CROP TOLERANCE TO HERBICIDES - A REVIEW

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Summary. The ultimate goal of selective weed control using herbicides is to achieve complete control of all weeds without sustaining crop injury. This is at present unattainable in the absolute sense and the state of the art of selective chemical weed control has reached a compromise based on economic considerations. The costs of alternative methods of weed control have to be weighed against the costs and benefits of using herbicides, including the direct effects of the herbicide on the crop. This paper is a brief review of the bases, problems and opportunities associated with crop tolerance to herbicides, with particular reference to the current situation in Australia.

INTRODUCTION

Crop tolerance to a herbicide may be defined as the ability of the plant to endure or resist the effects of the herbicide. Assessment of tolerance is dealt with later, but it must be said at this stage that we cannot directly measure tolerance, only intolerance, and that the herbicide effect may be negative or positive. In the first case damage has occurred, and in the second case enhancement has occurred, with the latter being either desirable or not depending on circumstances, particularly in relation to the exposure of non-target crops.

Many factors affect herbicide selectivity between plant species and account for crop tolerance to herbicides. Klingman and Ashton (1975) list seven factors as being important, namely plant age, plant growth rate, morphology, physiology,biophysical processes, biochemical processes and genetic inheritance. Field factors include edaphic and climatic factors, the circumstances of herbicide application and surfactants. This is neither the time nor place to consider mode of action of herbicides as a basis for tolerance, and the topic is covered comprehensively in Bovey and Young (1980), Audus (1976), Moreland (1980) and Kearney and Kaufman (1975). However, a systematic study of modes of action provides tools for selecting plants for herbicide tolerance even though the mechanisms involved are still not fully understood.

Commercial development of herbicides usually precedes understanding and can lead to problems. Toth, Milham and Kaldor report in this conference an experiment with paraquat which confirms field observations of toxicity to tomatoes following soil application. Paraquat was first introduced commercially in the 1960's and has been considered to be immobilised by adsorption onto soil particles and unavailable to plant roots except at very high dosage. They suggest that field damage followed root uptake due to the low clay content of the soil, superficial root development caused by the environment, and leaching of paraquat into the root zone.

The stages of crop development which are particularly sensitive to herbicides have been identified and this knowledge can be exploited to prevent crop injury. Lemerle, Fisher and Hinkley have examined this problem in wheat in relation to 2,4-D injury following application during critical stages of flower initiation. Where tillers at varying stages of development were present, spraying with 2,4-D had to be delayed to prevent crop injury. They showed an influence of plant density on crop tolerance in that in closely spaced plants the range of tiller maturities was narrower than in widely spaced plants, and earlier spraying was possible without injury to late developing tillers.

TOLERANCE OF CROP CULTIVARS TO HERBICIDES

Differential cultivar tolerance to herbicides has been known for many years, being noted for example in peas, broad beans and french beans in the early 1950's (King 1980). Active development of tolerant cultivars by plant breeders has been slow; most programmes involve screening breeding lines or cultivars rather than actively breeding for tolerance. Lupton (1980) considers that while it may be debated that it is the responsibility of the plant breeder to breed cultivars tolerant to existing herbicides, the chemist should also produce chemicals which have no harmful effects on the crops to which they are applied. The latter state of affairs currently applies to most crops, and the former can only proceed where genetically exploitable tolerance can be identified. However, Lupton states further than even where herbicide tolerance based on one or two genes may be established, plant breeders are reluctant to introduce another character to those for which they have to select, as it would reduce the overall efficiency of their programmes. In Europe, a wide range of herbicides is available for use in wheat for graminaceous weed control, and while not actively breeding for tolerance, Lupton has found that screening usually shows tolerance of at least one herbicide. To breed for tolerance to all herbicides is out of the question. This has particular significance for Australia where Plant Variety Rights are expected to be introduced soon. It will be interesting to see whether the fears of opponents of Plant Variety Rights will be borne out in any link between the specific herbicide tolerance of cultivars and the range of herbicides marketed by seedsmen's associated chemical companies.

Machado, Beversdorf and Switzer (1980) have reported a novel approach to inroducing herbicide tolerance to brassica crops. They identified triazine resistance in a weedy biotype of rapeseed (*Brassica campestris*) and have successfully incorporated it into potentially commercial lines through crossing with existing sensitive cultivars. Since the tolerance is transmitted only by the female parent, transmission of the characteristic back to sensitive weeds in the crop is not likely.

Three papers refer to cultivar tolerance to herbicides. Lemerle, Fisher and Hinkley suggest that as a result of their work on the timing of 2,4-D application in wheat, that plant breeders should select cultivars with more synchronous tiller development patterns so that 2,4-D can be applied early to minimise weed competition. In another paper, they demonstrated differential tolerance of durum wheat cultivars to the wild oat herbicides difenzoquat, flamprop-methyl and diclofop-methyl when applied at three times the normal rate. The commercial durum cultivar Durati was tolerant of flamprop-methyl, and several breeding lines showed tolerance to this and other herbicides indicating the potential to select for cultivar tolerance. Observation of grain yields indicates that the phytotoxicity observed in all plots following spraying had permanent effects on subsequent plant performance, since each plot yielded less than its control, though this was not always statistically significant. The authors conclude that variability in reports of herbicide and wheat cultivar interactions is probably due to differences in environment, herbicide rates, methodology and cultivars used.

In a study of the tolerance of some winter cereal, legume and oil seed crops to presowing applications of dicamba in a minimum tillage system, Morrow and Murrie applied four rates of dicamba at a range of times before sowing. The main objective was to establish minimum safe periods between applying the herbicide and planting the crop. Winter cereals tolerated a short period of seven days, depending on the application rate of dicamba, whereas legumes and oil seed crops required four to five weeks. Results are not presented for cultivars of each crop though it is indicated that there were no cultivar differences, and because the trial was variable large yield differences were recorded which were not statistically significant. The trial was of simple design with restricted randomisation and small datum areas were used for ease of assessment and harvesting. It seems that in this kind of trial operational simiplicity is achieved at the expense of precision.

Herbicide tolerance of crops leads to problems when crops become weeds, that is survive from preceeding crops or become contaminants in planting material. Cussans (1978) lists wheat, barley, rye, oats, potatoes, daffodils, horse radish, sugar beet, oilseed rape, mustard, ryegrasses, white clover and suckers from many fruit and vegetable crops. Australian experience would add sweet corn or maize, sorghum, peas, beans, tomatoes and capsicums. In this situation, diversity of crop and cultivar tolerance to a range of herbicides could provide an opportunity for selective crop control, but at this stage there is little information available to form the basis for such an approach. The extent of the problem in Australia is unknown, but is clearly causing concern in Europe.

INTERACTION BETWEEN CHEMICALS AND CROP TOLERANCE

Pesticide interactions in plants have been reviewed by Putnam and Penner (1974); they found examples of interactions between herbicides, fungicides and insecticides which could be synergistic, additive or antagonistic when applied together or possibly sequentially. Combinations of herbicides are usually used to improve weed control, but when they are applied in a crop selective situation crop tolerance may be affected. Combinations of herbicides with other pesticides or sprays seem to be becoming more common as a means of reducing application costs, and in the absence of adequate information on compatibility the practise proceeds by trial and error.

An interesting development of chemical interactions is the use of protectants or safeners to reduce or prevent herbicide injury. Protectants provide the opportunity to widen the selectivity or margin of safety of a herbicide so that higher doses can be used to (1) control a wide range of weed species including those closely related to the crop, (2) to increase the period over which weeds are controlled or (3) to provide greater reliability under varying environmental conditions. They may also allow the use of herbicides which would otherwise not be practicable for the crop, allowing cheaper and/or more efficient herbicides to be used. They consist of two groups, protectants (such as activitated carbon), and specific chemical antidotes.

Activated carbon has been used as a seed dressing or pellet, as root dips for transplants, incorporated in potting media as localised soil application, and for inactivating herbicide residues in soil. Localised soil application has had some application in northern Queensland in protecting direct-seeded tomatoes from damage from metribuzin at a stage of growth which is normally very sensitive to damage (Wright, personal communication, 1981). However the introduction of advanced seedling raising technology has displaced direct seeding and avoids the problem of early weed control.

Specific chemical antidotes act within the crop plant and endow it with increased tolerance to certain herbicides. Although they were mentioned in the literature in the early 1960's (for example Hoffman 1962) their application has been quite slow. Antidotes are available overseas as seed dressings or in mixture with herbicides for specific crop uses, and are just appearing commercially in Australia.

Crops provided with useful protection from herbicides by antidotes include sorghum, rice, maize, oats, wheat, barley, cotton and field bean. The other major broad leaved crops showed no useful response (Blair, Parker and Kasasian 1976). In New Zealand, Cox and Swain (1978) reported no benefit from applying antidote to tomatoes.

HERBICIDES AND CROP DISEASE

Interactions between herbicides and plant pathogens and their effects on host crops have been reviewed by Katan and Eshel (1973). The wide range encountered indicates that the phenonenom of increased or decreased incidence of disease following herbicide use is not restricted to a specific group of chemicals, pathogens or crops. Herbicides include 2,4-D, substituted ureas, triazines, trifluralin, 2,2-DPA, arsenates, DMPA, diphenamid, pebulate and TCA. Pathogens include fungi attacking aerial organs (such as *Alternaria solani* and *Erisyphe graminis*) and those causing root disorders (such as *Fusarium* spp. and *Rhizoctonia solani*), as well as viruses and nematodes. Crops include tomatoes, broadbean, wheat, flax, barley, bluegrass turf, cotton, pepper, sugar beet, black currant, corn, bean, cucumber, sugar cane, oats and lucerne.

Four mechanisms have been suggested that may be involved:

1. Direct stimulatory or inhibitory effect of the herbicide on the pathogen,
2. Effects of the herbicide on the virulence of the pathogen,
3. Effects on host susceptibility, and,
4. Effects on relationships between pathogens and other organisms.

The herbicide seems to shift the equilibrium between pathogen and host. This is exploited commercially in testing seed for the presence of seed-borne pathogenic fungi, when 2,4-D is used to reduce host resistance and vitality, thus favouring pathogen development (Kozlowski 1972). Peas grown in heavy soils in southern Queensland are often affected by root rots caused by a range of pathogens. Increased disease incidence and crop stunting has been observed following the application of metribuzin, whereas DNBP has been observed as reducing the problem.

Katan and Eshel (1973) suggest that herbicides that increase disease incidence should only be used if diseases are readily controlled, and that herbicides which increase the incidence of diseases difficult to control or of long persistence should be completely excluded.

TESTING FOR HERBICIDE TOLERANCE IN CROPS

Many variables affect herbicide tolerance, and evaluation trials should be designed to include variables such as cultivars, soil types and environments if they are to give useful results. Until recently there has been little specific guidance on herbicide evaluation available, but in 1979 the Australian Weeds Committee published "Guidelines for the Field Evaluation of Herbicides". This gives suggestions for experimental procedures appropriate to the different crop situations likely to be encountered in Australia. While visual assessment of crop damage may be useful the only real measure is that of yield; trials to determine crop tolerance must be conducted in weed free conditions to prevent differential weed competition occuring or interacting with crop injury. In reality, crop phytotoxicity ratings are of little value when subsequent crop growth compensates for earlier damage or conversely when potentially damaging treatments escape detection (Rep. Weed Res. Org. 1978-79). Yield measurements should include weight (e.g. grain) and if appropriate, number (e.g. cabbage), maturity (e.g. peas, beans) and other quality assessment (e.g. oil content of oilseed crops).

Testing of herbicide tolerance of crop cultivars may be by screening techniques, where statistical tests are unnecessary to determine gross differences in response or where visual assessment is adequate. However, such trials are not adequate in cases where a no effect result is required. In such cases statistical analysis is essential and experimental error should be minimised. Bearing in mind the variation found in field experiments, and the consequent loss in time and resources, other methods of testing could be considered. For example, Clay (1980) used pot studies in which shoots and roots of strawberry plants were treated separately to determine crop tolerance, and reduced the normal time for field testing from about five years to a few months. Clay claims a good correlation between pot and field tests and cites a case where in pot studies severe leaf damage but no root damage followed treatment with oxadiazon; in field tests severe leaf damage was followed by complete recovery.

HERBICIDE TOLERANCE OF NON TARGET CROPS

Crop damage from spray drift and herbicide residues in soils and plants is a major problem in many areas of the world and is aggravated where tolerant target crops and sensitive non target crops are grown in close proximity. Rawson and Schrodter report a preliminary study of the effects of herbicide drift on cotton to determine whether dicamba was less toxic than 2,4-D. They give an example from the literature of the sensitivity of cotton to 2,4-D, citing 0.11 g ha^{-1} as causing a total loss of seed yield. Their results show reduced plant weight at 0.1 as opposed to 0.001 kg ha^{-1} of herbicide but not in proportion to the yield depression they cite, whereas particular plant organs (in this case the squares) were sensitive to 2,4-D but not to dicamba. The authors propose further work under field conditions. This problem is obviously serious and highly emotive, particularly when spray drift occurs between adjacent properties, with one receiving pollution (or more prosaically, drift) from the other. The fact that this work needs to be done suggests that crop contamination has became accepted as almost inevitable, and that judicious choice of herbicides may minimise non-target crop injury.

A further problem is interpreting injury symptoms. A number of attempts have been made to illustrate and describe herbicide injury including Lockerman *et al.* (1975), Skroch and Sheets (1977), Jennings and Nyvall (1978) and Gage and Munro (1979), but none are complete and comprehensive for any one crop, herbicide or other causes of injury.

LITERATURE CITED

Audus, L.J. 1976. Herbicides: Physiology, Biochemistry, Ecology. Academic Press, London.

Australian Weeds Committee. 1979. Guidelines for field evaluation of herbicides. Australian Govt. Publishing Service, Canberra.

Blair, A.M., C. Parker and L. Kasasian. 1976. PANS 22: 65-74.

Bovey, R.W. and A.L. Young. 1980. The Science of 2,4,5-T and associated phenoxy herbicides. John Wiley and Sons, New York.

Clay, D.V. 1980. Weed Res. 21: 97-102.

Cox, T.I. and D.J. Swain. 1978. Proc. 1st Conf. Council Aust. Weed Sci. Soc. Melbourne p. 77-82.

Cussans, G.W. 1978. Proc. 14th Brit. Weed Cont. Conf. 3: 915-921.

Gage, J.F. and H. Munro. 1979. Herbicide effects on crop plants. Queensland Department of Primary Industries, Brisbane.

Hoffman, O.L. 1962. Weeds 10: 322-323.

Jennings, V.M. and R.F. Nyvall. 1978. Misapplied row crop herbicides - symptoms and problems. Cooperative Extension Service. Iowa State University, PM 738.

Katan, J. and Y. Eshel. 1973. Residue Rev. 45: 145-177.

Kearney, P.C. and D.D. Kaufman. 1975. Herbicides: Chemistry, Degradation, and Mode of Action. Dekker, New York.

King, J.M. 1980. Proc. 15th Brit. Weed Cont. Conf. 2: 453-460.

Klingman, G. and F.M. Ashton. 1975. Weed Science: Principles and Practises. John Wiley and Sons, New York.

Kozlowski, T.T. (Ed). 1972. Seed Biology. Academic Press, New York. p. 396.

Lockerman, R.H., A.R. Putnam, R.P. Rice and W.F. Meggitt. 1975. Diagnosis and prevention of herbicide injury. Cooperative Extension Service, Michigan State University Extension Bulletin E-809.

Lupton, F.G.H. 1980. Proc. 15th Brit. Weed Cont. Conf. 3: 873-876.

Machado, V.S., W. Beversdorf and C. Switzer. 1980. Proc. 15th Brit. Weed Cont. Conf. 3: 855-860.

Moreland, D.E. 1980. Ann. Rev. Plant Physiol. 31: 597-638.

Putnam, A.R. and D. Penner. 1974. Residue Rev. 50: 73-109.

Rep. Weed Res. Org. 1978-79. 1980.

Skroch, W.A. and T.J. Sheets. 1977. Herbicide Injury Symptoms and Diagnosis. The North Carolina Agricultural Extension Service, Raleigh.