AMITROLE, TRIAZINE, METRIBUZIN AND SUBSTITUTED UREA RESISTANCE IN ANNUAL RYEGRASS, *LOLIUM RIGIDUM*

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Abstract. Herbicides are now an integral part of western agriculture. A foreseen but, until recently, unimportant consequence of their use is the appearance of herbicide resistance in weeds that were once controlled. In Australia, annual ryegrass, *Lolium rigidum* Gaud., has displayed a propensity to develop resistance. Populations resistant to many important agricultural herbicides including the aryloxyphenoxypropionates, sulfonylureas, and cyclohexanediones have been described. In this contribution we report a biotype of annual ryegrass resistant to the herbicides amitrole and atrazine. This biotype became a severe problem on railway tracks in Western Australia after amitrole and atrazine had been applied in combination for ten years. The biotype is also resistant to metribuzin, the substituted urea herbicides and other triazine herbicides.

Amitrole and atrazine are applied in combination because they are mildly synergistic and provide knockdown and residual control respectively. Amitrole affects a variety of biological processes but is thought to exert its major herbicidal action via the inhibition of carotenoid production. Newly formed tissues are photo-bleached due to the absence of the protective carotenoids. Amitrole also interacts with metalloproteins such as peroxidases and catalase. The lack of a specific site of action for amitrole precludes the study of the active site as a possible cause of resistance. The triazines, substituted ureas and metribuzin are inhibitors of photosystem II. Resistance to these herbicides, particularly atrazine, is often the product of mutations of the psb A gene which codes for protein D1 in PS II. The altered D1 protein is not bound by atrazine. Thylakoids from the annual ryegrass biotype under study do not display reduced sensitivity to atrazine or diuron when compared with those of a susceptible biotype. This indicates that the mechanism of triazine and substituted urea resistance is not related to changes at the target site of these herbicides.

Maize, wheat and many other plants tolerant to triazines or substituted ureas are tolerant because they rapidly metabolise the herbicides to non-phytotoxic metabolites. When treated with a pulse of such herbicides, these plants characteristically show a brief cessation of photosynthetic activity before resuming normal levels after metabolising the herbicide. The biotype of annual ryegrass under study displays a similar pattern of photosynthetic recovery when treated with chlortoluron, a substituted urea herbicide.

The resistant annual ryegrass is able to metabolise simazine at a greater rate than the susceptible biotype. No biotype specific difference was observed in the uptake of simazine from nutrient solution. Similarly, there was no difference in the amount of ^{14}C label retained in the roots of plants from either biotype. The distribution of the herbicide within foliar tissue has not been studied, however, the observed differences in metabolism indicate that enhanced metabolism is probably a major component of the resistance mechanism.