

A mathematical model for population dynamics in honeybee colonies infested with *Varroa destructor* and the Acute Bee Paralysis Virus

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Abstract

We present a simple SIR-Ross-MacDonald like model of the infestation of a honeybee (*Apis mellifera*) colony by the Acute Bee Paralysis Virus (ABPV), which is transmitted by parasitic varroa mites (*Varroa destructor*) as vector. This is a four dimensional system of nonlinear ordinary differential equations for the dependent variables healthy and virus infected bees, number of mites in the colony overall and number of mites that carry the virus. In the autonomous case we study the model with analytical techniques deriving conditions under which the bee colony can fight off a ABPV epidemic. These results are then used to design and discuss numerical simulations of the more realistic case with periodic coefficient functions that mimic the seasonal changes in bee colonies.

keywords: honeybees, varroa destructor, acute bee paralysis virus, mathematical model

MSC: 92D25, 92D30

1 Introduction

Western honeybees (*Apis mellifera*) are very important for sustaining life on Earth by contribution to pollination of crops and other plants. The estimated value of honeybees for crop pollination is over \$2 billion annually[8]. Honeybees have been estimated to account for at least 80% of all pollinators [7]. In addition to pollination, bees play an important, age-old role as producer of honey and wax, which in turn find various nutritional and industrial uses.

According to [32], honeybees are, in economical terms, the third most important domestic animal after cattle and pigs, and before poultry.

A honeybee colony usually consists of a single reproductive queen and, depending on the season, up to around 60 000 adult female worker bees, 10 000-30 000 individuals at the brood stage (egg, larvae and pupae) and up to hundreds of male drones [20]. The only fertile individual of the colony is the queen with an average life span of 2-3 years [30]. She lays fertilized eggs that produce worker bees, or much more rarely queens, while drones develop from non-fertilized eggs. A large population of workers is needed to carry out the tasks of the bee colony, including foraging, pollination, honey production and in particular to care for the brood and rear the next generation of bees. In northern temperate locales the queen bee usually begins laying eggs in February at a rate that increases until about mid-summer. From August until mid-October the rate of egg laying declines. It comes to a halt in the mid of October [24]. Therefore, during winter as bees die, honeybee colonies decline in size [3]. The life span of an adult worker bee depends on the season. The average life span of worker honeybees in June is 28.3 days and in July 32.4 days. In winter, honeybees live for almost 154.1 days and the longevity of post wintering bees is 23.4 days [13]. The adult drone life span is around 59 days under optimal colony conditions [13].

A rapid decrease in the number of honeybee colonies has been observed since 2006 in North America [16, 23]. The syndrome, which is characterized by the disappearance of adult bees while the capped brood and honey remains in the hive, is known as Colony Collapse Disorder (CCD). It was first diagnosed in USA. There is an insufficient workforce present in the collapsing colony to care for the brood present. This workforce consists primarily of young adult bees. The bees that are present in the colony are reluctant to consume the stored honey and pollen. The queen may be present in a collapsing colony. Throughout U.S, CCD is spreading rapidly. In other parts of the world, the symptoms are not exactly the same as in CCD but huge losses, in particular wintering losses in Canada have been reported [16]. The exact reasons and the triggering factors for CCD have not been understood yet. Several possible stressors causing the decline of bee colonies have been proposed, including pesticides, intensive agriculture, harsh winter conditions, and the parasitic mites *Varroa destructor*, which are also vectors of viral diseases.

In Canada, varroa mites have been found to be the main reason behind wintering losses of bee colonies [14]. In the years subsequent to the introduction of *Varroa destructor* into Canada, normal long-term overwintering mortality is regarded as being 15%. In 2008-2009, the mortality from wintering losses and spring dwindling was 33.9%, or 2.3 times the normal rate [9]. This loss is similar to the 2007-08 winter mortality figure of 35.0 % and exceeds the 2006-07 rate of 29.0% . In recent years, much scientific research in this area has focused on infestation of honey bee colonies by the mite *V. destructor*. These parasites not only ectoparasitically feed on bees, but also vertically transmit a number of deadly viruses to the bees [16]. Many beekeepers have reported that honeybee colonies die if the mite population is not controlled, see also [29]. Thus, varroa mites have a marked economic impact on the beekeeping industry.

Varroa destructor's natural host is the Asiatic honey bee *Apis cerana*. In the late 1950s and 1960s it shifted host to the Western honey bee *A. mellifera* [2]. Subsequently *V. destructor* has spread quickly all over the Western world. Mite reproduction can occur only if honeybee brood is available. The female mite reproduces within the honey bee sealed brood cell. It enters into the cell just prior to it being capped. After the capping of the cell, the mite feeds on the developing bee. It lays a single male egg and several female eggs at 30-hour intervals [18]. The mother mite prepares a site on the host bee so that the offspring can feed [11], mature and mate within the cell. When the host bee leaves the cell, the mature female mites leave the cell with male and immature female mites, if present. Immature female mites die as they come out of the cell; they cannot survive outside the sealed cell. The adult female mite becomes attached to the adult bee. This is known as phoretic phase. It feeds on the bee's haemolymph by piercing the inter segmental membrane of the bees [1, 5], harming the host. Thus, mites affect the life span of honeybees directly.

Indirectly, they affect honeybees as vectors of viral diseases. There have been at least 14 viruses found in honeybee colonies [4, 16], which can differ in intensity of impact, virulence, etc. for their host. For example, the Acute Bee Paralysis Virus (ABPV) affects the larvae and pupae which fail to metamorphose to adult stage, while in contrast the Deformed Wing Virus (DWV) affects larvae and pupae, which can survive to the adult stage [31]. As the mites are the main cause for vectoring viruses between bees, these viruses are transmitted to the bees when mites feed on bees. When a virus carrying mite attach to a healthy bee during its phoretic phase, it can transmit the virus to the bee [5, 20, 26]. Thus, the previously uninfected bee becomes infected. A virus free phoretic mite can begin carrying a virus when it moves from an uninfected to an infected bee [26, 21].

In this study we focus on one honeybee virus, the Acute Bee Paralysis Virus. It belongs to the family Dicistroviridae, like the Kashmir Bee Virus, Black Queen Cell Virus or the Israeli Acute Paralysis Virus, to name but a few members of this group. These viruses share a number of biological characteristics, such as principal transmission routes, and primary host life stages [25]. ABPV is a common infective agent of honeybees that is frequently detected in apparently healthy colonies. Bees affected by this virus are unable to fly, lose the hair from their bodies and tremble uncontrollably. The virus has been suggested to be a primary cause of bee mortality. Infected pupae and adults suffer rapid death. ABPV is associated with Varroa mites and has been implicated in colony collapse disorder; it is highly relevant for the beekeeping industry. Because mites and virus appear simultaneously under field conditions it is difficult to separate the effects of both pathogens [16]. Therefore, they should be studied together and mathematical models of the disease dynamics should include both pathogens simultaneously.

The course of many infectious diseases can be predicted using mathematical models, which have been developed over many years and experienced a huge surge in activity in the last decade [6, 10, 17]. Although most of these research efforts are driven by diseases of human populations, the underlying concepts can

be adapted to diseases of animals as well. However, not many predictive models for honeybee and varroa mite population dynamics have been published in the literature. The most relevant for our study are [19, 20, 31]. In particular the latter presents a first model of the honeybee-mite-virus system using a traditional SIR-like modeling approach. In this model the number of mites infesting the colony overall is a given parameter but the number of mites carrying the virus is a dependent variable. The authors consider the constant coefficient case and give a stability analysis of the infestation equilibrium, using the number of mites in the colony as bifurcation parameter. The main result is an explicit formula for the dependence of the critical mite load on model parameters, for which the colony is able to survive the disease. That sheds light on the interplay between bee biology and infection dynamics, and on the effect on the fate of the colony if this balance tips. In [12] that model was modified for ABPV by including brood maintenance terms that reflect that a certain number of worker bees is always required to care for the brood in order for new bees to be born. Because the model of [31] assumed a constant rate of birth of bees, it does not permit a trivial equilibrium and allows one only to study whether or not a virus epidemic can be fought off. The extended model of [12], on the other hand permits a trivial (collapse) equilibrium, which is shown to be locally unconditionally, but not globally, stable. Thus our extended model also enables to study under which circumstances the colony will vanish. This happens once the healthy population size drops below a certain threshold (which depends on the brood maintenance terms), which explains e.g. wintering losses. On the other hand, the stability of the infestation equilibrium is qualitatively the same as in [31], but including brood maintenance terms shows that the maximum mite load for which the disease can be fought off, as computed by [31] is an overestimation.

Both, [31] and [12] assumed the mite load to be a given model parameter. In the current study we give up this restriction and couple the disease model with a simple logistic population growth model for the varroa mites. This not only requires us to add another equation to the system but also to modify the equations describing the growth of the bee population, by adding mite induced death terms.

The model that we obtained after this modification is a system of four nonlinear ordinary differential equations. Because essential features of bee population dynamics, such as birth rates and death rates vary drastically with the seasons, this is a non autonomous system, which is difficult to analyze. We first use well established methods for autonomous systems to study the special case of constant coefficients and then investigate in computer simulations whether or not these findings carry over to the more general transient case. In particular we are interested in the question, and if so, under which conditions a proper working, stable bee colony that becomes infested with varroa mites can fight off an epidemic of the Acute Bee Paralysis Virus.

2 Governing equations

We formulate a mathematical model for the honeybee-varroa-ABPV complex in terms of the dependent variables

x : number of honeybees that are virus free,

y : number of honeybees that are infected with the virus,

M : number of mites that infest the colony,

m : number of mites that carry the virus.

Given that bee and mite populations are large, consisting of thousands of individuals, we can consider these variables as continuous variables which allow us to use traditional SIR-like Ross-MacDonald differential equations to describe the progression of the vector borne disease. Our proposed mathematical model is based on [12] and extends this model by adding for the mite population an additional simple logistic equation with bee population size dependent carrying capacity, whereas in the previous studies the mite population strength was treated as a given parameter. Thus the modified model reads

$$\frac{dm}{dt} = \beta_1(M - m)\frac{y}{x + y} - \beta_2m\frac{x}{x + y} \quad (1)$$

$$\frac{dx}{dt} = \mu g(x)h(m) - \beta_3m\frac{x}{x + y} - d_1x - \gamma_1Mx \quad (2)$$

$$\frac{dy}{dt} = \beta_3m\frac{x}{x + y} - d_2y - \gamma_2My \quad (3)$$

$$\frac{dM}{dt} = rM \left(1 - \frac{M}{\alpha(x + y)} \right) \quad (4)$$

The parameter μ in (2) is the maximum birth rate, specified as the number of worker bees born per day.

The function $g(x)$ expresses that a sufficiently large number of healthy worker bees is required to care for the brood. We think of $g(x)$ as a switch function. If x falls below a critical value, which may seasonally depend on time, essential work in the maintenance of the brood cannot be carried out anymore and no new bees are born. If x is above this value, the birth of bees is not hampered. Thus $g(0, \cdot) = 0$, $\frac{dg(0)}{dx} \geq 0$, $\lim_{x \rightarrow \infty} g(x) = 1$. A convenient formulation of such switch like behavior is given by the sigmoidal Hill function

$$g(x) = \frac{x^n}{K^n + x^n} \quad (5)$$

where the parameter K is the size of the bee colony at which the birth rate is half of the maximum possible rate and the integer exponent $n > 1$. If $K = 0$ is chosen, then the bee birth terms of the original model of [31] is recovered. Then the brood is always reared at maximum capacity, independent of the actual bee population size, because $g(x) \equiv 1$.

Table 1: Seasonal averages of model parameters, derived from the data presented in [31].

Parameter	Spring	Summer	Autumn	Winter
β_1	0.1593	0.1460	0.1489	0.04226
β_2	0.04959	0.03721	0.04750	0.008460
β_3	0.1984	0.1460	0.1900	0.03384
d_1	0.02272	0.04	0.02272	0.005263
d_2	0.2	0.2	0.2	0.005300
μ	500	1500	500	0
k	0.000075	0.00003125	0.000075	N/A
r	0.0165	0.0165	0.0045	0.0045

The function $h(m)$ in (2) indicates that the birth rate is affected by the presence of mites that carry the virus. This is in particular important for viruses like ABPV that kill infected pupae before they develop into bees. The function $h(m)$ is assumed to decrease as m increases, $h(0) = 1$, $\frac{dh}{dm}(m) < 0$ and $\lim_{m \rightarrow \infty} h(m) = 0$; [31] suggests that this is an exponential function $h(m) \approx e^{-mk}$, where k is non-negative. We will use this expression in the computer simulations later on.

The parameter β_1 in (1) is the rate at which mites that do not carry the virus acquire it. The rate at which infected mites lose their virus to an uninfected host is β_2 . The rate at which uninfected bees become infected is β_3 , in bees per virus carrying mite and time.

Finally, d_1 and d_2 are the death rates for uninfected and infected honeybees. We can assume that infected bees live shorter than healthy bees, thus $d_2 > d_1$.

The newly added equation (4) is a logistic growth model for varroa mites. By r we denote the maximum mite birth rate. The carrying capacity for the mites changes with the host population size, $x + y$, and is characterized by the parameter α which indicates how many mites can be sustained per bee on average. This assumption is in agreement with [13].

Mites contribute to an increased mortality of bees. This is considered in (2) and (3) by including death terms that depend on M ; the parameters $\gamma_{1,2}$ are the rate at which mites kill bees.

The parameters μ , k , α , β_i , d_i , γ_i , $g(x)$, $h(m)$, r are assumed to be non-negative. They can change with time. In particular major differences may be observed between seasons. For example, the life span of a worker bee in summer is much shorter than in winter [1, 27]; the birth rate for bees is higher in Summer than in Spring and Autumn, and it drops down to 0 in winter [32]. Seasonal averages for the model parameters $\beta_{1,2,3}$, μ , $d_{1,2}$, k can be derived from the data in [31]. These are summarized in Table 1 and will be used in the simulations below.

In order to investigate the fate of a honeybee colony after mite/virus infestation over several years, the complete non-autonomous model with time dependent coefficients must be studied. This is not easily possible with purely

analytical techniques and we will resort to numerical simulations for this purpose below. In preparation for this, it is useful to study the governing equations in the autonomous case, i.e. assuming constant parameters, using qualitative analytical methods. For one, this will provide insight into the disease dynamics that will be helpful later on to discuss simulation results. Secondly, the analysis of the autonomous case will allow us to determine critical parameters and estimates for their numerical values which can be used to design the numerical simulation study.

3 Analysis in the autonomous case

In order to prepare for the analysis of the complete four dimensional model (1)-(4), we start our investigation by studying smaller, easier accessible sub-models. We begin by discussing the model for a healthy bee colony without mites and virus. In a second preliminary step we will introduce mites but not the virus. The analysis of the complete model builds on the results of these simpler special cases.

3.1 The one-dimensional healthy bee sub-model

In the absence of parasites and viruses, the model becomes

$$\frac{dx}{dt} = \mu g(x) - d_1 x. \quad (6)$$

It is easily verified that the solutions of this equation also give parasite and virus free solutions of (1)-(4) for initial data $m(0) = y(0) = M(0)$.

This system satisfies a Lipschitz condition for all $x \geq 0$, i.e. the initial value problem with $x(0) \geq 0$ has a unique solution. It follows by comparison with the trivial solution $x \equiv 0$, that its solution will be non-negative.

The dynamics of one-dimensional autonomous systems is simple: either the solutions converge to an asymptotically stable equilibrium or they diverge to $\pm\infty$. Because of $g(0) = 0$, we always find the trivial equilibrium $x_0^* = 0$, because of $g'(0) = 0$ it is asymptotically stable for all positive parameter values. The intersections of the line $y = \frac{d_1}{\mu}x$ with the function $g(x)$ gives further non-trivial equilibria. Since g is a sigmoidal function, it is easily verified that no such intersection for positive x exists for large d_1/μ , and two such intersections exist if d_1/μ is small enough, which we denote by x_1^* and x_2^* with $x_1^* > x_2^*$. With geometrical arguments it follows that in the latter case x_2^* is unstable, while x_1^* is stable. Assuming $g(x)$ to be a Hill function (5) we make this statement more precise:

Lemma 3.1. *The differential equation (6) with (5) has an asymptotically stable equilibrium at $x_0^* = 0$. If $\frac{d_1}{\mu} > \frac{n-1}{n} \frac{1}{K \sqrt[n]{n-1}}$ then this is the only steady state. If $\frac{d_1}{\mu} < \frac{n-1}{n} \frac{1}{K \sqrt[n]{n-1}}$ then two positive steady states x_1^*, x_2^* exist with $x_1^* > x_2^*$. Equilibrium x_1^* is asymptotically stable, x_2^* is unstable.*

Proof. The non-trivial equilibria of (6) with (5) are obtained as roots of the polynomial

$$G(x) = x^n - \frac{\mu}{d_1}x^{n-1} + K^n.$$

Descarte's Rule of Signs immediately implies that there are either two or none positive roots. No such equilibrium exists if the line $f(x) = \frac{d_1}{\mu}x$ lies above $g(x)$ for all $x > 0$. We denote by Δ the critical value of d_1/μ for which $f(x)$ is tangential to $g(x)$ in a point \hat{x} . Then

$$\Delta = \frac{g(\hat{x})}{\hat{x}} = \frac{\hat{x}^{n-1}}{K^n + \hat{x}^n}, \quad \text{and} \quad \Delta = g'(\hat{x}) = \frac{nKx^{n-1}}{(K^n + \hat{x}^n)^2},$$

whence

$$\hat{x} = K \sqrt[n]{n-1}.$$

Thus we calculate the critical value for Δ as

$$\Delta = \frac{n-1}{n} \frac{1}{K \sqrt[n]{n-1}}.$$

If $d_1/\mu > \Delta$, then $f(x)$ and $g(x)$ have no intersection, i.e. no non-trivial equilibrium of (6) exists. If $d_1/\mu < \Delta$, then $f(x)$ intersects with $g(x)$ twice, in points $x_{1,2}^*$, w.l.o.g. with $x_1^* > x_2^*$. The stability of these equilibria is determined from the signs of $g'(x_i^*) - f'(x_i^*)$. Since both functions start in $f(0) = g(0) = 0$ and $g'(0) < f'(0)$, the line $f(x)$ intersects with $g(x)$ in x_2^* from above, i.e. $f'(x_2^*) < g'(x_2^*)$ and in x_1^* from below, i.e. $f'(x_1^*) > g'(x_1^*)$. \square

Remark 1. The solutions of the initial value problem (6) with (5) and $x(0) \geq 0$ are bounded from above by $\max\{x(0), x_2^*\}$, thus they exist globally. If x_2^* does not exist they are monotonously decreasing and thus bounded by $x(0)$.

Remark 2. In the special case $n = 2$, we can explicitly calculate the two positive equilibria as

$$x_{1,2}^* = \frac{1}{2} \left(\frac{\mu}{d_1} \pm \sqrt{\frac{\mu^2}{d_1^2} - 4K^2} \right). \quad (7)$$

The unconditional stability of the trivial equilibrium reflects that a certain number of honeybees is required to care for the brood. If the initial value $x(0) < x_2^*$ the colony dies out, if $x(0) > x_2^*$, a healthy colony can establish itself attaining equilibrium x_1^* . For the data in Table 1, the non-trivial equilibria $x_{1,2}^*$ exist in Spring, Summer, and Fall, but not in Winter, when no new bees are born. Thus in Winter all solutions converge to the trivial one, however, as this is not reached in finite time.

3.2 The two-dimensional bee-mite sub-model

We investigate now how the stability of the equilibria $x_{0,1,2}^*$ changes when parasitic mites are considered in the colony that does not carry the virus. To this end we study the bee-mite subsystem of (1)-(4),

$$\frac{dx}{dt} = \mu g(x) - d_1 x - \gamma_1 M x \quad (8)$$

$$\frac{dM}{dt} = rM \left(1 - \frac{M}{\alpha x} \right). \quad (9)$$

where we again assume a sigmoidal Hill function as in (5) for the brood maintenance function $g(x)$.

It suffices to consider the case of strictly positive initial data. If initially $M = 0$ then $M = 0$ for all t and the model reduced to (6). Because bees are required as hosts for mites the case of initial data $M(0) \geq 0$ and $x(0) = 0$ is irrelevant.

Proposition 3.1. *The initial value problem of (8),(9) with $x(0) = x_0, M(0) = M_0$ and $(x_0, M_0) \in D := \{(x, M) : x > 0, M > 0\}$ possesses a unique solution in D , which is non-negative. Moreover, the set $D_{\tilde{x}} = \{(x, M) : 0 \leq x \leq \tilde{x}, 0 \leq M \leq \alpha \tilde{x}\}$ is positively invariant for all $\tilde{x} > \frac{\mu}{d_1}$.*

Proof. Local existence and uniqueness follow from standard arguments, since (8), (9) satisfy a Lipschitz condition in D . That positive initial data lead to positive solutions follows from the tangent criterion in the usual form.

To show the boundedness of x , we note that from equation (8) follows

$$\frac{dx}{dt} = \mu g(x) - d_1 x - \gamma_1 M x < \mu - d_1 x.$$

This means

$$x(t) < \frac{\mu}{d_1} - \left(\frac{\mu}{d_1} - x_0 \right) e^{-d_1 t},$$

whence, $x(t) < \max\{x_0, \frac{\mu}{d_1}\}$. In order to show the boundedness of M , we pick a $\tilde{x} \geq x$. We have

$$\frac{dM}{dt} = rM \left(1 - \frac{M}{\alpha x} \right) \leq rM \left(1 - \frac{M}{\alpha \tilde{x}} \right)$$

Hence, $M(t)$ is bounded from above by the solution of the logistic equation

$$\frac{d\tilde{M}}{dt} = r\tilde{M} \left(1 - \frac{\tilde{M}}{\alpha \tilde{x}} \right)$$

with carrying capacity $\alpha \tilde{x}$. Thus, if $M_0 \leq \alpha \tilde{x}$, then $M(t) \leq \alpha \tilde{x}$ for all $t > 0$. \square

To study the longtime behavior of (8), (9) and its stability, we investigate its equilibria. The system admits, under some conditions on parameters, the following equilibria:

- A:** the trivial equilibrium $(x^*, M^*) = (0, 0)$ exists for all choices of parameters
- B:** the mite-free equilibrium $(x^*, M^*) = (x_2^*, 0)$, where x_2^* is the unstable equilibrium of (6), according to Lemma 3.1; it exists under the conditions discussed there.
- C:** the mite-free equilibrium $(x^*, M^*) = (x_1^*, 0)$, where x_1^* is the stable equilibrium of (6), according to Lemma 3.1; it exists under the conditions discussed there.
- D,E:** mite infested equilibria of the form $(x^*, M^*) = (x^*, \alpha x^*)$ with $x^* > 0$, the existence of which is discussed below.

Proposition 3.2. *The set $D_{\tilde{x}} = \{(x, M) : 0 < x < \tilde{x}, 0 < M < \alpha \tilde{x}\}$ is positively invariant for all $\tilde{x} < x_2^*$ according to Lemma 3.1. All solutions entering such a $D_{\tilde{x}}$ converge to the trivial equilibrium A.*

Proof. From (8) it follows that for all $x < \tilde{x} < x_2^*$

$$\frac{dx}{dt} = \mu g(x) - (d_1 + \delta_1(M))x \leq \mu g(x) - d_1 x < 0.$$

Moreover, for all $M > \alpha x$ we have

$$\frac{dM}{dt} = rM \left(1 - \frac{M}{\alpha x}\right) < 0.$$

Furthermore, with standard arguments, the M - and x -axis are positively invariant (and all solutions on the axes converge to A). Thus, $D_{\tilde{x}}$ is positively invariant with inward pointing flux along the boundaries $x = \tilde{x}$ and $M = \alpha \tilde{x}$.

In $D_{\tilde{x}}$, for all solutions $\frac{dx}{dt} < 0$. On the other hand, in the lower triangle $M < \alpha x$, M is increasing, in the upper triangle $M > \alpha x$ it is decreasing; see Figure ?? for an illustration. Because the trivial equilibrium A is the only equilibrium in the closure of $D_{\tilde{x}}$, all solutions entering the upper triangle, therefore converge to A. Solutions in the lower triangle increase in M until they enter the upper triangle. \square

Proposition 3.3. *If they exist, the equilibrium $B = (x^*, 0) = (x_1^*, 0)$ of (8),(9) is an unstable node, while the equilibrium $C = (x^*, 0) = (x_2^*, 0)$ is an unstable saddle.*

Proof. The Jacobian of the right hand side of the differential equation is

$$J(x, M) = \begin{bmatrix} \mu g'(x) - (d_1 + \gamma_1 M) & -\gamma_1 x \\ \frac{rM^2}{\alpha x^2} & r \left(1 - \frac{2M}{\alpha x}\right) \end{bmatrix}. \quad (10)$$

Equilibria B, C exist if the equilibria $x_{1,2}^*$ of the one-dimensional model (6) exist. In this case we have for B, C that

$$J(x_{1,2}^*, 0) = \begin{bmatrix} \mu g'(x_{1,2}^*) - d_1 & -\gamma_1 x_{1,2}^* \\ 0 & r \end{bmatrix}$$

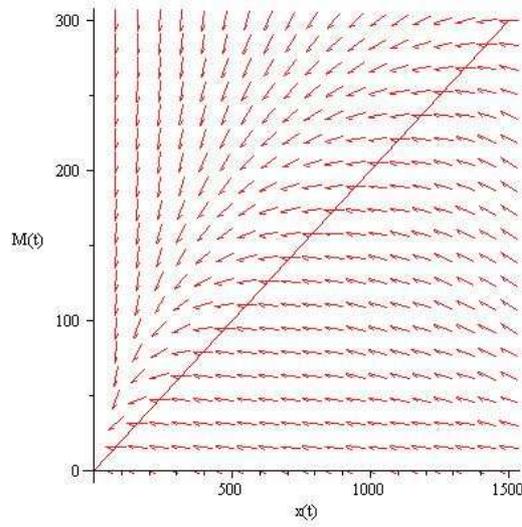


Figure 1: Direction field for the system (8), (9) for $0 < x < \tilde{x}, 0 < M < \alpha < \tilde{x}$ with $\tilde{x} < x_2^*$, the unstable equilibrium of (6). We have used the summer parameter values with $K = 8000$ and $\alpha = 0.2$. The initial values used are $x(0) = 30$ and $M(0) = 20$

The eigenvalues are $\lambda_1 = \mu g'(x_{1,2}^*) - d_1$ and $\lambda_2 = r$. Eigenvalue λ_2 is positive for all parameter sets and both equilibria B, C . With Lemma 3.1, we have $\lambda_1 > 0$ for x_2^* , and $\lambda_1 < 0$ for x_1^* . Thus, C is a saddle and B is an unstable node. \square

Remark 3. In the special case $n = 2$, the eigenvalues λ_1 of B and C are obtained as

$$\lambda_1 = \mp d_1 \sqrt{1 - 4K^2 \left(\frac{d_1}{\mu}\right)^2}.$$

Proposition 3.4. *There are at most two mite-infested equilibria D, E , with $x^* > 0, M^* = \alpha x^* > 0$. We denote the one with the smaller x^* by D , the other one by E . The point D is always unstable. If $x^* > \sqrt[n]{n-1}K$ for E , then E is stable; if $x^* < \sqrt[n]{n-1}K$ for E , then E is stable for small enough r , otherwise it is unstable.*

Proof. The points D and E are the intersections of the x -nullcline

$$\hat{M}(x) = \alpha x$$

with the M -nullcline

$$\tilde{M}(x) = \frac{\mu}{\gamma_1} \frac{g(x)}{x} - \frac{d_1}{\gamma_1}.$$

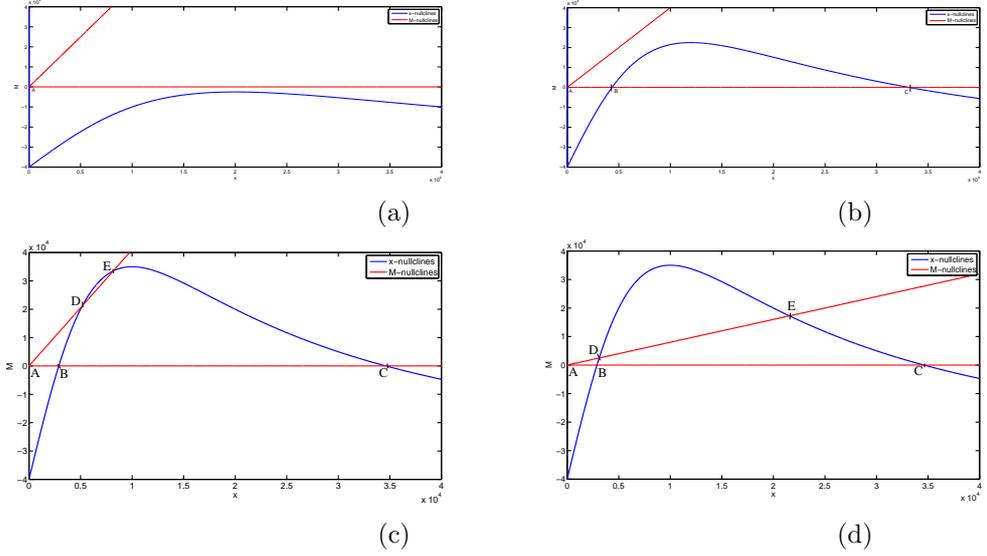


Figure 2: Nullclines and equilibria: (a) the trivial equilibrium A is the only equilibrium; (b) only the mite free equilibria A,B,C exist; (c), (d) two additional equilibria D, E with $M^* = \alpha x^* > 0$ exist, in (c) $x_E^* < \hat{x}$, in (d) $x_E^* > \hat{x} = \sqrt[n]{n-1}K$.

Whether or not these intersections exist depends on the values of the model parameters. Using $\hat{M}(x^*) = \tilde{M}(x^*)$ and (5), we find x^* as the roots of the polynomial

$$-\gamma\alpha x^{n+1} - d_1 x^n + \mu x^{n-1} - \gamma\alpha x K^n - d_1 K^n = 0$$

Descartes's Rule of Signs implies that this polynomial has at most two positive roots. For $n = 2$ also $\mu > \gamma\alpha K^2$ must be satisfied, i.e. the birth rate must be sufficiently high or the maximum sustainable mite load and brood maintenance coefficient sufficiently low.

For $x > 0$, the function $\tilde{M}(x)$ is a continuous, differentiable function with

$$\tilde{M}(0) = -\frac{d_1}{\gamma_1}, \quad \text{and} \quad \lim_{x \rightarrow \infty} \tilde{M}(x) = -\frac{d_1}{\gamma_1}.$$

With the usual calculus arguments we find that it has a single extremum, namely a maximum at

$$\hat{x} = \sqrt[n]{n-1}K.$$

It is strictly monotonically increasing for $x < \hat{x}$ and strictly decreasing for $x > \hat{x}$. We consider now the case where D and E exist. The stability of these equilibria is investigated with the help of the Jacobian (10). Substituting $M = \hat{M} = \alpha x$ into the second row and the equivalent $M = \tilde{M} = \frac{\mu}{\gamma_1} \frac{g(x)}{x} - \frac{d}{\gamma_1}$ into the first row,

we find

$$J(x^*, \alpha x^*) = \begin{bmatrix} \mu \left(g'(x^*) - \frac{g(x^*)}{x^*} \right) & -\gamma_1 x^* \\ r\alpha & -r \end{bmatrix}.$$

We introduce the notation

$$z(x) = \mu \left(g'(x) - \frac{g(x)}{x} \right)$$

and note that

$$\tilde{M}'(x) = \frac{z(x)}{\gamma_1 x}.$$

The stability of the equilibria is assessed from the trace and determinant of $J(x^*, \alpha x^*)$. We have

$$\det = -r(z^* - \alpha\gamma_1 x^*), \quad \text{tr} = z^* - r,$$

where we used the shorthand notation $z^* = z(x^*)$. In equilibrium point D , the x -nullcline $\tilde{M}(x)$ intersects with the M -nullcline $\hat{M}(x)$ from below, thus

$$\tilde{M}'(x^*) = \frac{z^*}{\gamma_1 x^*} > \alpha = \hat{M}'(x^*),$$

whence $z^* > \gamma_1 \alpha x^*$ and, therefore, $\det < 0$. Thus, the equilibrium D is unstable.

In equilibrium point E , the x -nullcline $\tilde{M}(x)$ intersects with the M -nullcline $\hat{M}(x)$ from above, thus

$$\tilde{M}'(x^*) = \frac{z^*}{\gamma_1 x^*} < \alpha = \hat{M}'(x^*),$$

whence $z^* < \gamma_1 \alpha x^*$. Thus $\det = -r(z^* - \alpha\gamma_1 x^*) > 0$. To analyse the stability of equilibrium E further, we have to distinguish between two possibilities:

If $x_E^* < \hat{x} = K \sqrt[n]{n-1}$, then $z^* > 0$. In this case $\text{tr} = z^* - r < 0$ for all $r < z^*$. Thus E is stable for small enough r but unstable if $r > z^*$.

If $x_E^* > \hat{x} = K \sqrt[n]{n-1}$, then $z^* < 0$. In this case $\text{tr} = z^* - r < 0$ for all r . Thus E is stable.

□

Remark 4. The value of z in the proof of the above proposition depends only on the parameters of the birth term, μ and K . It does not depend on bee death or mite parameters.

Remark 5. If α is sufficiently small or μ is sufficiently large the two intersections of $\hat{M}(x)$ and $\tilde{M}(x)$ exist. The possible cases are illustrated in Figure 2.

Note that in the proposed model, a mite infestation can never be fought off by the bee colony. This is an immediate consequence of the logistic growth function but agrees with observations reported by beekeepers.

3.3 The complete bee-mite-virus model

We investigate now the question whether a stable, mite infested honeybee colony can fight off the virus. To this end we consider the complete four-dimensional model (1)-(4) with (5).

Proposition 3.5. *The initial value problem of (1)-(4) with $(m_0, x_0, y_0, M_0) \in D := \{(m, x, y, M) : m > 0, x > 0, y > 0, M > 0\}$ possesses a unique solution, which is non-negative. Moreover, the set*

$$Z_{\tilde{x}} = \{(m, x, y, M) : 0 \leq m \leq \alpha\tilde{x}, 0 \leq x, 0 \leq y, (x + y) \leq \tilde{x}, 0 \leq M \leq \alpha\tilde{x}\}$$

is positively invariant for all $\tilde{x} > \frac{\mu}{d_1}$.

Proof. Existence, uniqueness and non-negativity of the solution follow with standard arguments that can be found in [33]. For boundedness of $x + y$, we note that from equations (2) and (3),

$$\begin{aligned} \frac{dx}{dt} + \frac{dy}{dt} &= \mu g(x) - d_1 x - d_2 y - \gamma_1 M x - \gamma_2 M y \\ &\leq \mu g(x) - d_1 x - d_2 y \\ &\leq \mu g(x + y) - d(x + y) =: G(x + y) \end{aligned}$$

where $d = \min\{d_1, d_2\}$. Here we used that $g(x)$ is a monotonously increasing function. Therefore, we have

$$G(x + y) \leq 0 \quad \forall \quad (x + y) \geq \tilde{x} > \frac{\mu}{d_1}.$$

In order to show the boundedness of M , we assume that $x + y \leq \tilde{x}$.

$$\frac{dM}{dt} = rM \left(1 - \frac{M}{\alpha(x + y)} \right) \leq rM \left(1 - \frac{M}{\alpha\tilde{x}} \right)$$

Thus, if $M(0) < \alpha\tilde{x}$, $M(t)$ is bounded from above by the solution of the logistic equation

$$\frac{dz}{dt} = rz \left(1 - \frac{z}{\alpha\tilde{x}} \right)$$

with carrying capacity $\alpha\tilde{x}$, in particular it is bounded by $\alpha\tilde{x}$. In order to show the boundedness of m , we have

$$\frac{dm}{dt} = \frac{\beta_1(M - m)y}{x + y} - \frac{\beta_2 m x}{x + y} \leq \beta_1(M - m) \leq \beta_1(\alpha\tilde{x} - m)$$

Therefore, m is bounded from above by the solution of the linear equation

$$\frac{d\tilde{m}}{dt} = \beta_1(\alpha\tilde{x} - \tilde{m}),$$

i.e. in particular by the constant $\alpha\tilde{x}$, if $m(0) < \alpha\tilde{x}$. □

It is easy to verify from (1) and (3) that equilibria with $m^* = 0$ imply $y^* = 0$ and *vice versa*. Moreover, we find that to every equilibrium $A, \dots, E : (x^*, M^*)$ of the two-dimensional bee-mite model, there corresponds a disease free equilibrium $A_4, \dots, E_4 : (0, x^*, 0, M^*)$ of the complete four-dimensional bee-mite-virus model. The question of whether or not a virus free colony at equilibrium can fight off a virus infection is primarily of interest for the only non-trivial stable equilibrium of the two-dimensional model, namely E . The equilibrium E can occur in two distinctively different types, either $x^* < \sqrt[n]{n-1}K$ or $x^* > \sqrt[n]{n-1}K$. In the former the bee colony is small, and while its population may be stable under the conditions outlined above, it is not a properly working bee colony, i.e. it might be able to maintain itself but is not able to produce sufficient honey, etc. More interesting is the other case. It depends on the specific parameters, such as α or γ_1 how strong the colony is. In many cases it might be very strong, i.e. closer to the disease and mite free equilibrium x_1^* than to $\hat{x} = \sqrt[n]{n-1}K$. We investigate the stability of the equilibrium $E_4 : (0, x^*, 0, \alpha x^*)$ under (1)-(4).

Proposition 3.6. *Let $E : (x^*, \alpha x^*)$, $x^* > 0$, be an asymptotically stable equilibrium of (8),(9). Then $E_4 : (0, x^*, 0, \alpha x^*)$ is an asymptotically stable equilibrium of (1)-(4) if*

$$\beta_3 \beta_1 \alpha < \beta_2 (d_2 + \gamma_2 \alpha x^*). \quad (11)$$

Proof. We analyse the Jacobian in E_4 ,

$$J(0, x^*, 0, \alpha x^*) = \begin{bmatrix} -\beta_2 & 0 & \beta_1 \alpha & 0 \\ \mu g(x^*) h'(0) - \beta_3 & \mu g'(x^*) - d_1 - \gamma_1 \alpha x^* & 0 & -\gamma_1 x^* \\ \beta_3 & 0 & -d_2 - \gamma_2 \alpha x^* & 0 \\ 0 & r \alpha & r \alpha & -r \end{bmatrix}.$$

Its eigenvalues are

$$\begin{cases} \lambda_1 = -\frac{1}{2} \left[d_2 + \gamma_2 \alpha x^* + (\beta_2) - \sqrt{(d_2 \gamma_2 \alpha x^* - \beta_2)^2 + 4\beta_3 \beta_1 \alpha} \right] \\ \lambda_2 = -\frac{1}{2} \left[(\beta_2 + d_2 + \gamma_2 \alpha x^*) + \sqrt{(\gamma_2 \alpha x^* - \beta_2 + d_2)^2 + 4\beta_3 \beta_1 \alpha} \right] \\ \lambda_3 = \frac{1}{2} (\mu g'(x^*) - d_1 - \gamma_1 \alpha x^* - r) + \frac{1}{2} \sqrt{(\mu g'(x^*) - d_1 - \gamma_1 \alpha x^* + r)^2 - 4r \gamma_1 \alpha x^*} \\ \lambda_4 = \frac{1}{2} (\mu g'(x^*) - d_1 - \gamma_1 \alpha x^* - r) - \frac{1}{2} \sqrt{(\mu g'(x^*) - d_1 - \gamma_1 \alpha x^* + r)^2 - 4r \gamma_1 \alpha x^*} \end{cases}$$

Since all parameters are non-negative, it follows immediately that λ_1 and λ_2 are real. The eigenvalue λ_2 is always negative. The first eigenvalue λ_1 is

$$\lambda_1 = -\frac{1}{2} \left[d_2 + \gamma_2 \alpha x^* + (\beta_2) - \sqrt{(d_2 \gamma_2 \alpha x^* - \beta_2)^2 + 4\beta_3 \beta_1 \alpha} \right]$$

For λ_1 to be negative we require

$$(d_2 + \gamma_2 \alpha x^* + \beta_2) > \sqrt{(d_2 \gamma_2 \alpha x^* - \beta_2)^2 + 4\beta_3 \beta_1 \alpha},$$

or, equivalently

$$4\beta_3\beta_1\alpha < (d_2 + \gamma_2\alpha x^* + \beta_2)^2 - (d_2 + \gamma_2\alpha x^* - \beta_2)^2,$$

which is the same as

$$\beta_3\beta_1\alpha < \beta_2(d_2 + \gamma_2\alpha x^*). \quad (12)$$

We will use the results of the two-dimensional model to investigate λ_3 and λ_4 . At equilibrium, we have

$$\mu \frac{g(x^*)}{x^*} = d_1 + \gamma_1\alpha x^*.$$

This allows us to rewrite λ_3 in terms of the function $z(x) = \mu \left(g'(x) - \frac{g'(x)}{x} \right)$ which was introduced above in the proof of Proposition 3.4. This gives

$$\lambda_3 = \frac{1}{2} \left[(z^* - r) + \sqrt{(z^* + r)^2 - 4\gamma_1\alpha x^* r} \right].$$

Defining

$$c := -4r(z^* - \gamma_1\alpha x^*),$$

this is equivalent to

$$\lambda_3 = \frac{1}{2} \left[(z^* - r) + \sqrt{(z^* - r)^2 - c} \right].$$

Recall from the proof of Proposition 3.4 that $z^* - r < 0$ (this is the trace of the stability matrix of E in the 2D case) and $c > 0$ (this is four times the determinant of the stability matrix of E in the 2D case), because E is stable for $x^* > \sqrt[n]{n-1}K$. If $c > (z^* - r)^2$, then λ_3 is complex with negative real part $z^* - \lambda$. Otherwise, $|z^* - r| > \sqrt{(z^* - r)^2 - c}$, implying that λ_3 is negative.

The last eigenvalue λ_4 is rewritten as

$$\lambda_4 = \frac{1}{2} \left[(z^* - r) - \sqrt{(z^* - r)^2 - c} \right].$$

Again, if $c > (z^* - r)^2$ then λ_4 is complex with negative real part $z^* - \lambda$. Otherwise, λ_4 is negative. \square

Remark 6. Keeping in mind that at equilibrium E the number of mites is $M^* = \alpha x^*$, the stability criterion (12) is a straightforward extension of the criterion for the model of [12], which treated the mite load as a known constant, to the model (1)-(4) in which the mites are a dependent variable.

4 Computational investigation of the periodic coefficient case

4.1 Computational setup and parameters

The study of the nonlinear non-autonomous model is more complicated than the autonomous case and an analytical treatment is not easily possible. Therefore, we study the model in computer simulations. The focus of this numerical study is to verify whether or not the various types of system behaviour that were found with analytical techniques for the autonomous case can also be observed in the non-autonomous case with seasonally fluctuating coefficients (periodic over the year). The result of the autonomous analysis are thereby used to design the computations that we execute numerically.

For the computer simulations we use the software package MATLAB. The ordinary differential equations are integrated by the built in routine `ode15s`, a variable order solver based on numerical differentiation formulas. In all cases, we run the computer simulations for a period of 7000 days (approx 20 years), or until the colony vanishes, whichever comes first.

Numerical values for the seasonal averages of the parameters β_i, d_i, μ, k are obtained from [31], see Table 1. Lacking more detailed information, we use these values to construct piecewise constant time varying parameter functions, assuming four equally long seasons of 91.25 days. For simplicity, we fix the Hill coefficient in the growth maintenance terms as $n = 2$, in accordance with [12]. The values of γ_1 and γ_2 are estimated to be 10^{-7} for every seasons, based on order of magnitude considerations. The parameter r is obtained from [19, 22].

We assume the above parameters to be given and investigate the behaviour of the model with respect to the remaining parameters K and α . The former affects the bee birth rate, the latter the mite population that can be established. There is no fixed value of the parameter α available in the literature. In [31], it has been found that 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. From this we we conclude $\alpha \approx 0.3$. On the other hand, in [5], the values 0.1321 (Oct-Dec) and 0.4785 (Jan-Feb) are reported, while an average value of 0.5 is found in [28]. In our simulations, we vary α over the range that covers these values.

In order to estimate values for the brood maintenance constant K , we look at the bee-model (6). The number of bees in an established colony, $x^* = \frac{\mu}{d_1}$, is greater than the brood maintenance constant K . The value of $\frac{\mu}{d_1}$ for Spring and Fall is computed as 22007 and for Summer 37500. This gives upper bounds on the values that we will choose for K .

4.2 The bee-mite sub-model

We start our investigation by considering the sub-model that is comprised by bees and mites only, without taking the virus into account, i.e. model (8), (9) In the autonomous case, we found that bee colonies that are infested by mites

either collapse (equilibrium A is attained) or that an endemic equilibrium, in which mites and bees co-exists is attained (equilibrium E). The latter was the case if $x^* < K^n \sqrt[n]{n-1}$ or if the mite birth rate r is small enough. In a first simulation experiment, we investigate whether this carries over to the transient, non-autonomous case with seasonally changing coefficients.

In the two-dimensional case, a bee population can vanish either because the initial population are too small for a colony to establish itself even in the absence of mites, or because the equilibrium E is unstable, or although E might be stable the solution can be attracted by the stable trivial equilibrium. In our experiment, we vary the brood maintenance coefficient K and the mite carrying capacity α . Both parameters affect the steady state population size x^* and thus the stability criterion for E . Moreover, we will compare the fate of the bee population in these cases in the absence and presence of parasitic mites.

We start with the following simulations, the results of which are plotted in the corresponding Figures. In this first set of simulations, the mite carrying capacity α was set to 0.4784, 0.5, 0.5, 0.4784 for Spring, Summer, Fall, Winter, respectively. See Table 1 for the remaining parameters. These simulations were conducted:

Figure 3(a): low brood maintenance coefficient for K for Spring, Summer, Fall, Winter as 6000, 8000, 6000, 6000, respectively; and initial data $x(0) = 20000$ and $M(0) = 0$ (no mites)

Figure 3(b): brood maintenance coefficient K as in 3(a), but with initial data $x(0) = 20000$ and $M(0) = 100$ (mite infestation)

Figure 3(c): high brood maintenance coefficient for K for Spring, Summer, Fall, Winter as 11800, 12000, 11800, 6000, respectively; and initial data $x(0) = 20000$ and $M(0) = 0$ (no mites)

Figure 3(d): brood maintenance coefficient K as in 3(c), but with initial data $x(0) = 20000$ and $M(0) = 100$ (mite infestation)

Comparison between 3(a) and 3(b). Figures 3(a) and 3(b) show the development of a honeybee colony with and without mites. In Figure 3(a), in the absence of mites, the bee population increases in Spring and in Summer, it reaches a level of approximately 35,000. It decreases in Fall and Winter to approximately 14,000. This pattern repeats annually. In Figure 3(b), in the presence of mites, the bee population behaves similarly. However, after mites are established, in our simulations from year two on, it will attain lower values. The mite population starts at a very small value, increasing steadily until the end of the second summer. Then it declines as the bee population declines, and thus the number of hosts. In this stage, the mite population size is above the seasonal carrying capacity. From then on the mite population shows the same oscillatory behaviour as the bee population. From the third year on, it reaches in Summer a maximum value approximately 12000. On the other hand, in winter the mite population drops to minimum values around 8000. Thus, the behaviour of the mite population is determined essentially by the development

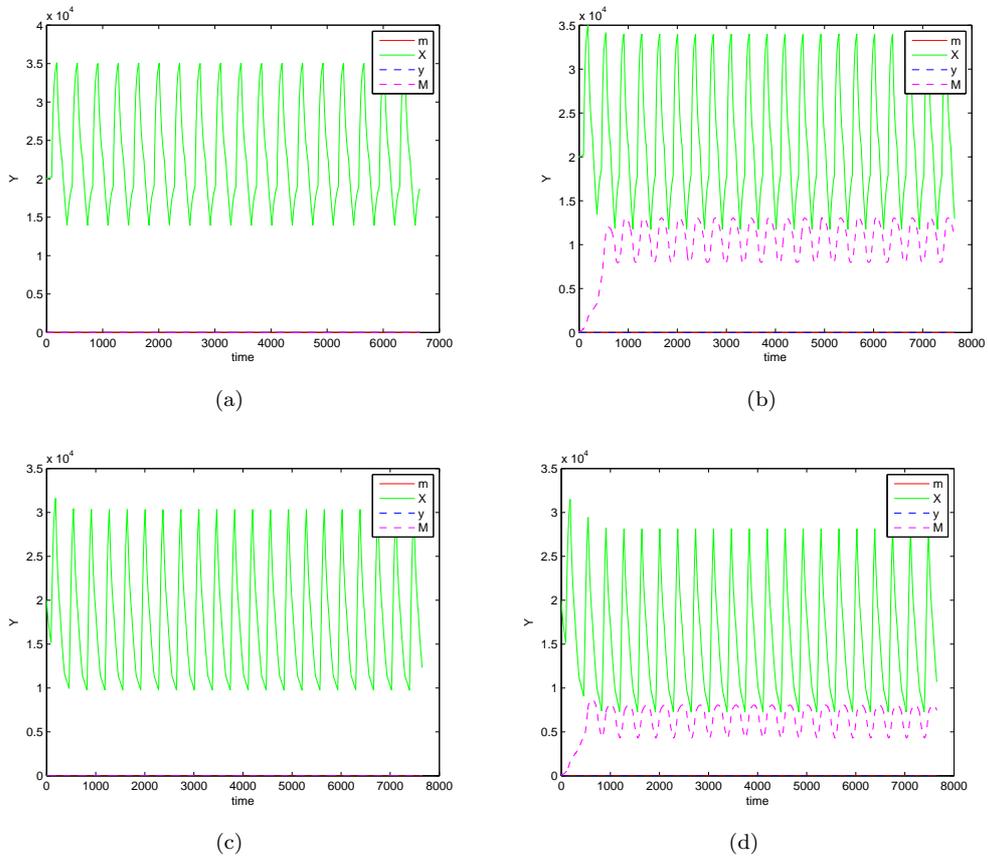


Figure 3: Simulation of bee-mite population dynamics: periodic solutions for varying brood maintenance coefficients K : low [top row, (a), (b)] versus high [bottom row (c),(d)] values, without mites [left column (a), (c)] and with mites [right column (b), (d)]. Time is given in days. See text for details of parameter values.

of the host population. The seasonally varying simulation of Figure 3(a) corresponds to the stable equilibrium x_1^* of the autonomous one-dimensional bee population model. The seasonally varying simulation in Figure 3(b) corresponds to the stable equilibrium E of the autonomous two-dimensional model.

Comparison between 3(a) and 3(c). Figures 3(a) and 3(c) show honeybee colonies in the absence of mites. The simulations differ with respect to the brood maintenance coefficient K that was used, which is lower in Figure 3(a) than in Figure 3(c). In both cases a periodic solution for the bee population is attained approximately from the second year on. In case 3(c) more bees are required to rear the brood at full capacity. Therefore, the bee population

reaches a lower size, fluctuating over the seasons between 10,000 and 30,000, compared to 14,000 \sim 35,000 in case 3(a).

Comparison between 3(c) and 3(d). Figure 3(c) shows a properly working honeybee colony in the absence of mites for higher values of K . It has been described in the previous paragraph. In Figure 3(d), the colony is infested by mites. After an initial transient period of three complete cycles during which the bee population decreases, compared to the data in Figure 3(c), a stable periodic solution is attained after the mite population establishes itself. The bee population size in the mite infested case Fig 3(d), however, is smaller than in the mite free case of Figure 3(c). The Summer maximum decreased from around 31,000 to 28,000, the Winter minimum from around 10,000 to around 8,000. The periodic solution in Figure 3(d) corresponds to the equilibrium of type E of the two-dimensional model in the autonomous case.

Comparison between 3(b) and 3(d). We compare the stable mite infested colonies for two different sets of brood maintenance coefficient K . Both figures have been explained above in detail. The maximum value of honeybees in summer is much more in 3(b) as compared to 3(d). The reason is that fewer worker bees are required to obtain bee birth at maximum capacity. A similar effect is also observed in the mite population as well. The difference in bee population values are also reflected in the differences of the sizes of the mite populations.

In the first simulation experiment we noticed that drastic changes in K can affect the fate of the bee population, but for relatively low values, the effect of mites on the bee population was less pronounced than for higher values.

In a second simulation experiment, we investigate whether or not mite infestation can lead to the extinction of 11,600 and 12,200 in Spring and Fall and between 12,000 and 14,000 in Summer. The relative carrying capacity parameter α is fixed as 0.4784, 0.1321, 0.1321, 0.4784 in Spring, Summer, Fall, and Winter. I.e. we assume a smaller tolerance of bees for mites in Summer and Spring compared to the previous simulations. The initial data used in the experiment is $x(0) = 20000$ and $M(0) = 100$. The following simulations are conducted:

Figure 4(a): brood maintenance coefficient for K for Spring, Summer, Fall, Winter as 11600, 12000, 11600, 6000, respectively

Figure 4(b): brood maintenance coefficient K for Spring, Summer, Fall, Winter as 11700, 14000, 11700, 6000, respectively

Figure 4(c): brood maintenance coefficient for K for Spring, Summer, Fall, Winter as 11900, 14000, 11900, 6000, respectively

Figure 4(d): brood maintenance coefficient for K for Spring, Summer, Fall, Winter as 12200, 14000, 12200, 6000, respectively

In the first case, Figure 4(a), as in the previous case we find a periodic co-existence solution that corresponds to the equilibrium E of the autonomous case, as in the simulations before. In the three other simulations, the bee colony vanishes after four [Fig. 4(b)], three [Fig. 4(c)], or two [Fig. 4(d)] years. In all

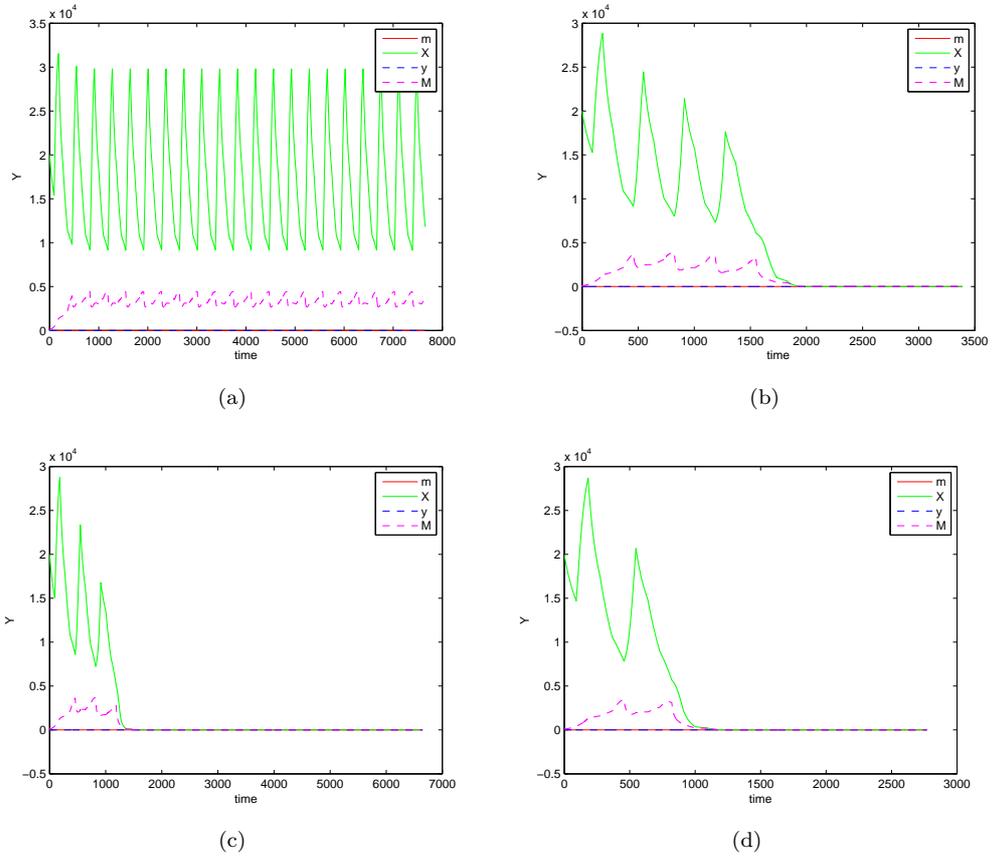


Figure 4: Simulation of bee-mite dynamics: disappearing bee colonies for brood maintenance coefficients K varying over the range $K = 11600$ and $K = 14000$ in Spring, Summer, Winter. Time is given in days; see text for details on model parameters.

three cases the collapse happens at the onset of Spring, when the bee population comes out of Winter too weak to care for the brood, i.e. the bee population falls during Winter below the brood maintenance coefficient. However, the decline in the bee population is visible from year one on as the maximum and minimum values clearly decrease from year to year.

4.3 The complete bee-mite-virus model

In our final simulation experiment we investigate the effect of the virus on bee populations that are infested by mites but attain a stable equilibrium. The analysis of the autonomous case showed that the disease can be fought off if

inequality (11) is satisfied. In our simulations we keep the disease transmission parameters $\beta_{1,2,3}$ at the given value and investigate whether result of the autonomous case carries over to the none autonomous case by varying parameter α . This also has an effect on x^* that enters (11). The values for the brood maintenance coefficient K used in all simulations are [8000, 12000, 8000, 6000] for Spring, Summer, Fall and Winter respectively. The following cases are considered.

Figure 5(a): high value of α for Spring, Summer, Fall, Winter as 0.4784, 0.5, 0.5, 0.4784, respectively; and initial data $x(0) = 20000$, $M(0) = 100$ and $m(0) = 0$ (no virus)

Figure 5(b): value of α as in 5(a), but with initial data $x(0) = 20000$, $M(0) = 100$ and $m(0) = 80$ (virus present)

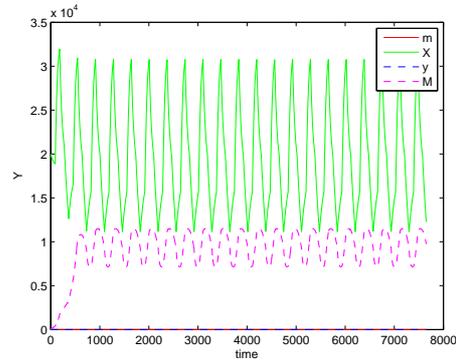
Figure 5(c): low value of α for Spring, Summer, Fall, Winter as 0.1, 0.1, 0.1, 0.1, respectively; and initial data $x(0) = 20000$, $M(0) = 100$ and $m(0) = 80$ (virus present)

Comparison between Figures 5(a), 5(b) and 5(c). The mite infested colony can be seen in the figure 5(a). Figure 5(b) shows that the colony collapses after 4 years due to virus and mites. It is interesting to see that the colony is working properly for the first 4 years and the mites are also in equilibrium with the bees. The virus is present in the colony for several years without being noticeable. After almost 6 years, the virus starts growing slowly. Due to this the bee population decreases in spring but it again starts increasing in summer. Now the virus grows rapidly, therefore the colony is not able survive the winter in the next year and it vanishes. This simulation experiment shows the collapse equilibrium of the four dimensional bee-mite-virus model. This kind of behaviour was also observed by Eberl et. al(2010). Therefore it is observed that the honeybee colony(with virus) vanishes for high values of the parameter α which is in agreement with [13].

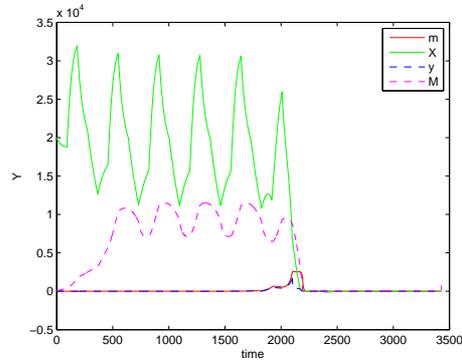
For lower values of α in Summer and Fall, we observe in Figure 5(c) that the disease is fought off and that a mite infested but stable honeybee colony survives. The parameter α is only reduced in two of the four seasons, compared to the previous simulations. Nevertheless the number of mites remains at much lower levels than in Figure 5(a) throughout.

5 Summary and Conclusion

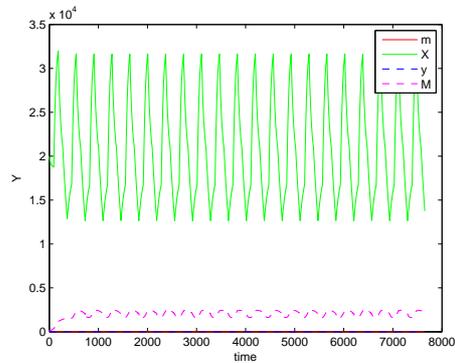
In Spring, Summer, and Autumn, in the absence of parasitic mites honey bee colonies can attain a healthy stable population size, which results from a balance of growth and natural death. Because workers are not produced in Winter born, the colony decreases in size. The lifespan of Winter bees is much longer than of Summer bees, so this decline in population size is small. However, if at the end of the Winter season the bee colony is too small to care for the new brood, however, the colony will not recover in Spring and die off. This phenomenon



(a)



(b)



(c)

Figure 5: Simulation of the bee-mite-virus complex: Panel (a) bees and mites co-exist in the absence of disease. In panel (b) the simulation is repeated with mites carrying the virus, leading to collapse during the 6th summer after introduction of the disease. In panel(c) the relative carrying capacity α is decreased in Summer and Spring compared to panel (b); the disease is fought off, the number of parasites is lower than in panel (a). Time is in days; see text for details on model parameters.

is known as *Wintering Losses* and has been described as a major contributor to the decline of honeybee colonies in colder climates. This is represented well by the bee growth model that underlies the more complex model of the honeybee-varroa-ABPV complex presented here.

In our model that describes mite growth by a simple logistic equation with host population dependent carrying capacity, parasitic varroa mites that infest a bee colony cannot be fought off. Depending on the severity of the original infestation and on seasonal parameters such as bee birth and death rates, mite birth rates, tolerance of bees for mites (as expressed by the relative carrying capacity α), mites might lead to a complete decline and die off of the bee colony, or an endemic equilibrium might be established, in which a stable bee and a stable mite population co-exist. In which case the bee colony often would be slightly weaker but still function well. However, depending on parameters, also the possibility was observed that a mite infested bee population co-exists with the parasite at a population strength that is far from that of a stable healthy, uninfested population. When a bee population vanishes as a consequence of a mite infestation, this can be a process of declining over several years (in our simulations we observed periods of 2-4 years), however, from year to year the bee population will be remarkably smaller.

The picture can change when the parasitic varroa mites become vectors for bee diseases, such as the Acute Bee Paralysis Virus. Even if in the absence of the disease the bees can co-exist with their parasite in healthy, strong numbers, this balance might tip as a consequence of the virus epidemic, and an eventual die out of the bee colony might be observed. This process can stretch over several years that it takes until the virus infestation has grown strong enough. During this transient period, virtually no sick bees are observed and it can appear as if bees and mites co-exist in a stable endemic equilibrium. The decline in the bee population from year to year can be very small and difficult to detect. The eventual collapse is then sudden.

Whether or not the bee population can fight off of a virus epidemic depends on model parameters describing disease transmission and how fast infected bees die, as well as on the size of the mite population in the stable bee-mite co-existence equilibrium. The latter depends on the growth and death rates of bees as well as on the tolerance level of the bees for the parasite (expressed in terms of parameter α). It appears to be independent of the birth rate of the mites. For given disease transmission rates (expressed in terms of $\beta_{1,2,3}$ and tolerance toward mites, faster death of infected bees helps the colony.

Although the model we present here yields qualitative insight into the progression of the bee disease, one has to realize that this is a simple, bare-bones model that neglects certain effects that can be of relevance. Therefore, its primary value has to be seen in the context of qualitative understanding rather than quantitative prediction.

For example, it is assumed here that the queen bee is always unaffected by mites and virus and that an old queen bee is replaced in the hive without swarming, i.e. loss of worker bees. We also assume that one year is identical to the next, i.e. that the parameters are periodic without accounting for random

modifications, e.g. due to weather, or for systematic changes of the environment (e.g. due to human development activities). Also, we conducted our simulation using seasonally averaged parameter values and it remains to be investigated whether the qualitative results remain the same if we consider continuously changing parameters instead. Nevertheless, despite this simplicity the model presented here might be a good starting point, for example to investigate efficacy of various varroa treatment strategies.

The results here were obtained using analytical techniques for the constant coefficient case and then verifying numerically whether or not the qualitative features of the autonomous case can be observed also in the non-autonomous case. We did not yet investigate whether or not the non-autonomous case can show additional behaviour that is not observable in the autonomous case. This needs to be explored computationally. Considering the high dimensionality of the parameter space and that the parameters are time dependent functions that is an ambitious undertaking that warrants a dedicated study in its own right.

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