North Carolina Flower Growers Bulletin

VOL. 26 NO. 5
OCTOBER 1982

OFFICIAL PUBLICATION OF THE N. C. COMMERCIAL FLOWER GROWERS' ASSOCIATION

# The Leafminer *Liriomyza trifolii*

## A Case of Developed Resistance to Insecticides

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The following is based on a presentation at the 35th Annual North Carolina Commercial Flower Growers Short Course, September 27, 1982, in Raleigh, N.C.

Resistance to insecticides is a phenomenon in which pest populations survive in spite of the application of a pesticide. There are two kinds of resistance: natural and developed (Brown and Pal, 1971). Natural resistance is shown by insects which are not susceptible to a pesticide. For example, aphids are susceptible to Pirimor but other insects are naturally resistant to it. Developed resistance is the case in which a pesticide gives good control for a period of time, usually several years, and then loses its effectiveness because of genetic changes in the pest population.

How does resistance to pesticides actually work? Pesticides affect insects in a variety of ways (Kohn, 1974), and resistance may develop in a variety of ways (Brown and Pal, 1981). For example, if a pesticide blocks a certain metabolic process, some individuals in the insect population may have a particular gene which causes an enzyme to break down the pesticide into harmless components. Insects with the gene will increase by continued pesticide application until few insects are killed by the pesticide.

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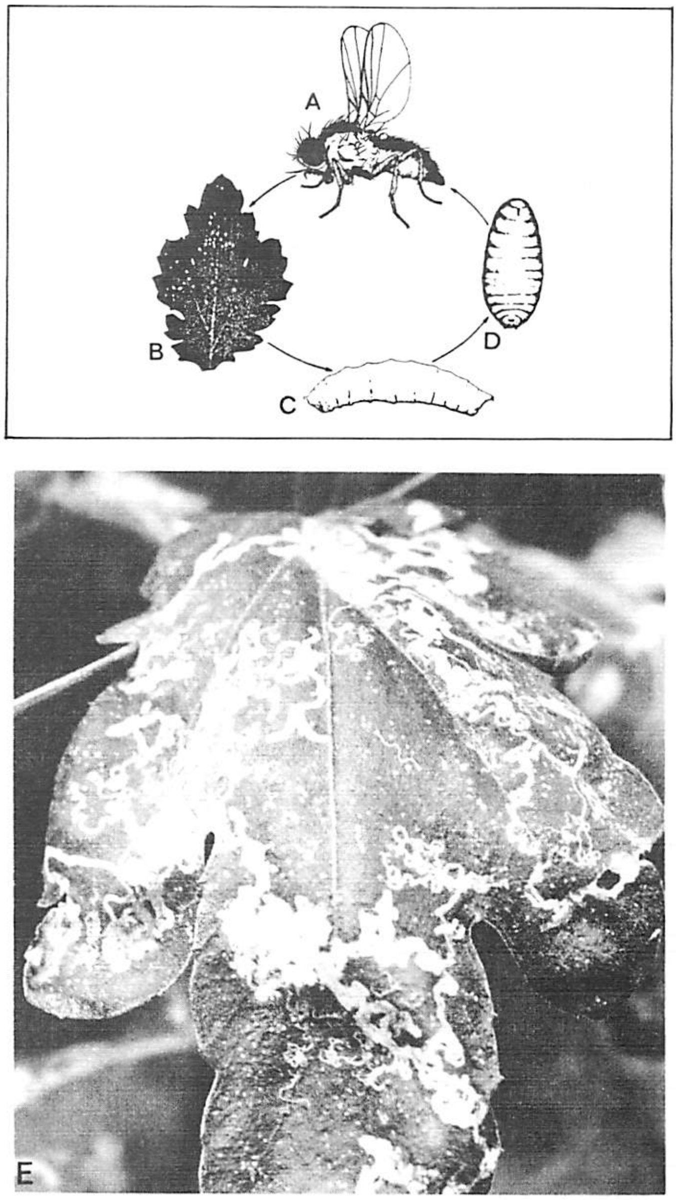


*Liriomyza trifolii*. A, Adult. B, Egg and feeding punctures. C, Larva. D, Puparium. E, Damage to chrysanthemum leaf.

The life history of Liriomyza trifolii is typical of many leafmining flies. An unusual feature seems to be that although chrysanthemums are at times heavily damaged by L. trifolii, these plants are poor hosts for this insect and many of the larvae do not survive, even if no pesticide is applied (Dr. Ralph Webb, personal communication, 1982). According to Parrella et al. (1981) chrysanthemum is not a preferred host for L. trifolii. Aster, calendula, cineraria, gerbera, baby's breath and snapdragon are other flowering crops also infested by this pest. Beans and celery are also infested.

Adult flies feed at punctures made by females with their ovipositors. About 17 times a day an egg is inserted into a leaf. Oviposition occurs for about 30 days. Hatch occurs in 3 or 4 days and the larvae mine in the leaf for 5 to 7 days. The mature larva cuts through the leaf and drops to the soil surface to transform within the skin of the mature larva into a developmental stage called a puparium. Adults emerge from the puparia in about 10 to 12 days. At warm temperatures, these insects develop from egg to adult in about three weeks (Parrella, et al., 1981). The pest develops through about 12-15 generations in North Carolina greenhouses. Thus L. trifolii is a pest which has many generations per year and which produces large numbers of offspring and which attacks crops of high commercial value.

The development of resistance to insecticides by L. trifolii is evident but poorly documented. Resistance to DDT and other chlorinated hydrocarbons was first noticed in Florida as early as 1947 on vegetables. Permethrin (Pramex®, Ambush®, Pounce®) gave very good control when first used in California, but by 1980 it was no longer effective (Parrella et al., 1981). Experience has shown that L. trifolii has become resistant to Temik® 10G, Pramex®, Penncap M®, and other insecticides in various greenhouses in North Carolina. However, there is another explanation for the sudden appearance of resistant L. trifolii populations. Chrysanthemum leafminers, Phytomyza syngenesiae; pea leafminers, Liriomyza huidobrensis; and vegetable leafminers, Liriomyza sativae, also infest chrysanthemums. These flies are fairly similar in appearance to L. trifolii but are less resistant to insecticides. When leafminers become resistant in a greenhouse, it may be that L. trifolii has replaced a pesticide susceptible species.

Because of the large numbers of pests of greenhouse floral crops, numerous kinds of pesticides are used, often at close intervals, to achieve a blemish-free crop. This use of insecticides and fungicides eliminates many insect and fungal parasites and predators of plant pests. Consequently, reproduction of plant pests in the greenhouse is hindered only by suitability of a host plant and tolerance to pesticides employed for control.

The short time required for development and the high reproductive potential of L. trifolii contribute to its tendency to develop resistance. The actual genetic mechanisms of resistance in L. trifolii are not known. However, if a single, dominant gene for resistance which doubles the survival rate occurs once in a million individuals of a pest population, after 15 generations of treatment with pesticides the gene would occur in one out of 300. In only seven more generations, half of the pests would be resistant (Hussey et al., 1969)! If the population has more than one gene for resistance, and if the genes are recessive or located on different chromosomes, the selection for resistance may take more generations although the eventual manifestation of resistance may seem abrupt (Hussey et al., 1969)

Another facet of developed resistance to insecticides is cross resistance. Some insects and mites which develop resistance to one insecticide often show some resistance to other insecticides in the same chemical group. For example, the predatory mite Amblyseius fallacis which was exposed to organophosphate Guthion® until it developed resistance was also found to be exceptionally resistant to Cygon®, parathion, and diazinon. This resistant mite population also showed some resistance to 13 other organophosphates (Croft, 1977).

Control of L. trifolii has been greatly confounded by resistance. There is one philosophy of control which involves alternation or rotation of insecticides at short intervals in an effort to prevent the occurrence of resistance. Although there are arguments in favor of short term rotation, it should not be practiced because the flies may develop multiple resistance to each chemical used in the short rotation scheme and cross resistance to related pesticides. The pear psylla, Psylla pyricola, has developed such complete multiple and cross resistance in some areas that growers at least temporarily abandoned all chemical control efforts against the pest. There is no evidence that short term rotation of insecticides delays the onset of resistance to any of the chemicals used in the rotation (Brader, 1977).

There is another aspect of developed resistance: genes that show resistance are not as suitable for the existence of L. trifolii as were the genes they replaced (otherwise the population would have been naturally resistant initially. Unless the population is continually subjected to the same selective pressure pesticide, non-resistant genes will gradually replace resistant genes as time goes by. This is called recession of resistance (Brader, 1977). Complete recession may occur in as few as 13 generations (Croft, 1977). However, in some pests resistance to a pesticide may persist through 45 or more generations (Glass, 1960). There is always the possibility the non-resistant genes might be eliminated completely in an isolated greenhouse population, a phenomenon called genetic drift (Grant, 1963). Hussey et al. (1969) even think it would be possible for a pest such as the green peach aphid to be susceptible to a certain pesticide in one range and resistant to it in a different range of the same greenhouse operation! In a case of circumstantial evidence of recession of resistance, one grower recently reported he was getting good control of L. trifolli with diazinon, a pesticide he had not used "for years."

I believe the best control regime involves the following course of action: Think ERADICATION! Treat THOROUGHLY! Select the most effective pesticide for the control of your L. trifolii population. Use only that pesticide (use Pentac®, Plictran® or Morestan® for mites during this eradication procedure). Treat at least twice a week. When the population gets low, DON'T STOP treating twice or three times a week. Treat until two weeks after there are no more leafminer flies in the house or maggots in the leaves. Use the same pesticide as long as your population is declining. Hussey et al. (1969) recommend using the same pesticide for at least one season. The rationale is that by using the most effective insecticide at short intervals, the flies will be killed before genetic recombination can fix the resistant genes in the population. At the same time relaxing the selective pressure of other insecticides may allow recession of resistance (if there are still susceptible genes in the population). If your population is not declining after several weeks of conscientious treatment, try rotating to a chemical which used to work but which you have not used in years. Perhaps recession of resistance to that chemical will be advanced enough to allow successful eradication.

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