. The molecular basis was investigated in more detail.

After incubation of the algae with [^{14}C]oleic acid and saponification, the remaining pellet was solubilized and fractionated consecutively with chloroform/methanol, phosphate buffer, amylase, pronase, and finally with dioxane/HCl. By acid hydrolysis in dioxane a part of the cell wall residue was solubilized showing inhibition of exogenously applied oleic acid and other labelled precursors such as stearic acid, palmitic acid, and acetate. After extraction of this dioxane-soluble subfraction with hexane, HPLC could separate labelled metabolites less polar than oleic acid. Their formation was completely inhibited by chloroacetamides, e.g. 1 μM metazachlor. This effect was also observed with the herbicidally active *S*-enantiomer of metolachlor while the inactive *R*-enantiomer had no influence. These strongly inhibited metabolites could be characterized by radio-HPLC/MS as *very long chain fatty acids* (VLCFAs) with a carbon chain between 20 and 26. Incubating a metazachlor-resistant cell line of *S. acutus* (Mz-1) with [^{14}C]oleic acid, VLCFAs could not be detected in the dioxane/HCl-subfraction. Furthermore, comparing the presence of endogenous fatty acids in wild-type and mutant Mz-1 the VLCFA content of the mutant is very low, while the content of long chain fatty acids (C16–18) is increased, particularly oleic acid.

Obviously, the phytotoxicity of chloroacetamides in *S. acutus* is due to inhibition of VLCFA synthesis. The resistance of the mutant to metazachlor has a bearing on the higher amount of long chain fatty acids replacing the missing VLCFAs in essential membranes or cell wall components.

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