The evolution of herbicide resistance

Author Malcolm McEwen

This essay was originally submitted for a course in “Weed Ecology” as part of a BSc in Habitat and Soil Management, University of Reading (2001).

Introduction

All life on this planet survives through constant evolution and adaptation to the environment. Changes in the abiotic and biotic environment need to be responded to if an organism is to survive. It is therefore not surprising that the most abundant and successful species are those who possess the greatest ability to adapt and hence exploit a changing environment. These rules apply to all living things with, in the plant kingdom, weeds being the masters of these strategies and adaptations to new and changing environments.

Origin of herbicides

Farmers have always faced weed problems with their management and control being an essential aspect of agriculture. Prior to the advent of herbicides the control of weeds was a costly labour intensive exercise that achieved "acceptable" control rather than complete removal. With different crops having different weed associations cultural techniques resulted in selective factors favouring the proliferation of associated weeds. Consequently farmers utilised rotations and ley farming to alter the environment sufficiently to prevent weed regeneration from a previous crop. Although copper and sulphuric acid were in use as herbicides by the beginning of the 20th century, the growth in herbicide use did not begin until the development of the contact herbicide DNOC (3,5-dinitro-o-cresol) in France during the 1930's. Following the Second World War the development of the translocated herbicides 2,4-D and MCPA coupled with the introduction of the tractor began to revolutionise weed management. Chemical weed control was now easier and cost effective (Coleman-Cooke, 1965; Hathway, 1989). Since then herbicide use has increased exponentially with current usage now exceeding both fungicide and pesticide use combined (Gressel, 1991).

Herbicide mechanisms

The earlier contact herbicides have now been replaced by the selective translocated herbicides. These herbicides act by inhibiting electron-transport or the production or function of an essential synthesis within targeted plants. The effectiveness of these herbicides however is dependent on the ability of the pesticide to be adsorbed, translocated to the relevant site and then activated. Differences in species ability to adsorb and the subsequent transport and rate of motabolisation of the active principle influence the susceptibility and resistance of the plant to the herbicide. Herbicides can be the origin of the active principle or may catalyse an existing plant metabolite so as to make it phytotoxic. Where a herbicide is the progenitor of the active principle the toxicity is dependent on the rate of enzyme activity associated with bioactivation, whilst conversely enzyme activity associated with detoxification contributes to herbicide resistance. Consequently differences in rates of activation and detoxification correspond with susceptibility and resistance between plant species (Hathway 1989).

The nature of resistance

Herbicide use has undoubtedly improved crop production by eliminating both competition and making mechanical harvesting far easier. With herbicides farmers have been able to concentrate on growing only the most profitable crops without variation. The effectiveness of herbicides coupled with fertiliser and pest management improvements have largely made traditional rotations redundant. Consequently in many agricultural systems herbicide use acts as the sole selective pressure on the weed population, and whilst a chemical may be phytotoxic at a given concentration or physiological state, at a lower concentration or applied during an indifferent physiological stage, a treated plant may survive. Hence to be effective, a herbicide must reach the biochemically or physiologically sensitive site or sites of action in the phytoxic state, at a concentration sufficient to cause the death of the target plant whilst leaving the crop plant unaffected (Hathway, 1989). In this way the herbicide acts as a selective pressure that favours the crop plant over it's weeds. Herbicides are therefore chosen for which the crop species exhibits a high degree of resistance whilst the target species exhibits a high degree of susceptibility. However differences in morphological, physiological and metabolic state of individual plants influence the susceptibility of a plant to survive. Plants which receive treatment but at insufficient concentration to kill may survive to sexual maturity and produce seed. This is not resistance, it is a consequence of insufficient concentration of the active principle at the biochemically or physiologically sensitive site to kill the target plant. To be resistance the concentration of the chemical at the active site must be at a point at which death would normally or previously have occurred. To be of consequence however this resistance must be inheritable at a rate above that normally expressed within an untreated population. Research and prediction models suggest that inherited resistance be as a result of one or two inherited genes (monogenetic) and not polygenic inheritance (Gressel 1991). It is therefore likely that within a given weed population genotypic expression of herbicide resistance exists both in monogenic and polygenic forms but that polygenic resistance is not directly inheritable and has little influence on the resistance of subsequent populations. Polygenic resistance would appear to be as a result of a superior "combination" of genes within an individual whereas monogenic resistance is the result of the presence of one or two "superior" genes within the whole population.

Evolution of resistance

The continued survival of most organisms depends on the presence of specific genetic systems to maintain diversity in the face of a changing environment. Plants are constantly being subjected to external stresses from both the biotic and abiotic environment and many have developed a suite of strategies to overcome them. In an ever changing environment plants have needed to possess mechanism flexible enough to respond and deal with toxins originating from new or mutated pathogens (Richter & Ronald, 2000) and whilst the development and use of herbicides is a relatively recent event, organic toxins, originating from pathogens and competitors (alleopathy) have always needed to be dealt with. Consequently the presence of herbicide resistance within a given population may be as a result of activation of these established defence mechanisms. The subsequent development or evolution of resistance within the whole population is then dependent on the survival of the resistant genotypes and inheritance of the trait by the following generation. Once a population has acquired resistance, repeated exposure will continue to exercise selective pressures in favour of the resistant biotypes. The rate at which resistance proliferates is then dependent on the influence of the resistant population on the whole breeding population and the dominance of the genetic expression on subsequent generations. The persistence of herbicide resistance within a population is therefore dependent on both the use of the herbicide, which reinforces the selective factor, and the relative fitness of resistant

biotypes to non resistant types. The relative fitness of resistant and susceptible biotypes has therefore important consequences on the management and evolution of herbicide resistance (Radosevich et al 1991).

Resistance mechanisms

With the effectiveness of a herbicide being dependent on sufficient concentrations of the active principle at the necessary site then if a plant is to survive the effects it must metabolise and or detoxify the active principle before concentrations and resulting damage reach an irreversible threshold. With the mechanisms involved in developing resistance dependent on the nature of the herbicide and the physiology of the target plant in relation to all the selective pressures operating, modelling of resistance is made highly complicated. Given that many of the herbicide resistant biotypes so far encountered are less fit than their susceptible counterparts (Conrad & Radosevich 1979), it is likely that in the absence of the herbicide, resistant populations will be replaced by their fitter susceptible counterparts (Radosevich et al. 1991). Consequently herbicide resistance may be a temporary strategy with the potential for a weed population to become resistant and the rate at which that resistance becomes established likely to be greatest in weed populations that exhibit a higher level of genetic diversity; however there is a lack of current evidence to correlate genetic diversity with evolutionary adaptations or herbicide resistance (O'Hanlon et al. 2000). Consequently herbicide resistance should be studied in relation to the whole plant genome and not just in isolated populations.

Weed avoidance strategies

Within Grimes ecological classification of plants weeds are predominantly ruderals. As such weeds utilise changes and opportunities in the environment to establish and complete their life-cycle. Environments may be subject to sudden or periodic disturbance events to which plants must adapt to if they are to survive. Equally to migrate into new environments plants need to adjust their strategies to take advantage of or minimise the impact of the new environmental factors. Avoidance of stress can be achieved through adjustment of life style. In environments where abiotic factors impact adversely on the plant it may adjust it's life-cycle by adopting a different habit or morphology, i.e. a prostrate habit in an exposed environment, or hasten a physiological aspect, i.e. shortening or omitting a vegetative stage thus completing it's life-cycle earlier, before the onset of the stress. Since plants have no means of predicting the stresses that they may encounter development of avoidance must be established in advance of encountering the stress. Therefore within a population the ability to cope with different stresses must be present prior to encountering the stress if the species is to survive.

Dormant resistance

As mentioned above resistant biotypes will often exist within a weed population but in the absence of the herbicide, their lower degree of relative fitness prevents their proliferation within the population. However where the influence of the herbicide alters the selective factors significantly enough to favour the survival and sexual maturity of the resistant biotypes then proliferation of resistance is likely. Resistance however may not subsequently appear within following weed populations due to the dampening effect of the seed bank. Given that the role of the seed bank in maintaining the persistence of a weed population is well documented in the literature it is somewhat surprising that it's role in the evolution and persistence of herbicide resistance has not been equally studied. The ecological role of the seed bank, to ensure the survival of the species through successive stressful environments, supports the premise of the traditional farming saw "one years seed equals seven years weed". It is therefore likely that where resistance may evolve in a population, the influence of recruitment

from a non-resistant seed bank initially dilutes the presence of resistance. Resistant biotypes produce seed which is then incorporated into the seed bank which in turn provides the subsequent weed population. Consequently successive generations of weeds contain individuals originating from seed produced by both resistant and susceptible biotypes however replenishment of the seed bank is dominated by the progeny of surviving resistant biotypes. Resistance within a population will therefore only become significant once the seedbank has first become dominated by the resistant biotypes. It is therefore likely that once resistance has become evident in a weed population that the seedbank has already become dominated by resistant biotypes. The rate at which the evolution occurred is therefore an expression of the turnover within the seed bank and for the resistance to be reversed the seed bank must first be exhausted of the resistant biotypes. The assumption therefore that cessation of the use of the herbicide will result in the rapid replacement of susceptible biotypes, due to their fitness advantage, may not hold true since the seed bank will ensure adequate recruitment of resistant biotypes until the seed bank has been completely replaced. The longevity of the seedbank therefore acts as a lag to the development of resistance, a lag that is equalled by the time taken to subsequently lose resistance.

Conclusions

Molecular biology and in particular molecular evolution may help to develop a better understanding of the mechanisms involved in the complex ecological interactions within eco-systems. Herbicide resistance should not be viewed as distinct from evolution theory; just as plants have evolved and adapted to natural biotic and abiotic changes so they evolve and adapt to anthropogenic ones. However molecular evolution is now beginning to challenge the "reactive" contention of classical evolutionary theory and support the contention that evolution is pro-active. In general it is not luck that maintains the extant but design, mutation and experimentation within a genome may well be deliberate and not random or accidental. In order to gain a true competitive advantage an organism must anticipate change if it is to exploit a new environment or maintain it's foothold in an existing. This process of anticipation is therefore likely to be best expressed in plants, such as weeds, that have evolved to take advantage of disturbance events. Whilst the causes of change may be different the mechanisms and strategies that plants use to adapt and cope with herbicides are likely to be the same as those currently utilised in overcoming natural changes. Just as they are important in natural evolution so selection pressure, competitiveness, fitness and negative cross-resistance are also key factors in the development of herbicide resistance (Gressel, 1989).

References

Coleman-Cooke, J. (1965). "The harvest that kills". Odhams, London. pp 208.

Conrad, S.G. & Radosevich, S.R. (1979). Ecological fitness of Seneceo *vulgaris* and *Amaranthus retroflexus* biotypes susceptible or resistant triazine. Journal of applied ecology, 16, 171-177. Cited In Radosevich et al (1991).

Gressel, J. (1989). Why get resistance? It can be prevented or delayed. In Herbicide resistance in weeds and crops. Caseley, J.C., Cussans, G. W., Atkin, R. K. (Eds.) (1991). Butterworth-Heinemann, Oxford. 513pp.

Hathway, D.E. (1989). Molecular mechanisms of herbicide selectivity. Oxford Uni. Press. pp 214.

Radosevich, S.R., Maxwell, B.D., Roush, M.L. (1991). Managing herbicide resistance through fitness and gene flow. In Herbicide resistance in weeds and crops. Caseley, J.C., Cussans, G. W., Atkin, R. K. (Eds.) (1991). Butterworth-Heinemann, Oxford. 513pp.

Richter, T.E. & Ronald, P.C. (2000). The evolution of disease resistance genes. Plant molecular biology 42: 195-204.