Triaziflam and Diaminotriazine Derivatives Affect Enantioselectively Multiple Herbicide Target Sites

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Enantiomers of triaziflam and structurally related diaminotriazines were synthesized and their herbicidal mode of action was investigated. The compounds caused light and dark-dependent effects in multiple test systems including heterotrophic cleaver and photoautotrophic algal cell suspensions, the Hill reaction of isolated thylakoids and germinating cress seeds. Dose-response experiments revealed that the *(S)*-enantiomers of the compounds preferentially inhibited photosystem II electron transport (PET) and algae growth with efficacies similar to that of the herbicide atrazine. In contrast, the *(R)*-enantiomers of the diaminotriazines were up to 100 times more potent inhibitors of growth in cleaver cell suspensions and cress seedlings in the dark than the *(S)*-enantiomers. The most active compound, the *(R)*-enantiomer of triaziflam, inhibited shoot and root elongation of cress and maize seedlings at concentrations below 1 μM. The meristematic root tips swelled into a club shape which is typical for the action of mitotic disrupter herbicides and cellulose biosynthesis inhibitors. Microscopic examination using histochemical techniques revealed that triaziflam *(R)*-enantiomer blocks cell division in maize root tips 4 h after treatment. The chromosomes proceeded to a condensed state of prometaphase but were unable to progress further in the mitotic cycle. Disruption of mitosis was accompanied by a loss of spindle and phragmoplast micotubule arrays. Concomitantly, cortical microtubules decreased which could lead to isodiametric cell growth and consequently to root swelling. In addition, a decline in cellulose deposition in cell walls was found 24 h after treatment. Compared to the *(R)*-form, triaziflam *(S)*-enantiomer was clearly less active. The results suggest that triaziflam and related diaminotriazines affect enantioselectively multiple sites of action which include PET inhibitory activity, mitotic disruption by inhibiting microtubule formation and inhibition of cellulose synthesis.