Journal of Animal Ecology 2004
73, 51–63

The dynamics of virus epidemics in *Varroa*-infested honey bee colonies

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Summary

1. When the parasitic mite *Varroa* jumped species from the eastern to the western honey bee, millions of infested bee colonies died. Recent work has revealed that the probable cause of this collapse is that mites provide a new route of transmission, by acting as a vector, for certain bee viruses.
2. Using a mathematical model parameterized by recently collected data on bee viruses, we investigate the relationship between the mite load in a colony and the possibility of a virus epidemic occurring within a bee colony.
3. The model suggests that the balance of coexistence between mite, virus and bee in the eastern honey bee, has been lost in the western bee host, not simply because of the new transmission route, but also because mite populations in western honey bee colonies has exceeded a critical epidemic threshold. We quantify the critical epidemic mite load for two well-studied bee viruses, acute paralysis virus and deformed wing virus, through the colony's yearly life cycle.
4. As well as providing practical insights into mite control strategies, the model allows us to disentangle the relative importance of different bee and mite behaviours in virus spread. We consider the evolutionary aspects of the new route of virus transmission, looking in particular at how changes to social organization might bring about collective resistance.

Key-words: collective resistance, honey bee, mathematical model, *Varroa*, virus dynamics.

Journal of Animal Ecology (2004) **73**, 51–63

Introduction

The population dynamics of invertebrates can be significantly influenced by pathogens such as viruses, fungi and bacteria (Anderson & May 1981). Recent experiments have revealed a system in which a group of naturally occurring honey bee viruses, that normally occur at low, non-epidemic levels, have become epidemic now that a new route of transmission has appeared (Carrek, Ball & Wilson 2002; Martin 2001a) This new route of transmission arose when a parasitic mite *Varroa jacobsoni* Oud. jumped species from the eastern honey bee *Apis cerana* F., its natural host, to the closely related western honey bee *A. mellifera* L. Various differences between the two honey bee species and possibly the mites – which have been identified as a distinct species *Varroa destructor* in *A. mellifera* (Anderson & Trueman 2000) – has led to much larger mite populations in *A. mellifera* than in *A. cerana*. In turn, these large mite populations have been responsible for vectoring bee viruses between honey bees. The viruses, which previously spread relatively slowly and caused colony mortality extremely rarely, are now thought to be responsible for the world-wide death of millions of mite-infested honey bee colonies.

While the link between mites, viruses and colony collapse has been established, there is not as yet a full understanding of why and when a *Varroa*-infested honey bee colony will collapse. In particular, the important question in determining the mite load that will cause a virus epidemic and colony collapse has remained largely unanswered. In this paper, we address this question by developing a mathematical model of the complex interactions between bees, mites and virus. To this end, we extend previous models of insect host and single pathogen interactions (reviewed by Briggs *et al.* 1995) to include vectored transmission of viruses (May & Anderson 1979). We are thus able to predict, for different seasons, the mite load at which a virus epidemic occurs within a bee colony, and consider the outcome of these epidemics. Such predictions provide practical insight into when various treatments should be applied to reduce mite populations.

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Since much is known about the biology of honey bees, their viruses and *Varroa*, this system provides an ideal test case for many of theoretical ideas about how parasites and viruses effect insect societies and how these societies might mount collective responses to resist disease (Hart, Bot & Brown 2002; Schmid-Hempel 1998). Our model allows us to investigate how different collective responses by the bees, such as hygienic behaviour or increased division of labour, may or may not provide collective resistance to virus infections. In particular, the richness of data collected on the reproduction and movement of *Varroa* through a honey bee colony allows us to quantify how change in bee behaviour might affect virus spread. Our analysis also sheds new light on the question of why mite, virus and *A. cerana* can coexist in the east, while mite-infested colonies of *A. mellifera* collapse in the west.

Biological background

HOST: THE HONEY BEE *APIS MELLIFERA*

In temperate climates during spring and summer, when nectar and pollen are abundant, a honey bee colony consists of a single reproductive queen, 20 000–60 000 adult workers, 10 000–30 000 worker brood (eggs, larvae and pupae), and several hundred drone brood. Brood are reared in individual wax cells, which are open during the egg and larval stage and sealed during the late larval and pupal stages. The cell remains sealed until the fully formed adult releases itself by chewing a hole in the wax capping of the cell. Brood production slows in autumn, after which the queen and 8000–15 000 adult workers over-winter, feeding solely on honey stored during the summer.

VECTOR: THE MITE *VARROA DESTRUCTOR* ANDERSON

An adult female *Varroa* mite lives either attached to an adult honey bee, known as the phoretic phase, or within a sealed brood cell where it reproduces. During the phoretic phase, mites remain attached to adult bees, occasionally moving between adult bees. To reproduce, a female mite enters a brood cell just prior to it being sealed, lays up to six eggs of which only the first develops into a male and the rest develop into females. Mating, typically between brother and sisters, occurs within the sealed cell (Martin 2001b). Adult female mites are then released into the colony, either when the developed bee emerges from the cell or adult bees remove the dead brood (Martin 2000). Most of the female mites move to a new adult worker bee soon after being released from the cell (Kuenen & Calderone 1997). All mite stages feed on bee haemolymph, which is obtained by piercing the bee cuticle of the developing or adult bee using specialized mouth-parts (Bowen-Walker, Martin & Gunn 1997).

VIRAL PATHOGENS

At least 14 small (17–60 nm) RNA viruses are known from the honey bee (Bailey & Ball 1991). Acute paralysis virus (APV) and deformed wing virus (DWV) normally reside at a common low-level inapparent infection in seemingly healthy bees (Bailey & Ball 1991), such that most bees in most colonies appear to carry a small number of 'undetectable' viral particles. Very little is known about the transmission routes of bee viruses at these low inapparent levels. Despite the high prevalence of inapparent APV in honey bee populations, APV has only rarely been reported in *Varroa*-free colonies at levels sufficient to kill a honey bee colony, i.e. with many bees in the colony exhibiting 'detectable' levels of APV (Bailey, Ball & Perry 1981 report on one isolated case). For DWV, there is no published information associating the virus with colony deaths prior to the arrival of *Varroa*, suggesting that, like APV, DWV occurs rarely in an overt form. Occasionally, bee viruses may multiply rapidly within a single bee so that the infection goes from being inapparent to being overt and 'detectable'. Such periodic bursts of virus production are a common strategy among viruses (Crawford 2000) and KBV can be activated by injecting adult bees with potassium buffer or insect ringer (Anderson & Gibbs 1988). Since the overt form of APV normally kills its host within a few days but very rarely kills entire *Varroa*-free colonies, we can assume that, in the absence of mites, transmission of overt virus is very low.

The feeding activities of the *Varroa* mite provides a new route of transmission for some bee viruses, earlier found only in inapparent form. It has been shown that APV (Ball 1989; Batuev 1979) DWV (Ball 1989; Bowen-Walker, Martin & Gunn 1999) slow paralysis virus (SPV) (Ball 1989) and Kashmir bee virus (KBV) (B. Ball, pers. comm) can all be successfully transmitted between honey bees during mite feeding activities. These four viruses are also known to multiply rapidly when artificially injected into bee pupae. Other viruses like cloudy winged virus (CWV) do not multiply when injected into adult bees or pupae (Bailey *et al*. 1981) and it also appears that *Varroa* cannot successfully transmit CWV (Nordstrom *et al.* 1999). Despite extensive investigations there is no good evidence to suggest that the feeding of *Varroa* triggers any inapparent viruses to multiply (Denholm 1999). Rather, in mite-infested colonies it is just a matter of time until a mite encounters a bee exhibiting spontaneous overt infection. A transmission cycle then begins as the mite vectors the overt virus through the colony.

Studies on DWV (Martin 2001a; S. Martin, B. Ball & N. Carreck, unpublished data) have shown that when transmitted via *Varroa* to pupae, pupal mortality increases and its subsequent adult longevity is reduced. However, there is no evidence that adult longevity is affected if bees become infected after the pupal stage. All infected bees are reservoirs of viable virus until they die. This is in contrast to the more virulent viruses such

as KBV, SPV and APV, which normally kill the infected bee in only a few days. KBV, the most virulent of all honey bee viruses, can kill a bee in 3 days (Bailey, Carpenter & Woods 1979).

Model derivation

We consider a honey bee colony consisting of b virus free adult workers, a adult workers which acquired the virus as adults and p adult workers which acquired the virus at the pupal stage. The colony contains a population of m virus carrying mites and u mites which are virus free. Each mite is either in a brood cell, a proportion γ of its time, or phoretic, the other $1 - \gamma$ proportion of its time. The bee colony contains a total of $c_w\mu_w$ worker brood cells and $c_d\mu_d$ drone brood cells within which the $\gamma(m + u)$ 'brood' mites are distributed. c_w and c_d are, respectively, the worker and drone sealed brood development times (in days, which we choose as a convenient time unit) for a single cell and μ_w and μ_d are, respectively, the number of worker and drone brood developing into adult bees per day in a virus-free colony. The virus can be transmitted both through the movement of mites between adult hosts, and through the infection of brood.

TRANSMISSION BETWEEN ADULT BEES

Mites only carry viruses so long as they are attached to an overtly infected bee. When a virus-free phoretic mite moves from an uninfected to an infected adult bee, it will begin carrying the virus (Nordstrom 2000; S.J. Martin, B. Ball & N. Carreck, unpublished data). Mites move between adult bees both spontaneously and just prior to the death of their host bee (Bowen-Walker & Gunn 1998). We assume that a mite moves spontaneously at rate λ per day and that healthy adult bees die at rate d_b per day. Ritter, Kerkhoff & Patzold (1980) showed that the distribution of number of mites per adult bee was consistent with a Poisson distribution, while Bowen-Walker & Gunn (1998) found it differed only at very high mite infestation levels. We can thus assume that each uninfected adult bee carries $(1 - \gamma)u/b$ virus-free mites on average. The rate at which mites acquire the virus through movement from uninfected to infected bees is then

$$(d_b + \lambda)(1 - \gamma)u \frac{a + p}{a + b + p} \qquad \text{eqn 1}$$

mites per day, where the probability a phoretic mite moves to an infected adult bee is $(a + p)/(a + b + p)$.

When virus-carrying mites move from an infected to an uninfected adult bee, they transmit the virus to their new host in a proportion we will denote q of cases (Bowen-Walker *et al*. 1999; Martin 2001a; Nordstrom 2000). In the $1 - q$ cases the mites fail to transmit the virus, the bee remains uninfected and the newly attached mite becomes virus-free. Virus-carrying mites are distributed both on bees which were infected with the virus as adults and those infected as pupae. The distribution of virus-carrying mites over these bees is not likely to be uniform. Indeed, bees which contracted the virus as adults have necessarily had a mite attached as an adult, while the mites initially attached to pupae infected bees will usually have moved to another adult bee (see below for details of how these movements are modelled). It is thus a reasonable approximation to assume that bees infected as adults carry $(1 - \gamma)m/a$ mites and those bees infected as pupae carry no mites. As with healthy bees, mites change host both spontaneously and just prior to the death of their host. The rate at which mites stop carrying the virus through moving host is thus

$$(d_a + \lambda)(1 - \gamma)m \frac{(1 - q)b}{a + b + p} \qquad \text{eqn 2}$$

mites per day, where d_a is the rate at which adult-infected bees die. Similarly, adult bees become infected through mites moving host at a rate

$$(d_a + \lambda)(1 - \gamma)m \frac{qb}{a + b + p} \qquad \text{eqn 3}$$

infections per day. In order to test the effect of the above assumption about the distribution of mites across infected bees we have repeated all of what follows using the alternative rule that virus-carrying mites are distributed uniformly at random across *all* infected bees, and the results obtained differ only slightly, and not qualitatively, from those presented below.

TRANSMISSION IN BROOD CELLS

The number of mites in worker brood cells follows a Poisson distribution (Martin 1995; Salvy *et al.* 1999). Distributing the mites over all available worker brood cells accordingly means that the rate of production of uninfected, b, and pupal-infected adult workers, p, are

$$\mu_w \exp\left(-\frac{\gamma m\rho}{c_w\mu_w}\right) \quad \text{and} \quad p_w\mu_w\left(1 - \exp\left(-\frac{\gamma m\rho}{c_w\mu_w}\right)\right) \qquad \text{eqn 4}$$

respectively, where p_w is the probability that a developing worker bee with a virus survives to adulthood and ρ is the probability that a mite enters a worker rather than a drone cell.

Drone brood cells are more attractive to mites than worker cells, with drone brood cell being 10 times as likely to contain a mite than a worker brood cell (Boot *et al.* 1995; Calis, Boot & Beetsma 1999). The mites in drone cells are also distributed according to a Poisson distribution (Salvy *et al.* 1999). Though drone brood cells play an important role in mite reproduction, we do not need to account for adult drones in our model since 90% of mites attached to newly hatched drone bees move to worker bees within 1 day of hatching and mites

seldom move back to an adult drone bee (Kuenen & Calderone 1997). We do, however, necessarily account for the mite movements from newly hatched drones and adult workers. We let β_w and β_d, respectively, denote the number of offspring per mite in worker and drone brood cells and s_w or s_d the probability a newly emerged mite will move to another adult worker for mite in worker and drone cells, respectively. We thus set

$$\alpha_u = \alpha_m = \alpha = \frac{\rho s_w(\beta_w + 1)}{c_w} + \frac{(1-\rho)s_d(\beta_d + 1)}{c_d} \quad \text{eqn 5}$$

to be the number of movements which occur when a virus-free (α_u) or a virus-carrying mite (α_m) emerges from a cell. Note that we set α_u and α_m equal since even if the bee in the cell occupied by a virus-carrying mite dies, the mite and its offspring emerge from the cell and attach themselves to adult bees. Mites start carrying the virus and adult bees become infected with rate

$$\gamma\alpha_u u\frac{a+p}{a+p+b} \quad \text{and} \quad \gamma\alpha_m m\frac{qb}{a+p+b} \quad \text{eqn 6}$$

respectively. Furthermore, virus-carrying mites leaving a cell and moving to an uninfected adult bee will stop carrying the virus with rate

$$\gamma\alpha_m m\frac{(1-q)b}{a+p+b} \quad \text{eqn 7}$$

mites per day.

COMPLETE MODEL

Our model addresses a specific question: whether, for various mite loads, a small amount of virus introduced into the colony will spread through the bees and how many bees will be infected if this epidemic occurs. To this end, in the analysis which follows, we assume that the total mite population, $u + m = M$, is constant. We will thus ignore the effects of births and deaths of mites. In reality, a mite population will grow exponentially with a daily population growth rate of between 0·021 (Calatayud & Verd 1995) and 0·026 (Martin 2001a) whenever bee brood is present. In a virus-free bee colony, the mite population will increase until the mites are controlled using acaricides. In the absence of chemical control, it is possible for mites to overwhelm a virus-free colony by shear numbers, though this requires mite numbers well above those considered in our virus model. Populations have been reported to reach 40 000 mites in some apparently healthy colonies (Allsopp 2000). The assumption of a fixed mite population is justified since virus-carrying mites are unaffected by the virus and will switch host if the bee they are attached to dies. The mite population is thus, at least up until the possible collapse of the bee colony, largely independent of the level of virus in the colony.

Combining the three ways in which a virus-carrying mite can acquire a virus – through movement (equation 1) and through swapping host after reproduction (equation 6) – with the three ways in which a virus-carrying mite can recover from the virus – again through spontaneous movement, host death and after reproduction (equations 2 and 7) – gives the following rate of change for the mite population with the virus,

$$\frac{dm}{dt} = ((\lambda + d_b)(1-\gamma) + \alpha_u\gamma)(M-m)\frac{a+p}{a+b+p} - ((\lambda + d_a)(1-\gamma) + \alpha_m\gamma)m\frac{(1-q)b}{a+b+p} \quad \text{eqn 8}$$

mites per day. The corresponding rate equations for the contraction of the virus by adult worker bees are, from equations 3, 4 and 6:

$$\frac{db}{dt} = \mu_w \exp\left(-\frac{\gamma m\rho}{c_w\mu_w}\right) - ((d_a + \lambda)(1-\gamma) + \alpha_m\gamma)\, m\frac{qb}{a+b+p} - d_b b \quad \text{eqn 9}$$

$$\frac{da}{dt} = ((d_a + \lambda)(1-\gamma) + \alpha_m\gamma)\, m\frac{qb}{a+b+p} - d_a a \quad \text{eqn 10}$$

$$\frac{dp}{dt} = p_w\mu_w\left(1 - \exp\left(-\frac{\gamma m\rho}{c_w\mu_w}\right)\right) - d_p p \quad \text{eqn 11}$$

where d_b, d_a and d_p are the rates of death of healthy, adult infected and pupal infected bees. Note that possible measures of virus virulence are $1 - d_b/d_a$ and $1 - d_b/d_p$, giving the proportional reduction in life span resulting from infection at the adult and pupal stages, respectively.

The theory of virus spread

We say an *epidemic* occurs or, interchangeably, the virus *spreads* if the injection of a small amount of virus into a single mite results in a persisting positive viral load. Under natural conditions such an injection would correspond to a mite feeding on a bee which exhibited the virus in overt form. An epidemic can have a range of severities, an epidemic may mean the virus persists at a low level with only a small proportion of bees being infected, i.e. $b >> p + a$, or it may mean the virus has spread through the whole colony, i.e. $b = 0$. Mathematically speaking, an epidemic occurs if and only if the virus-free equilibrium is unstable. The virus-free equilibrium for equations 8 to 11 is $\{m = 0, b = \mu_w/d_b, a = 0, p = 0\}$, i.e. when the population is virus-free, though not necessarily free of *Varroa* mites, the population of adult bees reaches an equilibrium, $b = \mu_w/d_b$, where the rate of emergence of new adult bees is balanced by death of adult bees. Determining whether an equilibrium is unstable is a standard mathematical

technique (details of which may be found in, for example, Diekmann & Heesterbeek 2000). We first determine the Jacobian matrix of partial derivatives then evaluate whether any of the eigenvalues of this matrix are greater than zero. If any of these eigenvalues are greater than zero then the virus-free equilibrium is unstable and an epidemic occurs.

In our model, the disease free equilibrium is unstable when the number of mites in the colony, $M >$

$$M_{\text{crit}} = \frac{c_w(1-q)d_a d_p(\alpha\gamma+(\lambda+d_a)(1-\gamma))}{d_b(qc_w d_p(\alpha\gamma+(\lambda+d_a)(1-\gamma))+\gamma p_w \rho d_a)(\alpha\gamma+(\lambda+d_b)(1-\gamma))}\mu_w \quad \text{eqn 12}$$

M_{crit} is thus the *critical mite load* at which an epidemic is possible. The complexity of equation 12 reflects the variety and complex interaction of the factors which determine how the virus can spread. However, we can see immediately that M_{crit} is given in terms of a fraction multiplied by μ_w, where μ_w is the rate of production of worker bees. This fraction is composed of rates (d_p, d_b, d_a and λ), proportions (q, ρ, p_w and γ) and constants (α and c_w). It is the relative magnitudes of these which determine how much larger or smaller the vector population size, M, must be relative to the healthy host population size, μ_w/d_b, for the virus to spread.

Determining whether a specific virus will spread involves evaluating equation 12 for particular biological scenarios. As a simple example, consider a vector which does not feed on early life stages of its host (i.e. $\gamma = 0$). This case reduces to an oversimplified model of malaria, similar to one discussed by May & Anderson (1979) and gives a critical vector load of

$$M_{\text{crit}} = \frac{d_a(1-q)}{d_b(d_b+\lambda)q}\mu_w \quad \text{eqn 13}$$

vectors. In this case the number of vectors required to spread the decreases as the probability of transmission, q, increases but increases as the virulence of the disease, measured by $1 - d_b/d_a$, increases.

Bee viruses through the seasons

We now consider models of the deformed wing and acute paralysis viruses, the two best studied bee viruses, at various stages of the honey bee colony's yearly life-cycle. Each virus and stage in the colony cycle gives different parameters for equation 12. Table 1 gives 'standard' parameter values, estimated from the literature, that, unless stated otherwise, we will use in parameterizing our model. Since the link between bee viruses has only recently been established, some of the parameter estimates relating to virus spread, in particular for q, p_w and d_a, must be considered as provisional. In order that the conditions for an epidemic can be updated as improved parameter estimates become available, we

Table 1. Standard parameter values

	Units		Summer	Autumn	Winter	Spring	Reference
Bees							
Number of workers produced	per day	μ_w	1500	500	0	500	1
Number of drones produced	per day	μ_d	50	5	0	5	2
Worker sealed brood development time	days	c_w	12	12	N/A	12	1
Drone sealed brood development time	days	c_d	14	N/A	N/A	14	1
Healthy adult bee death	per day	d_b	1/25	1/44	1/190	1/44	3, 4
Mites							
Proportion of time in brood		γ	0·75	0·5	N/A	0·5	5
Movement between hosts	per day	λ	0·2	0·2	0·037	0·2	6
Probability of infection		q	0·8	0·8	0·8	0·8	7
Probability enter worker cell		ρ	0·75	0·90	N/A	0·90	8
Offspring in worker cell	mites	β_w	1·0	1·0	N/A	1·0	9
Offspring in drone cell	mites	β_d	2·25	N/A	N/A	2·25	9
Probability move host on leaving worker cell		s_w	0·5	0·5	N/A	0·5	10
Probability move host on leaving drone cell		s_d	0·9	0·9	N/A	0·9	From 10
APV							
Infected as adult bee death	per day	d_a	0·2	0·2	0·2	0·2	11
Probability infected worker pupa emerges		p_w	0	0	N/A	0	7, 12
Infected as pupae bee death	per day	d_p	N/A	N/A	N/A	N/A	7, 12
DWV							
Infected as adult bee death	per day	d_a	0·0400	0·0227	0·0053	0·0227	7
Probability infected worker pupa emerges		p_w	0·8	0·8	N/A	0·8	7, 13
Infected as pupae bee death	per day	d_p	0·2	0·2	N/A	0·2	7, 13

References for these values are (1) Snodgrass 1925; (2) Seeley 2002; (3) Fukuda & Sekiguchi 1966; (4) Free & Spencer-Booth 1959; (5) Martin 1998a; (6) Bowen-Walker & Gunn, 1998; (7) Martin 2001a; (8) Boot *et al*. 1995; (9) Martin 2001b; (10) Kuenen & Calderone 1997; (11) Bailey 1965; (12) Bailey & Gibbs 1964; (13) Nordstrom 2000.
'N/A' signifies that the parameter value is 'not applicable.

give full mathematical expressions for M_{crit} for each season and virus throughout.

ACUTE PARALYSIS VIRUS IN SUMMER AND AUTUMN

A biological observation immediately simplifies the expression for M_{crit} for APV. If a mite infects a bee pupa with APV in a brood cell then the pupa will die before reaching adulthood and thus in the model $p_w = 0$. The virus-carrying mites in the brood cell will attach themselves to adult bees after they emerge from the brood cell, thus vectoring the virus further. Substituting $p_w = 0$ into equation 12 gives:

$$M_{APV} = \frac{d_a(1-q)}{d_b q(1-\gamma)(\lambda + d_b) + \alpha\gamma}\mu_w \qquad \text{eqn 14}$$

is the critical number of mites for APV to spread. Since bees infected at a pupal stage do not emerge, they do not spread APV and the terms in the denominator of equation 14 all relate to movement of mites between hosts, rather than to spread by bees infected as pupae.

Equation 14 shows that the number of mites required for virus spread is higher for more virulent viruses, i.e. M_{APV} increases as d_a increases but decreases as d_b increases. APV is virulent: as well as killing pupae, APV kills bees infected as adults after 5 days (i.e. $d_a = 1/5$) while a healthy bee will live, on average, for 25 days during the summer and 44 days during the autumn. The virulence of APV thus means that a large mite population is required for an epidemic. Table 2 shows the critical mite load at which an APV epidemic can start, for the standard parameter values. For a summer colony with 37 500 bees, 12 289 mites are required to start an epidemic, while an autumn population of 22 000 bees requires 6830 mites for an epidemic. Such large mite populations are only usually seen in areas where mites have recently become established and treatment using acaricides has not yet begun. In one study by Ritter, Leclercq & Koch (1984) where colonies with a mite population of around 20 000 were studied, high levels of APV were found (Ball & Allen 1988).

Although high virulence means large critical mite load, the virulence of APV does have a downside as far as colony survival is concerned. Figure 1 shows the equilibrium bee population, for both summer and autumn, as the mite population is increased, obtained by numerical solution of equations 8–11, starting with one infected adult carrying a single mite. Once M_{APV} is exceeded, the colony size is drastically reduced. Above the critical mite load, a bee colony in autumn will invariably collapse from an APV epidemic, while in summer the population of healthy bees will be drastically reduced. This prediction is supported by the observations of Ritter *et al.* (1984): colonies with 20 000 mites and high levels of APV all died within 4 months.

DEFORMED WING VIRUS IN SUMMER AND AUTUMN

DWV is less virulent in its effects on individual bees than APV. It is not thought to effect the life span of bees infected as adults ($d_a = d_b$) and infected pupae have a large probability of emerging ($p_w = p_d = 0{\cdot}8$) but with a life span reduced by around 70% (Martin 2001a). The fact that $d_a = d_b$ introduces a symmetry into the model so that equation 12 is simplified for DWV and

$$M_{DWV} = \frac{c_w(1-q)d_p}{qc_w d_p(\alpha\gamma + (\lambda + d_b)(1-\gamma)) + \gamma p_w \rho d_b}\mu_w \qquad \text{eqn 15}$$

Substituting our standard parameter values into this function gives $M_{DWV} = 2315$ mites in a summer colony of 37 500 bees and $M_{DWV} = 737$ mites in an autumn colony of 20 000 bees.

The lower virulence of DWV means that a much smaller mite population is required for an epidemic of DWV to occur than for APV. However, the lower virulence also means that colonies that acquire the virus do not necessarily collapse. Figure 2 shows the equilibrium bee population as the mite population is increased, obtained by numerical solution of equations 8–11, starting with one infected adult carrying a single mite. Although there is an epidemic of DWV above the critical mite load, the bee population is not greatly affected; for example, in the autumn for the total healthy adult bee population to remain greater than

Table 2. Number of mites required for an epidemic and for colony collapse for the two viruses. An epidemic occurs when the number of mites exceeds M_{crit}. Values in parentheses for summer and autumn show the number of mites per bee at the critical load. Note that since bees die faster than mites, the viral load will always increase during winter though very slowly, thus $M_{crit} = 0$. The mite load for colony collapse is defined dependent on the season, and in each case is an estimate of the maximum number of mites which a colony with the virus can sustain. See text for details. Parameter values for these estimates are given in Table 1

Virus	State of colony	Summer	Autumn	Winter	Spring
Acute paralysis	Epidemic	12289 (32·7%)	6830 (31·1%)	0	6830
	Collapse	13000 (34·7%)	6830 (31·1%)	9000	7000
Deformed wing	Epidemic	2315 (6·2%)	737 (3·4%)	0	737
	Collapse	12500 (33·3%)	4000 (18·1%)	N/A	3000

N/A, not applicable.

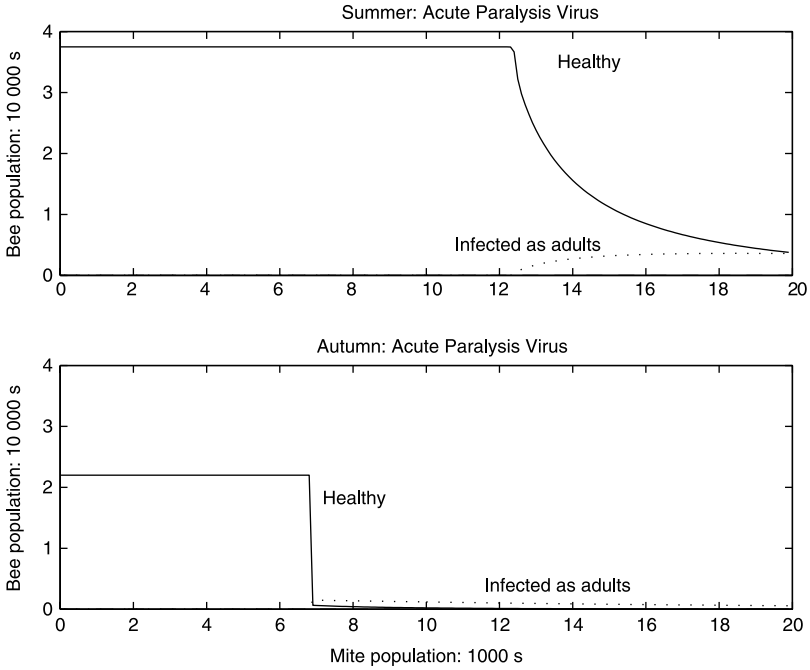

Fig. 1. The spread of the acute paralysis virus in a honey bee colony. Predicted number of healthy and infected bees in a colony during summer (top panel) and autumn (bottom panel) as a function of the number of mites in the colony. Levels are obtained from the equilibrium numerical solution of equations 8–11 for the parameter values given in Table 1.

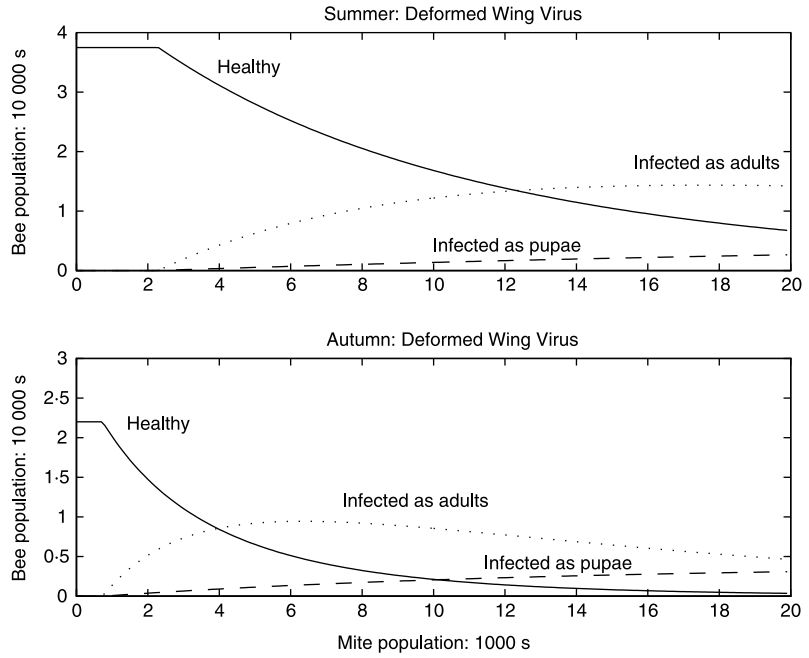

Fig. 2. The spread of the deformed wing virus in a honey bee colony. Predicted number of healthy and infected bees in a colony during summer (top panel) and autumn (bottom panel) as a function of the number of mites in the colony. Levels are obtained from the equilibrium numerical solution of equations 8–11 for the parameter values given in Table 1.

10 000 the mite population must be less than 3362. At around 4000 mites the number of bees carrying DWV becomes greater than the number of healthy bees. Such high levels of DWV will begin to reduce colony efficiency as at least 10% of the worker brood will die from the virus. In nature, colonies experiencing such reductions in efficiency are prone to collapse.

VIRUSES DURING WINTER

During the winter, honey bees produce little or no brood ($\gamma = \mu_w = \mu_d = 0$ and $p = 0$) and the rate at which mites spontaneously move between bees drops significantly ($\lambda = 0{\cdot}037$) (Bowen-Walker & Gunn 1998). The rate of change of the mite and bee populations are then

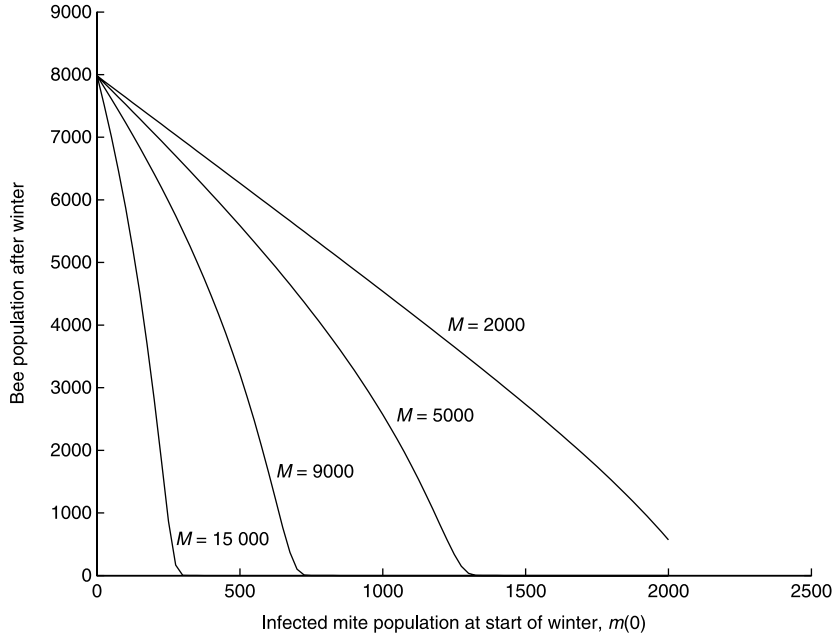

Fig. 3. Predicted colony size at the end of the winter given a start of winter population of 15 000 bees, as a function of the number of mites in the colony with APV at the start of the winter, $m(0)$. Levels are obtained from the numerical solution of equations 16–18 over 120 days.

$$\frac{dm}{dt} = (d_b + \lambda)(M - m)\frac{a}{a+b} - d_a m\frac{(1-q)b}{a+b} \quad \text{eqn 16}$$

$$\frac{da}{dt} = d_a m\frac{qb}{a+b} - d_a a \quad \text{eqn 17}$$

$$\frac{db}{dt} = -d_a m\frac{qb}{a+b} - d_b b \quad \text{eqn 18}$$

For DWV $d_b = d_a = 1/190$ so since $\lambda > 0$ the proportion of mites and bees with virus in the colony will increase over winter, though more slowly than during the other seasons.

For APV, the death of bees with the virus is much quicker than for DWV, so the possibility exists for the virus to spread quickly through the colony. Figure 3 shows how the number of bees in a colony after winter, from a before winter population of 15 000 bees, depends on the number of mites in the colony carrying APV at the start of the winter, $m(0)$ and the total number of mites M. The larger the number of mites carrying the virus at the start of the winter, the smaller the population of bees at the end of the winter. If there is only a low level of APV in the colony, a very large number of mites are required for there to be any significant effect on the after-winter colony size. It is known that a colony must have at least 3000 bees after the winter to rear brood (Fukuda & Sakagami 1968). Thus, given an initial population of 500 virus-carrying mites, it would take a total population of at least 9000 mites to reduce the colony to a below viable size.

VIRUSES IN SPRING

In spring, the colony grows from its after-winter level of 5000–10 000 bees up to the summer population of 20 000–45 000 individuals (Winston 1987). Although brood production and bee life span are the same as in autumn ($\mu_w = 500$ and $d_b = 1/44$), in spring, viruses may pose a more serious threat to a colony of reduced size that is attempting to grow. Furthermore, any virus load in the colony before winter is likely to have increased over winter. Figure 4 shows the time course of the growth of a colony of 5000 bees for different mite loads over 3 months for both viruses. In all these simulations $m(0) = 200$ mites initially carried the virus. For both viruses, as the number of mites increases, the resultant bee population decreases. Figure 5 shows how number of mites in the colony determines start of summer colony size. If we set a target of 15 000 bees for a viable start of summer colony we find that there must be less than 7000 mites in a colony with APV and 3000 for a colony with DWV in order for the colony to arrive at that target. Care must be taken in interpreting these results since, unlike summer and autumn, the initial number of virus-carrying mites plays an important role in determining the ultimate level of infection. Given estimates of these two parameters, initial level of infection and the number of mites in a colony, it is possible to use our model to estimate colony growth from the start of spring to early summer.

Discussion

TREATMENT STRATEGY

Studies both in the UK (Martin 1999) and USA (Delaplane & Hood 1997) recommended treatment of honey bee hives with acaricide for *Varroa* at a thresholds of between 2000 and 3500 mites. These strategies are based on preventing the mite population in a colony

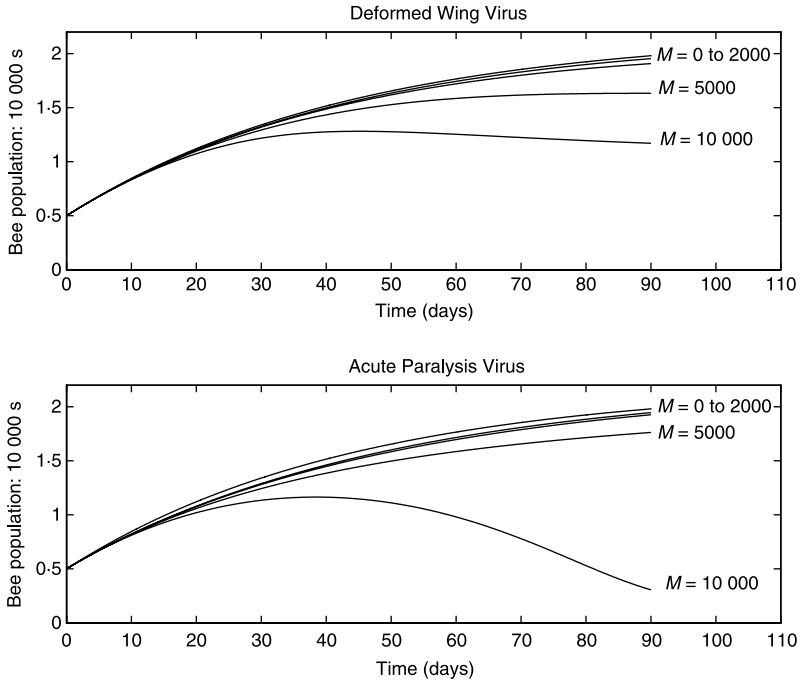

Fig. 4. Change in colony size over time from the start of spring to the start of summer from an initial population of 5000 bees, for APV (top panel) and DWV (bottom panel). Initially $m(0) = 200$ mites carry the virus. Levels are obtained from the numerical solution of equations 8–11 over 90 days. Parameter values are given in Table 1.

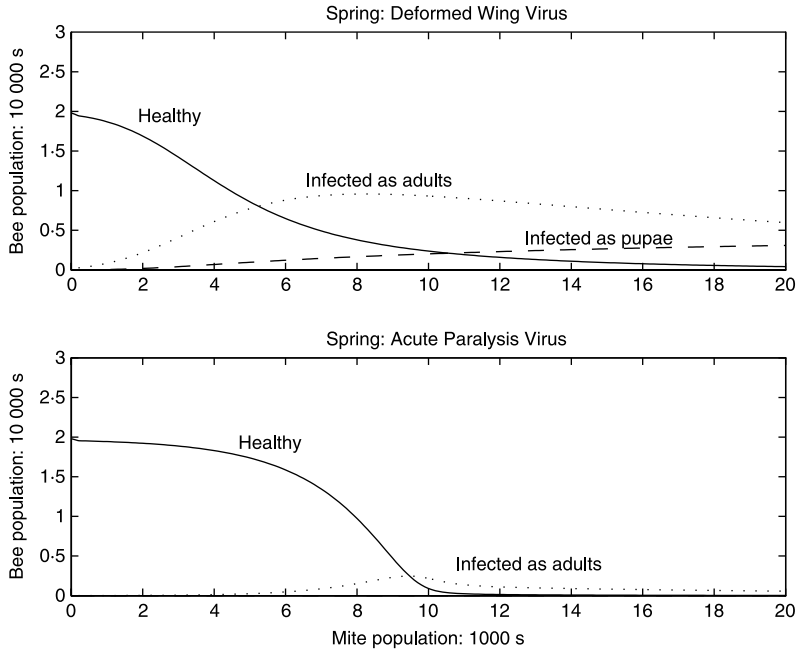

Fig. 5. Predicted number of healthy and infected bees at the start of summer given a start of spring population of 5000 bees, for APV (top panel) and DWV (bottom panel) as a function of the number of mites. Initially, $m(0) = 200$ mites carry the virus. Levels are obtained from the numerical solution of equations 8–11 at the end of 90 days. Parameter values are given in Table 1.

exceeding 2500 during the year (Martin 1998b). Our study shows that the current policy will help control viral diseases in bee colonies (see Table 2 for a summary of the results). Autumn is the time at which the colony is at greatest risk to viruses and maintaining a mite population below 2500 will prevent APV epidemics and confine DWV to low levels. To prevent DWV epidemics would require treatment at smaller mite populations, i.e. reducing the mite population to below 700. If prevention of DWV is an aim then this may be more easily achieved at the start of the summer when the threshold mite load required to eliminate DWV is larger. It is, however, difficult to eliminate completely any bee virus from a colony, since the reduction of the mite population does not affect the normal cycle of bee to bee transmissions that allows the virus to persist at

inapparent levels within the colony. Sporadic appearance of overtly infected bees will always occur and if sufficient mites are present the viral-mite transmission route will become re-established.

In general, APV is only a threat to a colony of bees treated to reduce mite populations immediately before winter, when the introduction of virus-carrying mites will reduce a colony's size over the winter (see Fig. 3) and result in a failure to grow in spring (see Fig. 4). Similar predictions as those for APV may also hold for other virulent viruses such as slow paralysis virus (SPV) and Kashmir bee virus (KBV). For example, SPV appears seasonally, leading to accelerated death of the colony (Ball 1997).

EVOLUTION OF COLLECTIVE RESISTANCE

In the absence of acaricide treatment, the European honey bees' only defence against virus epidemics would be the evolution, either by natural selection or artificial breeding, of some form of resistance. Such resistance may take the form of individual immunity, where bees evolve an individual immune response that lowers virus infection. For social insects, resistance may also take the form of a *collective response*, where the behaviour of bees or the social structure of the colony evolves to prevent virus epidemics. Examples of behaviour that may have evolved as part of a collective response to disease include: uncapping and removal of diseased (Arathi, Burns & Spivak 2000; Rothenbuhler 1964) and parasitized (Spivak & Reuter 2001a) brood; *Nosema*-infected honey bees defecating outside of the hive (Schmid-Hempel 1998); alarm responses by termites in contact with spores of pathogenic fungi (Rosengaus *et al.* 2001); and an increase in the number of leaf-cutter ants working on the garbage midden in colonies with parasitized fungus gardens (Hart *et al*. 2002). Apart from the notable exception of honey bee resistance to American foul-brood through hygienic behaviour (Spivak & Reuter 2001b), it has been difficult to quantify the advantages, in terms of colony fitness, of collective responses to disease and parasitism. We now use our model to give quantitative predictions as to the effect of certain collective responses on critical mite load and virus epidemics.

Since a major factor in the large critical number of mites required for an epidemic of APV was the fact that the virus was virulent enough to kill pupae with the virus, one possible control strategy for DWV might be to breed bees that destroy all brood in cells containing virus-carrying mites. Extending on work by Rothenbuhler (Spivak & Gilliam 1998), Spivak and co-workers have investigated the mechanisms whereby honey bees bred for hygienic behaviour (the removal of dead and/or unhealthy bees and brood) are effective against infections of American foul-brood and infestation by *Varroa* mites (Arathi *et al*. 2000; Arathi & Spivak 2001; Spivak & Reuter 2001a,b) We can test the effect of removing infected brood by setting $p_w = \alpha_m = 0$ in equation 15, giving a critical mite load, M_{crit}, of

$$\frac{(1-q)}{q(\alpha_u\gamma + (d_b+\lambda)(1-\gamma))}\,\mu_w \qquad \text{eqn 19}$$

which gives M_{crit} equal to 2458 and 776 for summer and autumn, respectively. M_{crit} is thus altered very little from the values, given in Table 2 for DWV under 'standard' conditions. The removal of infected cells before adult bees emerge makes little difference to the critical mite load.

The conclusion that a breed of hygienic bees, which destroy virus-carrying brood, will have no effect on the spread of the virus for a given mite load is counter-intuitive. In interpreting this result, it should be remembered that we are looking at the spread of viruses as a function of mite population. A breed of bees that keep mite populations below the critical mite load will prevent DWV epidemics simply because there will be insufficient mites to spread the virus. Indeed, current breeding programmes for *Varroa*-resistant bees concentrate on selecting bees that destroy all mite-occupied cells, rather than just virus-infected cells. Our model predicts that a breed of hygienic bees which, although reducing the number of mites, cannot keep the mite numbers below the critical load, will be no more resistant to DWV epidemics than standard 'unhygienic' colonies. This prediction needs to be tested empirically, but is consistent with current experimental observations: Spivak & Reuter (2001b) showed that colonies of hygienic bees with initial mite infestations of less than 15% mites per bee maintained low levels of mite infestation, had less brood disease, and were thus less likely to collapse than 'unhygienic' control colonies. This result contrasted strongly with hygienic colonies with mite infestations of greater than 15% mites per bee, where the majority of the colonies required treatment to prevent collapse.

Other biologically plausible collective responses also have little or no effect on disease spread. For example, if infected bees ensure that they die outside the nest, thus preventing virus-carrying mites from moving to other uninfected bees (i.e. in the model we omit the term $d_a m$ from equations 9 and 10) then M_{crit} for DWV is increased only slightly to 2467 and 790 in the summer and autumn, respectively. Another possible response is for bees to remove drone cells, which are more attractive to mites (i.e. by setting $\alpha_m = \alpha_u = \alpha = \rho s_w (\beta_w + 1)/c_w$ in equation 5). Drone removal would prove somewhat effective during the summer, with the critical mite load for DWV rising to 3169 mites, but ineffective in autumn, with a critical mite load of 788 mites.

The most important factors in determining critical mite load differ for summer and autumn. Removing the effect of spontaneous movement of mites between hosts, by setting $\lambda = 0$ in equation 15 gives a critical mite load

$$\frac{c_w(1-q)d_p}{qc_w d_p(\alpha\gamma + d_b(1-\gamma)) + \gamma p_w \rho d_b}\,\mu_w$$

and M_{crit} equal to 3349 and 1796 for summer and autumn, respectively. The absence of spontaneous movement would thus double the critical mite load during the autumn. During summer it is the movement of mites emerging from brood cells which has the greatest effect on M_{crit}. Setting $\alpha = 0$ (and leaving $\lambda = 0{\cdot}2$ unchanged from the 'standard' parameter values) in equation 15 gives a critical mite load of

$$\frac{c_w(1-q)d_p}{qc_w d_p(\lambda + d_b)(1-\gamma) + \gamma p_w \rho d_b}\,\mu_w$$

and M_{crit} equal to 5405 and 1042 for summer and autumn, respectively. Furthermore, if we set $\alpha_u = 0$, while leaving α_m unchanged, M_{crit} becomes even larger, 5888 and 1066 for summer and autumn, respectively.

It is thus the early switch of host by *virus-free* mites, where they attach themselves to possibly infected bees soon after emerging, that produces virus epidemics during summer. A possible collective response, which could reduce transmission at this time, would be a tendency of virus-infected bees to switch task to foraging or any activity that reduces their contact with brood cells. Such 'disease avoidance' has been observed in leaf-cutting ants, where contact between disease-prone garbage heap workers and other workers is minimized through strict division of labour and spatial segregation (Hart & Ratnieks 2001). However, honey bee communication and organization relies on repeated direct contacts between individuals, and it is difficult to envisage how the required spatial segregation could be achieved through natural selection.

EVOLUTION OF VIRULENCE

In insect societies, virulence can be viewed both at the level of the individual and at the level of the colony. When a virus reaches epidemic levels within a colony and ultimately leads to colony collapse, we can think of the virus as being virulent at a colony level. In this sense, DWV and APV have, without any known change in genotype, 'evolved' phenotypically from being relatively non-virulent to become virulent viruses that, without treatment, threaten the existence of an entire species. Before the arrival of *Varroa* in western honey bee colonies, bee RNA viruses had evolved a relatively benign relationship with western honey bees, and likewise *Varroa* had evolved a relatively benign relationship with the eastern honey bee. While possibly reducing the efficiency of a few individual workers or drones, neither viruses nor mites had any great effect on the fitness of bee colonies. It is worth noting therefore that there are two factors which have been essential in causing DWV, in particular, to become virulent at the colony level in *A. mellifera*. The first is the change in the method of transmission provided by the mites. The second less obvious factor, which our study has highlighted, is that critically large populations of mites observed in western honey bee colonies may be required for virus spread.

The critically large mite load required for spread of DWV is not usually observed in eastern honey bee colonies, where mite populations in *A. cerana* are usually well below 800 and mite reproduction is restricted to drone brood (Rath & Drescher 1990) Although *Varroa* mites are present in most *A. cerana* colonies and DWV has been detected in *A. cerana* (Bailey & Ball 1991), colony collapse due to *Varroa* has never been reported. If viral transmission via *Varroa* occurs in *A. cerana*, which appears highly likely, then a very fine balance between pathogen, vector and host has evolved in the eastern honey bee. Mite populations are self-limiting – due to the removal of infected cells by *A. cerana* workers, the fact that mites may only reproduce in drone cells, and that more than two mites in a drone cell usually kill the host (Oldroyd 1999; Rath 1999; Sumpter & Broomhead 2001) – and the level at which the mite population is limited is just below that which would allow a DWV virus epidemic to occur. Indeed, bearing in mind that the vector *Varroa* is itself a parasite of *A. cerana*, an increase in virus virulence with mite population is a plausible evolutionary explanation of the self-limiting mite populations (Ewald 1983, 1995). Whether such a speculative explanation for the previous balance in the east between mite and bee is justified or not, it is clear that this balance has been lost in the west and, without intervention, bee, mite and virus populations would follow a path towards epidemic and possible extinction.

Acknowledgements

We thank Brenda Ball for extensive comments on this document, and Madeleine Beekman, Ben Oldroyd and two anonymous referees for their comments and criticism. This work was possible through the kind hospitality to the authors of the Newton Institute in December 2001 at the 'Individual to Collective Behaviour in Biological Systems' programme. D.J.T.S. was funded by Engineering and Physical Sciences Research Council (EPSRC) and Stiftelsen für internationalisering av hügre utbildning och forskning (STINT).

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Received 12 February 2003; accepted 27 June 2003