# Honey Bees and Pesticides

## Guidelines for Future Honey Bee/Pesticide Research

**(Concluded From January Issue)**

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### Pesticides in the Honey Bee Food Chain

Far too little attention has been given to the fate by relative quantity, of pesticides in the hive. To be sure, we know from analyses (see Part II) that dead bees, pollen, nectar and wax normally all contain some pesticide residues when bees forage in a contaminated area. But, of the total quantity carried into the hive, what percentage of a given poison is accounted for in each of the above? A fraction, most or all of it?

Much has been written about the incidence of pesticides in pollen and bee bread. To be sure they are there, both as free chemical residues and bound up with the lipids (fats) that are prevalent in pollens. But, previous concepts regarding larval diets have created confusion relative to the significance of contaminated pollen. We now know that bee larvae are not fed bee bread. Rather, newly emerged adult bees, up to 10-12 days of age, consume this stored pollen which they convert into at least two glandular products that are fed in varying proportions to larvae of all ages. Hence, we must ask which, if any, of the many pesticides used are passed along to larvae in their diets? Existing knowledge would tell us that some may be while probably others are not. Of those that are, what are the relative short and long term hazards to the individual? Will the larva die? The pupa? Will the emergent adult be fully functional? Three-fourths of a larva consists of fat bodies — principal sites for sequestering (storing) or detoxification of harmful chemicals. What is the role of these fat bodies? What happens at pupation?

Of those pesticides not passed along in the diet, what is the fate of each in the nurse bees body? Are they sequestered in fat bodies or cuticle (exoskeleton), are they detoxified, passed along through the digestive tract and deposited with the feces, or do they remain within the bee and kill it? If so, how long does this take and at what temperature and humidity? What effect does this have on worker bee efficiency and the ability to produce brood food of full nutritive value? What might the effect be on the aging process in bees and conversely what is the effect of individual bee age on the efficiency of each of these mechanisms? Obviously, if we knew that some man-made toxins are sequestered or detoxified by bees in the same way that they or other insects manage naturally occurring plant toxins, we might capitalize on this when developing pest control recommendations. We do know that many pesticides are specifically formulated to insure that they are rapidly absorbed and concentrated in living systems.

Often it is said that pesticide residues seldom occur in honey, and indeed recent studies seem to bear this out. Certainly, when they do occur they are usually at comparatively small levels. But, as Elbert Jaycox pointed out in 1964, we can no longer accept, without proof, the often repeated statements that the purity of

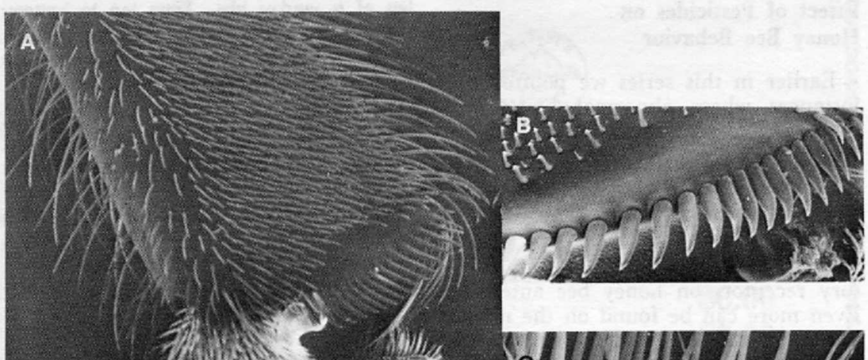


**Figure 3. A) Inside surface of worker bee hind leg showing pollen press at center and pollen comb on the basitarsus below. B) Enlargement showing rastellum (rake) above the press. This row of spines is used to clean the comb of the opposite leg. C) A higher magnification of the floor of the pollen press. Pollen and pesticide contaminated particles are gathered and processed with these structures.**

honey is assured because bees gathering poisoned nectar are killed in the field or die in the hive before giving up their contaminated load. The question is, what is the protective (cleaning) mechanism for honey and how can we use it to further protect the colony?

There are other related questions to be asked. We know, for example, that in the honey bee intestinal tract certain microflora are essential for normal digestion of food. But we don't know what the effects of pesticides are on these microorganisms or to what extent pesticides act as biochemical inhibitors — limiting digestion and assimilation of nutrients. For example, pesticides may slow protein production by the bees, thus slowing their recovery from the effects of toxins. Occasionally, complete protein inhibition may occur. There is some data on the effects of these toxins on the central nervous system, but one must also ask what the effects of pesticides are on other functions such as egg production, sperm viability and mating. Finally, we must learn how toxins affect hormone and pheromone production, either those necessary for normal colony development and behavior or for mating and reproduction. Certainly, these and all other bee/pesticide interactions are dosage dependent. It is in issues such as these that sublethal effects of pesticides might be most important.

### Effect of Pesticides on Honey Bee Behavior

Earlier in this series we pointed out instances where abnormal honey bee foraging and malformed adult bees resulted from certain pesticide exposures. But what about effects on other essential activities and other abnormalities less easily detected?

There are thousands of tiny sensory receptors on honey bee antennae. Even more can be found on the mouth parts and elsewhere on the bee's body. These function variously to assist the insect through its daily activities such as locating food, eating, locating and feeding hungry larvae, regulating the hive environment (e.g. temperature and humidity), cleaning cells, foraging, mating, etc. If the sensory system of the insect is fouled by a toxin, will the individual starve, incorrectly or inadequately rear young, create an unbalanced drone to worker bee ratio, or maintain improper cluster conditions (e.g. temperature and humidity) for normal activity and brood rearing? In short, at what point will the bees' bodily functions become erratic, inefficient or incomplete? Almost certainly, we should expect to see some or all of these effects.

We have often discussed the likelihood that undetected sublethal effects of pesticides play a role in reducing or altering normal colony activity such that honey production or pollination efficiency is reduced. We have also speculated on the role of pesticides in altering the protective layer of fatty acids (cuticular lipids) on the insect's body surface. If these are changed or destroyed, the insect becomes vulnerable to attack by microorganisms and to other problems such as dessication. The difficult part is devising ways to measure these sublethal effects.

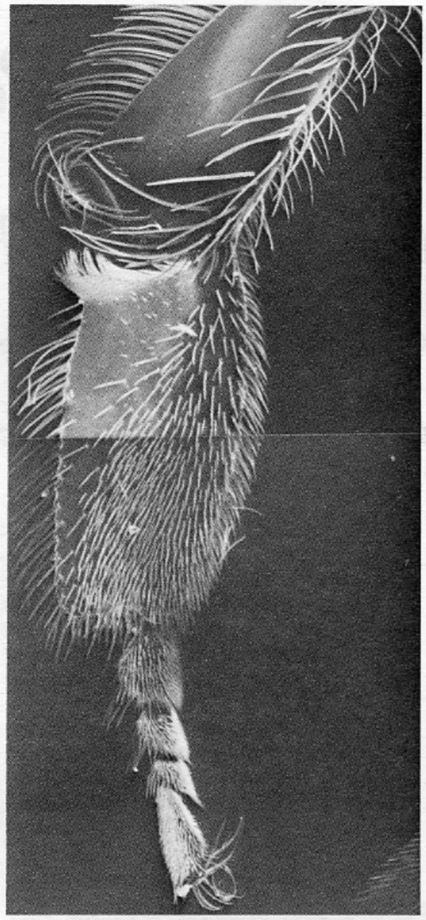

**Figure 4. Outside surface of the hind leg of a worker bee. From top to bottom: the pollen basket, pollen press, basitarsus, remaining tarsal segments including the terminal claw bearing foot.**

### Stress and Pesticide Susceptibility

There is a need to develop a stress index (stress model) for honey bee colonies as a way of more accurately interpreting the highly variable and often contradictory results now coming out of studies of pesticides and honey bees. We at Madison have initiated such an effort. Colony size is a consideration in any stress model as is food availability, weather and climate and the efficiency of the bees' natural defense against toxic chemicals. Stress thresholds must be determined for each major factor in colony development and behavior. For example, a small cluster of bees has a greater surface to volume ratio than a large cluster. Hence, one must expect that the smaller population has a greater struggle for survival under stress (e.g. temperature and humidity extremes) than a large one. It may in fact be that stress limits population size more often than we recognize. This undoubtedly happens during winter in the northern climates, and in the hot desert of the Southwest. It probably happens elsewhere in less discernable fashion. In these circumstances the bees struggle to maintain temperature and, perhaps more importantly, humidity in the hive. The ability of the bees to overcome obstacles imposed by the environment depends in large part upon limitations imposed by food reserves, age and their overall physical well being as affected by stress factors including pesticides.

There is a great need to study the composition of colonies of bees by age group. A study of mortality by age due to a given pesticide is of even greater importance. Some data like this exists but what is needed is data at least for families of insecticides (carbamates, organophosphates, etc.) so that the effect of a pesticide on age groups of bees can be predicted. This would then allow some implementation of preventive measures.

Again, as we pointed out in Part II, residues of most pesticides persist in the hive for long periods of time — primarily in the wax and pollen: A thread of evidence for the sublethal effects of these residues is common throughout all reported bee losses. It is easy to see that when contaminated stored pollen (bee bread) is eaten further toxic effects result. But, it is much more difficult to determine the extent of which each pesticide is permanently stored in the wax such that it poses no threat to bees and how much continuously emanates from the wax imposing a chronically toxic environment. We don't know if there is any fumigant action from insecticides absorbed into wax comb and stored pollen. Nor do we know whether antidotes might be used to neutralize the chemical thus absorbed. There has only been a small amount of unsuccessful research on this aspect. We do know that methyl parathion has been detected in wax following simulated commercial wax processing. Similarly, samples of commercially refined beeswax and wax foundation have been shown to contain pentachlorophenol. Whether these levels are harmful to bees is unknown. We need to develop decay curves for pesticide residues in bees and bee products.

For various reasons, some beekeepers have advocated replacing old dark combs every 6-8 years. Those who do this now report that the benefits in terms of stronger, healthier, more productive colonies outweigh the loss in terms of beeswax and comb construction. Could it be that this removal of old combs also eliminates persistent pesticide residues along with other undesirable elements? Or might it be that newly constructed comb contains some natural detoxification (protective) mechanism that is depleted over time? Similar stimulatory effects have been noted following ethylene oxide fumigation of hives. Could this powerful reducing agent have a similar effect by detoxifying harmful chemicals?

**Insecticide Resistence in Honey Bees**

Generally, there are at least three types of heritable resistance to insecticides that occur in insects. Those systems that enable insects to cope with toxic chemicals in their environments are: 1) metabolic degradation; 2) differential penetration or sequestering and 3) insensitivity. In the first, various enzyme systems have been shown to detoxify pesticides before they can harm the insect. As previously noted, certain of these systems in honey bees have been studied and we now have some knowledge of their effectiveness. However, much work remains to be done.

Inherited differences in the ease of insecticide penetration through the insect's cuticle have also been shown to exist. While little is known about this phenomenon, certain morphological characteristics such as differences in body hairs and the insect cuticle have been shown to be related to this type of insecticide resistance. As previously noted, insects have the ability to sequester toxic substances in specialized glands or body tissue which prevent the toxin from interacting with the metabolic processes that would be otherwise affected.

The third type, insect insensitivity to the chemical, is notable because its incidence is on the rise. One measure of insensitivity is something called knockdown resistance (KDR). While the mode of action here is unknown, we do know that insects have a high KDR to DDT and the Permethrins. Perhaps this is why they have been identified as "less hazardous to bees" when acute immediate mortality is determined. Nevertheless, this phenomenon has no known effect on residues in the hive nor on the effect of these residues on colony performance.

The most obvious question that must be asked in light of the foregoing is, can we develop insecticide resistant bees based on any one of the three types of resistance? Presently, there is no good answer. However, procedures for breeding bees for insecticide resistance have been identified. The limitation may be the difficulty of holding specific desirable germplasm for long periods of time. However, newly conceived closed populations may be an effective means of doing this.

All studies to date concerned with pesticide residues in bees (as opposed to bee products) focus on dead bees i.e. those dead in front of the hive or taken from dead bee traps. To our knowledge little concern has been paid to pesticide residues in live bees. So when LD50's are established for a given pesticide, we need to ask several questions such as: Why did 50% of the bees not die? What pesticide residues are found in live bees from exposed colonies? And, did these bees survive when bees from the same colony and perhaps with the same level of pesticide residue did not?

If a breeding program were undertaken, perhaps screening for resistance would best be carried out using drones. Since drones are haploid (come from unfertilized eggs), we would know that the full complement of any observed resistance came from the queen. Hence, a well organized breeding program could be started from this well defined genetic base. Maintenance of the resultant germ plasm could be accomplished by sperm storage (once perfected) or within a closed population system. Then we would only have to worry about that old bugaboo, environmental variability.

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### Analytical Procedures

Simple inexpensive pesticide detection methods that can handle large numbers of samples of bees and bee products relatively inexpensively are needed to make possible the research needed for fully understanding the interaction(s) of bees and pesticides. The lack of such methods is presently a major research limitation. One such method, immunoassay, offers great promise in this regard. Immunoassay employs highly specific and sensitive antibodies, which have been produced in response to certain pesticides. Methodology has already been developed for several insecticides and insect growth regulators. Further research is now needed to expand immunoassay procedures to the bee-pesticide complex: We at Madison have undertaken such cooperative studies. Perhaps the greatest contribution of these procedures will be the ability to simultaneously analyze for several toxins rapidly and inexpensively. This new tool will also open up many new avenues of investigation.

### Epilogue

In this paper we have raised many important and thought provoking questions in the hope that others might use them as springboards for their own research efforts. Most of these questions remain unanswered just as many more questions could be raised. We have suggested that future research objectives emphasize studies designed to look for differences that might stimulate research in other environs or build basic concepts applicable to a wide range of pesticides. If in so doing we have contributed in some small way to the development of lasting and meaningful solutions to the bee/pesticide problem complex, we will feel that the effort was worth it, whatever the contribution.

Changes in pesticide use practices are evolving constantly. New and more efficient ways to kill pest insects are being developed. Some of this comes about by an earnest desire to protect bees. With these changes and from research advances will come a need to periodically reassess some of the perspectives presented in this series. We would strongly encourage others to provide the beekeeping industry and the scientific community with appropriate updates on the issue of bees and pesticides. ●