ENDOSULFAN RESISTANCE IN *HELICOVERPA ARMIGERA*.

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The insecticide, endosulfan, is still an effective weapon in the arsenal against Heliothis pest species. It is used extensively in spring and summer as a cheap alternative to the synthetic pyrethroids. This is despite the recurrence of endosulfan resistance first observed in the 1970's (Kay *et al.* 1983). According to the monitoring data obtained by Neil Forrester (1992), NSW Agriculture & Fisheries, endosulfan resistance was present at levels of 10-30% in the Namoi Valley during years 1986-1989. However, in the past two years resistance has undergone a sudden increase, with frequencies of up to 50% during summer. A similar pattern was observed in Emerald, although resistance frequencies are higher than in the Namoi Valley (Forrester 1992).

I have been investigating the ecology and genetics of endosulfan resistance. Of particular interest is why the frequency of resistance was low until recently even though up to 50% of insecticide applications for Heliothis control per season are with this pesticide. There are a number of possibilities:

(1) The exposure of *H. armigera* to endosulfan may be limited because most applications are in springtime against *H. punctigera.*

(2) The genetic basis of resistance may impede the rate at which resistance develops.

(3) The insecticide may still kill resistant larvae when applied at commercial rates in cotton fields.

I have investigated the last two areas.

The Genetics of Endosulfan Resistance.

When resistance is determined by a single gene there are usually three kinds of individuals in the populations: resistant homozygotes that have two copies of the resistance gene; resistant heterozygotes that have only one copy; and susceptible individuals that have no copies.

The rate at which resistance develops depends, in part, on the relative dominance of the heterozygotes compared with the fully resistant homozygotes. If the heterozygotes resemble the susceptible individuals then the gene is recessive and resistance develops very slowly, if at all. The rate is maximised when the heterozygotes resemble the resistant homozygotes; the gene is said to be dominant.

Endosulfan resistance is sex-linked (Daly & Fisk 1991), so that the gene(s) conferring resistance is located on the sex chromosomes. This means that females have only one copy of the gene associated with resistance, either the susceptible or resistant one. Males have two copies of the gene so that they have all three type of individuals described above. In endosulfan, females with the resistance gene are as resistant (21- to 34-fold) as males which have two copies. Heterozygous males have an intermediate form of resistance (10-fold).

This mode of inheritance is particularly important when resistance is at low frequency, say when less than 10% of individuals are resistant, a situation that occurs when resistance is first evolving. At these frequencies, the pattern of inheritance depends on which parent is resistant. Resistant mothers give rise to susceptible daughters and heterozygous sons; heterozygous resistant fathers produce fully resistant daughters and susceptible sons. The rate at which resistance develops can be maximised in

such a genetic system because even at low frequencies, fully resistant individuals (all females) are present in the population.

Thus, the genetics of endosulfan resistance would appear to favour rapid evolution of resistance.

Discrimination between resistant and susceptible individuals in cotton fields.

Relative dominance relationships, described above, were derived in laboratory conditions under carefully controlled conditions. These relationships may differ in the cotton field under realistic conditions. Such a situation was observed with pyrethroid resistance in which very small larvae were killed at commercial application rates even if they are resistant (Daly et al. 1988).

Resistant and susceptible larvae were exposed to cotton leaves treated under commercial conditions with aerial applications of endosulfan. Progeny from the cross between the resistant and susceptible parents were also tested. These individuals are referred to as the F_1 progeny. Leaves were placed in petri dishes on nutrient agar. Larvae of two ages, 0-d old and 2-d old were examined. The results are illustrated in the Figure.

When neonate larvae were exposed to freshly sprayed leaves, mortality of all individuals was high. There was very little difference in the mortality of the three types (susceptible, F_1 progeny, and resistant). This was still true in 2-d old larvae, although the proportion killed was slightly lower. However, when larvae were exposed to leaves 2 days after the leaves had been sprayed, mortality was very low (<10%). Again there was little difference in mortality between the three types in either neonates or 2-d old larvae. The rapid drop

in the efficacy of endosulfan over two days presumably is because of its short residual activity.

These results may explain why endosulfan resistance has not become a major problem in field populations. The window of time in which endosulfan can selectively kill susceptible versus resistant insects appears very limited, possibly to larvae 1-d old, or in larvae exposed to spray deposits the day after application, or to adults.

Why then do frequencies of resistance increase each year when there is little evidence that endosulfan can lead to selective mortality of susceptible but not resistant individuals? I have calculated that even small differences in mortality between types can lead to large changes in gene frequency in *H. armigera*, because larvae of this size are very abundant in the crop. Although most 0-d and 2-d larvae are killed by endosulfan, the gene frequency in the small number of survivors can be double that of the original population.

Thus, to the grower, endosulfan appears to retain its efficacy, even when resistance is present in the population because most small larvae can still be killed with this pesticide. However, the continued use of endosulfan leads to a detectable increase in resistance frequencies.

Conclusions

Endosulfan resistance has the potential to become a problem in field populations. However, its use on spring populations of *H. armigera* and the short residual activity of endosulfan appear to limit the risk of field failures. Future studies will determine under what conditions these failures can occur. In particular, they will focus on selection in 1-d old larvae and in adults.

References

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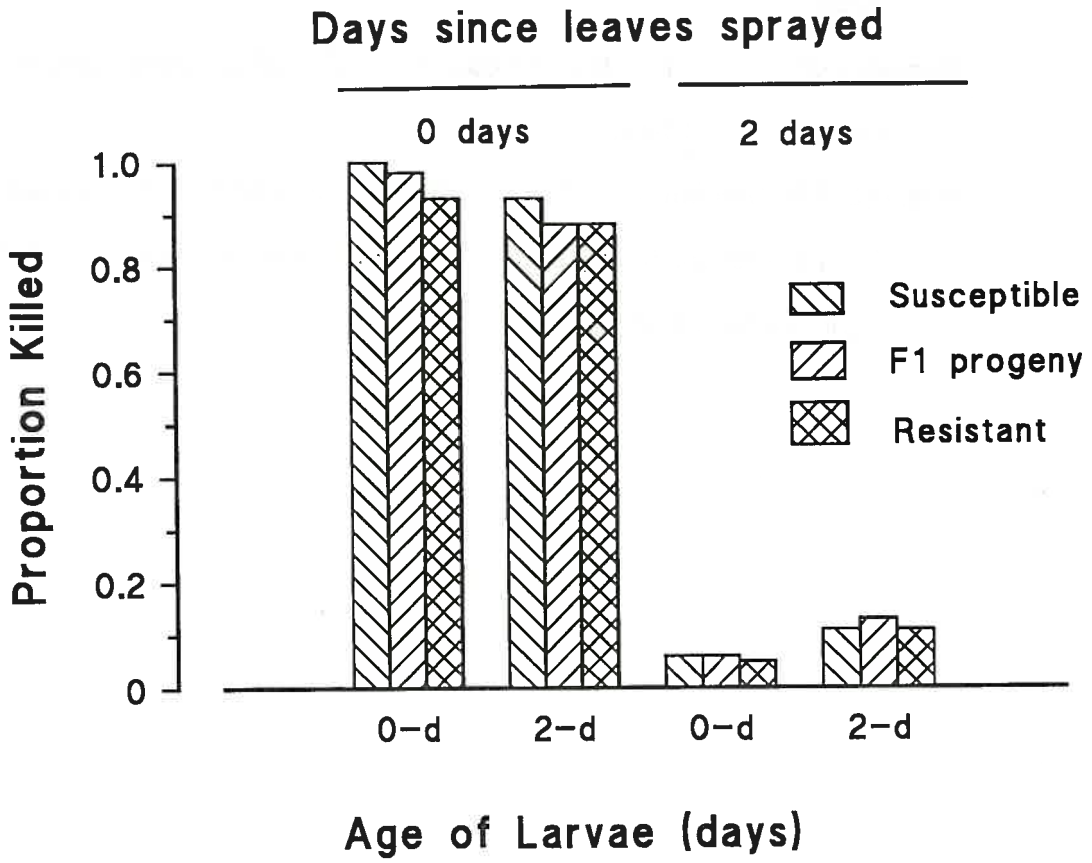

Fig. Mortality of larvae when exposed to leaves sprayed 0 or 2 days before.