

Disease dynamics of Honeybees with *Varroa destructor* as parasite and virus vector

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Abstract

The worldwide decline in honeybee colonies during the past 50 years has often been linked to the spread of the parasitic mite *Varroa destructor* and its interaction with certain honeybee viruses carried by Varroa mites. In this article, we propose a honeybee-mite-virus model that incorporates (1) parasitic interactions between honeybees and the Varroa mites; (2) five virus transmission terms between honeybees and mites at different stages of Varroa mites: from honeybees to honeybees, from adult honeybees to the phoretic mites, from brood to the reproductive mites, from the reproductive mites to brood, and from adult honeybees to the phoretic mites; and (3) Allee effects in the honeybee population generated by its internal organization such as division of labor. We provide completed local and global analysis for the full system and its subsystems. Our analytical and numerical results allow us have a better understanding of the synergistic effects of parasitism and virus infections on honeybee population dynamics and its persistence. Interesting findings from our work include: (a) Due to Allee effects experienced by the honeybee population, initial conditions are essential for the survival of the colony. (b) Low adult honeybees to brood ratios have destabilizing effects on the system which generate fluctuating dynamics that lead to a *catastrophic event* where both honeybees and mites suddenly become extinct. This catastrophic event could be potentially linked to Colony Collapse Disorder (CCD) of honeybee colonies. (c) Virus infections may have stabilizing effects on the system, and parasitic mites could make disease more persistent. Our model illustrates how the synergy between the parasitic mites and virus infections consequently generates rich dynamics including multiple attractors where all species can coexist or go extinct depending on initial conditions. Our findings may provide important insights on honeybee **viruses** and parasites and how to best control them.

Keywords: Allee Effects; Honeybees; Extinction; Virus; Parasite; Colony Collapse Disorder (CCD)

1. Introduction

Honeybees are the world's most important pollinators of food crops. It is estimated that one third of food that we consume each day mainly relies on pollination by bees. For example, in the United States, honeybees are major pollinators of alfalfa, apples, broccoli, carrots and many other crops, and hence are of economic **importance**. Honeybees have an estimated monetary value between \$15 and \$20 billion dollars annually as commercial pollinators in the U.S [29]. There are growing concerns both locally and globally that despite a 50% growth in honeybee stocks, the supply cannot keep up with the over 300% increase in agricultural demands [75]. Therefore, the recent sharp declines in honeybee populations have been considered as a global crisis. The most recent data from the 2012-2013 winter has shown an average loss of 44.8% of hives in the

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6 U.S., and a total of 30.6% loss of commercial hives [65]. Some beekeepers have reported a loss of 90% of
7 their hives [20, 46].
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9 Between 1972 and 2006, the wild honeybee populations declined severely and are now considered vir-
10 tually nonexistent [44, 73]. Hence the use of commercial honeybees for pollination is extremely important.
11 Beginning in 2006, beekeepers began to report an unusual phenomenon in dying bee colonies. Worker bees
12 would leave the colony to forage and never return, leaving the queen and the young behind to die. No
13 dead worker bees were found at the nest sites; they simply disappear [13, 68]. This phenomenon is known
14 as Colony Collapse Disorder (CCD), which is a serious problem threatening the health of honeybees and
15 therefore the economic stability of commercial beekeeping and pollination operations.
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17 The exact causes and triggering factors for CCD have not been completely understood yet. Researchers
18 have proposed several possible causes of CCD including stress on nutritional diet, harsh winter conditions,
19 lack of genetic diversity, exposure to certain pesticides, diseases, and parasitic mites *Varroa destructor* which
20 are also vectors of viral diseases of honeybees [29, 52]. Even before CCD was detected in honeybee colonies,
21 studies showed that most of the losses could be generally attributed to two main causes: the vampire mite,
22 *Varroa destructor*, which feeds on host haemolymph, weakens host immunity and exposes the bees to a
23 variety of viruses, and the tracheal mite, which infests the breathing tubes of the bee, punctures the tracheal
24 wall and sucks the bee's blood and also exposes the bee to a variety of viruses [57, 63, 41]. Since then,
25 *Varroa* mites have been implicated as the main culprit in dying colonies. For example, in Canada, *Varroa*
26 mites have been found to be the main reason behind wintering losses of bee colonies [25], and more generally
27 studies have shown that if the mite population is not properly controlled, the honeybee colony will die [62].
28 Recent studies also suggest that the *Varroa* mite could be a contributing cause of CCD since they not only
29 ectoparasitically feed on bees, but also vertically transmit a number of deadly viruses to the bees [37, 35].
30 There have been at least 14 viruses found in honeybee colonies [4, 37], which can differ in intensity of impact,
31 virulence, etc. for their host. For example, the Acute Bee Paralysis Virus (ABPV) affects the larvae and
32 pupae which fail to metamorphose to adult stage, while in contrast the Deformed Wing Virus (DWV) affects
33 larvae and pupae, which can still survive to the adult stage [69].
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36 Mathematical models are powerful tools that can provide insights on potential ecological processes that
37 link to CCD with other factors that contribute to colony mortality. There are several models of honey-
38 bee colony population dynamics that include the effects of *Varroa* mites (see refs. in Becher *et al.* [6]).
39 DeGrandi-Hoffman *et al.* [16] produced the first time-based honeybee colony growth model. Later, the pop-
40 ulation dynamics of *Varroa* mites were added [15]. In this model, the impact of mites on colony population
41 growth is based on reductions in adult worker longevity following parasitism during immature stages. Martin
42 [40] developed a simulation model consisting of ten components, that connected various aspects of honeybee
43 and mite biology and later included effects of viral pathogens to explain the link between the *Varroa* mite and
44 the collapse of colonies [41]. Wilkinson and Smith [74] developed a difference equation model of *Varroa* mites
45 reproducing in a honeybee colony. Their study focused on parameter estimations and sensitivity analysis.
46 Simulation models are useful but may be too complex to study mathematically and obtain general predictions.
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49 More recently, mathematical models have been formulated to explore potential mechanisms causing CCD
50 in honeybees. Eberl *et al.* [18] developed a model connecting *Varroa* mites to CCD by including brood main-
51 tenance terms. They found an important threshold for the number of hive worker bees needed to maintain
52 and care of the brood. Houry *et al.* [32, 31] developed differential equations models to study different death
53 rates of foragers and the impact it had on colony growth and development. They then linked their results
54 to CCD. Martin [41] constructed a *Varroa* population model that included mortality from virus and later
55 modeled the effects of a constant population of *Varroa* mites on the brood and on adult worker bees, and
56 found that sufficiently large mite infestations may make hives vulnerable to collapse from viral epidemics
57 [66]. Betti *et al.* [8] constructed a model that combines the dynamics of the spread of disease within a
58 bee colony with the underlying demographic dynamics of the colony to determine its ultimate fate under
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6 different scenarios. Their results suggest that the age of recruitment of hive bees to foraging duties is a good
7 early marker for the survival or collapse of a honeybee colony in the face of infection. Kribs-Zaleta *et al.* [34]
8 created a model to account for both healthy hive dynamics and hive extinction due to CCD by modeling
9 CCD via a transmissible infection brought into the hive by foragers. Perry *et al.* [50] examined the social
10 dynamics underlying dramatic colony failure. Their model includes bee foraging performance varying with
11 age, and displays dynamics of colony population collapse that are similar to field reports of CCD. These
12 models, no doubt, are insightful and provide us a better understanding on the potential mechanisms that
13 link to CCD. However, most of these models only account for the honeybee population dynamics with mites
14 or viruses but not both.

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16 The host-parasite relationship between honeybees and Varroa mites is complicated by the mite's close
17 association with a wide range of honeybee viral pathogens. In order to understand how Varroa mite infes-
18 tations and the related viruses transmitted to honeybees affect honeybee population dynamics, and which
19 may link to CCD, there is a need to develop realistic and mathematically tractable models that include both
20 virus and mite population dynamics. The goal of our work is to develop a useful honeybee-mite-virus system
21 to obtain a better understanding on the synergistic effects of honeybee-mite interactions and honeybee-virus
22 interactions on the honeybee population dynamics, thus develop good practices to control these parasites
23 to maintain or increase honeybee populations. The most relevant modeling papers for our study purposes
24 are by Sumpter and Martin [66], and Ratti *et al.* [52] whose work examined the transmission of viruses
25 via Varroa mites, using the susceptible-infectious (SI) disease modeling framework with mites as vectors for
26 transmission. However, Sumpter and Martin assumed that the mites' population is constant while Ratti *et*
27 *al.* took no account of the fact that virus transmissions occur at different biological stages of Varroa mites
28 and honeybees.

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31 In this article, we follow both approaches of Sumpter and Martin [66] and Ratti *et al.* [52], and propose a
32 honeybee-mite-virus model that incorporates (1) parasitic interactions between honeybees and Varroa mites;
33 (2) different virus transmission terms that account for the virus transmissions among honeybees, between
34 honeybees and mites at two different stages of Varroa mites; and (3) Allee effects in the honeybee population
35 generated by the internal organization of honeybees (e.g., division of labor). Our proposed model will allow
36 us explore the following questions:

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1. What are the dynamics of a system only consisting of honeybees and the virus?
 2. What are the dynamics of a system only consisting of honeybees and Varroa mites?
 3. What are the synergistic effects of Varroa mites and the virus on the honeybee population, and how
44 may these synergistic effects contribute to CCD?
 4. How can we maintain honeybee populations?
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49 The structure of the remainder of the article is organized as follows: In Section 2, we first provide
50 the biological background of honeybees, Varroa mites, and the associated virus transmission routes in the
51 honeybee-mite system; then we derive our SI-type model for honeybees co-infected with the mite and virus.
52 In Section 3, we perform local and global analysis of the proposed model and the related subsystems. The
53 results from the analysis are then connected to biological contexts and implications. Additionally, we also
54 explore numerical simulations of the subsystems and the full system to obtain the effects of each parameter
55 in our system. In Section 4, we summarize our results and the related biological implications of our studies
56 in finding potential causes of Colony Collapse Disorder. We also provide potential projects for future work.
57 The detailed mathematical proofs of our theoretical results are provided in the last section.

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2. Biological background and model derivations

Honeybee colony: During the spring and summer, a honeybee colony typically consists of a single reproductive queen, 20,000 – 60,000 adult worker bees, 10,000 – 30,000 individuals at the brood stage (egg, larvae and pupae) and up to hundreds of male drones. During the winter, the colony typically reduces in size and consists of a single queen and somewhere between 8,000 – 15,000 worker bees [41]. A large population of workers carry out the tasks of the bee colony, which include foraging, pollination, honey production and, in particular, caring for the brood and rearing the next generation of bees. The queen is the only fertile individual of the colony and has an average life span of 2 – 3 years [66]. During the peak season (in the summer), the queen lays up to 2000 eggs per day, where fertilized eggs produce female worker bees, or much more rarely queens, while drones develop from non-fertilized eggs [8]. The bees go through the following stages in development: egg (about 3 days), larvae (about 7 days), pupae (about 14 days), and adult. The life span of an adult worker bee also depends on the season. Workers usually have a lifespan of 3 – 6 weeks during the spring and summer, and are reported to live as long as 4 months during the winter [47]. The adult drone life span is typically 20 – 40 days, with reports of drone living up to 59 days under optimal colony conditions [47, 27].

Let $N_h(t)$ be the total number of honeybees in the colony, including the larvae, pupae and adult bees (both hives and foragers) at time t . The subscript h means honeybee for all future notations. Define $\xi_h \in [0, 1]$ as the **proportion** of adult honeybee population in the colony, then $(1 - \xi_h)N_h$ is the brood population and $\frac{\xi_h}{1 - \xi_h}$ is the ratio of adult honeybees to brood in colony. Empirical study shows that the successful honeybee colony should have $\frac{\xi_h}{1 - \xi_h} > 2$ (see [59]). We should expect that the value of ξ_h varies with time. In our current model, instead of employing the explicit age structure model, we let ξ_h be a parameter. **Though brood and adult numbers change throughout the year, the ratio ξ_h remains at the steady state of the explicit age structure model which serves as a limiting factor in proportion of eggs that are reared into larvae and emerge as adults.** Under this simplification, we are able to obtain analytical results on how ξ_h affects dynamics of the model with essential biological components. In the absence of mites and virus, the population dynamics of honeybees $N_h(t)$ is described by the following nonlinear equation:

$$N_h' = \frac{r(\xi_h N_h)^2}{K + (\xi_h N_h)^2} - d_h N_h \quad (1)$$

where $'$ is the sign of the derivative with respect to time; the parameter r is specified as the average number of eggs laid by the queen per day; the parameter \sqrt{K} is the size of the bee colony at which the egg laying rate is half of r ; and d_h is the average death rate of the workers. The term $\frac{(\xi_h N_h)^2}{K + (\xi_h N_h)^2}$ describes that the successful survival of an egg which will develop into a worker bee needs the care of adult workers ($\xi_h N_h$) inside the colony and also needs food brought in by foragers. This term also implicitly assumes that more adult workers inside the colony can increase the survival of an egg and the development into an adult that is supported by empirical evidence reported in [59]. Our modeling approach is similar to Eberl *et al* [18] for honeybee diseases and Kang *et al* [30] for the population dynamics of leaf-cutter ants. This assumption includes the internal organization of the colony, division of labor. Model (1) implies that the colony is able to survive when ξ_h is above a threshold, i.e., $\xi_h > \frac{2d_h\sqrt{K}}{r}$. More detailed analysis and related results are provided in the next section.

Varroa mites: Varroa mites were first reported in Kentucky in the Bluegrass region of the Commonwealth in 1991 in U.S. They have since spread to become a major pest of honeybees in many states of U.S. [7]. Varroa mites are external honeybee parasites that attack both adult honeybees and brood, with a distinct preference for drone brood [48]. They suck the blood from both adult workers and the developing brood, weakening them and shortening the life span of the bees which they feed on. Emerging brood may be born with deformed wings. Untreated infestations of Varroa mites can cause honeybee colonies to collapse [39].

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6 The mites go through a series of stages: larva, protonymph, deutonymph and then adult. Adult mites
7 (all female) undergo two phases in their life cycle, the phoretic and reproductive phases. During the phoretic
8 phase, Varroa feed on adult bees and are passed from bee to bee as they pass one another in the colony.
9 During the phoretic phase, the mites live on adult bees and can usually be found between the abdominal
10 segments of the bees. The mites puncture the soft tissue between the segments and feed on bee hemolymph,
11 harming the host [54, 9]. Mite reproduction can occur only if brood is available. A mature female mite
12 enters the brood cell about one day before capping and will be sealed in with the larva. After the capping
13 of the cell, it lays a single male egg and several female eggs at 30-hour intervals [70], and the mite feeds
14 and develops on the maturing bee larva. When the host bee leaves the cell, the mature female mites leave
15 the cell. The male mite dies after mating with his sisters, and if immature female mites are present they
16 die as they come out of the cell, as they cannot survive once outside the cell. The adult female mite begins
17 searching for other bees or larvae to parasitize.
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20 The phoretic period of the mite appears to contribute to the mite's reproductive ability, which may last
21 4.5 to 11 days when brood is present in the hive; or as long as five to six months during the winter when
22 little or no brood is present in the hive. Consequently, mites living when brood is present in the colony
23 have an average life expectancy of 27 days, yet in the absence of brood, they may live for many months.
24 In the average temperate climate, mite populations can increase 12-fold in colonies which have brood half
25 of the year and 800-fold in colonies which have brood year-round. This period usually begins in late winter
26 when brood rearing resumes from a winter period when little or no brood is present. The period of mite
27 population increase continues through the spring and summer and peaks in the fall when brood rearing is
28 nearly done. This makes the mites very difficult to control, especially in warmer climates where colonies
29 maintain brood year-round [21].
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31 Let $N_m(t)$ be the number of adult mites in the honeybee colony in the absence of a virus where the
32 subscript m means mite for all future notations. Varroa mites feed on the haemolymph of brood and adult
33 honeybees, and their reproduction depends on the availability of the brood and the population of the re-
34 productive mites. Similarly to the case of honeybees, we incorporate an implicit age structure model of
35 Varroa mites by defining a parameter $\xi_m \in [0, 1]$ as the **proportion** of mites at the phoretic stage. This
36 implies that **the reproductive mite population has size of $(1 - \xi_m)N_m$ and the phoretic mite population is**
37 **of size $\xi_m N_m$.** This simplification still allows us to investigate the parasitic interactions between mites and
38 honeybees rigorously with essential biological components.
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40 We model the parasitic interactions between mites and honeybees by using the Holling Type I functional
41 responses, i.e. $\hat{\alpha}(1 - \xi_h)N_h(1 - \xi_m)N_m = \alpha N_h N_m$ where $\hat{\alpha}$ is the parasitism rate; the term $(1 - \xi_h)N_h$ is the
42 brood population; the term $(1 - \xi_m)N_m$ is the reproductive mites population; and let $\alpha = \hat{\alpha}(1 - \xi_h)(1 - \xi_m)$.
43 Therefore, in the presence of mites N_m , the dynamics of the honeybee population N_h can be described as:
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$$45 \quad 46 \quad 47 \quad N'_h = \frac{r(\xi_h N_h)^2}{K + (\xi_h N_h)^2} - d_h N_h - \alpha N_h N_m = \frac{r(\xi_h N_h)^2}{K + (\xi_h N_h)^2} - (d_h + \alpha N_m)N_h$$

48 which implies that the parasitism decreases the life span of the honeybee, i.e., the average life span has been
49 reduced to $\frac{1}{d_h + \alpha N_m}$ after parasitism. Here we do not assume that parasitism would lead to certain death
50 of the honeybees. The Varroa mite population depends on the nutrient obtained from honeybees, thus, the
51 dynamics of mites population can be described as:
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$$53 \quad 54 \quad N'_m = c\alpha N_h N_m - d_m N_m$$

55 where the parameter α measures the parasitic rate of Varroa mites; c is the conversion rate from nutrient
56 consumption obtained from honeybees to sustenance for Varroa mites reproduction; and d_m is the natural
57 death rate of Varroa mites. Therefore, in the absence of virus, the population dynamics of mites and
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honeybees can be described by the following two nonlinear equations:

$$\begin{aligned} N'_h &= \frac{r(\xi_h N_h)^2}{K + (\xi_h N_h)^2} - d_h N_h - \alpha N_h N_m \\ N'_m &= c\alpha N_h N_m - d_m N_m \end{aligned} \quad (2)$$

The modeling approach of Model (2) has applied the traditional host-parasite modeling framework including non-lethal parasites [1, 2]. Model (2) also implies that Varroa mites population N_m goes extinct if the population of honeybees N_h goes extinct.

Varroa mites act as a virus-vector for virus transmissions: Varroa mites reproduce in cells with developing worker or drone larvae. Just before a brood cell is sealed, a female mite (i.e., foundress) will invade and parasitize the larvae thus beginning the reproductive phase (for details see Rosenkranz *et al.* [54]). The offspring (i.e., daughter mites) will mate in the cells. When the adult bee emerges, the foundress mother mite and her daughters will leave the cell. The mites will attach to adult bees (phoretic phase) until they find a new cell where they can invade and reproduce. The mites can feed on the adult bee during the phoretic phase. To model the virus transmissions between mites and honeybees during these two phases, we let $S_h(t)$, $S_m(t)$ be the susceptible population of honeybees and mites, respectively; and $I_h(t)$, $I_m(t)$ be the virus infected population of honeybees and mites, respectively. Then the total population of honeybees is $N_h(t) = S_h(t) + I_h(t)$, and the total population of mites is $N_m(t) = S_m(t) + I_m(t)$.

The virus transmissions between female mites and honeybees can occur in the following two phases of the mite life cycle:

1. The honeybee colony has $\xi_h S_h$ susceptible adult honeybees; $\xi_h I_h$ virus infected adult honeybees, $\xi_m S_m$ susceptible phoretic mites; and $\xi_m I_m$ virus infected phoretic mites. In the phoretic phase, mites move between adult bees both spontaneously and just prior to the death of bee [66]. Following the approach of [41], we assume that virus transmissions are frequency dependent, i.e.,
 - We model the rate at which the susceptible adult honeybees are virus infected by infected phoretic mites (IPFM) based on the approach of [41, 66, 18, 52]. This rate can be described as follows:

$$\begin{aligned} & \underbrace{\beta_{mh}}_{\text{probability being infected after contacts}} \times \underbrace{\xi_m I_m}_{\text{population of infected IPFM}} \times \underbrace{\frac{\xi_h S_h}{\xi_h S_h + \xi_h I_h}}_{\text{probability contacts susceptible adult honeybees}} \\ &= \frac{\beta_{mh} \xi_m S_h I_m}{S_h + I_h}. \end{aligned}$$

which also implies that susceptible honey bees become virus infected at a rate proportional to the ratio of the infected phoretic mite population to the total honeybee population.

- The rate at which susceptible phoretic mites (SPFM) are virus infected by virus infected adult honeybees (IAH) is:

$$\begin{aligned} & \underbrace{\beta_{hm}}_{\text{probability being infected after contacts}} \times \underbrace{\xi_m S_m}_{\text{population of SPFM}} \times \underbrace{\frac{\xi_h I_h}{\xi_h S_h + \xi_h I_h}}_{\text{probability being contacted by IAH}} \\ &= \frac{\beta_{hm} \xi_m S_m I_h}{S_h + I_h}. \end{aligned}$$

2. The honeybee colony has $(1 - \xi_h) S_h$ susceptible brood; $(1 - \xi_h) I_h$ virus infected brood, $(1 - \xi_m) S_m$ susceptible reproductive mites; and $(1 - \xi_m) I_m$ virus infected reproductive mites. We do not assume that brood will die from parasitism, thus, parasitised brood or daughter mites will face virus infection

if either brood or the mother mite is virus infected. Chen *et al.* found a direct relationship between virus frequency and the number of mites to which brood were exposed, i.e., the more donor mites that were introduced per cell, the greater the incidence of virus that was detected in brood [11, 12]. This implies that the virus transmission rate between mites and brood during the reproductive phase of mites is density dependent, i.e., similar to the term that describes the parasitic interaction between mites and honeybee. Therefore, we have follows:

- A developing bee becomes virus-infected if it is parasitized by an infected foundress mite. Thus, the rate at which the susceptible brood is infected by virus infected reproductive mites (IRFM), i.e., infected foundress mites, is:

$$\underbrace{\beta_{mh2}}_{\text{probability being infected after contacts}} \times \underbrace{(1 - \xi_h)S_h}_{\text{population of healthy honeybee brood}} \times \underbrace{\hat{\alpha}(1 - \xi_m)I_m}_{\text{parasitism by IRFM}}$$

$$= \beta_{mh2}\alpha S_h I_m.$$

- The reproduction of mites depends on brood. Daughter mites can become virus infected if either brood or their mother is virus infected. Thus, based on the formulation of the host-parasite interaction model (2), the rate at which daughter mites (INFM) become virus infected depends on the parasitic interaction between mites and honeybees described as $c\alpha [I_h(S_m + I_m) + S_h I_m]$ where the term $I_h(S_m + I_m)$ is the daughter mites infected with virus infected brood; and the term $S_h I_m$ is the daughter mites infected with virus via their mothers.

The virus transmission among honeybees: The proportion of honeybees which can infect themselves is also dependent on the total number of susceptible and virus infected bees present in the colony, and hence frequency-dependent transmission is used [41], which is described as follows:

$$\underbrace{\beta_h}_{\text{probability being infected after contacts}} \times \underbrace{S_h}_{\text{the healthy honeybee population}}$$

$$\times \underbrace{\frac{I_h}{S_h + I_h}}_{\text{probability of contacting or being contacted by infected honeybees}} = \frac{\beta_h S_h I_h}{S_h + I_h}.$$

The reduced fitness of honeybees due to virus infections: The virus infections contribute to morphological deformities of honeybees such as small body size, shortened abdomen and deformed wings, which reduce vigor and longevity, and they can also influence flight duration and the homing ability of foragers [35]. To include the effects of virus infections in our model, we assume that the virus infected adult honeybee population $\xi_h I_h$ affects brood rearing rates and colony population growth with a reduced fitness measured by $\rho \in (0, 1)$. The healthy honeybee population S_h (i.e., un-infected honeybees) can be modeled as follows,

$$S'_h = \underbrace{\frac{r\xi_h^2 (S_h + \rho I_h)^2}{K + \xi_h^2 (S_h + \rho I_h)^2}}_{\text{reproduction of honeybees}} - \underbrace{\frac{\beta_h S_h I_h}{S_h + I_h}}_{\text{honeybee infected by nestmates}} - \underbrace{\alpha S_h (S_m + I_m)}_{\text{parasitism by mites}}$$

$$- \underbrace{\frac{\beta_{mh}(\xi_m S_h) I_m}{S_h + I_h}}_{\text{adult honeybees infected by the phoretic mites}} - \underbrace{\beta_{mh2}\alpha S_h I_m}_{\text{brood infected by the reproductive mites}} - d_h S_h \quad (3)$$

And the virus infected honeybee population can be modeled by the following equation,

$$\begin{aligned}
I'_h = & S_h \left[\frac{\beta_h I_h}{S_h + I_h} + \frac{\beta_{mh} \xi_m I_m}{S_h + I_h} + \beta_{mh2} \alpha I_m \right] - \underbrace{\alpha I_h (S_m + I_m)}_{\text{Consumed by mites}} \\
& - \left(\underbrace{d_h}_{\text{natural honeybee mortality rate}} + \underbrace{\mu_h}_{\text{additional death due to virus infections}} \right) I_h .
\end{aligned} \tag{4}$$

Let μ_m be the additional death rate of mites due to virus infections. In the case that virus infections do not cause additional mortality of mites, the value of μ_m is zero. **The population** of healthy mites S_m and the virus infected mites I_m can be described by the following set of nonlinear equations:

$$\begin{aligned}
S'_m &= S_m \left[c\alpha S_h - \underbrace{\frac{\beta_{hm} \xi_m I_h}{S_h + I_h}}_{\text{the phoretic mites infected by adult honeybees}} - \underbrace{d_m}_{\text{natural mortality rate of mites}} \right] \\
I'_m &= c\alpha \left[\underbrace{I_h (S_m + I_m) + S_h I_m}_{\text{mites born with virus infections}} + \frac{\beta_{hm} I_h (\xi_m S_m)}{S_h + I_h} - (d_m + \underbrace{\mu_m}_{\text{additional death due to virus infections}}) I_m \right] .
\end{aligned} \tag{5}$$

Based on the above, the full model of honeybee-mites-virus population dynamics **is modeled by** the following system of differential equations:

$$\begin{aligned}
S'_h &= \frac{r\xi_h^2 (S_h + \rho I_h)^2}{K + \xi_h^2 (S_h + \rho I_h)^2} - d_h S_h - \frac{\beta_h S_h I_h}{S_h + I_h} - \frac{\beta_{mh} (\xi_m S_h) I_m}{S_h + I_h} \\
&\quad - \beta_{mh2} \alpha I_m - \alpha S_h (S_m + I_m) \\
I'_h &= S_h \left[\frac{\beta_h I_h}{S_h + I_h} + \frac{\beta_{mh} \xi_m I_m}{S_h + I_h} + \beta_{mh2} \alpha I_m \right] - \alpha I_h (S_m + I_m) - (d_h + \mu_h) I_h . \\
S'_m &= S_m \left[c\alpha S_h - \frac{\beta_{hm} \xi_m I_h}{S_h + I_h} - d_m \right] \\
I'_m &= c\alpha [I_h (S_m + I_m) + S_h I_m] + \frac{\beta_{hm} I_h (\xi_m S_m)}{S_h + I_h} - (d_m + \mu_m) I_m
\end{aligned} \tag{6}$$

For convenience, let $\hat{K} = \frac{K}{\xi_h^2}$, $\hat{\beta}_{mh} = \beta_{mh} \xi_m$, $\tilde{\beta}_{mh} = \beta_{mh2} \alpha$, $\hat{\beta}_{hm} = \beta_{hm} \xi_m$. Then the full model (6) can be rewritten as the following model

$$\begin{aligned}
S'_h &= \frac{r(S_h + \rho I_h)^2}{\hat{K} + (S_h + \rho I_h)^2} - d_h S_h - \frac{\beta_h S_h I_h}{S_h + I_h} - \frac{\hat{\beta}_{mh} S_h I_m}{S_h + I_h} - \tilde{\beta}_{mh} S_h I_m - \alpha S_h (S_m + I_m) \\
I'_h &= S_h \left[\frac{\beta_h I_h}{S_h + I_h} + \frac{\hat{\beta}_{mh} I_m}{S_h + I_h} + \tilde{\beta}_{mh} I_m \right] - \alpha I_h (S_m + I_m) - (d_h + \mu_h) I_h \\
S'_m &= S_m \left[c\alpha S_h - \frac{\hat{\beta}_{hm} I_h}{S_h + I_h} - d_m \right] \\
I'_m &= c\alpha [I_h (S_m + I_m) + S_h I_m] + \frac{\hat{\beta}_{hm} I_h S_m}{S_h + I_h} - (d_m + \mu_m) I_m
\end{aligned} \tag{7}$$

where $\alpha > 0, c > 0, \rho \in [0, 1]$ and the virus transmission rates $\beta_h, \hat{\beta}_{mh}, \hat{\beta}_{hm}, \tilde{\beta}_{mh} \in (0, 1)$. In summary, the full honeybee-mite-virus model (7) incorporates (1) Allee effects of honeybees due to the cooperation of the internal organization; (2) parasitism interactions between honeybee and mites; (3) the vertical **virus** transmission mode modeled by the frequency-dependent **virus** transmission function during the mites' phoretic phase; (4) the horizontal **virus** transmission mode modeled by the density-dependent **virus** transmission func-

tion during the mites' reproductive phase; and (5) the reduced fitness of honeybees due to virus infections. Model (7) allows us to investigate the following scenarios:

1. Population growth of colonies in the absence of mites and virus.
2. In the absence of the virus, how the Varroa mites may affect the population dynamics of colonies.
3. In the absence of the mites, how virus infections may affect the population dynamics of colonies. This case can apply to situations where honeybees are infected by virus through ecological processes such as foraging.
4. In the presence of both the mites and virus infections, which conditions can lead to the extinction of mites, virus infections, and colony population; and which conditions can guarantee colony survival.

The remainder of this manuscript will focus on the dynamics of Model (7) and the related subsystems.

3. Mathematical analysis

Let $\frac{S_h I_h}{S_h + I_h} \Big|_{S_h = I_h = 0} = 0$ and $\frac{I_m}{S_m + I_m} \Big|_{S_m = I_m = 0} = 0$. Define $X = \{(S_h, I_h, S_m, I_m) \in \mathbb{R}_+^4 : S_h + I_h > 0 \text{ and } S_m + I_m > 0\}$, then X can be considered as the state space of our model (7). To continue the analysis, let us define $N_h = S_h + I_h$, $N_m = S_m + I_m$ and $N = cN_h + N_m$ as the population of honeybees, the population of mites, and the sum of the population of honeybees and mites, respectively. In addition, we let $d = \min\{d_h, d_m\}$, and define N^* as the upper bound of the sum of the population of honeybees and mites and N^c as the corresponding threshold, where

$$N^c = c \frac{\frac{r}{d} - \sqrt{\left(\frac{r}{d}\right)^2 - 4\hat{K}}}{2}, \quad N^* = c \frac{\frac{r}{d} + \sqrt{\left(\frac{r}{d}\right)^2 - 4\hat{K}}}{2}.$$

We let \bar{N}_h^* , \underline{N}_h^* be the upper bound, lower bound of the population of honeybees, respectively, and \bar{N}_h^c , \underline{N}_h^c be the corresponding thresholds, where

$$\bar{N}_h^c = \frac{\frac{r}{d_h} - \sqrt{\left(\frac{r}{d_h}\right)^2 - 4\hat{K}}}{2}, \quad \bar{N}_h^* = \frac{\frac{r}{d_h} + \sqrt{\left(\frac{r}{d_h}\right)^2 - 4\hat{K}}}{2}$$

$$\underline{N}_h^c = \frac{\frac{r}{d_h + \mu_h + \alpha N^*} - \sqrt{\left(\frac{r}{d_h + \mu_h + \alpha N^*}\right)^2 - 4\hat{K}/\rho^2}}{2}, \quad \underline{N}_h^* = \frac{\frac{r}{d_h + \mu_h + \alpha N^*} + \sqrt{\left(\frac{r}{d_h + \mu_h + \alpha N^*}\right)^2 - 4\hat{K}/\rho^2}}{2}$$

And we let S_h^* be the lower bound of the population of susceptible honeybees, and S_h^c be the corresponding threshold, where

$$S_h^c = \frac{\frac{r}{d_h + \beta_h + \frac{\beta_m h N^*}{N_h^*} + (\beta_m h + \alpha)(N^* - cN_h^*)} - \sqrt{\left(\frac{r}{d_h + \beta_h + \frac{\beta_m h N^*}{N_h^*} + (\beta_m h + \alpha)(N^* - cN_h^*)}\right)^2 - 4\hat{K}}}{2},$$

$$S_h^* = \frac{\frac{r}{d_h + \beta_h + \frac{\beta_m h N^*}{N_h^*} + (\beta_m h + \alpha)(N^* - cN_h^*)} + \sqrt{\left(\frac{r}{d_h + \beta_h + \frac{\beta_m h N^*}{N_h^*} + (\beta_m h + \alpha)(N^* - cN_h^*)}\right)^2 - 4\hat{K}}}{2}$$

Define $f^b(x, y) = \frac{\frac{r}{x} + \sqrt{\left(\frac{r}{x}\right)^2 - 4\hat{K}/y}}{2}$ and $f_b(x, y) = \frac{\frac{r}{x} - \sqrt{\left(\frac{r}{x}\right)^2 - 4\hat{K}/y}}{2}$, then we have

$$\frac{\partial f^b(x, y)}{\partial x} < 0, \quad \frac{\partial f^b(x, y)}{\partial y} < 0, \quad \frac{\partial f_b(x, y)}{\partial x} > 0, \quad \frac{\partial f_b(x, y)}{\partial y} > 0$$

which imply the following inequalities

$$S_h^c < S_h^c < S_h^* < \bar{N}_h^*, \quad N^c \leq \bar{N}_h^c < \underline{N}_h^c < \underline{N}_h^* < \bar{N}_h^* \leq N^*/c.$$

Theorem 3.1 (Basic dynamical properties). *Assume that all parameters are strictly positive and $\rho \in [0, 1]$. The model (7) is positively invariant and bounded in the state space X , which is attracted to the following compact set*

$$C = \{(S_h, I_h, S_m, I_m) \in \mathbb{R}_+^4 : 0 \leq c(S_h + I_h) + (S_m + I_m) = cN_h + N_m \leq N^*\}$$

provided that $\frac{r}{d} > 2\sqrt{\hat{K}}$ and time is large enough. Moreover, the following statements hold for Model (7):

- If $\frac{r}{2\sqrt{\hat{K}}} > d_h$, then the total population of honeybees N_h is bounded by \bar{N}_h^* , i.e.,

$$\limsup_{t \rightarrow \infty} N_h(t) \leq \bar{N}_h^*.$$

If $\frac{r}{2\sqrt{\hat{K}}} > \frac{d_h + \mu_h + \alpha N^*}{\rho}$ and $N_h(0) > \underline{N}_h^c$ hold, then the total population of honeybees N_h is persistent, i.e.,

$$\underline{N}_h^* \leq \liminf_{t \rightarrow \infty} N_h(t) \leq \limsup_{t \rightarrow \infty} N_h(t) \leq \bar{N}_h^* \leq \limsup_{t \rightarrow \infty} N(t)/c = N^*/c.$$

- If the inequalities $\frac{r}{2\sqrt{\hat{K}}} > \max \left\{ d_h + \beta_h + \frac{\hat{\beta}_{mh} N^*}{\bar{N}_h^*} + (\tilde{\beta}_{mh} + \alpha)(N^* - c\underline{N}_h^*), \frac{d_h + \mu_h + \alpha N^*}{\rho} \right\}$ with $N_h(0) \geq S_h(0) > S_h^c$ hold, then S_h is persistent with the following properties:

$$S_h^* \leq \liminf_{t \rightarrow \infty} S_h(t) \leq \liminf_{t \rightarrow \infty} N_h(t) \leq \liminf_{t \rightarrow \infty} N(t)/c \leq \limsup_{t \rightarrow \infty} N(t)/c \leq N^*/c.$$

- The extinction equilibrium $E_0 = (0, 0, 0, 0)$ is always local stable. Moreover, the system (7) converges to E_0 globally if $d_h > \frac{r}{2\sqrt{\hat{K}}}$ holds; and it converges to E_0 locally if the initial population satisfies either $N(0) < N^c$ or $N_h(0) < \bar{N}_h^c$.

Notes: The positive invariance and boundedness results from Theorem 3.1 imply that our model is well-defined biologically. In addition, the results of Theorem 3.1 indicate follows:

1. Initial conditions are important determinants of colony survival.
2. The inequality $\frac{r}{2\sqrt{\hat{K}}} > d_h$ is a necessary condition for colony survival, i.e., the large intrinsic growth rate r , the small half saturation \hat{K} , and the small death rate of honeybees d_h .
3. Low rates of virus transmission and parasitism $\beta_h, \hat{\beta}_{mh}, \tilde{\beta}_{mh}, \alpha$ are also important for the persistence of the healthy honeybee population (i.e., uninfected) S_h .

Recall that $d = \min\{d_h, d_m\}$, $N^c = c \frac{\frac{r}{d} - \sqrt{(\frac{r}{d})^2 - 4\hat{K}}}{2}$, $N^* = c \frac{\frac{r}{d} + \sqrt{(\frac{r}{d})^2 - 4\hat{K}}}{2}$ and

$$\underline{N}_h^* = \frac{\frac{r}{d_h + \mu_h + \alpha N^*} + \sqrt{\left(\frac{r}{d_h + \mu_h + \alpha N^*}\right)^2 - 4\hat{K}/\rho^2}}{2}.$$

Theorem 3.1 implies that under proper initial conditions, colonies will survive if $\frac{r}{2\sqrt{K}} > \frac{d_h + \mu_h + \alpha N^*}{\rho}$ and $N_h(0) > \frac{N_h^c}{\xi_h}$ holds. Notice that ξ_h is the ratio of adult honeybees in the colony, and one of sufficient conditions that guarantee colony survival is the following inequality:

$$\frac{r}{2\sqrt{K}} = \frac{r\xi_h}{2\sqrt{K}} > \frac{d_h + \mu_h + \alpha N^*}{\rho} \Leftrightarrow \frac{r\xi_h}{2\sqrt{K}} - \frac{\alpha\sqrt{\left(\frac{r}{d}\right)^2 - 4\frac{K}{\xi_h^2}}}{2\rho} > \frac{d_h + \mu_h + \alpha\frac{r}{2d}}{\rho}.$$

Thus, Theorem 3.1 provides a critical function of the hives population ξ_h such that colony will survive. In addition, notice that $\frac{r\xi_h}{2\sqrt{K}} - \frac{\alpha\sqrt{\left(\frac{r}{d}\right)^2 - 4\frac{K}{\xi_h^2}}}{2\rho}$ is an increasing function of ξ_h , this implies that the larger adult workers to brood ratio ξ_h , the better colony growth and survival. This is supported by empirical evidence reported in [59]. In the following theorem, we provide theoretical results on the sufficient conditions that lead to the persistence and the extinction of **virus** population or the mites population.

Theorem 3.2 (Persistence and extinction of **virus** or mites). *The following statements hold*

- If $N^* < \frac{d_m}{\alpha}$, then the total population of mite N_m goes extinct, i.e.,

$$\limsup_{t \rightarrow \infty} N_m(t) = 0$$

where system (7) is attracted to the mite-free invariant set $MF = \{(S_h, I_h, S_m, I_m) \in \mathbb{R}_+^4 : S_m + I_m = 0\}$, and its dynamics is equivalent to the following two-D model (8)

$$\begin{aligned} S'_h &= \frac{r(S_h + \rho I_h)^2}{K + (S_h + \rho I_h)^2} - d_h S_h - S_h \frac{\beta_h I_h}{S_h + I_h} \\ I'_h &= S_h \frac{\beta_h I_h}{S_h + I_h} - (d_h + \mu_h) I_h \end{aligned} \quad (8)$$

- If $\frac{r}{2\sqrt{K}} > d_h$, $\bar{N}_h^* < \frac{d_m}{\alpha c}$, and $S_h(0) > S_h^c$, then the total population of honeybees persists while the healthy mite population S_m goes extinct, i.e.,

$$\limsup_{t \rightarrow \infty} S_m(t) = 0$$

where the system (7) is attracted to the healthy-mite-free invariant set $HMF = \{(S_h, I_h, S_m, I_m) \in \mathbb{R}_+^4 : S_m = 0\}$ and its dynamics is equivalent to the following three-D system (9):

$$\begin{aligned} S'_h &= \frac{r(S_h + \rho I_h)^2}{K + (S_h + \rho I_h)^2} - d_h S_h - \frac{\beta_h S_h I_h}{S_h + I_h} - \frac{\hat{\beta}_{mh} S_h I_m}{S_h + I_h} - \tilde{\beta}_{mh} S_h I_m - \alpha S_h I_m \\ I'_h &= S_h \left[\frac{\beta_h I_h}{S_h + I_h} + \frac{\hat{\beta}_{mh} I_m}{S_h + I_h} + \tilde{\beta}_{mh} I_m \right] - \alpha I_h I_m - (d_h + \mu_h) I_h \\ I'_m &= c\alpha I_m \left[I_h + S_h - \frac{d_m + \mu_m}{c\alpha} \right]. \end{aligned} \quad (9)$$

- Assume that $\frac{r}{2\sqrt{K}} > d_h + \beta_h + \frac{\hat{\beta}_{mh} N^*}{N_h^*} + (\tilde{\beta}_{mh} + \alpha)(N^* - \frac{N_h^*}{\xi_h})$ and $S_h(0) > S_h^c$. Then the **virus**

$I = cI_h + I_m$ persists if the inequality $\frac{\min\left\{\beta_h, c\hat{\beta}_{mh} + c\tilde{\beta}_{mh} + c\alpha S_h^*\right\}}{\max\left\{(d_h + \mu_h), (d_m + \mu_m)\right\}} \geq 1$ holds.

- Assume that $\frac{r}{2\sqrt{K}} > \frac{d_h + \mu_h + \alpha N^*}{\rho}$ and $N_h(0) > \frac{N_h^c}{\xi_h}$. Then the **virus** $I = cI_h + I_m$ goes extinct if the

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6 following inequality holds $\frac{\max \left\{ \beta_h + \frac{\hat{\beta}_{hm} N^*}{N_h}, c\hat{\beta}_{mh} + c\tilde{\beta}_{mh} + c\alpha\tilde{N}_h^* \right\}}{\min \left\{ (d_h + \mu_h), (d_m + \mu_m) \right\}} < 1$. Under this condition, the system (7)

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8 is attracted to the virus-free invariant set $DF = \{(S_h, I_h, S_m, I_m) \in \mathbb{R}_+^4 : I_h + I_m = 0\}$ and its dynamics is equivalent to the following two-D model (10)

$$\begin{aligned} S'_h &= \frac{rS_h^2}{K+S_h^2} - d_h S_h - \alpha S_h S_m \\ S'_m &= c\alpha S_h S_m - d_m S_m \end{aligned} \quad (10)$$

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17 **Notes:** The results of the reduced dynamics in Theorem 3.2 can be easily obtained by the theory of asymptotically autonomous systems [10]. The detailed proof of our results are provided in the last section.

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19 According to Theorem 3.2, the condition $N^* = c \frac{\frac{r}{d_h} + \sqrt{\left(\frac{r}{d_h}\right)^2 - 4\tilde{K}}}{2} < \frac{d_m}{\alpha}$ can lead to the extinction of the entire mite population. Therefore, we can conclude that large values for the death rate of mites, d_m , can lead to the extinction of the colony; and large values of the death rate of mites d_m , low rates of parasitism α , and its energy conversion rate c , can lead to either the extinction of the entire mite population N_m or the extinction of the healthy mite population S_m . Here we would like to point out that it is possible to have the persistence of virus infected mites while the healthy mite goes extinct (see the resulting dynamics (9) when the healthy mite goes extinct). In addition, the results of Theorem 3.2 also suggest that: **1.** the persistence of the virus requires a large value for the **virus** transmission rate between adult honeybees, β_h , or the **virus** transmission rate between brood and the reproductive mites, $\tilde{\beta}_{mh}$; or the small values of the total death rates of honeybees, $d_h + \mu_h$, and mites, $d_m + \mu_m$; **2.** the extinction of the virus requires the small values of all **virus** transmission rates, i.e., the small values of $\beta_h, \hat{\beta}_{mh}, \tilde{\beta}_{mh}, \hat{\beta}_{hm}$; or the large values of the total death rates of honeybees and mites.

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34 Theorem 3.2 provides sufficient conditions that the full system (7) reduces to the virus-free subsystem (10), the mite-free subsystem (8), and the healthy-mite-free subsystem (9). In the following three subsections, we explore the global dynamics of these subsystems.

3.1. Dynamics of the virus-free subsystem: only parasitism by mites

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40 Theorem 3.2 in previous section suggests that either low virus transmission rates or large values of the total death rates of honeybees and mites can lead to the extinction of the virus infected honeybees and mites, which gives the following virus-free dynamics (10):

$$\begin{aligned} S'_h &= \frac{rS_h^2}{K+S_h^2} - d_h S_h - \alpha S_h S_m \\ S'_m &= c\alpha S_h S_m - d_m S_m \end{aligned}$$

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49 The dynamics of the virus-free **system** (10) (i.e., the dynamics of the parasitism interactions between honeybees and mites) can be summarized by the following theorem:

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53 **Theorem 3.3** (Dynamics of the virus-free subsystem). Let $H^* = \frac{d_m}{\alpha c}$, $M^* = \frac{1}{\alpha} \left[\frac{rH^*}{K+(H^*)^2} - d_h \right]$.

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57 The virus-free subsystem (10) can have one, three, or four equilibria. The existence and stability conditions for these equilibria are listed in Table 1. The global dynamics of the virus-free subsystem (10) can be summarized as follows:

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65 1. The system (10) converges to extinction (0, 0) for almost all initial conditions if $\frac{r}{2\sqrt{K}} < d_h$ or $\frac{d_m}{\alpha c} < \tilde{N}_h^c$.

Equilibria	Existence Condition	Stability Condition
$(0, 0)$	Always exists	Always locally stable
$(\bar{N}_h^c, 0)$	$\frac{r}{2\sqrt{\hat{K}}} > d_h$	Saddle if $\bar{N}_h^c < \frac{d_m}{\alpha c} = H^*$; Source if $\bar{N}_h^c > \frac{d_m}{\alpha c} = H^*$
$(\bar{N}_h^*, 0)$	$\frac{r}{2\sqrt{\hat{K}}} > d_h$	Sink if $\bar{N}_h^* < \frac{d_m}{\alpha c} = H^*$; Saddle if $\bar{N}_h^* > \frac{d_m}{\alpha c} = H^*$
(H^*, M^*)	$\bar{N}_h^c < \frac{d_m}{\alpha c} = H^* < \bar{N}_h^*$	Sink if $H^* > \sqrt{\hat{K}}$; Source if $H^* < \sqrt{\hat{K}}$.

Table 1: The existence and stability of equilibrium for the virus-free subsystem (10), where $\bar{N}_h^c = \frac{r}{d_h} - \sqrt{\left(\frac{r}{d_h}\right)^2 - 4\hat{K}}$, $\bar{N}_h^* = \frac{r}{d_h} + \sqrt{\left(\frac{r}{d_h}\right)^2 - 4\hat{K}}$ and $H^* = \frac{d_m}{\alpha c}$, $M^* = \frac{1}{\alpha} \left[\frac{rH^*}{\hat{K} + (H^*)^2} - d_h \right]$.

2. If $\bar{N}_h^* < \frac{d_m}{\alpha c}$, depending on initial condition, the trajectory of (10) converges to either $(0, 0)$ or $(\bar{N}_h^*, 0)$.
3. If $\bar{N}_h^c < \frac{d_m}{\alpha c} < \bar{N}_h^*$ then Model (10) has a unique interior equilibrium (H^*, M^*) which is locally asymptotically stable when $\frac{d_m}{\alpha c} > \sqrt{\hat{K}}$ and is a source when $\frac{d_m}{\alpha c} < \sqrt{\hat{K}}$.

Notes: Theorem 3.3 provides us a global picture on the dynamics of the virus-free subsystem (10), i.e., the colony virus infected with only mites but not the virus. By applying the results in [67, 71], we can conclude that the virus-free subsystem (10) undergoes a subcritical Hopf-bifurcation at $\frac{d_m}{\alpha c} = \sqrt{\hat{K}}$. The subsystem (10) has a unique **unstable** limit cycle around (H^*, M^*) whenever $\frac{d_m}{\alpha c} < \sqrt{\hat{K}}$. In this case, the periodic orbits expand until it touches the stable manifold of the boundary equilibrium $(\bar{N}_h^c, 0)$ which leads to the extinction of both honeybees and the parasitic mites. We refer to this phenomena as a *catastrophic event* which is similar to CCD. Our theoretical results also suggest that a small death rate for mites and a large parasitism rate can destabilize the system.

Linking to CCD: To illustrate the *catastrophic event*, we use reasonable parameters from [66, 52]. Let queen's daily egg laying rate in summer be $r = 1500$; and the population size of the colony at which queen's daily egg laying rate is half of r be $\sqrt{\hat{K}} = 2000$; the natural death rate of honeybees is $d_h = 0.01$; the parasitism rate is $\alpha = 0.005$; the energy conversion rate is $c = 0.01$; and the natural death rate of mites is $d_m = 0.1$. This set of parameter values gives $\frac{d_m}{\alpha c} < \sqrt{\hat{K}}$ which implies that a *catastrophic event* will occur (see Figure 1; the population of honeybees is in red and collapses around time=200 day).

Stochastic effects and oscillations: Theorem 3.3 implies that if the inequality $\frac{d_m}{\alpha c} < \sqrt{\hat{K}}$ holds, then the virus-free subsystem (10) has a unique **unstable** limit cycle around (H^*, M^*) where for all initial conditions either the system converges to $(0, 0)$ quickly or the system experiences the expanding oscillations leading to eventual extinction. The oscillating extinction in the later type is driven by the deterministic dynamics. The extinction fate of the system cannot be prevented by introducing stochastic effects, however, introduced stochastic effects may cause extinction more quickly without expanding oscillations.

Note that $\hat{K} = \frac{K}{\xi_h^2}$ and $\alpha = \hat{\alpha}(1 - \xi_h)(1 - \xi_m)$ where ξ_h, ξ_m are ratio of adult bees and ratio of phoretic mites in the colony, respectively. The *catastrophic event* occurs when

$$\frac{d_m}{\alpha c} < \sqrt{\hat{K}} \Leftrightarrow \frac{d_m}{\hat{\alpha}(1 - \xi_m)c\sqrt{K}} < \frac{(1 - \xi_h)}{\xi_h} \Leftrightarrow \frac{\xi_h}{(1 - \xi_h)} < \frac{\hat{\alpha}(1 - \xi_m)c\sqrt{K}}{d_m}.$$

This inequality provides a critical low hive to brood ratio that can destabilize the system and cause colony sudden extinction.

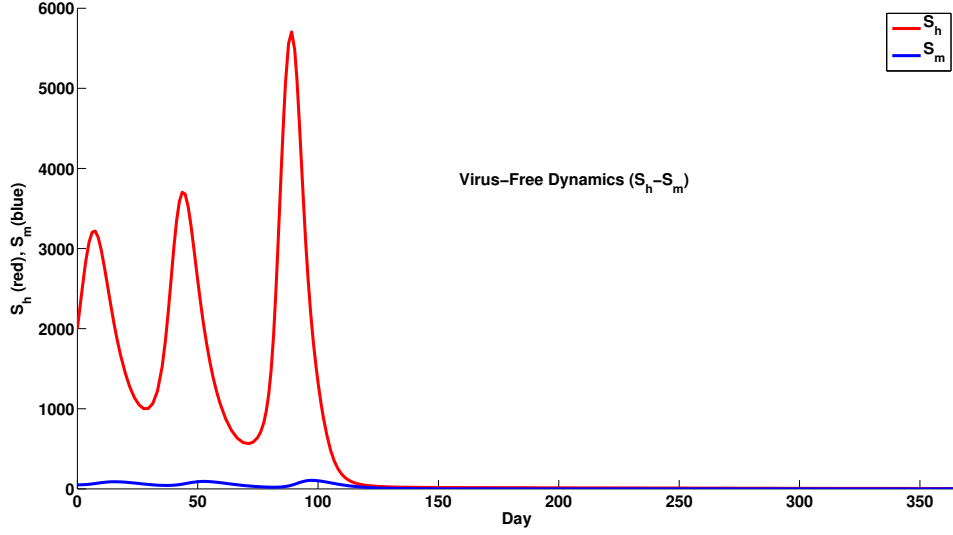


Figure 1: Time series (in days) of Model (10) when $r = 1500$, $\alpha = 0.005$, $c = 0.01$, $d_h = 0.01$, $d_m = 0.1$: population of honeybees is in red while Varroa mites is in blue.

3.2. Dynamics of the mite-free subsystem: only virus infections

According to Theorem 3.2, if the honeybee population is too small, e.g., $N^* < \frac{d_m}{\alpha c}$, then the dynamics of (7) is equivalent to the following mite-free dynamics (8)

$$\begin{aligned} S'_h &= \frac{r(S_h + \rho I_h)^2}{K + (S_h + \rho I_h)^2} - d_h S_h - S_h \frac{\beta_h I_h}{S_h + I_h} \\ I'_h &= S_h \frac{\beta_h I_h}{S_h + I_h} - (d_h + \mu_h) I_h \end{aligned}$$

To continue studying the dynamics of the mite-free system (8), we define $a = \frac{1}{\frac{\beta_h}{d_h + \mu_h} - 1}$ as the ratio of the susceptible honeybee population to the virus infected honeybee population; $\mathcal{R}_0^V = \frac{\beta_h}{d_h + \mu_h}$ as the basic reproduction number, i.e., the number of secondary cases which one case would produce in a completely susceptible population; $\tilde{d} = (a + 1)d_h + \mu_h = d_h \left(\frac{\mathcal{R}_0^V}{\mathcal{R}_0^V - 1} \right) + \mu_h$ as the updated average death of the honeybee due to virus infections. In addition, we let $S_h^k = a I_h^k$, $k = 1, 2$ and

$$I_h^1 = \frac{\frac{r}{d} - \sqrt{\left(\frac{r}{d}\right)^2 - 4 \frac{\tilde{K}}{(a+\rho)^2}}}{2}, \quad I_h^2 = \frac{\frac{r}{d} + \sqrt{\left(\frac{r}{d}\right)^2 - 4 \frac{\tilde{K}}{(a+\rho)^2}}}{2}.$$

The dynamics of the mite-free system (8) can be summarized by the following theorem:

Theorem 3.4 (Dynamics of the mite-free subsystem). *The mite-free subsystem (8) can have one, three, or five equilibria. The existence and stability conditions for these equilibria are listed in Table 2. In addition, the global dynamics of the mite-free subsystem (8) can be summarized as follows:*

1. The trajectory of (8) converges to extinction $(0, 0)$ for all initial conditions in \mathbb{R}_+^2 if one of the following conditions hold:

- $\frac{r}{2\sqrt{\tilde{K}}} < d_h$, or

Equilibria	Existence Condition for Existence	Stability Condition
$(0, 0)$	Always exists	Always locally stable
$(\bar{N}_h^c, 0)$	$\frac{r}{2\sqrt{K}} > d_h$	Saddle if $\mathcal{R}_0^V < 1$; Source if $\mathcal{R}_0^V > 1$
$(\bar{N}_h^*, 0)$	$\frac{r}{2\sqrt{K}} > d_h$	Sink if $\mathcal{R}_0^V < 1$; Saddle if $\mathcal{R}_0^V > 1$
(S_h^1, I_h^1)	$\mathcal{R}_0^V > 1$ and $\frac{r}{2\sqrt{K}} > \frac{d}{a+\rho}$	Always a saddle.
(S_h^2, I_h^2)	$\mathcal{R}_0^V > 1$ and $\frac{r}{2\sqrt{K}} > \frac{d}{a+\rho}$	Always locally asymptotically stable

Table 2: The existence and stability of equilibrium for the mite-free subsystem (8). We have $\mathcal{R}_0^V = \frac{\beta_h}{d_h + \mu_h}$, $a = \frac{1}{\mathcal{R}_0^V - 1}$, $\tilde{d} = (a+1)d_h + \mu_h = d_h \left(\frac{\mathcal{R}_0^V}{\mathcal{R}_0^V - 1} \right) + \mu_h$, $\bar{N}_h^c = \frac{\frac{r}{d_h} - \sqrt{\left(\frac{r}{d_h}\right)^2 - 4\tilde{K}}}{2}$, $\bar{N}_h^* = \frac{\frac{r}{d_h} + \sqrt{\left(\frac{r}{d_h}\right)^2 - 4\tilde{K}}}{2}$, and $I_h^1 = \frac{\frac{r}{d} - \sqrt{\left(\frac{r}{d}\right)^2 - 4\frac{\tilde{K}}{(a+\rho)^2}}}{2}$, $I_h^2 = \frac{\frac{r}{d} + \sqrt{\left(\frac{r}{d}\right)^2 - 4\frac{\tilde{K}}{(a+\rho)^2}}}{2}$, $S_h^k = aI_h^k, k = 1, 2$.

- $\mathcal{R}_0^V > 1$ and $d_h < \frac{r}{2\sqrt{K}} < \frac{\tilde{d}}{a+\rho}$.

2. The trajectory of (8) converges to either $(0, 0)$ or $(\bar{N}_h^*, 0)$ for almost all initial conditions in \mathbb{R}_+^2 if the inequalities $\mathcal{R}_0^V < 1$ and $\frac{r}{2\sqrt{K}} > d_h$.

3. The trajectory of (8) converges to either $(0, 0)$ or (S_h^2, I_h^2) for almost all initial conditions in \mathbb{R}_+^2 if the inequalities $\mathcal{R}_0^V > 1$, $\frac{r}{2\sqrt{K}} > \max\left\{d_h, \frac{\tilde{d}}{a+\rho}\right\}$ hold.

Notes: Theorem 3.4 implies that the mite-free subsystem (8) has relatively simple dynamics, i.e., no limit cycle. The results show the following interesting findings:

1. Honeybees can persist with proper initial conditions if the virus transmission rate among honeybees β_h is not large, i.e., $\mathcal{R}_0^V < 1$.
2. Both honeybees and the virus can coexist if β_h is in the medium range, i.e. $\mathcal{R}_0^V > 1$ and $\frac{\tilde{d}}{a+\rho} < \frac{r}{2\sqrt{K}}$
3. However, the large virus transmission rate among honeybees β_h can cause colony extinction. This occurs when the inequalities $\mathcal{R}_0^V > 1$ and $d_h < \frac{r}{2\sqrt{K}} < \frac{\tilde{d}}{a+\rho}$ hold.

3.3. Dynamics of the healthy-mite-free subsystem

According to Theorem 3.2, if $\frac{r}{2\sqrt{K}} > d_h$, $\bar{N}_h^* < \frac{d_m}{\alpha c}$, and $S_h(0) > S_h^c$, then the total population of honeybees persists while the healthy mite population S_m goes extinct, i.e.,

$$\limsup_{t \rightarrow \infty} S_m(t) = 0$$

where the system (7) is attracted to the healthy-mite-free invariant set $HMF = \{(S_h, I_h, S_m, I_m) \in \mathbb{R}_+^4 : S_m = 0\}$ and its dynamics is equivalent to the following three-D system (9):

$$\begin{aligned} S_h' &= \frac{r(S_h + \rho I_h)^2}{K + (S_h + \rho I_h)^2} - d_h S_h - \frac{\beta_h S_h I_h}{S_h + I_h} - \frac{\hat{\beta}_{mh} S_h I_m}{S_h + I_h} - \tilde{\beta}_{mh} S_h I_m - \alpha S_h I_m \\ I_h' &= S_h \left[\frac{\beta_h I_h}{S_h + I_h} + \frac{\hat{\beta}_{mh} I_m}{S_h + I_h} + \tilde{\beta}_{mh} I_m \right] - \alpha I_h I_m - (d_h + \mu_h) I_h \\ I_m' &= c\alpha I_m \left[I_h + S_h - \frac{d_m + \mu_m}{c\alpha} \right] \end{aligned}$$

Let

$$f_1(I_h) = \frac{r \left(\frac{d_m + \mu_m}{c\alpha} - I_h + \rho I_h \right)^2}{\hat{K} + \left(\frac{d_m + \mu_m}{c\alpha} - I_h + \rho I_h \right)^2} - d_h \frac{d_m + \mu_m}{c\alpha} - \mu_h I_h$$

$$f_2(I_h) = \frac{\left(\frac{d_m + \mu_m}{c\alpha} - I_h \right) \left[\frac{\beta_h}{d_m + \mu_m} - (d_h + \mu_h) \right] I_h}{\left[\alpha + \frac{c\alpha \hat{\beta}_{mh}}{d_m + \mu_m} + \hat{\beta}_{mh} \right] I_h - \frac{d_m + \mu_m}{c\alpha} \left(\frac{c\alpha \hat{\beta}_{mh}}{d_m + \mu_m} + \hat{\beta}_{mh} \right)}$$

The dynamics of the healthy-mite-free subsystem (9) can be summarized by the following theorem:

Theorem 3.5 (Dynamics of the healthy-mite-free subsystem). *If $\frac{r}{2\sqrt{\hat{K}}} > d_h$ and $\bar{N}_h^* < \frac{d_m + \mu_m}{c\alpha}$, then the population of virus infected mites goes extinct in the subsystem (9), i.e.,*

$$\limsup_{t \rightarrow \infty} I_m(t) = 0$$

which reduces to the following mite-free model (8). In addition, the following statements hold

1. *If (S_h, I_h, I_m) is an interior equilibrium of the healthy-mite-free subsystem (9), then I_h is a positive intercept of $f_1(I_h)$ and $f_2(I_h)$ subject to $0 < I_h < \frac{d_m + \mu_m}{c\alpha}$, $S_h = \frac{d_m + \mu_m}{c\alpha} - I_h$ and $I_m = f_1(I_h)$.*
2. *The healthy-mite-free subsystem (9) has no interior equilibrium if the inequality $rc\alpha < d_h(d_m + \mu_m)$ holds.*
3. *Assume that $\frac{r}{2\sqrt{\hat{K}}} > \frac{d_h + \mu_h + \alpha N^*}{\rho}$ and $N_h(0) > \underline{N}_h^c$. Then both virus infected honeybee population I_h and virus infected mites I_m persist if the inequalities $\bar{N}_h^* > \frac{d_m + \mu_m}{c\alpha}$ and $\mathcal{R}_0^V = \frac{\beta_h}{d_h + \mu_h} < 1$ hold.*

Notes: Theorem 3.5 implies that, under the condition of $\bar{N}_h^* < \frac{d_m + \mu_m}{c\alpha}$, the virus infected mite population I_m goes extinct in the healthy-mite-free subsystem (9) which reduces to the mite-free subsystem (8) that we studied in the previous subsection. In addition, Theorem 3.5 shows that the subsystem (9) has no interior equilibrium if the inequality $rc\alpha < d_h(d_m + \mu_m)$ holds. Therefore, we could expect the extinction of I_m for small values of r, c, α and large values of d_h, d_m, μ_m . This has been confirmed by numerical simulations. The population of honeybees and virus infected mites (9) experiences sudden collapse when we (1) increase the values of c, α, \hat{K} , and the related virus transmission rates, or (2) decrease the values of d_h, ρ, r, μ_m . The biological implications are that increasing or decreasing the values of these parameters destabilizes the system and generates fluctuating dynamics. The destabilizing effects generate unstable oscillations. The amplitudes of oscillations increase until they touch the stable manifold of the extinction equilibrium, which cause the collapse of the colony. The destabilizing effects of c, α, \hat{K}, d_m can be explained through the dynamics of the virus free subsystem (10) that we have studied in Theorem 3.3. In addition, our simulations provide important insights on the complicated dynamic of (9), which suggest the following:

- Decreasing the values of c, α, \hat{K} can stabilize the system; small values of c, α can cause the extinction of the virus infected mite population I_m , and lead to the coexistence of S_h and I_h .
- Increasing the value of μ_h can stabilize the system but large values of μ_h can cause extinction of the colony due to the initial oscillations.
- Decreasing μ_m can destabilize the system; while increasing it can stabilize the system; large values of μ_m can lead to the extinction of I_m and the persistence of S_h, I_h .
- Decreasing the value of ρ could destabilize the system, thus causing the extinction of the colony.

- Increasing the virus transmission rates (i.e., $\beta_h, \hat{\beta}_{hm}, \tilde{\beta}_{mh}, \hat{\beta}_{hm}$) can stabilize the system, while decreasing their values can destabilize the system and cause the extinction of all species.

Synergistic effects of parasitic mites and virus infections: If there are no mites in the system, according to Theorem 3.4, the mite-free subsystem (8) reduces to the healthy honeybee population, i.e., **virus** goes extinct whenever the initial population of honeybees is above \bar{N}_h^c , $\frac{r}{2\sqrt{K}} > \frac{d_h + \mu_h + \alpha N^*}{\rho}$, and the basic reproduction number $\mathcal{R}_0^V = \frac{\beta_h}{d_h + \mu_h} < 1$ (see two figures in the first row of Figure 2 where virus infected honeybees go extinct (the black curve in right) and the healthy honeybees persist (the red curve in red)). However, when there are virus infected mites in the colony, both **virus** and mites can persist under proper conditions (see Figure 3 where virus infected honeybees (the black curve), the healthy honeybees (the red curve), and the virus infected mites persist (the cyan curve) with the healthy mites going extinct (the blue curve)). For example, the synergistic effects of parasitic mites and virus infections have been illustrated in Figure 2-3 when $r = 1500; K = 1000000; \rho = 0.9; d_h = .15; \mu_h = 0.1; \alpha = 0.005; d_m = 0.1; \mu_m = 0.01; c = 0.005; \beta_h = .24; \hat{\beta}_{mh} = 0.03; \tilde{\beta}_{mh} = .005; \hat{\beta}_{hm} = 0.03$. These are reasonable parameter values derived from [66, 52].

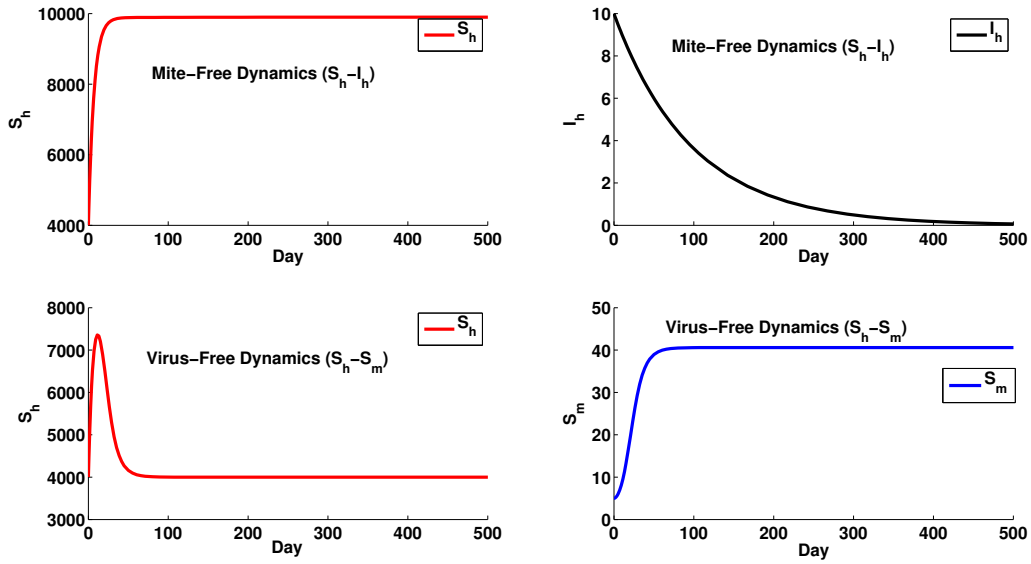


Figure 2: Population dynamics of the subsystems of the the honeybee-mite-virus model (7) when $r = 1500; K = 1000000; \rho = 0.9; d_h = .15; \mu_h = 0.1; \alpha = 0.005; d_m = 0.1; \mu_m = 0.01; c = 0.005; \beta_h = .24; \hat{\beta}_{mh} = 0.03; \tilde{\beta}_{mh} = .005; \hat{\beta}_{hm} = 0.03$. The left figure in the first row is the healthy honeybee population (the red curve) and the right figure in the first row is the virus infected honeybee population (the black curve) in the mite-free subsystem (10) when $S_h(0) = 4001, I_h(0) = 10$. The left figure in the second row is the healthy honeybee population (the red curve) and the right figure in the second row is the healthy mite population (the blue curve) in the virus-free subsystem (8) when $S_h(0) = 4001, S_m(0) = 5$.

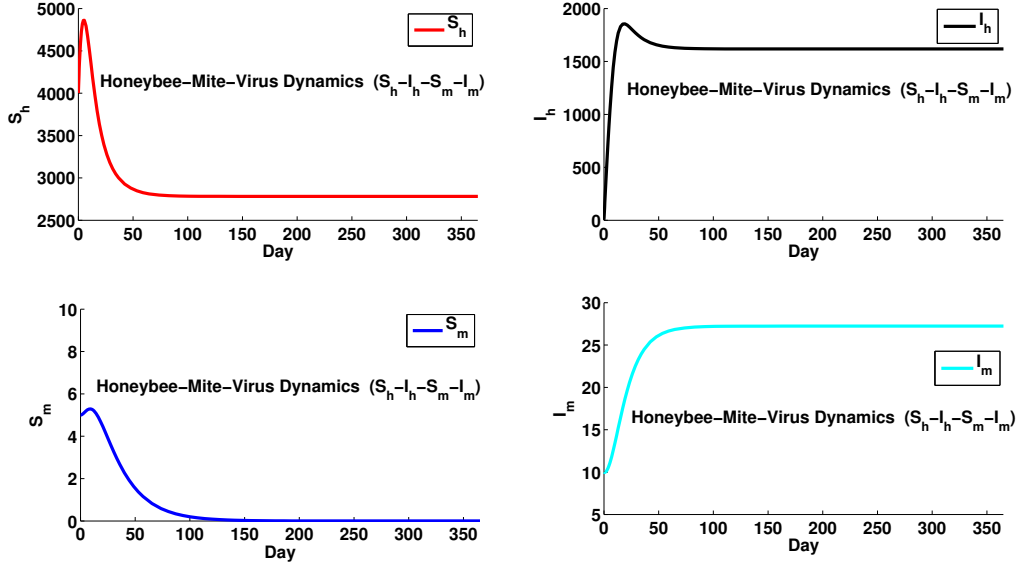


Figure 3: Population dynamics of the honeybee-mite-virus model (7) when $r = 1500$; $K = 1000000$; $\rho = 0.9$; $d_h = .15$; $\mu_h = 0.1$; $\alpha = 0.005$; $d_m = 0.1$; $\mu_m = 0.01$; $c = 0.005$; $\beta_h = .24$; $\hat{\beta}_{mh} = 0.03$; $\tilde{\beta}_{mh} = .005$; $\hat{\beta}_{hm} = 0.03$ and $S_h(0) = 4001$, $I_h(0) = 10$, $S_m(0) = 5$, $I_m(0) = 10$. The healthy honeybee population S_h is in red; the virus infected honeybee population I_h is in black; the healthy mite population S_m is in blue; and the virus infected mite population I_m is in cyan.

3.4. Dynamics of the full system

Recall that the full system (7) of honeybee-mite-virus interactions can be described by the following set of equations:

$$\begin{aligned}
S'_h &= \frac{r(S_h + \rho I_h)^2}{K + (S_h + \rho I_h)^2} - d_h S_h - \frac{\beta_h S_h I_h}{S_h + I_h} - \frac{\hat{\beta}_{mh} S_h I_m}{S_h + I_h} - \tilde{\beta}_{mh} S_h I_m - \alpha S_h (S_m + I_m) \\
I'_h &= S_h \left[\frac{\beta_h I_h}{S_h + I_h} + \frac{\hat{\beta}_{mh} I_m}{S_h + I_h} + \tilde{\beta}_{mh} I_m \right] - \alpha I_h (S_m + I_m) - (d_h + \mu_h) I_h \\
S'_m &= S_m \left[c\alpha S_h - \frac{\hat{\beta}_{hm} I_h}{S_h + I_h} - d_m \right] \\
I'_m &= c\alpha [I_h (S_m + I_m) + S_h I_m] + \frac{\hat{\beta}_{hm} I_h S_m}{S_h + I_h} - (d_m + \mu_m) I_m
\end{aligned}$$

The results from the previous section provide us a complete picture of the dynamics of the subsystems of the full system (7). In this subsection, we explore the dynamics of the full system as the following theorem.

Theorem 3.6 (Colony survival). *Assume that $\frac{r}{2\sqrt{K}} > d_h$. If $N^* < \frac{d_m}{\alpha}$ and $\mathcal{R}_0^V = \frac{\beta_h}{d_h + \mu_h} < 1$, the full system (7) converges to the virus-mite-free set $DMF = \{(S_h, I_h, S_m, I_m) \in \mathbb{R}_+^4 : S_m + I_h + I_m = 0\}$ where the system (7) is reduced to the following one-D system (11):*

$$S'_h = \frac{rS_h^2}{K + S_h^2} - d_h S_h \quad (11)$$

whose dynamics can be summarized as follows:

1. If the inequality $\frac{r}{2\sqrt{K}} < d_h$ holds, then (11) converges to 0.

- 1
2
3
4
5
6 2. If the inequalities $\frac{r}{2\sqrt{K}} > d_h$ and $S_h(0) > \bar{N}_h^c$ hold, then (11) converges to \bar{N}_h^* . If the initial condition
7 falls below \bar{N}_h^c , i.e., $S_h(0) < \bar{N}_h^c$, then (11) also converges to 0.
8

9 Moreover, the following statements hold

- 10
11 1. If the inequalities $\frac{r}{2\sqrt{K}} > \max \left\{ d_h + \beta_h + \frac{\hat{\beta}_{mh}N^*}{\underline{N}_h^*} + (\tilde{\beta}_{mh} + \alpha)(N^* - c\underline{N}_h^*), \frac{d_h + \mu_h + \alpha N^*}{\rho} \right\}$ with $N_h(0) \geq$
12 $S_h(0) > S_h^c$ hold, then S_h is persistent, i.e., $\liminf_{t \rightarrow \infty} S_h(t) \geq S_h^*$.
13
14 2. Assume that $\frac{r}{2\sqrt{K}} > d_h + \beta_h + \frac{\hat{\beta}_{mh}N^*}{\underline{N}_h^*} + (\tilde{\beta}_{mh} + \alpha)(N^* - \underline{N}_h^*)$ with $S_h(0) > S_h^c$ and $\bar{N}_h^* < \frac{d_m}{c\alpha}$ hold.

15
16 Then the **virus** $I = cI_h + I_m$ persists if the inequality $\frac{\min \left\{ \beta_h, c\hat{\beta}_{mh} + c\tilde{\beta}_{mh} + c\alpha\bar{N}_h^* \right\}}{\max \left\{ (d_h + \mu_h), (d_m + \mu_m) \right\}} \geq 1$ holds.
17
18
19

- 20 3. The full system (7) has no interior equilibrium if one of the following inequalities hold:

21
22 $\frac{\beta_h}{d_h + \mu_h} > \frac{\hat{\beta}_{hm}}{d_m + \hat{\beta}_{hm}}, \frac{\hat{\beta}_{mh}}{\frac{\alpha\mu_m\hat{\beta}_{hm}}{(d_m + \hat{\beta}_{hm})} + c\alpha\hat{\beta}_{mh} - \tilde{\beta}_{mh}\hat{\beta}_{hm}} > \frac{d_m + \mu_m}{c\alpha(d_m + \hat{\beta}_{hm} + \mu_m)}$. Or

23
24 $\frac{\alpha\mu_m\hat{\beta}_{hm}}{\hat{\beta}_{hm} + d_m} > \tilde{\beta}_{mh}\hat{\beta}_{hm} - c\alpha, 0 < \frac{\frac{\beta_h}{d_h + \mu_h} - \frac{\hat{\beta}_{hm}}{d_m + \hat{\beta}_{hm}}}{\beta_h(d_m + \hat{\beta}_{hm})(d_h + \mu_h)} < d_m$. Or

25
26 $\frac{\beta_h}{d_h + \mu_h} < \frac{\hat{\beta}_{hm}}{d_m + \hat{\beta}_{hm}}, 0 < \frac{\hat{\beta}_{mh}(d_m + \hat{\beta}_{hm})}{\frac{\alpha\mu_m\hat{\beta}_{hm}}{(d_m + \hat{\beta}_{hm})} + c\alpha\hat{\beta}_{mh} - \tilde{\beta}_{mh}\hat{\beta}_{hm}} < \frac{d_m}{c\alpha}$. Or

27
28 $\frac{\alpha\mu_m\hat{\beta}_{hm}}{\hat{\beta}_{hm} + d_m} < \tilde{\beta}_{mh}\hat{\beta}_{hm} - c\alpha, \frac{\frac{\beta_h}{d_h + \mu_h} - \frac{\hat{\beta}_{hm}}{d_m + \hat{\beta}_{hm}}}{\beta_h(d_m + \hat{\beta}_{hm})(d_h + \mu_h)} > \frac{(d_m + \mu_m)(d_m + \hat{\beta}_{hm})}{d_m + \hat{\beta}_{hm} + \mu_m}$.

- 29
30 4. Assume that $\frac{r}{2\sqrt{K}} > \frac{d_h + \mu_h + \alpha N^*}{\rho}$ and $N_h(0) > \underline{N}_h^c$. Then the total mite population $N_m = S_m + I_m$
31 persists if the inequalities $\bar{N}_h^* > \frac{d_m + \mu_m}{c\alpha}$ and $\mathcal{R}_0^V = \frac{\beta_h}{d_h + \mu_h} < 1$ hold.
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37 **Notes:** Theorem 3.6 along with Theorem 3.1 - 3.3, we can conclude that the extinction of **virus** occurs when
38 all values of all **virus** transmission rates, $\beta_h, \hat{\beta}_{mh}, \tilde{\beta}_{mh}, \hat{\beta}_{hm}$ are small; with the consequence that the full
39 system (7) converges to either $(0, 0, 0, 0)$ or $(\bar{N}_h^*, 0, 0, 0)$ when $\mathcal{R}_0^M = \frac{\bar{N}_h^*}{H^*} < 1$ while (7) converges to either
40 $(0, 0, 0, 0)$ or $(H^*, 0, M^*, 0)$ when $1 < \mathcal{R}_0^M < \frac{\bar{N}_h^*}{S_h^c}$. The persistence of **virus** or mites indicates the colony
41 survival even though the population may be low under the influence of **virus** or mites. Theorem 3.6 provides
42 a summary on sufficient conditions when honeybees can persist in the full (7) alone, with mites, or with
43 **virus**. The item 4 of Theorem 3.6 is consistent with the results from Theorem 3.5 regarding the synergistic
44 effects of parasitic mites and virus infections: If there are no mites in the system, according to Theorem 3.4,
45 the mite-free subsystem (8) reduces to the only healthy honeybee population, however, in the presence of
46 mites, both **virus** and mites in the honeybee-mite-virus system (7) can persist under proper conditions (see
47 Figure 2-3 for more details).
48
49

50 The dynamics of the full system (7) can be extremely complicated. We are unable to obtain an explicit
51 form of the interior equilibrium and the related stability. We perform a series of numerical simulations to
52 explore how different parameters affect the population dynamics. The effects of $r, c, \alpha, d_h, d_m, \mu_m, \mu_h, \rho$ and
53 virus transmission rates are similar to our observations for the subsystem (9). More specifically, we have the
54 following observations:
55

- 56 1. Effects of r : Increasing r can stabilize the system, but increasing it too much can drive healthy mites
57 S_m to extinction while the population of virus infected mites I_m increases. Decreasing the values of
58 r can destabilize the system, and cause the extinction of mites. And very small values can cause the
59 colony to perish.
60
61
62
63
64
65

2. Effects of c : Increasing can destabilize the system and cause extinction of all species. Decreasing its value can stabilize the system but extremely small values can drive the extinction of mites.
3. Effects of α : Increasing can destabilize the system and cause extinction of all species while decreasing can stabilize the system and increase the healthy honeybee population. Small values of α can drive the mite population to extinction, and extremely small values can drive both mites and virus to extinction, and only healthy honeybees are left.
4. Effects of d_m : Increasing d_m can stabilize the system, and drive S_m to extinction first. Increasing it further can lead to the extinction of mites, and the system approaches the limiting mite-free system (8). Decreasing can destabilize the system and drive the extinction of the virus. Extremely small values can cause the colony extinction.
5. Effects of d_h : Increasing can stabilize the system. Large values can drive virus extinctions, however, extremely large values can lead to the extinction of the colony. Decreasing can destabilize the system which may cause the extinction of the colony under certain conditions.
6. Effects of μ_m : Increasing can drive virus extinction. Extremely large values can lead to the extinction of the colony. Decreasing can destabilize the system. Small values can drive the healthy mites extinct, and extremely small values may cause the extinction of the colony.
7. Effects of μ_h : Increasing can cause the extinction of virus. Decreasing can stabilize the system, and small values can drive healthy mites to extinction.
8. Effects of virus transmission rates: Increasing can destabilize the system and cause the extinction of healthy mites S_m , while extremely large values may drive all populations extinct. Decreasing can cause the extinction of virus.

3.5. Mechanisms of collapse dynamics and synergistic effects

Let $r = 1500$; $K = 1000000$; $\rho = 0.9$; $d_h = .15$; $\mu_h = 0.1$; $\alpha = 0.005$; $d_m = 0.1$; $\mu_m = 0.01$; $c = 0.005$; $\beta_h = .24$; $\hat{\beta}_{mh} = 0.03$; $\tilde{\beta}_{mh} = .005$; $\hat{\beta}_{hm} = 0.03$ (Figure 2-3) and $r = 1500$; $\rho = 0.9$; $\hat{K} = 1600001$; $d_h = 0.15$; $\mu_h = 0.1$; $\alpha = 0.05$; $c = 0.005$; $d_m = 0.1$; $\mu_m = 0.01$; $\beta_h = 0.3$; $\hat{\beta}_{mh} = 0.08$; $\tilde{\beta}_{mh} = 0.001$; $\hat{\beta}_{hm} = 0.03$ (Figure 4-5). These are reasonable parameter values derived from [66, 52]. We use these two sets of parameters as illustrations to explore the synergistic effects of parasitic mites and virus infections as well as potential mechanisms linking to CCD (see Figure 1 and Figure 4-5). These comparisons suggest the following:

1. Synergistic effects of parasitic mites and virus infections: Based on the two sets of parameters, we have the following two typical scenarios:

- (a) Under the parameter values of $r = 1500$; $K = 1000000$; $\rho = 0.9$; $d_h = .15$; $\mu_h = 0.1$; $\alpha = 0.005$; $d_m = 0.1$; $\mu_m = 0.01$; $c = 0.005$; $\beta_h = .24$; $\hat{\beta}_{mh} = 0.03$; $\tilde{\beta}_{mh} = .005$; $\hat{\beta}_{hm} = 0.03$:

If there are no mites, the mite-free system (8) (i.e., the dynamics of the healthy (uninfected) honeybee and the virus infected honeybee) converges to only the healthy honeybee with virus infected honeybees becoming extinct (see the first row of Figure 2).

If there is no virus, the virus-free system (10) (i.e., the parasitism dynamics of the healthy honeybee and the healthy mites) converges to a stable equilibrium where both the healthy honeybee and the healthy mites can persist (see the second row of Figure 2).

However, if honeybees, mites, and virus are all present in the system (i.e., the full system (7)),

then both virus infected honeybees (black curve) and virus infected mites (cyan curve) can persist (see Figure 3).

This implies that the presence of mites can promote the persistence of **virus**.

- (b) Under the parameter values of $r = 1500$; $\rho = 0.9$; $\hat{K} = 1600001$; $d_h = 0.15$; $\mu_h = 0.1$; $\alpha = 0.05$; $c = 0.005$; $d_m = 0.1$; $\mu_m = 0.01$; $\beta_h = 0.3$; $\hat{\beta}_{mh} = 0.08$; $\tilde{\beta}_{mh} = 0.001$; $\hat{\beta}_{hm} = 0.03$:

If there are no mites, the mite-free system (8) (i.e., the dynamics of the uninfected honeybee and the virus infected honeybee) converges to a stable equilibrium where both infected and uninfected bees can persist (see the first row of Figure 4).

If there is no virus, the virus-free system (10) (i.e., the parasitism dynamics of uninfected bees and mites) converges to the extinction of both species through *catastrophic event* (see the second row of Figure 2).

However, if honeybees, mites, and virus are all presented in the system (i.e., the full system (7)), then both honeybees and mites go extinct (see Figure 3).

This implies that the presence of the unstable mite population can lead to the extinction of honeybees.

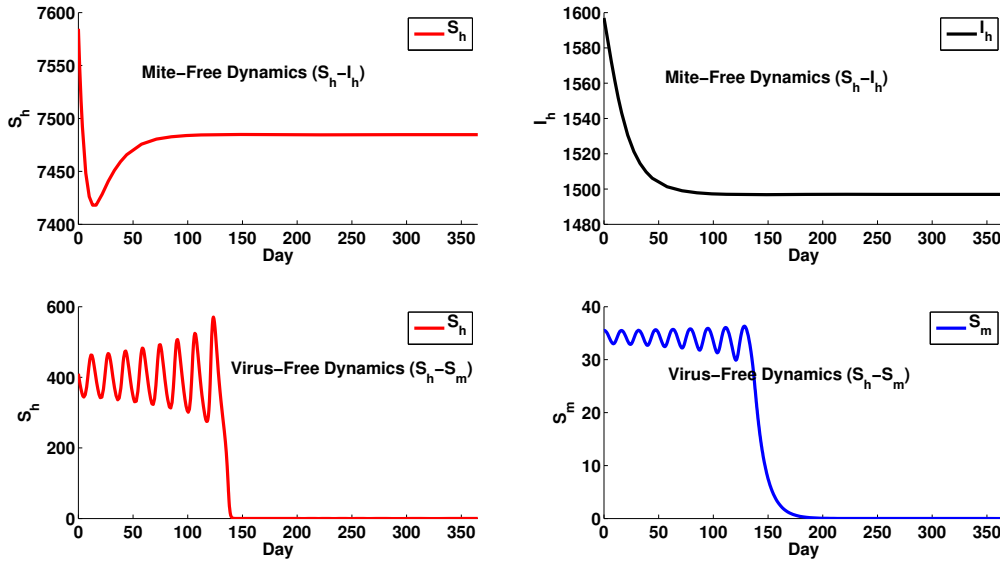


Figure 4: Population dynamics of the subsystems of the the honeybee-mite-virus model (7) when $r = 1500$; $\rho = 0.9$; $\hat{K} = 1600001$; $d_h = 0.15$; $\mu_h = 0.1$; $\alpha = 0.05$; $c = 0.005$; $d_m = 0.1$; $\mu_m = 0.01$; $\beta_h = 0.3$; $\hat{\beta}_{mh} = 0.08$; $\tilde{\beta}_{mh} = 0.001$; $\hat{\beta}_{hm} = 0.03$. The left figure in the first row is the healthy honeybee population (the red curve) and the right figure in the first row is the virus infected honeybee population (the black curve) in the mite-free subsystem (10) when $S_h(0) = 7684$, $I_h(0) = 1700$. The left figure in the second row is the healthy honeybee population (the red curve) and the right figure in the second row is the healthy mite population (the blue curve) in the virus-free subsystem (8) when $S_h(0) = 410$, $S_m(0) = 35$.

2. Linking to CCD: In the absence of virus infections, the subsystem (10) goes through the *catastrophic*

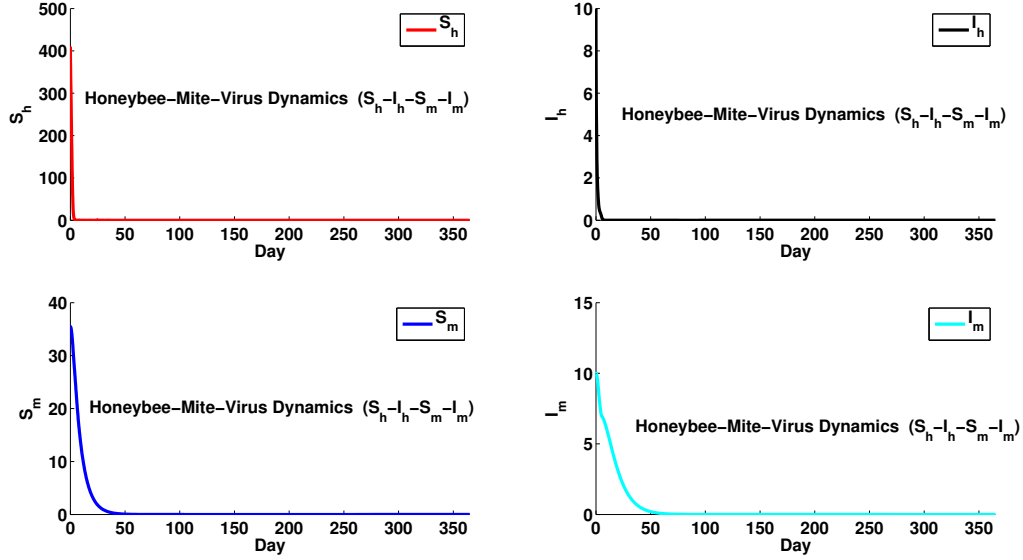


Figure 5: Population dynamics of the honeybee-mite-virus model (7) when $r = 1500$; $K = 160001$; $\rho = 0.9$; $d_h = .15$; $\mu_h = 0.1$; $\alpha = 0.05$; $d_m = 0.1$; $\mu_m = 0.01$; $c = 0.005$; $\beta_h = .3$; $\hat{\beta}_{mh} = 0.08$; $\tilde{\beta}_{mh} = .001$; $\hat{\beta}_{hm} = 0.03$ and $S_h(0) = 410$, $I_h(0) = 10$, $S_m(0) = 35$, $I_m(0) = 10$. The healthy honeybee population S_h is in red; the virus infected honeybee population I_h is in black; the healthy mite population S_m is in blue; and the virus infected mite population I_m is in cyan.

event which causes the extinction of honeybees (see the honeybee population in the red curve of Figure 1). This property has been inherited by the full system (7) as honeybee population goes extinct suddenly for most initial conditions (see the honeybee population in the red curve of the first left figure in 5).

Our analysis and simulations suggest that it is important to include both mites and virus in studying the population dynamics of honeybees as we proposed the full system (7) due to the synergistic effects of parasitism induced by mites and the virus infections as well as the *catastrophic event* from the parasitism interactions between mites and honeybees. In other words, if we only consider the honeybee versus virus dynamics as described by the subsystem (8), or only consider the parasitism interactions between mites and honeybees as described by the subsystem (10), we are not able to capture the full mechanics that can lead to colony death or the biological implications on the persistence of virus in colonies. In addition, the full system (7) has rich dynamics which can possess multiple attractors. Thus, depending on initial conditions, the full system (7) can either experience extinction or have coexistence of both honeybees and mites. More sophisticated mathematical analysis is needed in order to understand the detailed dynamics.

4. Discussion

The association of virus infection with Varroa mites infestation in honeybee colonies causes great concern for researchers and beekeepers [12]. Many studies have suggested that Varroa mite infestations could be a key explanatory factor for the widespread increase in annual honeybee colony mortality and has been implicated as a contributing factor leading to CCD [43]. In this paper, we derive and study a honeybee-virus-mite model by using the susceptible-infectious (SI) disease framework. Our proposed model uses frequency-dependent transmission functions to model horizontal virus transmissions between the phoretic mites and honeybees, a Holling Type I functional response to model parasitic interactions between mites

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6 and honeybees, a density-dependent transmission function to model the vertical virus transmission between
7 mites and honeybees during the mite reproductive phase and a frequency-dependent transmission function
8 to model the horizontal virus transmission among honeybees. Our analytical and numerical results of the
9 full system suggest:

- 10 1. Initial honeybee populations play an important role in its persistence since its dynamics exhibits strong
11 Allee effects in the absence of both parasites and virus (see Theorem 3.1). In addition, patterns of
12 population dynamics are sensitive to initial conditions as suggested by our numerical results, i.e., de-
13 pending on initial conditions, the full system can experience the *catastrophic event* where honeybees
14 collapse dramatically, or both mites and honeybees can coexist but may exhibit different dynamical
15 patterns.
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- 18 2. In the absence of Varroa mites, the honeybee and **virus** system has only equilibrium dynamics (see
19 Theorem 3.4 and the first row of Figure 2-3). In the presence of Varroa mites, the synergistic effects
20 of parasitism and virus make **virus** and mites more persistent (see Theorem 3.5-Theorem 3.6), and
21 thus, difficult to control (see Theorem 3.2 and the related arguments). In addition, the synergistic ef-
22 fects could stabilize the system, but could also drive both honeybees and mites go extinct (see Figure 5).
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- 25 3. In the absence of virus infections, the mite and honeybee system can be destabilized by the low adult
26 workers to brood ratio in the colony where the system has oscillating dynamics leading to a sudden
27 extinction of all species (see Theorem 3.3 and Figure 1). This dynamical property is called the *catas-*
28 *trophic event* which has been inherited by the full system when the virus infection is present (see Figure
29 5). This phenomenon could be linked to CCD which has been observed in honeybee colonies where
30 the low hive to brood ratio could be a contributing factor.
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- 33 4. Our numerical simulations suggest that large values of virus transmission rates can drive the extinction
34 of healthy mites while extremely large values can lead to the extinction of all species.
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38 **Identify the contribution to the broader field:** Our current work provides many useful insights
39 regarding the complicated synergistic effects that Varroa and associated virus infections have on honeybee
40 population dynamics. The model generates scenarios frequently seen in colonies and provides insights into
41 the limitations of certain Varroa control strategies in preventing colony death especially over winter or in
42 early spring. Our theoretical results imply that maintaining the adult workers to brood ratio above a critical
43 threshold during brood rearing periods (e.g, spring and summer) is pivotal for colony survival. These results
44 are similar to previous reports (e.g., [58]). Our work suggests that increasing the values of the queen egg
45 laying rate and the mortality rates of mites can stabilize the system. However, a unique feature to our
46 model is that increase colony population growth also potentially increase Varroa and virus populations. The
47 behavior of the full model generates dynamics commonly seen in the field where large colonies die over-
48 winter even when mite populations are reduced with miticide treatments in the fall [54]. In our model,
49 virus transmission can occur during both contacts with nestmates and parasitism events so viruses do not
50 go extinct in the absence of Varroa as in Ratti *et al.* [52]. The addition of vertical transmission of virus
51 between nestmates generates dynamics that differ from those in previous models where it can take several
52 years for colonies to die. Those models assume that reductions in Varroa populations result in simultaneous
53 reductions in virus levels. However, this is not realistic since the bees can harbor viruses and transmit
54 them among nestmates. Our model generates dynamics seen in the field where colonies collapse overwinter
55 due to high virus transmission rates and the persistence of virus even in the absence of Varroa [26]. These
56 findings suggest that colony losses are due to the combination of virus transmission between nestmates in
57 addition to parasitism and that late season Varroa treatments might not insure overwintering colony survival.
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6 An important insight obtained from our model is that colonies can survive for relatively long periods
7 of time if they are infected with either viruses or Varroa mites. However, the combination of Varroa mites
8 and viruses inevitably leads to colony collapse as shown in the full model. Parasites and pathogens that
9 ultimately kill their hosts must adapt strategies that enable them to disperse before the host dies. Mites
10 cannot move between colonies on their own, but they can disperse by attaching to foragers leaving the hive.
11 If the forager drifts to another colony, the mite and virus have successfully dispersed. Viruses transmitted
12 by Varroa mites (e.g., Deformed wing virus and Israeli acute paralysis virus) affect learning and memory and
13 might affect the return rate of infected foragers to their colonies [36, 28, 33]. Mite migration into colonies on
14 drifting foragers and from the robbing of collapsing colonies has been documented [56, 24, 23, 22]. The syn-
15 ergistic effects revealed in our honeybee-mite-virus system that ultimately lead to colony extinction provide
16 a mathematical explanation for dispersal strategies that might include mite migration when both viruses
17 and Varroa mites are present.
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19
20 **Compare the findings with other works:** As we mentioned in our introduction, the work of Sumpter
21 and Martin [66] and Ratti *et al.* [52] are most relevant modeling papers for our study proposes. They have
22 done great work with interesting results. However, there are limitations on assumptions of their models.
23 Sumpter and Martin [66] assumed that the mites population is constant and the virus transmission occurs
24 only through Varroa mites. This assumption prevents us to study how mites population affect the virus
25 transmission and the honeybee population dynamics. The model of Ratti *et al.* [52] assumes that the virus
26 transmission does not transmit through honeybees themselves. This is not realistic. Because this assumption
27 implies that the extinction of Varroa mites can lead to the extinction of virus, which cannot reflect the fact
28 that honeybee colonies have viruses in the absence of mites. Our honeybee-mite-virus model has relaxed
29 these unrealistic assumptions used in the work of Sumpter and Martin [66] and Ratti *et al.* [52]. More
30 specifically, our proposed model includes the bee to bee virus transmission and both population dynamics
31 of virus and mites with implicit age structures. By including the bee to bee virus transmission, our model
32 allows us to explore the effects of virus and mites on honeybee population dynamics separately. This is one
33 improvement by comparing to the model of Ratti *et al.* [52]. This allows us to investigate the dynamics of
34 the mites-honeybee interactions (i.e., the virus-free dynamics (10)). Our study of the virus-free dynamics
35 (10) implies that the large values of the parasitism rate of mites and the nutrient conversion rate from
36 honeybee to mite and the small value of the mite natural mortality can destabilize the system to generate a
37 catastrophic event that is similar to CCD. Our model permits us to study the dynamics of the virus-honeybee
38 interactions (i.e., the mite-free dynamics (8)) which has only equilibrium dynamics. The comparison of the
39 virus-free dynamics (10) and the mite-free dynamics (8) suggests that the parasitism from mites should be
40 a key factor contributing CCD, not virus infections.
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43 The second improvement of our model is that our model includes both population dynamics of virus and
44 mites with implicit age structure. This improvement allows us to explore the synergistic effects of Varroa
45 mites and the associated virus infections on the honeybee population dynamics. Our study suggests that
46 (a) the presence of mites is able to promote the persistence of virus; (b) the presence of the unstable mite
47 population can lead to the extinction of honeybees while in the absence of mites, honeybee and virus are
48 able to coexist; (c) the presence of both mites and virus can lead to complicated dynamics which can possess
49 multiple attractors, i.e., depending on initial conditions, the full system can either experience extinction or
50 have coexistence of both honeybees and mites; and (d) the low hive to brood ratio could be a contributing
51 factor of CCD. In addition, our model is able to provide sufficient conditions that can guarantee the persis-
52 tence of honeybee population and conditions that lead to the extinction of honeybees.
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55 There are models where pathogens are introduced via the foragers and spread through the colony by
56 worker-worker interactions only [34, 8]. These models do not include sensitivity to Varroa mites. The behav-
57 iors of these models are similar to the case in our analysis examining population dynamics of the mite-free
58 system with virus only. The model by Betti *et al.* [8] simulates the interplay between the dynamics of the
59 spread of the pathogen and the changing demographics of the colony during the season. The model reveals a
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6 critical period just prior to the onset of winter where if a contagious pathogen is introduced, the colony will
7 collapse because the number of bees surviving the winter is not sufficient to support colony growth in the
8 spring. In our model, the mite free subsystem with only virus infections causes colony collapse when trans-
9 mission rates are high as they are during winter colony confinement. These models provide mathematical
10 explanations for the loss of colonies from pathogens alone over the winter due to adult bee to brood ratios
11 in the spring that are insufficient to generate colony growth.
12

13 Models simulating the effects on colony survival of Varroa alone generate predictions of a slow decline in
14 colony populations followed by a rapid collapse [15]. These predictions are similar to the catastrophic event
15 demonstrated in our model analysis with the virus-free system. The behaviors of the models are similar
16 to documented colony losses from Varroa where strong colonies die rapidly in the spring [54]. The initial
17 conditions of colony size, egg laying rates, and Varroa infestation levels strongly influence the length of time
18 required for colonies to collapse. If initial mite levels are low, it can take more than a year for colonies to
19 collapse. Models have been constructed to combine Varroa and viruses that can be spread throughout a
20 colony by Varroa feeding and reproductive behaviors [66, 52, 6]. In the model by Sumpter and Martin [66],
21 the Varroa population is constant. Other models simulate the growth of the Varroa population over time
22 either as a function of colony growth [15, 6] or with a simple logistic equation [52]. Mortality can occur from
23 the effects of parasitism or viral infection. These models reveal the interplay between life stages when death
24 can occur due to infection and colony survival, and capture a counterintuitive aspect of viral spread. Viruses
25 that are highly virulent require greater proportions of Varroa infected with the virus (because transmission
26 rates are reduced), or a longer time to reach epidemic levels that cause colony death [66, 52, 8]. An increase
27 in the transmission rate, simulated by bees being infected at an earlier age, also has a highly deleterious
28 effect on colony survival. Our model predicts colony death when virus transmission rates are high or colony
29 growth is limited by egg laying. In all models cited above, there is persistence in virus infection due to the
30 presence of Varroa.
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33 The results of our model analyses indicate that Varroa control strategies need to keep mite levels and
34 virus transmission rates low, while maintaining adult to brood ratios that insure colony growth. This can
35 be achieved by reducing mite populations in the early spring when brood rearing resumes. Initial conditions
36 strongly influence late season mite population size and virus levels, so mite treatments in early spring can
37 reduce mite population numbers and growth rates. Keeping mite and virus levels low will prevent high levels
38 of parasitism and viral infections later in the year particularly before colonies overwinter. The bees that
39 emerge prior to the end of the yearly brood rearing period should be free of parasitism and pathogen stress.
40 The longevity of bee comprising the overwintering population ultimately determines brood rearing rates and
41 colony growth in the spring. The model suggests that the effectiveness of miticide treatments applied late in
42 the fall when Varroa populations peak might be limited because virus levels and transmission rates will be
43 high in the winter cluster. In the analysis of the full model, high virus transmission rates reduce the adult
44 to brood ratio in the spring when brood rearing resumes leading to colony extinction.
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47 **Limitation of the current model:** In our current model, we assume that the ratios of brood and adult
48 are constants as a replacement for having the explicit dynamical age structure profile. This simplification
49 allows us to obtain rigorous results on how this ratio affect colony dynamics. This model is a good description
50 of the dynamics of the system at or near the steady-state of the age-structured system, but not far from
51 steady-state. This limitation implies that our current model may not be a good description of how colony
52 dynamics will progress if there is a sudden change in the colony demography, such as might occur during a
53 colony collapse event. In particular, the importance of a dynamics age profile of a colony to whether or not
54 it will collapse has been highlighted by the recent work by Perry *et al.* [50], in which the age of onset of
55 foraging is an important factor in determining colony survival. The work of Perry *et al.* [50] examined the
56 social dynamics underlying the dramatic colony failure with an aid of a honeybee population model. Their
57 model does not include population dynamics of Varroa mites or virus infections, but it does includes bee
58 foraging performance varying with age, and displays dynamics of colony population collapse that are similar
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6 to field reports of CCD. It is important to expand our current modeling framework to include the detailed
7 stage-structure model of honeybees with social dynamics, age of onset of foraging as the work by Perry *et*
8 *al.* [50].
9

10 **Suggest options for further research:** A common feature to all the models described above is that
11 equilibrium states between colony populations and pathogens are likely to be unstable in many situations.
12 Without beekeeper intervention, colonies infested with Varroa and infected with viruses die. If colonies
13 with Varroa and viruses are ultimately coursing toward extinction, the pathogens and parasites must adapt
14 dispersal strategies prior to complete colony collapse. The migration of mites among colonies is well docu-
15 mented and appears to be a significant factor in the growth of the Varroa populations particularly in the fall
16 [56, 24, 23, 22]. The inclusion of migration of mites with disease into and out of colonies is needed to provide
17 an added dimension of reality to models that simulate the growth of Varroa populations and the spread of
18 disease in colonies. Models that simulate mite population growth from reproduction alone might ultimately
19 overestimate the time required for colonies to collapse and be poor representation of present conditions par-
20 ticularly in commercial apiaries.
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23 A fixed ratio of adult workers to brood proposed in our current model is a simplification since this ratio
24 varies with the availability of nutrients, the quality and availability of pollen. The mortality of honey bees
25 depends on different risk factors such as parasites, pathogen, viruses, pesticides, nutrition and environmental
26 changes [64]. Among these factors, honey bee nutrition is one of the main factors which impacts honey bee
27 health and influences the capabilities of honey bees to combat different stressors. Nutrients such as pro-
28 tein from pollen is essential in fighting parasites and viruses, and in maintaining the high adult worker to
29 brood ratio in honeybees. This is because honeybees solely depend on protein (containing essential amino
30 acids, vitamins and mineral) and energy, respectively [3]. Cage studies showed that honey bees could sur-
31 vive for a long time without pollen provision [5], but pollen feeding significantly prolonged their lifespan
32 [38, 42, 61, 60]. Intensive research has investigated the connection between nutrition and honey bee virus
33 and stress resistance. Both cage studies and field studies indicated that bees with poor nutrition were under
34 more stress (Wang *et al.* unpublished data) [72], more susceptible to Nosema and Varroa destructor, and
35 had shorter lifespan [19, 53]. DeGrandi-Hoffman found a similar result that bees with good pollen or protein
36 nutrition were more resistant to many different viruses [14]. Furthermore, studies on molecular mechanisms
37 suggested that pollen nutrition may positively affect antimicrobial peptides and improve immune defensive
38 response to parasites [17]. The additions of nutrient/brood dynamics and explicit division of labor would
39 generate scenarios where nutritional stress could reduce immune function resulting in colony death from
40 the proliferation of viruses and other pathogens and thus more closely align with the general consensus that
41 colony losses have multifactorial origins [45]. Therefore, a more realistic model that includes nutrient/brood
42 dynamics with explicit division of labor are needed in addition to migration.
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45 Moreover, seasonal effects are also important. Models that simulate colony growth either assume a
46 constant egg laying rate or have sensitivity to seasonal patterns of brood production [15, 52, 55, 6, 8, 51]. If
47 seasonal effects on egg laying are included, the worker to brood ratio varies throughout the yearly cycle as
48 does the vulnerability of the colony. If parasites such as Varroa or pathogens such as viruses are components
49 in a model, then including seasonal effects on egg laying prevents an overestimation of the mite load and
50 population size of uninfected (i.e., healthy bees) that can be maintained without affecting colony survival.
51 In future work, a realistic model should also include seasonal effects.
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5. Proof

Proof of Theorem 3.1

Proof. According to Theorem A.4 (p.423) of Thieme (2003), we can conclude that Model (7) is positive invariant in X . Let $d = \min\{d_h, d_m\}$, $N_h = S_h + I_h$, $N_m = S_m + I_m$, and $N = cN_h + N_m$. Then we have

$$\begin{aligned} N' = cN'_h + N'_m &= \frac{rc(S_h + \rho I_h)^2}{\hat{K} + (S_h + \rho I_h)^2} - cd_h N_h - c\mu_h I_h - d_m N_m - \mu_m I_m \\ &\leq \frac{rc(N/c)^2}{\hat{K} + (N/c)^2} - \min\{d_h, d_m\}(cN_h + N_m) = \frac{rcN^2}{c^2\hat{K} + N^2} - \min\{d_h, d_m\}N = \frac{rcN^2}{c^2\hat{K} + N^2} - dN \end{aligned}$$

which implies that $\limsup_{t \rightarrow \infty} N(t) \leq c \frac{\frac{r}{d} + \sqrt{\left(\frac{r}{d}\right)^2 - 4\hat{K}}}{2} = N^*$ with implication that $\limsup_{t \rightarrow \infty} N(t) = 0$ if either $\frac{r}{d} < 2\sqrt{\hat{K}}$ or $N(0) < N^c = c \frac{\frac{r}{d} + \sqrt{\left(\frac{r}{d}\right)^2 - 4\hat{K}}}{2}$ holds. The arguments above also imply that if $\frac{r}{d} > 2\sqrt{\hat{K}}$, then we have

$$\limsup_{t \rightarrow \infty} N_h(t) \leq N^*/c \text{ and } \limsup_{t \rightarrow \infty} N_m(t) \leq N^*.$$

Similarly, we have follows for N_h :

$$\begin{aligned} N'_h = S'_h + I'_h &= \frac{r(S_h + \rho I_h)^2}{\hat{K} + (S_h + \rho I_h)^2} - d_h N_h - \mu_h I_h - \alpha N_h N_m \\ &\leq \frac{rN_h^2}{\hat{K} + N_h^2} - d_h N_h \end{aligned}$$

which implies that

$$\limsup_{t \rightarrow \infty} N_h(t) \leq \frac{\frac{r}{d_h} + \sqrt{\left(\frac{r}{d_h}\right)^2 - 4\hat{K}}}{2} = \bar{N}_h^* \text{ if } \frac{r}{d_h} > 2\sqrt{\hat{K}}$$

and

$$\limsup_{t \rightarrow \infty} N_h(t) = 0 \text{ if } \frac{r}{d_h} < 2\sqrt{\hat{K}} \text{ or } N_h(0) < S_h^c = \frac{\frac{r}{d_h} - \sqrt{\left(\frac{r}{d_h}\right)^2 - 4\hat{K}}}{2}.$$

On the other hand, we have

$$\begin{aligned} N'_h = S'_h + I'_h &\geq \frac{r\rho^2(S_h/\rho + I_h)^2}{\hat{K} + \rho^2(S_h/\rho + I_h)^2} - (d_h + \mu_h + \alpha N_m)N_h \\ &\geq \frac{r\rho^2 N_h^2}{\hat{K} + \rho^2 N_h^2} - (d_h + \mu_h + \alpha N^*)N_h \\ &\geq \frac{rN_h^2}{\hat{K}/\rho^2 + N_h^2} - (d_h + \mu_h + \alpha N^*)N_h \end{aligned}$$

Therefore, apply the comparison theorem, we can conclude that

$$\liminf_{t \rightarrow \infty} N_h(t) \geq \frac{\frac{r}{d_h + \mu_h + \alpha N^*} + \sqrt{\left(\frac{r}{d_h + \mu_h + \alpha N^*}\right)^2 - 4\hat{K}/\rho^2}}{2} = \underline{N}_h^*$$

if the following inequalities hold

$$\frac{r}{d_h + \mu_h + \alpha N^*} > 2\sqrt{\frac{\hat{K}}{\rho^2}} \text{ and } N_h(0) > \underline{N}_h^c = \frac{\frac{r}{d_h + \mu_h + \alpha N^*} - \sqrt{\left(\frac{r}{d_h + \mu_h + \alpha N^*}\right)^2 - 4\hat{K}/\rho^2}}{2}.$$

The discussions above provide sufficient conditions that allow N_h being persistent, i.e., when the inequalities $\frac{r}{d_h + \mu_h + \alpha N^*} > 2\sqrt{\frac{\hat{K}}{\rho^2}}$ and $N_h(0) > \underline{N}_h^c$ hold. This implies that S_h is also persistent under this condition since all species go extinct if S_h goes extinct. However, this persistence condition does not provide an estimate of S_h . To explore an estimation of S_h , we look at the population of S_h .

Recall that if $\frac{r}{d_h + \mu_h + \alpha N^*} > 2\sqrt{\frac{\hat{K}}{\rho^2}}$ and $N_h(0) > \underline{N}_h^c$, then we have the following inequalities

$$\underline{N}_h^* \leq \liminf_{t \rightarrow \infty} N_h(t) \leq \liminf_{t \rightarrow \infty} N(t)/c \leq \limsup_{t \rightarrow \infty} N(t)/c \leq N^*/c.$$

If the inequalities $\frac{r}{d_h + \beta_h + \frac{\hat{\beta}_{mh} N^*}{\underline{N}_h^*} + (\tilde{\beta}_{mh} + \alpha)(N^* - c\underline{N}_h^*)} > 2\sqrt{\hat{K}}$ and

$$S_h(0) > \frac{\frac{r}{d_h + \beta_h + \frac{\hat{\beta}_{mh} N^*}{\underline{N}_h^*} + (\tilde{\beta}_{mh} + \alpha)(N^* - c\underline{N}_h^*)} - \sqrt{\left(\frac{r}{d_h + \beta_h + \frac{\hat{\beta}_{mh} N^*}{\underline{N}_h^*} + (\tilde{\beta}_{mh} + \alpha)(N^* - c\underline{N}_h^*)}\right)^2 - 4\hat{K}}}{2}$$

hold, then we have following inequalities

$$\begin{aligned} S_h' &= \frac{r(S_h + \rho I_h)^2}{\hat{K} + (S_h + \rho I_h)^2} - d_h S_h - \frac{\beta_h S_h I_h}{S_h + I_h} - \frac{\hat{\beta}_{mh} S_h I_m}{S_h + I_h} - \tilde{\beta}_{mh} S_h I_m - \alpha S_h (S_m + I_m) \\ &> \frac{r(S_h)^2}{\hat{K} + (S_h)^2} - d_h S_h - \beta_h S_h - \frac{\hat{\beta}_{mh} I_m}{\underline{N}_h^*} S_h - \tilde{\beta}_{mh} S_h N_m - \alpha S_h N_m \\ &> \frac{r(S_h)^2}{\hat{K} + (S_h)^2} - \left[d_h + \beta + \frac{\hat{\beta}_{mh} N^*}{\underline{N}_h^*} + (\tilde{\beta}_{mh} + \alpha) N_m \right] S_h \\ &= \frac{r(S_h)^2}{\hat{K} + (S_h)^2} - \left[d_h + \beta + \frac{\hat{\beta}_{mh} N^*}{\underline{N}_h^*} + (\tilde{\beta}_{mh} + \alpha)(N - c\underline{N}_h) \right] S_h \\ &> \frac{r(S_h)^2}{\hat{K} + (S_h)^2} - \left[d_h + \beta + \frac{\hat{\beta}_{mh} N^*}{\underline{N}_h^*} + (\tilde{\beta}_{mh} + \alpha)(N^* - c\underline{N}_h^*) \right] S_h \end{aligned}$$

which implies that

$$\liminf_{t \rightarrow \infty} S_h(t) \geq S_h^* = \frac{\frac{r}{d_h + \beta_h + \frac{\hat{\beta}_{mh} N^*}{\underline{N}_h^*} + (\tilde{\beta}_{mh} + \alpha)(N^* - c\underline{N}_h^*)} + \sqrt{\left(\frac{r}{d_h + \beta_h + \frac{\hat{\beta}_{mh} N^*}{\underline{N}_h^*} + (\tilde{\beta}_{mh} + \alpha)(N^* - c\underline{N}_h^*)}\right)^2 - 4\hat{K}}}{2}.$$

Therefore, we can conclude that S_h is persistent with the following properties:

$$S_h^* \leq \liminf_{t \rightarrow \infty} S_h(t) \leq \liminf_{t \rightarrow \infty} N_h(t) \leq \liminf_{t \rightarrow \infty} \frac{N(t)}{c} \leq \limsup_{t \rightarrow \infty} N(t)/c \leq N^*/c$$

if the following inequalities hold

$$1. \frac{r}{2\sqrt{\hat{K}}} > \max \left\{ d_h + \beta_h + \frac{\hat{\beta}_{mh} N^*}{\underline{N}_h^*} + (\tilde{\beta}_{mh} + \alpha)(N^* - \underline{N}_h^*), \frac{d_h + \mu_h + \alpha N^*}{\rho} \right\} \text{ with } N_h(0) \geq S_h(0) > S_h^c.$$

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6 It is easy to check that the extinction equilibrium $E_0 = (0, 0, 0, 0)$ is always local stable. We omit the details.
7 Based on the discussion of the upper bound of the total population N and the total population of honeybees
8 N_h , we can conclude that the system (7) converges to E_0 globally if $2\sqrt{\hat{K}} > \frac{r}{d_h}$ holds; while if the initial
9 population satisfies either $N(0) < N^c$ or $N_h(0) < \bar{N}_h^c$, then the system (7) converges to E_0 locally. \square

11
12 *Proof of Theorem 3.2*

13 *Proof.* Now we consider the population of N_m . From Model (7), we obtain the following inequalities:

$$14 \quad c\alpha N_h N_m - (d_m + \mu_m)N_m \leq N'_m = S'_m + I'_m = c\alpha N_h N_m - d_m N_m - \mu_m I_m \leq N_m (c\alpha N_h - d_m)$$

15
16 which implies that if $N^* < \frac{d_m}{\alpha}$, then we have

$$17 \quad \begin{aligned} 18 \quad N'_m &\leq N_m [c\alpha N_h - d_m] = N_m [c\alpha(N - N_m)/c - d_m] \\ 19 &\leq N_m [\alpha N^* - d_m - \alpha N_m] < N_m [\alpha N^* - d_m] \end{aligned}$$

20
21 This indicates that $\limsup_{t \rightarrow \infty} N_m(t) = 0$.

22
23 Assume that the following inequalities hold $\frac{r}{d_h} > 2\sqrt{\hat{K}}$, and $S_h(0) > S_h^c$. Then according to Theorem
24 3.1, we have

$$25 \quad \limsup_{t \rightarrow \infty} S_h(t) \leq \limsup_{t \rightarrow \infty} N_h(t) \leq \bar{N}_h^*.$$

26
27 Now let us focus on the population of S_m . Notice that we have the following inequalities when time is large
28 enough,

$$29 \quad S'_m = S_m \left[c\alpha S_h - \frac{\hat{\beta}_{hm} I_h}{S_h + I_h} - d_m \right] \leq S_m [c\alpha N_h - d_m] \leq S_m [c\alpha \bar{N}_h^* - d_m].$$

30
31 This implies that if $\bar{N}_h^* < \frac{d_m}{c\alpha}$ and $\frac{r}{d_h} > 2\sqrt{\hat{K}}$, and $S_h(0) > S_h^c$, the healthy mite population S_m goes extinct
32 while the total population of honeybees persists, i.e.,

$$33 \quad \limsup_{t \rightarrow \infty} S_m(t) = 0.$$

34
35 Now we look at the population dynamics of I_m and I_h . Let $I = cI_h + I_m$. From Model (7), then we have
36 the following equations:

$$37 \quad \begin{aligned} 38 \quad I' &= (cI_h + I_m)' = S_h \left[\frac{c\beta_h I_h}{S_h + I_h} + \frac{c\hat{\beta}_{mh} I_m}{S_h + I_h} + c\tilde{\beta}_{mh} I_m \right] - c\alpha I_h (S_m + I_m) - c(d_h + \mu_h) I_h \\ 39 &\quad + c\alpha [I_h (S_m + I_m) + S_h I_m] + \frac{\hat{\beta}_{hm} I_h S_m}{S_h + I_h} - (d_m + \mu_m) I_m \\ 40 &= I_h \left[\frac{c\beta_h S_h}{N_h} + \frac{c\hat{\beta}_{hm} S_m}{N_h} \right] + I_m \left[\frac{c\hat{\beta}_{mh} S_h}{N_h} + c\tilde{\beta}_{mh} + c\alpha S_h \right] - c(d_h + \mu_h) I_h - (d_m + \mu_m) I_m \end{aligned}$$

41
42 This implies that

$$43 \quad I' \geq I \left[\min \left\{ \frac{\beta_h S_h}{N_h} + \frac{\hat{\beta}_{hm} S_m}{N_h}, \frac{c\hat{\beta}_{mh} S_h}{N_h} + c\tilde{\beta}_{mh} + c\alpha S_h \right\} - \max \left\{ (d_h + \mu_h), (d_m + \mu_m) \right\} \right].$$

44
45 Assume that $\frac{r}{2\sqrt{\hat{K}}} > d_h + \beta_h + \frac{\hat{\beta}_{hm} N^*}{N_h^*} + (\tilde{\beta}_{mh} + \alpha)(N^* - \underline{N}_h^*)$ and $S_h(0) > S_h^c$, then according to Theorem
46 3.1, we have

$$47 \quad \liminf_{t \rightarrow \infty} S_h(t) \geq S_h^*.$$

Therefore, if $\frac{\min \left\{ \beta_h, c\hat{\beta}_{mh} + c\tilde{\beta}_{mh} + c\alpha S_h^* \right\}}{\max \left\{ (d_h + \mu_h), (d_m + \mu_m) \right\}} \geq 1$, then we have

$$\begin{aligned} \frac{I'}{I} \Big|_{I_h=I_m=0} &\geq \left[\min \left\{ \frac{\beta_h S_h}{N_h}, c\hat{\beta}_{mh} + c\tilde{\beta}_{mh} + c\alpha S_h \right\} - \max \left\{ d_h + \mu_h, (d_m + \mu_m) \right\} \right] \Big|_{I_h=I_m=0} \\ &> \left[\min \left\{ \beta_h, c\hat{\beta}_{mh} + c\tilde{\beta}_{mh} + c\alpha S_h^* \right\} - \max \left\{ (d_h + \mu_h), (d_m + \mu_m) \right\} \right] \geq 0 \end{aligned}$$

which implies that the **virus** I persists by applying the average Lyapunov theorem (Hutson 1984).

On the other hand, we have the following inequalities:

$$\begin{aligned} I' &\leq I \left[\max \left\{ \frac{\beta_h S_h}{N_h} + \frac{\hat{\beta}_{hm} S_m}{N_h}, \frac{c\hat{\beta}_{mh} S_h}{N_h} + c\tilde{\beta}_{mh} + c\alpha S_h \right\} - \min \left\{ (d_h + \mu_h), (d_m + \mu_m) \right\} \right] \\ \frac{I'}{I} \Big|_{I_h=I_m=0} &< \left[\max \left\{ \beta_h + \frac{\hat{\beta}_{hm} S_m}{N_h}, \frac{c\hat{\beta}_{mh} S_h}{N_h} + c\tilde{\beta}_{mh} + c\alpha S_h \right\} - \min \left\{ (d_h + \mu_h), (d_m + \mu_m) \right\} \right] \end{aligned}$$

Assume that $\frac{r}{2\sqrt{K}} > \frac{d_h + \mu_h + \alpha N^*}{\rho}$, $N_h(0) > \underline{N}_h^c$. Then according to Theorem 3.1, we have the following inequalities:

$$\underline{N}_h^* \leq \liminf_{t \rightarrow \infty} N_h(t) \leq \limsup_{t \rightarrow \infty} N_h(t) \leq \bar{N}_h^* \leq \limsup_{t \rightarrow \infty} N(t)/c = N^*/c.$$

This implies the following inequality when $\frac{\max \left\{ \beta_h + \frac{\hat{\beta}_{hm} N^*}{N_h}, c\hat{\beta}_{mh} + c\tilde{\beta}_{mh} + c\alpha \bar{N}_h^* \right\}}{\min \left\{ (d_h + \mu_h), (d_m + \mu_m) \right\}} < 1$, then we have

$$\frac{I'}{I} \Big|_{I_h=I_m=0} < \left[\max \left\{ \beta_h + \frac{\hat{\beta}_{hm} N^*}{N_h}, c\hat{\beta}_{mh} + c\tilde{\beta}_{mh} + c\alpha \bar{N}_h^* \right\} - \min \left\{ (d_h + \mu_h), (d_m + \mu_m) \right\} \right] < 0$$

which implies that the **virus** goes extinct, i.e.,

$$\lim_{t \rightarrow \infty} I(t) = 0.$$

□

Proof of Theorem 3.3

Proof. If $M = 0$, the virus-free subsystem (10) reduces to the only healthy honeybee population:

$$S_h' = \frac{r S_h^2}{\hat{K} + S_h^2} - d_h S_h$$

which leads to the following three boundary equilibria if $\frac{r}{2\hat{K}} > d_h$:

$$(0, 0), (\bar{N}_h^c, 0), \text{ and } (\bar{N}_h^*, 0).$$

The local stability can be easily determined by the eigenvalues evaluated at its Jacobian matrix. Simple algebraic calculations show that $(\bar{N}_h^c, 0)$ is a saddle if $\bar{N}_h^c < \frac{d_m}{\alpha c}$ while it is a source if $\bar{N}_h^c > \frac{d_m}{\alpha c}$; and $(\bar{N}_h^*, 0)$ is a sink if $\bar{N}_h^* < \frac{d_m}{\alpha c}$ while it is a saddle if $\bar{N}_h^* > \frac{d_m}{\alpha c}$.

Now let (H, M) be an interior equilibrium of the virus-free subsystem (10), then we have the following

equations hold:

$$0 = c\alpha HM - d_m M \Leftrightarrow H = \frac{d_m}{c\alpha}$$

$$0 = \frac{rH^2}{K+H^2} - d_h H - \alpha HM \Leftrightarrow M = \frac{1}{\alpha} \left(\frac{rH}{K+H^2} - d_h \right)$$

which gives the unique interior equilibrium $(H^*, M^*) = \left(\frac{d_m}{c\alpha}, \frac{1}{\alpha} \left(\frac{rH^*}{K+(H^*)^2} - d_h \right) \right)$ provided that $\bar{N}_h^c < \frac{d_m}{\alpha c} < \bar{N}_h^*$.

The local stability of (H^*, M^*) is determined by the eigenvalues $\lambda_i, i = 1, 2$ of the following Jacobian matrix of (10):

$$J_{HM} = \begin{pmatrix} \frac{rH^*(\hat{K}-(H^*)^2)}{(\hat{K}+(H^*)^2)^2} & -\alpha H^* \\ \alpha c M^* & 0 \end{pmatrix} \quad (12)$$

which gives the following two equations:

$$\begin{aligned} \lambda_1 + \lambda_2 &= \frac{rH^*(\hat{K}-(H^*)^2)}{(\hat{K}+(H^*)^2)^2} \\ \lambda_1 \lambda_2 &= c\alpha^2 H^* M^* \end{aligned} \quad (13)$$

Therefore, we can conclude that (H^*, M^*) is a sink if $H^* > \sqrt{\hat{K}}$ while (H^*, M^*) is a source if $H^* < \sqrt{\hat{K}}$. The discussion above implies follows:

1. If $\frac{r}{2\sqrt{\hat{K}}} < d_h$, then the extinction equilibrium $(0, 0)$ is the only locally stable equilibrium for the virus-free subsystem (10). Thus it is globally stable.
2. If $\frac{r}{2\sqrt{\hat{K}}} > d_h$ and either $\bar{N}_h^c > \frac{d_m}{\alpha c}$ or $\frac{d_m}{\alpha c} > \bar{N}_h^*$, then the virus-free subsystem (10) has three boundary equilibria: $(0, 0)$, $(S_h^c, 0)$, $(\bar{N}_h^*, 0)$ where $(0, 0)$ is always locally stable.
 - If $\bar{N}_h^c > \frac{d_m}{\alpha c}$, then $(S_h^c, 0)$ is a source and $(\bar{N}_h^*, 0)$ is a saddle. Since (10) is a two-D ode system, it has global stability at the extinction equilibrium $(0, 0)$ according to the Poincare-Bendison theorem [49].
 - If $\bar{N}_h^c < \frac{d_m}{\alpha c}$, then $(S_h^c, 0)$ is a saddle and $(\bar{N}_h^*, 0)$ is a sink. This implies that (10) has two locally asymptotically stable boundary equilibria $(0, 0)$ and $(\bar{N}_h^*, 0)$ which are reserved as the only two attractors for the model.
3. If $\frac{r}{2\sqrt{\hat{K}}} > d_h$ and $\bar{N}_h^c < \frac{d_m}{\alpha c} < \bar{N}_h^*$, then (10) has three boundary equilibria $(0, 0)$, $(S_h^c, 0)$, $(\bar{N}_h^*, 0)$ and the unique interior equilibrium (H^*, M^*) where $(0, 0)$ is locally stable, both $(S_h^c, 0)$ and $(\bar{N}_h^*, 0)$ are saddle nodes. The local stability of (H^*, M^*) is determined by the sign of $H^* - \sqrt{\hat{K}}$: if $H^* - \sqrt{\hat{K}} > 0$, then (H^*, M^*) is locally asymptotically stable while if $H^* - \sqrt{\hat{K}} < 0$, then (H^*, M^*) is a source.

Define $\mathcal{R}_0^M = \frac{\bar{N}_h^*}{H^*}$. Then we can conclude that (10) has no interior equilibrium if $\mathcal{R}_0^M < 1$ or $\mathcal{R}_0^M > \frac{\bar{N}_h^*}{S_h^c} > 1$; and (10) has a unique interior equilibrium (H^*, M^*) if $1 < \mathcal{R}_0^M < \frac{\bar{N}_h^*}{S_h^c}$. More specifically, (H^*, M^*) is locally stable if $1 < \mathcal{R}_0^M < \frac{\bar{N}_h^*}{K}$ while it is a source if $\mathcal{R}_0^M > \max \left\{ 1, \frac{\bar{N}_h^*}{K} \right\}$. \square

1
2
3
4
5
6 *Proof of Theorem 3.4*

7 *Proof.* If $I = 0$, the mite-free subsystem (8) reduces to the only healthy honeybee population:

$$9 \quad S'_h = \frac{rS_h^2}{\hat{K} + S_h^2} - d_h S_h$$

10 which leads to the following three boundary equilibria if $\frac{r}{2\hat{K}} > d_h$:

$$11 \quad (0, 0), (\bar{N}_h^c, 0), \text{ and } (\bar{N}_h^*, 0).$$

12 The local stability can be easily determined by the eigenvalues evaluated at its Jacobian matrix. Define
13 $\mathcal{R}_0^V = \frac{\beta_h}{d_h + \mu_h}$. The simple algebraic calculations show that if $\mathcal{R}_0^V < 1$, $(\bar{N}_h^c, 0)$ is a saddle and $(\bar{N}_h^*, 0)$ is
14 a sink; while if $\mathcal{R}_0^V > 1$, then $(\bar{N}_h^c, 0)$ is a source and $(\bar{N}_h^*, 0)$ is a saddle. According to Theorem 3.1, the
15 extinction equilibrium $(0, 0)$ is always a sink.

16 Let (S, I) be an interior equilibrium of the mite-free subsystem (8), then it satisfies the following equations:

$$\begin{aligned} 17 \quad \frac{\beta_h S}{S+I} - (d_h + \mu_h) &= 0 & \Rightarrow \quad S &= \frac{d_h + \mu_h}{\beta_h - (d_h + \mu_h)} I = \frac{I}{\frac{\beta_h}{d_h + \mu_h} - 1} = aI \\ 18 \quad \frac{r(S+\rho I)^2}{\hat{K} + (S+\rho I)^2} - d_h S - S \frac{\beta_h I}{S+I} &= 0 & \Rightarrow \quad \frac{rI}{\frac{\hat{K}}{(a+\rho)^2} + I^2} - a \left(d_h + \frac{\beta_h}{1+a} \right) &= 0 \\ 19 \quad \frac{r(S+\rho I)^2}{\hat{K} + (S+\rho I)^2} - d_h S - S \frac{\beta_h I}{S+I} &= 0 & \Rightarrow \quad \frac{r(S+\rho I)^2}{\hat{K} + (S+\rho I)^2} - d_h S - (d_h + \mu_h) I &= 0 \\ 20 & & \Rightarrow \quad \frac{rI}{\frac{\hat{K}}{(a+\rho)^2} + I^2} - ((a+1)d_h + \mu_h) &= \frac{rI}{\frac{\hat{K}}{(a+\rho)^2} + I^2} - \tilde{d} = 0 \\ 21 & & \Rightarrow \quad \frac{a\beta_h}{1+a} &= d_h + \mu_h. \end{aligned} \quad (14)$$

22 Therefore, if $\mathcal{R}_0^V = \frac{\beta_h}{d_h + \mu_h} > 1$ and $\frac{r}{d} > \frac{2\sqrt{\hat{K}}}{a+\rho}$, then we can conclude that the mite-free subsystem (8) can
23 have two interior equilibria (S_h^k, I_h^k) , $k = 1, 2$ where

$$24 \quad a = \frac{1}{\frac{\beta_h}{d_h + \mu_h} - 1}, \quad d = (a+1)d_h + \mu_h = d_h \left(\frac{\beta_h}{d_h + \mu_h} \right) + \mu_h,$$

25 and

$$26 \quad I_h^1 = \frac{\frac{r}{d} - \sqrt{\left(\frac{r}{d}\right)^2 - 4\frac{\hat{K}}{(a+\rho)^2}}}{2}, \quad I_h^2 = \frac{\frac{r}{d} + \sqrt{\left(\frac{r}{d}\right)^2 - 4\frac{\hat{K}}{(a+\rho)^2}}}{2}, \quad S_h^k = aI_h^k, \quad k = 1, 2.$$

27 Now we examine the local stability of these two interior equilibria provided that they exist. The Jacobian
28 matrix of (8) evaluated at the interior equilibrium $(S, I) = (aI, I)$ can be expressed as follows:

$$29 \quad J_{HV} = \begin{pmatrix} \frac{2r\hat{K}(a+\rho)I}{((a+\rho)^2 I^2 + \hat{K})^2} - d_h - \frac{\beta_h}{(1+a)^2} & \frac{2r\rho\hat{K}(a+\rho)I}{((a+\rho)^2 I^2 + \hat{K})^2} - \frac{a^2\beta_h}{(1+a)^2} \\ \frac{\beta_h}{(1+a)^2} & -\frac{a\beta_h}{(1+a)^2} \end{pmatrix} \quad (15)$$

whose two eigenvalues $\lambda_i, i = 1, 2$ satisfy the following equations:

$$\begin{aligned}
\lambda_1 + \lambda_2 &= \frac{2r\hat{K}(a+\rho)I}{((a+\rho)^2I^2+\hat{K})^2} - d_h - \frac{\beta_h}{1+a} = \frac{2\hat{K}}{((a+\rho)^2I^2+\hat{K})(a+\rho)} \frac{r(a+\rho)^2I}{(a+\rho)^2I^2+\hat{K}} - d_h - \frac{\beta_h}{1+a} \\
&= \frac{2\hat{K}}{((a+\rho)^2I^2+\hat{K})(a+\rho)} a \left(d_h + \frac{\beta_h}{1+a} \right) - d_h - \frac{\beta_h}{1+a} = \left(d_h + \frac{\beta_h}{1+a} \right) \left[\frac{2a\hat{K}}{((a+\rho)^2I^2+\hat{K})(a+\rho)} - 1 \right] \\
&< \left(d_h + \frac{\beta_h}{1+a} \right) \left[\frac{2\hat{K}}{(a+\rho)^2I^2+\hat{K}} - 1 \right]
\end{aligned} \tag{16}$$

and

$$\begin{aligned}
\lambda_1\lambda_2 &= \frac{\beta_h}{(1+a)^2} \left[-\frac{2ra\hat{K}(a+\rho)I}{((a+\rho)^2I^2+\hat{K})^2} + ad_h + \frac{a\beta_h}{(1+a)^2} - \frac{2r\rho\hat{K}(a+\rho)I}{((a+\rho)^2I^2+\hat{K})^2} + \frac{a^2\beta_h}{(1+a)^2} \right] \\
&= \frac{\beta_h}{(1+a)^2} \left[-\frac{2r\hat{K}(a+\rho)^2I}{((a+\rho)^2I^2+\hat{K})^2} + ad_h + \frac{a\beta_h}{1+a} \right] = \frac{\beta_h}{(1+a)^2} \left[-\frac{2r\hat{K}(a+\rho)^2I}{((a+\rho)^2I^2+\hat{K})^2} + \tilde{d} \right].
\end{aligned} \tag{17}$$

If $\mathcal{R}_0^V = \frac{\beta_h}{d_h+\mu_h} > 1$ and $\frac{r}{\tilde{d}} > \frac{2\sqrt{\hat{K}}}{a+\rho}$, then we have two interior equilibria $(S_h^k, I_h^k), k = 1, 2$ where

$$I_h^1 = \frac{\frac{r}{\tilde{d}} - \sqrt{\left(\frac{r}{\tilde{d}}\right)^2 - 4\frac{\hat{K}}{(a+\rho)^2}}}{2}, \quad I_h^2 = \frac{\frac{r}{\tilde{d}} + \sqrt{\left(\frac{r}{\tilde{d}}\right)^2 - 4\frac{\hat{K}}{(a+\rho)^2}}}{2}, \quad S_h^k = aI_h^k, k = 1, 2.$$

This implies that $\lambda_1(I_h^2)\lambda_2(I_h^1) < 0$, $\lambda_1(I_h^2)\lambda_2(I_h^2) > 0$ and

$$\begin{aligned}
\lambda_1(I_h^2) + \lambda_2(I_h^2) &< \left(d_h + \frac{\beta_h}{1+a} \right) \left[\frac{2\hat{K}}{(a+\rho)^2I^2+\hat{K}} - 1 \right] \\
&< \left(d_h + \frac{\beta_h}{1+a} \right) \left[\frac{2\hat{K}}{(a+\rho)^2\frac{\hat{K}}{(a+\rho)^2}+\hat{K}} - 1 \right] = 0
\end{aligned}$$

since

$$I_h^2 = \frac{\frac{r}{\tilde{d}} + \sqrt{\left(\frac{r}{\tilde{d}}\right)^2 - 4\frac{\hat{K}}{(a+\rho)^2}}}{2} > \frac{r}{2\tilde{d}} > \frac{\sqrt{\hat{K}}}{(a+\rho)} \Rightarrow (I_h^2)^2 > \frac{\hat{K}}{(a+\rho)^2}.$$

Therefore, we can conclude that if $\mathcal{R}_0^V = \frac{\beta_h}{d_h+\mu_h} > 1$ and $\frac{r}{\tilde{d}} > \frac{2\sqrt{\hat{K}}}{a+\rho}$, then the system has two interior equilibria $(S_h^k, I_h^k), k = 1, 2$ where (S_h^1, I_h^1) is always a saddle while (S_h^2, I_h^2) is always locally asymptotically stable.

Notice that $\frac{r}{\tilde{d}} > \frac{2\sqrt{\hat{K}}}{a+\rho} \Leftrightarrow \frac{r}{2\sqrt{\hat{K}}} > \frac{\tilde{d}}{a+\rho} = \frac{d_h(a+1)}{a+\rho} + \frac{\mu_h}{a+\rho} > d_h$. The discussions above show that the mite-free subsystem (8) has no interior equilibrium if either $\mathcal{R}_0^V < 1$ or $\frac{r}{2\sqrt{\hat{K}}} < \frac{\tilde{d}}{a+\rho}$ holds. This leads to the following two cases:

1. If either $\frac{r}{2\sqrt{\hat{K}}} < d_h$, then the mite-free subsystem (8) has only the extinction equilibrium $(0, 0)$ which is locally stable, thus it is globally stable.
2. If $\mathcal{R}_0^V > 1$ and $d_h < \frac{r}{2\sqrt{\hat{K}}} < \frac{\tilde{d}}{a+\rho}$, then the mite-free subsystem (8) has the following three boundary equilibria

$$(0, 0), (\bar{N}_h^c, 0), \text{ and } (\bar{N}_h^*, 0)$$

where $(0, 0)$ is locally asymptotically stable; $(\bar{N}_h^c, 0)$ is a source; and $(\bar{N}_h^*, 0)$ is a saddle. Then according to the Poincare-Bendixon theorem, we can conclude that all interior points of \mathbb{R}_+^2 converges to the

extinction equilibrium $(0, 0)$.

3. If $\mathcal{R}_0^V < 1$ and $d_h < \frac{r}{2\sqrt{\hat{K}}}$, then the mite-free subsystem (8) has the following three boundary equilibria

$$(0, 0), (\bar{N}_h^c, 0), \text{ and } (\bar{N}_h^*, 0)$$

where $(0, 0)$ is locally asymptotically stable; $(\bar{N}_h^c, 0)$ is a saddle; and $(\bar{N}_h^*, 0)$ is a sink. This implies that both $(0, 0)$, and $(\bar{N}_h^*, 0)$ are locally asymptotically stable. □

Proof of Theorem 3.5

Proof. Let $N_h = S_h + I_h$, then from Model (9), we obtain

$$\begin{aligned} N_h' &= \frac{r(S_h + \rho I_h)^2}{\hat{K} + (S_h + \rho I_h)^2} - d_h N_h - \mu_h I_h \\ &\leq \frac{r N_h^2}{\hat{K} + N_h^2} - d_h N_h \end{aligned}$$

which implies that when $\frac{r}{2\sqrt{\hat{K}}} > d_h$, we have

$$\limsup_{t \rightarrow \infty} (S_h(t) + I_h(t)) = \limsup_{t \rightarrow \infty} N_h(t) \leq \frac{\frac{r}{d_h} + \sqrt{\left(\frac{r}{d_h}\right)^2 - 4\hat{K}}}{2} = \bar{N}_h^*.$$

Therefore, we can conclude that if the inequalities $\frac{r}{d_h} > 2\sqrt{\hat{K}}$ and $\bar{N}_h^* < d_m + \mu_m$ hold, then we have $\limsup_{t \rightarrow \infty} S_m(t) = 0$ since

$$I_m' = c\alpha I_m \left[I_h + S_h - \frac{(d_m + \mu_m)}{c\alpha} \right] = c\alpha I_m \left[N_h - \frac{(d_m + \mu_m)}{c\alpha} \right] \leq c\alpha I_m \left[\bar{N}_h^* - \frac{(d_m + \mu_m)}{c\alpha} \right] < 0.$$

This implies that the limiting dynamics of Model (9) is reduced to the mite-free model (8).

An interior equilibrium (S_h, I_h, I_m) of the healthy-mite-free subsystem (9), satisfies the following equations:

$$\begin{aligned} c\alpha I_m \left[I_h + S_h - \frac{d_m + \mu_m}{c\alpha} \right] &= 0 \Rightarrow N_h = I_h + S_h = \frac{d_m + \mu_m}{c\alpha} \\ \frac{r(S_h + \rho I_h)^2}{\hat{K} + (S_h + \rho I_h)^2} - d_h N_h - \alpha N_h I_m - \mu_h I_h &= 0 \Rightarrow \frac{r(S_h + \rho I_h)^2}{\hat{K} + (S_h + \rho I_h)^2} - d_h N_h - \alpha N_h I_m - \mu_h I_h = 0 \\ \Rightarrow \frac{r(N_h - I_h + \rho I_h)^2}{\hat{K} + (N_h - I_h + \rho I_h)^2} - d_h N_h - \alpha N_h I_m - \mu_h I_h &= 0 \\ \Rightarrow I_m &= \frac{\frac{r\left(\frac{d_m + \mu_m}{c\alpha} - I_h + \rho I_h\right)^2}{\hat{K} + \left(\frac{d_m + \mu_m}{c\alpha} - I_h + \rho I_h\right)^2} - d_h \frac{d_m + \mu_m}{c\alpha} - \mu_h I_h}{\alpha \left(\frac{d_m + \mu_m}{c\alpha}\right)} = f_1(I_h) \end{aligned} \tag{18}$$

$$\begin{aligned}
S_h \left[\frac{\beta_h I_h}{S_h + I_h} + \frac{\hat{\beta}_{mh} I_m}{S_h + I_h} + \tilde{\beta}_{mh} I_m \right] &= \alpha I_h I_m + (d_h + \mu_h) I_h \\
\left(\frac{d_m + \mu_m}{c\alpha} - I_h \right) \left[\frac{\beta_h I_h}{d_m + \mu_m} + \frac{c\alpha \hat{\beta}_{mh} I_m}{d_m + \mu_m} + \tilde{\beta}_{mh} I_m \right] &= \alpha I_h I_m + (d_h + \mu_h) I_h \\
\Rightarrow I_m &= \frac{\left(\frac{d_m + \mu_m}{c\alpha} - I_h \right) \left[\frac{\beta_h}{d_m + \mu_m} - (d_h + \mu_h) \right] I_h}{\alpha I_h - \left(\frac{d_m + \mu_m}{c\alpha} - I_h \right) \left(\frac{c\alpha \hat{\beta}_{mh}}{d_m + \mu_m} + \tilde{\beta}_{mh} \right)} \\
\Rightarrow I_m &= \frac{\left(\frac{d_m + \mu_m}{c\alpha} - I_h \right) \left[\frac{\beta_h}{d_m + \mu_m} - (d_h + \mu_h) \right] I_h}{\left[\alpha + \frac{c\alpha \hat{\beta}_{mh}}{d_m + \mu_m} + \tilde{\beta}_{mh} \right] I_h - \frac{d_m + \mu_m}{c\alpha} \left(\frac{c\alpha \hat{\beta}_{mh}}{d_m + \mu_m} + \tilde{\beta}_{mh} \right)} = f_2(I_h)
\end{aligned} \tag{19}$$

The equations above imply that the interior equilibrium of (9) is the positive intercept of $f_1(I_h)$ and $f_2(I_h)$ subject to $0 < I_h < \frac{d_m + \mu_m}{c\alpha}$. The expression for the function $f_1(I_h)$ implies that the subsystem (9) has no interior equilibrium if

$$r < \frac{d_h(d_m + \mu_m)}{c\alpha} \Leftrightarrow r c\alpha < d_h(d_m + \mu_m)$$

since $I_m = f_1(I_h) < 0$ when this inequality holds.

Assume that $\frac{r}{2\sqrt{K}} > \frac{d_h + \mu_h + \alpha N^*}{\rho}$ and $N_h(0) > \underline{N}_h^c$, then according to Theorem 3.1 and 3.2, then we have

$$\underline{N}_h^c < \underline{N}_h^* \leq \liminf_{t \rightarrow \infty} N_h(t) \leq \limsup_{t \rightarrow \infty} N_h(t) \leq \bar{N}_h^*$$

which implies that the set $\{S_h + I_h \geq \underline{N}_h^c\}$ is invariant. If $I_m = 0$, then the subsystem (9) reduces to the mite-free system (8). According to Theorem 3.4, the omega limit set of the mite-free system (8) is $(\bar{N}_h^*, 0)$ when $\mathcal{R}_0^V = \frac{\beta_h}{d_h + \mu_h} < 1$ holds. If $\bar{N}_h^* > \frac{d_m + \mu_m}{c\alpha}$, then we have

$$\frac{I'_m}{I_m} \Big|_{I_m=0} = c\alpha \left(\bar{N}_h^* - \frac{d_m + \mu_m}{c\alpha} \right) > 0.$$

This implies that the virus I_m persists by applying the average Lyapunov theorem (Hutson 1984). Notice that

$$I_h \Big|_{I_h=0} = \frac{\hat{\beta}_{mh} S_h I_m}{N_h} > 0.$$

Therefore, the virus infected honeybees I_h also persists. \square

Proof of Theorem 3.6

Proof. The first part of Theorem 3.6 can be deduced directly from Theorem 3.1, 3.2, 3.3, 3.4, and 3.5. We focus on the sufficient conditions that lead to no interior equilibrium (Item 3) and the persistence of mites (Item 4).

If (S_h, I_h, S_m, I_m) is an interior equilibrium of the system (7), then it satisfies the following equations:

$$\begin{aligned}
0 &= S_m \left[c\alpha S_h - \frac{\hat{\beta}_{hm} I_h}{S_h + I_h} - d_m \right] \\
\Rightarrow I_h &= S_h \frac{c\alpha S_h - d_m}{d_m + \hat{\beta}_{hm} - c\alpha S_h}, \frac{d_m}{c\alpha} < S_h < \frac{d_m + \hat{\beta}_{hm}}{c\alpha} \\
\Rightarrow N_h &= S_h + I_h = \frac{\hat{\beta}_{hm} S_h}{d_m + \hat{\beta}_{hm} - c\alpha S_h} = g_1(S_h)
\end{aligned} \tag{20}$$

$$\begin{aligned}
0 &= \frac{r(S_h + \rho I_h)^2}{K + (S_h + \rho I_h)^2} - d_h(S_h + I_h) - \alpha(S_h + I_h)(S_m + I_m) - \mu_h I_h \\
&\Rightarrow N_m = S_m + I_m = \frac{\frac{r(S_h + \rho I_h)^2}{K + (S_h + \rho I_h)^2} - d_h(S_h + I_h) - \mu_h I_h}{\alpha(S_h + I_h)} \tag{21}
\end{aligned}$$

$$\Rightarrow N_m = \frac{rS_h(\hat{\beta}_{hm} + (d_m - c\alpha S_h))(\hat{\beta}_{hm} + (d_m - c\alpha S_h)(1-\rho))^2}{\alpha\hat{\beta}_{hm}\left[\hat{K}(\hat{\beta}_{hm} + (d_m - c\alpha S_h))^2 + S_h^2(\hat{\beta}_{hm} + (d_m - c\alpha S_h)(1-\rho))^2\right]} - \frac{d_h}{\alpha} - \frac{\mu_h(c\alpha S_h - d_m)}{\hat{\beta}_{hm}} = g_2(S_h)$$

$$\begin{aligned}
0 &= c\alpha(S_h + I_h)(S_m + I_m) - d_m(S_m + I_m) - \mu_m I_m \\
&\Rightarrow I_m = \frac{N_m(c\alpha(S_h + I_h) - d_m)}{\mu_m} = \frac{N_m[c\alpha\hat{\beta}_{hm}S_h - d_m(\hat{\beta}_{hm} + (d_m - c\alpha S_h))]}{\mu_m(\hat{\beta}_{hm} + (d_m - c\alpha S_h))} = \frac{N_m(c\alpha S_h - d_m)(\hat{\beta}_{hm} + d_m)}{\mu_m(\hat{\beta}_{hm} + (d_m - c\alpha S_h))} \tag{22} \\
&= N_m g_3(S_h) = g_2(S_h)g_3(S_h)
\end{aligned}$$

$$\begin{aligned}
0 &= S_h \left[\frac{\beta_h I_h}{S_h + I_h} + \frac{\hat{\beta}_{mh} I_m}{S_h + I_h} + \tilde{\beta}_{mh} I_m \right] - \alpha I_h (S_m + I_m) - (d_h + \mu_h) I_h \\
&\Rightarrow \frac{d_m + \hat{\beta}_{hm} - c\alpha S_h}{c\alpha S_h - d_m} \left[\frac{\beta_h(c\alpha S_h - d_m)}{\hat{\beta}_{hm}} + \hat{\beta}_{mh} \frac{N_m(c\alpha S_h - d_m)(\hat{\beta}_{hm} + d_m)}{\mu_m \hat{\beta}_{hm} S_h} + \tilde{\beta}_{mh} \frac{N_m(c\alpha S_h - d_m)(\hat{\beta}_{hm} + d_m)}{\mu_m(\hat{\beta}_{hm} + (d_m - c\alpha S_h))} \right] - \alpha N_m - (d_h + \mu_h) = 0 \\
&\Rightarrow \frac{\beta_h(d_m + \hat{\beta}_{hm} - c\alpha S_h)}{\hat{\beta}_{hm}} + \frac{\hat{\beta}_{mh} N_m(\hat{\beta}_{hm} + d_m)(d_m + \hat{\beta}_{hm} - c\alpha S_h)}{\mu_m \hat{\beta}_{hm} S_h} + \frac{\tilde{\beta}_{mh} N_m(\hat{\beta}_{hm} + d_m)}{\mu_m} - (d_h + \mu_h) = \alpha N_m \tag{23} \\
&\Rightarrow N_m = \frac{\frac{\beta_h(d_m + \hat{\beta}_{hm} - c\alpha S_h)}{\hat{\beta}_{hm}} - (d_h + \mu_h)}{\alpha - \frac{\hat{\beta}_{mh}(\hat{\beta}_{hm} + d_m)(d_m + \hat{\beta}_{hm} - c\alpha S_h) - \tilde{\beta}_{mh}(\hat{\beta}_{hm} + d_m)}{\mu_m \hat{\beta}_{hm} S_h}} = g_4(S_h)
\end{aligned}$$

Therefore, the interior equilibrium (S_h, I_h, S_m, I_m) are positive solutions of the following four equations:

$$\begin{aligned}
N_h &= S_h + I_h = g_1(S_h) = \frac{\hat{\beta}_{hm} S_h}{d_m + \hat{\beta}_{hm} - c\alpha S_h} \text{ subject to } S_h < \frac{d_m + \hat{\beta}_{hm}}{c\alpha} \\
\frac{I_m}{N_m} &= g_3(S_h) = \frac{(c\alpha S_h - d_m)(\hat{\beta}_{hm} + d_m)}{\mu_m(\hat{\beta}_{hm} + d_m - c\alpha S_h)} < 1 \text{ subject to } \frac{d_m}{c\alpha} < S_h < \frac{d_m + \hat{\beta}_{hm}}{c\alpha} \frac{d_m + \mu_m}{d_m + \hat{\beta}_{hm} + \mu_m} \\
N_m &= g_2(S_h) = \frac{rS_h(\hat{\beta}_{hm} + (d_m - c\alpha S_h))(\hat{\beta}_{hm} + (d_m - c\alpha S_h)(1-\rho))^2}{\alpha\hat{\beta}_{hm}\left[\hat{K}(\hat{\beta}_{hm} + (d_m - c\alpha S_h))^2 + S_h^2(\hat{\beta}_{hm} + (d_m - c\alpha S_h)(1-\rho))^2\right]} - \frac{d_h}{\alpha} - \frac{\mu_h(c\alpha S_h - d_m)}{\hat{\beta}_{hm}} \tag{24} \\
N_m &= g_4(S_h) = \frac{\frac{\beta_h(d_m + \hat{\beta}_{hm} - c\alpha S_h)}{\hat{\beta}_{hm}} - (d_h + \mu_h)}{\alpha - \frac{\hat{\beta}_{mh}(\hat{\beta}_{hm} + d_m)(d_m + \hat{\beta}_{hm} - c\alpha S_h) - \tilde{\beta}_{mh}(\hat{\beta}_{hm} + d_m)}{\mu_m \hat{\beta}_{hm} S_h}} \\
&= \frac{\mu_m S_h [\beta_h(d_m + \hat{\beta}_{hm}) - \hat{\beta}_{hm}(d_h + \mu_h) - c\alpha\beta_h S_h]}{[\alpha\mu_m \hat{\beta}_{hm} + c\alpha\hat{\beta}_{mh}(\hat{\beta}_{hm} + d_m) - \tilde{\beta}_{mh}\hat{\beta}_{hm}(\hat{\beta}_{hm} + d_m)]S_h - \hat{\beta}_{mh}(\hat{\beta}_{hm} + d_m)(d_m + \hat{\beta}_{hm})}
\end{aligned}$$

Thus, we can conclude the following statements regarding the sign of $g_4(S_h)$:

1. If $\beta_h(d_m + \hat{\beta}_{hm}) < \hat{\beta}_{hm}(d_h + \mu_h)$ and $\alpha\mu_m\hat{\beta}_{hm} + c\alpha\hat{\beta}_{mh}(\hat{\beta}_{hm} + d_m) < \tilde{\beta}_{mh}\hat{\beta}_{hm}(\hat{\beta}_{hm} + d_m)$, then $g_4(S_h) > 0$ for all $S_h > 0$.
2. If $\beta_h(d_m + \hat{\beta}_{hm}) > \hat{\beta}_{hm}(d_h + \mu_h)$ and $\alpha\mu_m\hat{\beta}_{hm} + c\alpha\hat{\beta}_{mh}(\hat{\beta}_{hm} + d_m) > \tilde{\beta}_{mh}\hat{\beta}_{hm}(\hat{\beta}_{hm} + d_m)$, then $g_4(S_h) > 0$ when

$$\frac{\hat{\beta}_{mh}(\hat{\beta}_{hm} + d_m)(d_m + \hat{\beta}_{hm})}{\alpha\mu_m\hat{\beta}_{hm} + c\alpha\hat{\beta}_{mh}(\hat{\beta}_{hm} + d_m) - \tilde{\beta}_{mh}\hat{\beta}_{hm}(\hat{\beta}_{hm} + d_m)} < S_h < \frac{\beta_h(d_m + \hat{\beta}_{hm}) - \hat{\beta}_{hm}(d_h + \mu_h)}{c\alpha\beta_h}$$

3. If $\beta_h(d_m + \hat{\beta}_{hm}) < \hat{\beta}_{hm}(d_h + \mu_h)$ and $\alpha\mu_m\hat{\beta}_{hm} + c\alpha\hat{\beta}_{mh}(\hat{\beta}_{hm} + d_m) > \tilde{\beta}_{mh}\hat{\beta}_{hm}(\hat{\beta}_{hm} + d_m)$, then $g_4(S_h) > 0$ when

$$S_h < \frac{\hat{\beta}_{mh}(\hat{\beta}_{hm} + d_m)(d_m + \hat{\beta}_{hm})}{\alpha\mu_m\hat{\beta}_{hm} + c\alpha\hat{\beta}_{mh}(\hat{\beta}_{hm} + d_m) - \tilde{\beta}_{mh}\hat{\beta}_{hm}(\hat{\beta}_{hm} + d_m)}.$$

4. If $\beta_h(d_m + \hat{\beta}_{hm}) > \hat{\beta}_{hm}(d_h + \mu_h)$ and $\alpha\mu_m\hat{\beta}_{hm} + c\alpha\hat{\beta}_{mh}(\hat{\beta}_{hm} + d_m) < \tilde{\beta}_{mh}\hat{\beta}_{hm}(\hat{\beta}_{hm} + d_m)$, then $g_4(S_h) > 0$ when

$$S_h > \frac{\beta_h(d_m + \hat{\beta}_{hm}) - \hat{\beta}_{hm}(d_h + \mu_h)}{c\alpha\beta_h}.$$

Notice that the interior equilibrium (S_h, I_h, S_m, I_m) requires $g_3(S_h) > 0$, i.e., $\frac{d_m}{c\alpha} < S_h < \frac{d_m + \hat{\beta}_{hm}}{c\alpha} \frac{d_m + \mu_m}{d_m + \hat{\beta}_{hm} + \mu_m}$. Therefore, the interior equilibrium (S_h, I_h, S_m, I_m) does not exist if one of the following inequalities hold

1. $\frac{\beta_h}{d_h + \mu_h} > \frac{\hat{\beta}_{hm}}{d_m + \hat{\beta}_{hm}}$, $\frac{\alpha\mu_m\hat{\beta}_{hm}}{\hat{\beta}_{hm} + d_m} > \tilde{\beta}_{mh}\hat{\beta}_{hm} - c\alpha$ and

$$\frac{\hat{\beta}_{mh}}{\frac{\alpha\mu_m\hat{\beta}_{hm}}{(d_m + \hat{\beta}_{hm})} + c\alpha\hat{\beta}_{mh} - \tilde{\beta}_{mh}\hat{\beta}_{hm}} > \frac{d_m + \mu_m}{c\alpha(d_m + \hat{\beta}_{hm} + \mu_m)} \text{ or } \frac{\frac{\beta_h}{d_h + \mu_h} - \frac{\hat{\beta}_{hm}}{d_m + \hat{\beta}_{hm}}}{\beta_h(d_m + \hat{\beta}_{hm})(d_h + \mu_h)} < d_m.$$

2. $\frac{\beta_h}{d_h + \mu_h} < \frac{\hat{\beta}_{hm}}{d_m + \hat{\beta}_{hm}}$, $\frac{\alpha\mu_m\hat{\beta}_{hm}}{\hat{\beta}_{hm} + d_m} > \tilde{\beta}_{mh}\hat{\beta}_{hm} - c\alpha$ and $\frac{\hat{\beta}_{mh}(d_m + \hat{\beta}_{hm})}{\frac{\alpha\mu_m\hat{\beta}_{hm}}{(d_m + \hat{\beta}_{hm})} + c\alpha\hat{\beta}_{mh} - \tilde{\beta}_{mh}\hat{\beta}_{hm}} < \frac{d_m}{c\alpha}$.

3. $\frac{\beta_h}{d_h + \mu_h} > \frac{\hat{\beta}_{hm}}{d_m + \hat{\beta}_{hm}}$, $\frac{\alpha\mu_m\hat{\beta}_{hm}}{\hat{\beta}_{hm} + d_m} < \tilde{\beta}_{mh}\hat{\beta}_{hm} - c\alpha$ and $\frac{\frac{\beta_h}{d_h + \mu_h} - \frac{\hat{\beta}_{hm}}{d_m + \hat{\beta}_{hm}}}{\beta_h(d_m + \hat{\beta}_{hm})(d_h + \mu_h)} > \frac{(d_m + \mu_m)(d_m + \hat{\beta}_{hm})}{d_m + \hat{\beta}_{hm} + \mu_m}$.

Assume that $\frac{r}{2\sqrt{K}} > \frac{d_h + \mu_h + \alpha N^*}{\rho}$ and $N_h(0) > \underline{N}_h^c$, then according to Theorem 3.1 and 3.2, then we have

$$\underline{N}_h^c < \underline{N}_h^* \leq \liminf_{t \rightarrow \infty} N_h(t) \leq \limsup_{t \rightarrow \infty} N_h(t) \leq \bar{N}_h^*$$

which implies that the set $\{S_h + I_h \geq \underline{N}_h^c\}$ is invariant. If $S_m = I_m = 0$, then the full system (7) reduces to the mite-free system (8). According to Theorem 3.4, the omega limit set of the mite-free system (8) is $(\bar{N}_h^*, 0)$ when $\mathcal{R}_0^V = \frac{\beta_h}{d_h + \mu_h} < 1$ holds. If $\bar{N}_h^* > \frac{d_m + \mu_m}{c\alpha}$, then we have

$$\frac{N'_m}{N_m} \Big|_{N_m=0} \geq c\alpha \left(\bar{N}_h^* - \frac{d_m + \mu_m}{c\alpha} \right) > 0.$$

This implies that the mite population N_m persists by applying the average Lyapunov theorem (Hutson 1984).

□

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