

VIRUS SUCCESSION IN HONEYBEE COLONIES INFESTED WITH *VARROA DESTRUCTOR*

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Introduction

When *Varroa destructor* nov. sp. (ANDERSON and TRUEMAN, 2000) was first discovered in Europe in the 1970s, it was rapidly found to be a devastating pest of *Apis mellifera*. It was soon apparent however that the damage to colonies was not solely related to the size of the mite population. It was therefore suggested that secondary infections by other pathogens might explain the observed variation in the effect of mite parasitism. Work in Germany and elsewhere on mainland Europe in the early 1980s (BALL, 1985; BALL and ALLEN, 1988) showed that the primary cause of mortality in severely infested colonies was acute paralysis virus (APV), a virus never previously known to cause mortality in nature (BAILEY and BALL, 1991; BALL and BAILEY, 1997). Further work demonstrated that the mite could act as a vector of a number of honey viruses (BALL, 1989), and led to the discovery of a previously undescribed virus, deformed wing virus (DWV) in infested colonies (BAILEY and BALL, 1991; BALL and BAILEY, 1997).

Varroa destructor in Britain

Varroa destructor was discovered in Devon in south west England early in 1992, and has since spread to most parts of the British Isles. Field studies initiated at this time aimed to determine the primary causes of colony mortality associated with mite infestation, and to identify significant changes in virus prevalence by comparison with data collected over many years prior to the arrival of the mite.

Investigations in Devon and Hertfordshire during 1992-6 determined that the death of severely infested colonies was associated with slow paralysis virus (SPV), an infection which had never previously been found to be responsible for mortality in nature (CARRECK, 1994; BALL, 1997). The occurrence of SPV in dead adult bees and brood seems to be analogous to that of APV in infested colonies on the European mainland, although, curiously, APV has rarely been found as a cause of mortality in infested colonies in Britain. Both of these viruses normally persist as inapparent or latent infections in bees, are infective by injection into the haemolymph, are rapidly fatal, and are efficiently transmitted to both brood and adult bees by the mite.

A number of other viruses, filamentous virus (FV), black queen-cell virus (BQCV), bee virus Y (BVY) and bee virus X (BVX), were also detected in dead adult bees from infested colonies. However, the prevalence of these infections was similar to previous results obtained from uninfested colonies and was independent of mite infestation levels (BALL, 1997). Significantly, these viruses are all only infective for adult bees by ingestion and would not therefore be expected to be transmitted by the feeding activities of mites. Cloudy wing virus (CWV) was frequently detected in infested colonies in Devon and showed an increased prevalence in comparison to earlier observations (BAILEY et al., 1981).

Detailed studies in a small number of colonies investigated the seasonal incidence of SPV and CWV and the possible role of the mite in the initiation and spread of virus infection. In contrast to CWV, the appearance and establishment of SPV was strongly seasonal and associated with large populations of mites. Towards the end of summer, as brood rearing declined, there was a progressive increase in the proportion of brood that was both infested with mites and infected with SPV. Subsequent increased transmission of virus to adult bees often resulted in the spectacular collapse of the colony over a matter of weeks. Although CWV was detected in a surprisingly large proportion of the brood in infected colonies, there was no difference in virus distribution between infested and non-infested cells, indicating that transmission was independent of the mite (CARRECK, 1996; BALL 1997; CARRECK, 1998; CARRECK et al., 1999a).

Although DWV was not found in the early field studies in Devon, during late 1996 we began to detect the virus in infested colonies at Rothamsted, and by 1998 it was virtually ubiquitous in our experimental colonies. (CARRECK et al., 1999b). DWV was known as an infection of adult bees in Britain prior to the arrival of *V. destructor* but a significant increase in the prevalence of this virus has occurred over the period of a few years.

In order to determine whether the changing pattern of virus prevalence observed in our experimental colonies was reflected throughout Britain, during the autumn of 2000 beekeepers were invited to send us mite samples. A total of 207 samples from 27 English counties were received and analysed for a range of viruses by enzyme-linked immunosorbent assay (ELISA). APV was detected in only one sample, confirming

its rare occurrence. SPV was also uncommon, being detected in 4% of samples. This low prevalence is probably a reflection of the use of acaricide treatments by beekeepers, limiting mite population development. The two most prevalent viruses detected in mites were CWV in 45% and DWV in 87% of samples. This suggests that DWV is now almost universally present in infested colonies in Britain.

Fig. 1 shows changes in the prevalence of CWV, SPV and DWV in experimental colonies over a number of years. The pre *V. destructor* data is derived from sampling carried out at Rothamsted between 1987 and 1986. Each month, dead bees were collected from approximately 25 undisturbed colonies and analysed for a wide range of pathogens. Over this nine year period, CWV was detected in an average of 14% of colonies, whilst SPV and DWV (which was only tested for in the last two years) were not detected at all (BAILEY et al., 1981; BALL and ALLEN, unpublished data). In contrast, in the first five years after *V. destructor* was detected in Britain SPV infections appeared in severely infested colonies, whilst towards the end of that period DWV began to be detected. In the second five-year period, SPV continued to increase in prevalence, and DWV became almost universal. The markedly greater prevalence of SPV in experimental colonies in comparison to the national average in infested colonies may be explained by the absence of mite control measures in these colonies and the natural spread of mites containing virus to neighbouring colonies also used in studies.

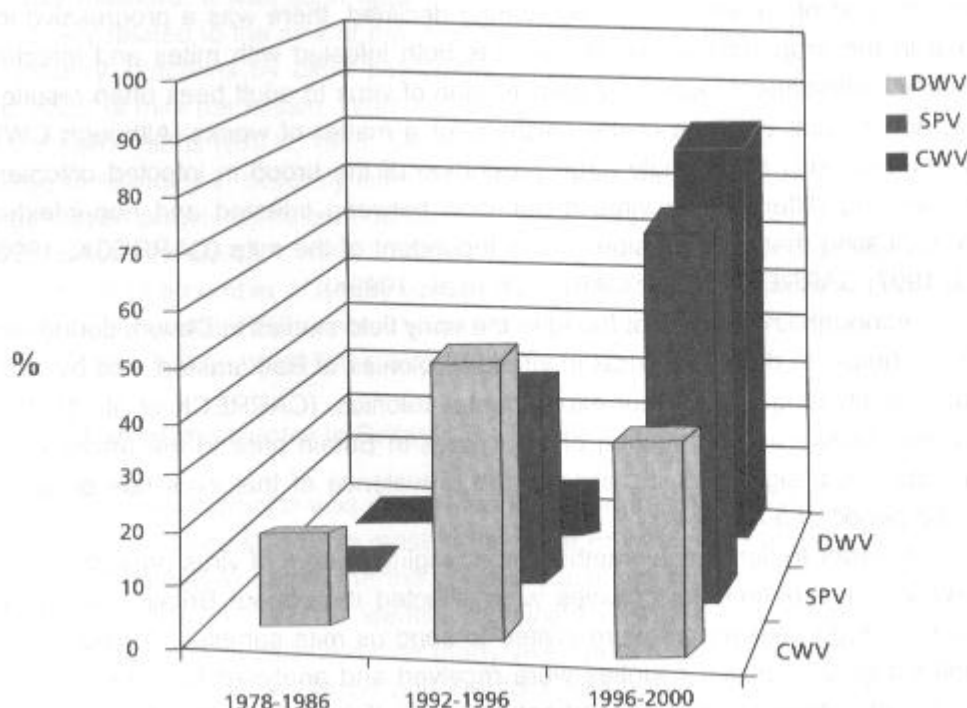


Fig. 1 – Changes in the % of colonies infected with viruses before (1978-1986) and after (1992-1996 and 1996-2000) the arrival of *Varroa destructor* in Britain

As DWV has now become the most widespread infection in association with *V. destructor* in honey-bee colonies in Britain, the epidemiology of the virus has been investigated in more detail. DWV was known previously only as an infection of adult bees, but the mite has been responsible for introducing the virus into a life stage of the bee that it would not normally infect. Unlike APV and SPV, DWV is not rapidly fatal. Bees infected at the pupal stage continue to develop and emerge, although all contain large amounts of virus and their longevity is significantly reduced. Large amounts of virus also accumulate in bees infected after emergence but their lives are not similarly shortened. Such individuals provide a persistent reservoir of infection for mites to acquire and transmit, which explains the eventual predominance of this infections.

CWV remains something of an enigma. The results from both the national mite survey and the Rothamsted colonies (Fig. 1) appear to show an increased prevalence of CWV since the arrival of *V. destructor*. However laboratory infection experiments have given equivocal results and field observations have failed to demonstrate that CWV is transmitted by *V. destructor*. Until the mode of infection, persistence and spread of CWV is better understood any possible association with the mite remains speculative.

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