COMPUTER MODELS FOR STUDYING THE DEVELOPMENT AND MANAGEMENT OF PESTICIDE RESISTANCE

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The ability of pest species to develop resistance to pesticides is now an accepted fact. Given the number of cases reported, surprisingly little is known about the phenomenon. This lack of knowledge reflects the difficulty of studying resistance. Mutant genes occur at random and cannot be detected at low frequencies and so the presence and behaviour of these initially rare genes is almost impossible to study. Normally the development of detectable resistance takes many years and by the time control failure occurs the resistance gene is firmly established in the population.

The study of resistant populations and, perhaps more importantly, how to evaluate strategies for preventing or delaying the development of pesticide resistance is a challenging problem. There is no shortage of tactics for affecting the rate at which resistance develops but such tactics cannot be tested in the field in the same way as those aimed solely at reducing pest damage. Decisions on which strategies are most likely to delay or prevent resistance have largely been the product of informed speculation.

Much of our present understanding of the dynamics of pesticide resistance is derived from simple mathematical models of genetic mechanisms (Comins 1977; Taylor and Georgiou 1979; Tabashnik and Croft 1982; Mani 1985). These models have given valuable insights into the importance of such factors as immigration, dose rate, dose frequency, differential fitness and reversion and the use of alternation/mixtures of pesticides. They have stimulated considerable informed debate and have served to focus expensive and difficult research. Although contributing significantly to our understanding of general principles, such models are limited in their ability to make predictions about specific instances of resistance.

The applicability of simple genetic models can be greatly enhanced by their incorporation into more complex population models for a particular pest species (e.g. Sutherst *et al* 1979; Sutherst and Comins 1979). A major advantage in this approach is that the pest model can be validated against field data and the ecological parameters relating to resistance development, with the exception of details of the genetic mechanism and the initial gene frequencies, can all be measured in the field. Experimental evidence on factors such as whether resistance is mono- or polygenic and the degree of heterozygote dominance can significantly improve confidence in model output.

We are currently developing a complex simulation model for the epidemiology of gastrointestinal nematodes of sheep in order to study the development of drench resistance. The model is based on many years of accumulated field and laboratory data and successfully reproduces the epidemiology of the parasites under a wide range of pesticide regimes (Brunsdon and Vlassoff 1982). The model has suggested times in the yearly cycle which may be sensitive to disruption, and may lead to improved nematode control strategies.

The model is still being developed, but as a first step a resistance mechanism based on monogenic inheritance has been incorporated. This has been used to investigate the influence of frequency and timing of drench application on the development of resistance. Not surprisingly, outputs suggest that selection for resistance increases with drench frequency (Fig. 1). However, what is unexpected is the suggestion that where drench frequency is low, there is a significant advantage in terms of resistance development from applying drenches in the early part of the season. These results are based on the same assumptions as other models (Sutherst and Comins 1979; Sutherst *et al* 1979), but must be considered preliminary in view of the fact that drench resistance in

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Fig. 1: Predicted annual increase in resistant gene frequency under different drench regimes. Assumptions are monogenic resistance, intermediate heterozygote dominance and no differential fitness.
START: First drench given x weeks after weaning.
DRENCHES: Number of drenches at 4 weekly intervals.
FREQR: Times increase in R gene.

nematodes is generally polygenic (N. Anderson, pers. comm.). Further work is in progress to examine the implications of the precise mechanism operating in this case.

The above conclusion is not inherently apparent from a knowledge of nematode epidemiology and, as it would be possible to test this hypothesis in the field, stands as an excellent example of how modelling can enhance research and stimulate debate on resistance management. Without knowledge of the genetic mechanism or initial gene frequency it is impossible to develop models which can predict exactly when a particular pest population will develop resistance. The objective of modelling is rather to derive relative time scales for different control options. In this case we know something of the genetic mechanisms involved which will enable us to be more confident in our conclusions. However, even if we don't know how the resistance mechanism works we still have the option of evaluating control strategies under a range of possible mechanisms and selecting only those which are sufficiently robust to deal with all possible mechanisms. Models therefore supply a mechanism for comparison of management strategies when there is very little in the way of alternatives.

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REFERENCES

- Brunsdon, R.V. and Vlasoff, A., 1982. Parasite control — A revised approach. pp. 53-64. In: Ross, A.D. (Ed) Internal Parasites of Sheep, Technical Handbook, Animal Industries Workshop, Lincoln College.
- Comins, H.N., 1977. The management of pesticide resistance. *J. theor. Biol.* 65: 399-420.
- Mani, G.S., 1985. Evolution of resistance in the presence of two insecticides. *Genetics* 109: 761-783.

- Sutherst, R.W. and Comins, H.N., 1979. The management of acaricide resistance in the cattle tick, *Boophilus microplus* (Canestrini) (Acari: Ixodidae), in Australia. *Bull. ent. Res.* 69: 519-537.
- Sutherst, R.W., Norton, G.A., Barlow, N.D., Conway, G.R., Birley, M. and Comins, H.N., 1979. An analysis of management strategies for cattle tick (*Boophilus microplus*) control in Australia. *J. of Appl. Ecol.* 16: 359-382.
- Tabashnik, B.E. and Croft, B.A., 1982. Managing pesticide resistance in crop-arthropod complexes: interactions between biological and operational factors. *Environ. Entomol.* 11: 1137-1144.
- Taylor, C.E. and Georgioui, G.P., 1979. Suppression of insecticide resistance by alteration of gene dominance and migration. *J. Econ. Entomol.* 72: 105-109.