Update on research on Varroa jacobsoni at Rothamsted.

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UPDATE ON RESEARCH ON *VARROA JACOBSONI* AT ROTHAMSTED

The last article in BBKA News 98 described the background to our current research at Rothamsted on Varroa.

In 1993, our initial studies (funded by MAFF) on *Varroa* infested British colonies in Devon identified slow paralysis virus (SPV) as the main cause of mortality. A number of other honey bee viruses were identified in both dead adult bees and brood from infested colonies but the incidence and prevalence of these infections was similar to that in uninfested colonies. Some mortality due to honey bee virus infection occurs in most colonies at some time during the year, but this occurs in both mite infested and uninfested colonies. The mortality of adult bees and brood due to APV or SPV infection in infested colonies was unexpected as neither of these two viruses has previously been found to be responsible for disease outbreaks in nature. Both APV and SPV normally persist in the adult bee population only as inapparent or latent infections, and the mite in some way induces the virus to multiply to lethal levels. Once the virus is actively multiplying in individuals, infection rapidly becomes systemic and the mite can then transfer virus to other adult bees or developing pupae within brood cells. If this transmission cycle is stopped (by acaricide treatment, or other means) at an early stage, and the rearing of healthy brood can resume, the colony has an excellent chance of recovery. If, however, the percentage of virus infected individuals in the bee population continues to increase, the spread of infection within the colony no longer depends on the mite. Acaricide treatment at this stage is too late to prevent the loss of the colony.

It is apparent that in 1995, for the first time in this country, substantial losses of colonies have occurred in apiaries infested by *Varroa*. Colony mortality has often occurred despite treatment with proprietary acaricides in the early spring, as advised. It is important to bear in mind, however, that colony losses occurred before *Varroa* became established in this country, and that all current losses cannot be ascribed to the mite. From the information we have gathered, a good indicator of virus associated mortality due to mite infestation is the unusual occurrence of dead brood late in the summer. Although there is adult bee mortality, piles of dead bees on the floorboard or beneath the entrance to colonies are rarely seen (i.e. there is no 'external' indication of problems at this stage).

During 1994, our research concentrated on six infested untreated colonies, which were intensively studied. Every three weeks, dead bees were collected from entrance traps, dead mites were collected from the floor debris, and total adult bee and brood populations were estimated. Approximately 1% of the live adult bees, and 1% of the sealed brood were also collected to measure their degree of infestation. This enabled the total mite population to be estimated, and to be compared with the bee and brood populations. Samples of live and dead bees, infested and uninfested brood and mites were then saved and analysed for the presence of a range of pathogens, especially viruses.

This information showed that from a small number of mites present in the early spring, populations can increase rapidly, during the period of maximum bee population growth. Mite populations peaked in late summer, and then rapidly declined, as has been observed in other colonies in the UK, ref. Dr. S. Martin's work. The decline occurs at the time when honey bee brood rearing is drastically reduced, and when there is excessive competition amongst mites for brood cells in which to reproduce. We, have observed that a large proportion of dead mites at this time of year are pale in colour, indicating immaturity and mortality before reproducing.

Our studies have suggested that the rate of increase of the bee population is a far more important factor affecting mite population growth than the number of mites initially present. In a weak colony, with erratic brood laying, or under poor weather conditions, mite numbers may increase very little over a season. In contrast, in a large colony with a young queen, and under good weather conditions, mite populations can increase rapidly. What appears to have happened in 1995 is that the exceptionally good summer favoured both the bee and the mite, allowing mite populations to increase rapidly from low levels to very high levels, prompting the appearance of virus infections and colony losses.

Virus analysis of the 1994 samples showed the SPV was a primary cause of adult bee and brood mortality, as in the previous year. Some dead brood samples were also found to contain much cloudy wing virus (CWV), previously known only as a disease of adult bees, and sacbrood virus (SBV) was also commonly found early in the

season in infested colonies.

SPV could not be detected by sensitive serological tests in live bees or brood any earlier than it was detected in dead bees. In contrast, SPV was detectable in mites two months before it could be detected in the bees, implying that early prediction of virus infections in colonies may be possible, which would allow timely treatment of the mites to prevent colony loss.

In 1995, our work has concentrated on laboratory studies in order to quantify the direct damaging effects of mites feeding on pupae and adult bees. Studies elsewhere have correlated increasing mite infestation with increasing mortality of pupae, but these did not take account of the possible presence of pathogens such as viruses. Our studies, using bees and mites in the absence of viruses, have shown that the *direct* effects of mite feeding on both adult bees and brood appear to be minimal. Bees emerging from infested cells initially have a lower body weight (20% less with five mites per cell) than those from uninfested cells, but these differences soon disappear, and we have found no significant differences between the longevity of bees emerging from infested and uninfested cells in cage experiments.

The mechanism of virus induction is not yet understood, but a postgraduate student, Colin Denholm, has recently begun a three-year MAFF funded PhD studentship at Rothamsted in collaboration with Dr. William Kirk at Keele University to study the problem.

Our other studies also have practical applications. More precise timing and targeting of acaricide treatments will reduce losses due to secondary infections and minimise chemical inputs into colonies. The mites themselves are probably sensitive indicators of virus presence in colonies, and the development of a predictive test could help to identify those colonies most likely to suffer damage. In addition, further work on the effect of the timing of acaricide treatment on virus epidemiology would define more accurately when action was required. As with most biological systems, the process of infestation by *Varroa* is affected by many conflicting factors. The growth of the mite population depends on the population of adult bees and brood, which in turn is dependant on weather conditions, forage availability, the degree of competition from other bees, the age of the queen, and presence of disease. Secondary virus infections add further variability to the system. We can, however, be certain that the final death of a colony is unlikely to be due to the effects of *Varroa* alone, or due to the effects of a single pathogen. Only by investigating and evaluating the factors involved can we hope to understand the process of infestation, which is essential to devise effective control strategies for the future.