

## GUEST EDITORIAL



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visiting Assoc. Prof., U. of California, Berkeley; Sept.–Oct. 1980, visiting Prof., U. of Queensland. *Awards and Honors, Recognitions, etc.:* Forbes (1953) and Huxley (1973) medals and prizes of Imperial College; Karl Escherich Medal of the German Society for General and Applied Entomology (DGaaE) (1993). President (1981–83) and Hon. Treasurer (1989–), Royal Entomological Society. Member of the editorial boards of *Insect Science and its Application*, *International Journal of Pest Management*, *Journal of European Entomology* and *Tropical Agricultural Research and Extension*. *Main Research Interests:* Aphids, host plant resistance and interactions of host plant resistance with other control methods.

### **Whatever Happened to Integrated Control?**

My appointment in October 1961 to teach applied entomology at The University of Reading approximately coincided with the publication of Rachel Carson's *Silent Spring* (3). I can still remember my disbelief at those first seven words of her last chapter ('The Other Road'), which read "*We stand now where two roads diverge.*" These words expressed Rachel Carson's passionate belief that, at the end of the 1950s, humanity was, in fact, at a fork of two roads. Now was the time to choose the narrow road to salvation as opposed to continuing on the broad highway of insecticides that led to damnation! The narrow road she identified was *via*, of all things, sterile male insect release and insect growth regulators as the principal methods – with biological control a poor last.

Why my disbelief? In one hand I was holding 'The Other Road', and in the other a copy of *Hilgardia*, volume 29. In the latter, already in 1959, Stern, Smith, van den Bosch and Hagen (11) had explained how reducing insecticide dose had solved the problem of

organophosphate resistant aphids on alfalfa in California. It had allowed considerable survival of natural enemies whenever pesticide had to be applied. Their paper was entitled 'The integrated control concept'. There had been other examples of Integrated Control. One was in the Cañete Valley of Peru, where pest resistance and resurgence disasters in a massive development scheme of irrigated cotton were brought under control in the late 1950s. For a short time, only a stomach poison (lead arsenate) was permitted, and the valley had then been repopulated with natural enemies. And what about the use of DDT specifically to improve biological control on coffee in Kenya in the mid 1950s (see later)? Surely Integrated Control was more immediately relevant and practical than the hope of finding 'The Other Road'? Yet, alas, researching this other road is exactly what many entomologists did in the early 1960s. Sadly also, the authors of 'The integrated control concept' revised their definition at an FAO conference in 1967 to stress integration (of methods in general) at the expense of the previous emphasis on selective use of broad spectrum insecticides. I have always felt that it was at this moment that the evolution of ideas about pest control lost its momentum for practical implementation. The concept became bogged down in terminology and moved from 'Integrated Control' via 'Pest Management' to 'Integrated Pest Management' by 1976. It had become more an activity in the comfort of the armchair (13) than a way of dealing with pest problems on the farm.

By 1970, we had another example of the proactive use of insecticides in relation to biological control. This was a mixed insecticide/biological control system for all-year-round glasshouse chrysanthemums in the UK, where the aphid *Myzus persicae* had developed a high level of resistance to organophosphate insecticides by the early 1960s. Ten years later, the carbamate pirimicarb became available. For biochemical reasons this is a selective compound. It does not kill the parasitoid *Encarsia formosa* used for the biological control of whitefly, nor the predatory mite *Phytoseiulus persimilis* which controls red spider mite. Yet this is only part of the story. Another pesticide, dichlorvos, became important in the *Phytoseiulus*/spider mite system. The predator was very easily transported around nurseries. It invaded glasshouses set aside for rearing the pest mite, needed to re-infest the commercial houses whenever the predator was in danger of exterminating its food supply. That dichlorvos was needed to kill the predator, but not the pest, in the pest-rearing houses is a twist that just emphasizes how ingenious pesticide/biological control systems can be.

Still in 1970, a meeting of the Association of Applied Biologists was held at The University of Reading, and included a debate in which several entomologists were asked to say what they regarded as the most important idea in pest control, with only one slide permitted. It sought to explain the basis of the potential selectivity inherent in pesticide dose reductions which was a key element in the early example of integrated control in alfalfa in California mentioned earlier. There is a greater spread of insecticide tolerance (expressed in a shallower slope of the probit line in an insecticide bioassay) in populations of herbivorous than of carnivorous insects. Like the Californians, we had found this phenomenon in our studies at Reading (4). A decade later, I found a paper (9) which offered a biochemical explanation. Herbivores have a better armory of enzymes for detoxifying pesticides than carnivores, evolved from the need to deal with the diverse secondary chemistry of plant food. In turn, a diversity of detoxifying enzymes leads to more heterogeneity in the response of individuals to pesticides.

For the last 15 years I and many co-workers at Reading have been studying the effect of partial host plant resistance as an additional element in Integrated Control (1959 defini-

tion). This produces what I call the Pest Management Triad (13). Part of this triad relates to direct benefits to biological control of partial plant resistance, but this topic is outside the scope of this editorial. More relevant is the repeated result in my laboratory (2,7) and elsewhere (1,10) that aphids and caterpillars show the same kill as when reared on susceptible plant varieties at a lower insecticide dose when reared on varieties that are partially resistant. Even where the host plant resistance is so slight that they carry as much as 85% of the pest population found on a susceptible variety, it may nevertheless be possible to reduce the dose of insecticide by one-third. A small part of this effect can be explained by the reduction in the size of the pest that is generally shown on resistant varieties, but more importantly there is a true physiological increase in susceptibility to pesticide of the insect. On the basis of my 1970 slide, this dose reduction is likely to lead to increased selectivity of the pesticide. However, it has to be remembered that the natural enemy will not be similarly affected by the plant resistance. Parasitoids developing in smaller insects may be smaller themselves (6), but the effect is slight. The potential increase in selectivity that may result with a decreased insecticide dose on a partially resistant variety is therefore dramatic: the dose response curve for the pest will shift towards lower pesticide concentrations, whereas the curve for the natural enemy remains stationary. Surprisingly, recent work (12) suggests that parasitoids of both aphids and ladybird larvae may show increased resistance to insecticide on partially aphid-resistant wheat cultivars. This further increases the selectivity of a reduced dose of pesticide on such cultivars.

Two reservations have been expressed by others about these beneficial interactions between partial plant resistance and other control methods. First, pesticide dose reduction is often criticized as accelerating resistance to pesticides by allowing it to survive in the heterozygous state and to breed into homozygosity. However, this argument can itself be countered. Survivors of a high kill strategy, as well as being homozygous, must by definition be resistant. The theoretical debate on the genetic merits of low vs high kill strategies is not resolved (14). Furthermore, opponents of dose reductions tend to forget that, in a pest management system, other mortality factors such as biological control will act on the survivors, whether or not they are heterozygous for pesticide resistance. The second reservation raised (5) is that there may be pest genotypes in the population that are not affected by the plant resistance mechanism. They will therefore not suffer greater biological control mortality or show high mortality at decreased pesticide doses. The selection for such genotypes will be accelerated. This is indisputable as a concept. However, the process will be very slow on a variety showing only partial resistance as well as, as often happens, several resistance mechanisms. Furthermore, the combination will certainly not lead to selection for a host plant resistance-breaking biotype as rapidly as would achieving the same level of control by a stronger level of plant resistance on its own.

The selectivity of pesticides achievable by the introduction of partial plant resistance can be increased with ingenious use of broad spectrum insecticides. Some examples have already been mentioned, but selectivity in time or space offers obvious additional possibilities. For example, ladybirds lay eggs in proportion to aphid density on the plant. If we allow eggs to be laid first, and then reduce aphid numbers with a nonpersistent pesticide (to which the developing ladybird embryos are not sensitive while protected by the egg shell), the emerging larvae will find themselves with a greatly reduced aphid population as food. This should lead to successful biological control (8). As one example of selectivity

in space, one can cite an Australian technique of restricting spray, as far as possible, to the bottom half of citrus trees. The incorporation of a bait brings the pest fruit flies, but not their natural enemies, down from the top of the tree and into contact with the pesticide.

What all this points to is that we should not worry about absolute selectivity between pests and natural enemies. The higher the proportion of the pests we destroy when we apply pesticide, the higher also is the proportion of beneficials that we can afford to kill. Provided the proportion of beneficials killed is reduced more than the proportion of pests killed by the modification to spraying we adopt, each pesticide application will improve biological control by a favorable change in the natural enemy:pest ratio (14). An excellent example is when DDT was the agent used to make successful what was otherwise inefficient biological control of coffee loopers (15). The DDT was painted as a band around the trunks of the coffee trees. Whenever caterpillars became too abundant, the tree foliage was sprayed with a concentration of natural pyrethrum sufficient only to stun, but not to kill, insects. Both pests and beneficials then fell to the ground. When they recovered, many beneficials returned to the leaves by flying. The caterpillars could reach the leaves only by traversing the DDT band and acquiring a lethal dose of pesticide. Ingenious? Yes, very. But what a simple biological basis – caterpillars can't fly!

Much of this editorial has referred to useful synergism between partial plant resistance and other pest control measures. Unfortunately, new crop varieties developed for resistance to pests by transgenic methods are unlikely to be suitable for Integrated Control. One reason is that the resistance is likely to be very high, leaving little room for integration with a second method. More disturbingly, direct gene transfer is likely to result in resistance based on a single toxin, transferred from perhaps unrelated plants or totally unrelated sources. The first transgenic resistances have been based largely on the toxin produced by *Bacillus thuringiensis*. Single gene/toxic chemical-based host plant resistance is likely to damage, not support, biological control and may challenge the pest to an increase in resistance, rather than in susceptibility, to pesticides. We already hear stories that *B. thuringiensis* toxin-based plant resistance in the USA may have been defeated by tolerant pests. One does wonder what is so special about transgenic plants. Can a change in the delivery system, a switch from the spraying nozzle to the food source of the pest, really be expected to alter the fundamental responses of insects to strong selection pressure from a toxic substance?

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