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# Epidemiological Modelling of the Population Dynamics of Bee Colonies

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**Abstract**

A mathematical model that combines the normal demographic dynamics of a bee colony, including the population of the broods, with the dynamics of *Nosema* infection affecting foragers during foraging duties was designed. The model included the brood population and determined how it affects the population dynamics of the colony. An epidemiological threshold called the basic reproduction number of the model was derived and a qualitative analysis of the model was carried out to investigate the asymptotic stability of both the disease-free and endemic equilibria. A locally asymptotically stable disease-free equilibrium at the basic reproduction number less than unity was proven via the analysis of characteristic equation. Furthermore, the existence of a locally asymptotically stable endemic equilibrium was established at the basic reproduction number greater than unity based on the use of the center manifold theory of bifurcation. In addition, a sensitivity analysis was performed to examine the contributory effects of the model parameters on the transmission and spread of the *Nosema* infection with respect to the basic reproduction number. Lastly, numerical simulations using Python were conducted to analyze the dynamics of the colony in the presence of infection.

## 1 Introduction

Most of the Philippine population relies in some way on agriculture for a living and food production is an essential industry for everyone. The Economic Value of Insect Pollination (EVIP) for the Philippines in 2009 was assessed by Food and Agriculture Organization methods to be $710 million (US). Crops associated with insect (mainly bee) pollination such as mangos, coconuts, coffee and cotton are producing significantly less than their potential compared to other ASEAN countries. The decline in many crop yields has coincided with the ongoing removal of the main insect pollinators from the agricultural regions. The major limiting factor for many Philippine crops is poor insect pollination due to insecticides and continuing destruction of the key beneficial insects, which are the honeybees and stingless bees [33].

In the Philippines 75% of the food crops need insect, mainly bees, pollinators for good fruit and seed production for sufficient successful pollination. This currently equates to 35% by volume largely due to the massive production of wind-pollinated staple crops such as cereals and

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sugarcane. Specifically, there are several types of bees, mostly native to the Philippines, which are particularly important. These are the *Apis cerana* (Asian Honeybees) and *Apis andreniformis* (Dwarf Honeybees) or collectively known as 'Ligwan', *Apis dorsata* (Wild Honeybees) or natively known as 'Putyokan', *Tetragonula species* (Stingless Bees) natively known as 'Kiwot' and the *Apis mellifera* (European Honeybees) [33]. Hence, it is evident that if bee colonies collapse, Philippine agriculture, specifically food production, will decline.

The widespread collapse of honey bee colonies has been the subject of much discussion and research in recent years [8, 16, 31]. Aside from their ecological importance [4], honey bee populations have a large economical impact on agriculture in North America, Europe, the Middle East, and Japan [8, 26, 31]. The focus of Betti et. al. [1] has been largely on environmental factors outside the hive, such as pesticides or insecticides, which may cause death or injury to foraging bees and jeopardize their return to the hive. The reduced number of foraging bees then leads to younger hive bees being recruited prematurely to perform foraging duties and this chain reaction ultimately leads to a disruption in the dynamics of the colony as a whole. Examples of this scenario would be produced by the effects of various pesticides to which foraging bees are exposed in the course of their duties [31, 14]. Other factors in the same category include possible disruptions to the bees' navigation system by mobile phones or other electronic devices, again to the effect of jeopardizing their return to the hive and thereby reducing their numbers [10].

A key element in this category of disruption to honey bee population dynamics is the untimely death of a certain proportion of foraging bees outside the hive and the consequences of this on the colony as a whole. An important question here concerns the threshold in the death rate of foraging bees that would determine the survival or collapse of the bee colony. This was examined recently in two papers by Khoury et. al. [20, 21].

In Betti et. al. [1], the researchers considered a different category of disruption to the healthy dynamics of a bee colony, namely one in which the key hazard is an infection by a communicable disease acquired by foraging bees outside the hive. The key difference there is that foraging bees that have been infected would then transport the disease into the hive and go on to infect other members of the colony within the hive. The affected bees will ultimately suffer an untimely death, but the effects on the dynamics of the colony are clearly more complex because the infection in this case may now involve all members of the colony. They researchers sought a model that would allow a comparison between the effects of these two categories of hazards on the ultimate fate of the bee colony.

The intention of this endeavor is to create an epidemiological model of the effects of infection on honeybee population dynamics following the study of Betti et. al. [1] and incorporating a more general model which combines the normal dynamics of a honey bee colony with the dynamics of an infectious disease which is acquired outside the hive but ultimately spreads to the rest of the colony which was developed by Khoury et. al [20, 21]. This study sought to develop a model that provides a possible mechanism for the linkage in terms of the interplay between the dynamics of the infection and the normal dynamics of the honey bee colony. However, in this paper, we include the population of the brood and explored the effects of this inclusion on the population dynamics of the colony.

## 2 Background of the Study

### 2.1 Normal Demographics of a Bee Colony

Honey bee colonies are complex societies in which different members of the colony have specialized functions that serve the entire colony, thus making members of the colony highly

dependent on each other [28].

The queen can live up to three years, is responsible for laying eggs, and during peak season may lay up to 2000 eggs per day [27]. In this function the queen is dependent on worker bees [32]. The worker bees emerge from fertilized eggs of the queen and consist of females who maintain the hive and gather resources, and males who mate with the queen to produce more eggs Drones are born from unfertilized eggs of the queen [28] and typically making up less than 5% of the hive population [28, 18]. Because they do not contribute to the colony work force, and because of their small numbers, they are generally neglected when considering the dynamics of the colony as a whole.

Female hive bees, following a transition period, leave the hive to start foraging duties and usually forage until their death. The age at which they start foraging duties is variable, depending on the state of the colony and its needs. If the number of forager bees is lower than is required for meeting the colony needs, hive bees will begin foraging duties at a younger age [17]. If the number of forager bees is higher than required, behavioral maturation of hive bees will be regulated by a pheromone, ethyl oleate, produced by the foragers. This process is usually referred to as *social inhibition* [22]. Similarly, if the number of hive bees is too low, it is possible for foragers to revert back to hive bee duties [17].

## 2.2 Methodology

Communicable diseases have always been an important part of human history as well as other living organisms. Mathematical analysis and modelling is central to infectious disease epidemiology. In this paper, we provide an intuitive extension of the researches of Betti et. al. [1] and Khoury et. al. [20, 21] to the process of disease transmission, how this process can be presented mathematically and how this mathematical representation can be used to analyze the emergent dynamics of the introduced infection to the bee colony. Below is the flow of methods and processes used in this paper:

(1) In the construction of the model, the preliminary concepts of Betti et. al. [1] and Khoury et. al. [20, 21] were followed. However, in this paper, we added another compartment and observed the effects of this added consideration to the population dynamics of bee colonies and formulated assumptions for the dynamics of the constructed model.

(2) Properties of the model namely the feasibility, positivity, existence and uniqueness of the solution were analyzed using known results and determine the biological meaning of the solutions of the constructed model equations.

(3) In the absence of the infectious disease, we solved for the disease free state by solving the simultaneous equations using elementary elimination and substitution. The solution were checked using the basic substitution and was verified using Python.

(4) The basic reproduction number was solved using the Next Generation Matrix (NGM) method and linearization technique by Diekmann et. al. [5, 6].

(5) The stability analysis of the disease-free steady state is done using the analysis of the characteristic equation, Routh-Hurwitz criterion, and the Descartes' rule of signs.

(6) Since the explicit form of the endemic equilibrium point of the model is burdensome to obtain, the existence and the stability of the endemic equilibrium was established instead, using the center manifold theory of bifurcation analysis described in Castillo-Chavez and Song [2].

(7) Following the basic sensitivity analysis done by Obabiyi et. al. [24], we performed the analysis on the sensitivity indices of the basic reproduction number to determine several parameters that have the most influence on the prevalence and transmission of the infection in the colony.

(8) Numerical simulations of the key scenarios that illustrates the main dynamics of the bee colony in the presence of the infectious disease were explored and investigated using Python.

## 3 Construction of the Mathematical Model

In this paper, we consider population of the adult bees (hive bees and foragers) including the surviving population of the broods that may also be infected through the food they consume or the infected hive bees that nurse them. Thus, in this model the hive population is divided into five compartments, namely susceptible broods $B_S$, susceptible hive bees $H_S$, infected hive bees $H_I$, susceptible foraging bees $F_S$ and infected foraging bees $F_I$. The dynamics of the model is formulated under the following assumptions:

(a) susceptible broods emerge from the eggs layed by the queen at rate $L$ considering the survival rate modelled by the function $S$;

(b) susceptible broods become infected only through the interaction of the infected hive bees that nurse them;

(c) susceptible broods that become infected will not survive the pupation stage and will suffer death due to the infection hence cannot become hive bees;

(d) susceptible hive bees emerge from the susceptible broods considering that the broods survive the pupation duration $\phi$;

(e) infected hive bees emerge from the susceptible hive bees through interacting with infected hive bees and foragers;

(f) infected hive bees cannot be recruited to foraging duties for it is assumed that they will suffer death due to infection;

(g) susceptible foragers are recruited from susceptible hive bees through the recruitment function $R$ and may suffer natural death during foraging duties; and

(h) foragers become infected only during foraging duties outside the hive and may suffer both natural death or death due to infection.

Let $f$ be the amount of food that is stored in the hive and available for the colony to use. Following Khoury *et. al.* [21], we also assumed that the survival of uncapped broods is dependent on the number of hive bees available to tend and feed them, on food availability and on the laying rate $L$ of the queen. Larvae become pupae inside cells that are capped by worker bees and we assume that pupation occurs at a constant rate proportional to the amount of brood present.

The rate of change of number of susceptible broods in time $t$ days is assumed to be governed by the equation

$$\frac{dB_S}{dt} = LS - \phi B_S - \beta_{BH} H_I B_S \tag{1}$$

where $L$ represents the queen's egg laying rate per day, $S$ is a function of food and hive bee numbers. We assume that $S$ becomes constant when $H_S + H_I$ become large and that the dependence of food and hive bee numbers is independent of one another. Also, it is assumed that $f$ is sufficient for consumption in the hive. With these assumptions, $S$ is given by

$$S = \frac{H_S + H_I}{w + H_S + H_I} \tag{2}$$

where $w$ is a parameter that determine how rapidly $S$ tends to one as $H_S + H_I$ increases. Again, following Khoury *et. al.* [13], $S$ is modelled the way that brood survival declines when food stores are low.

We assume that the broods entering the pupation stage are the same broods that will become adult hive bees excluding the bees that will become infected. The last term of the (1) determines the rate at which susceptible broods become infected. The transmission rate per day per susceptible brood is given by $\beta_{BH} H_I$ where $\beta_{BH}$ is interaction rate between broods and hive bees. It is assumed that the natural death of the broods is negligible compared to their survival rate to become adult bees.

Again, infected broods are assumed to die eventually due to the infection and cannot survive the pupation stage and therefore cannot become hive bees. The rate of change of susceptible hive bees is given by the equation

$$\frac{dH_S}{dt} = \phi B_S - RH_S - (\beta_{HH} H_I + \beta_{HF} F_I) H_S \tag{3}$$

where $RH_S$ represents the rate that hive bees become foragers.Since it is assumed that there is an abundance of food, we write the recruitment function as

$$R = R_b - \alpha_F \left( \frac{F_S + F_I}{N} \right) \tag{4}$$

where $N$ is the total population of adult bees. That is, $N = H_S + H_I + F_S + F_I$ and $R_b$ is the baseline recruitment rate in the absence of foragers but sufficient food stores. Social inhibition depends on the proportion of foragers in the adult bee population, and the strength of this inhibition is governed by $\alpha_F$. The forager-to-hive bee transition depends on food stores in a similar way to how brood survival depends on food stores. This implicitly assumes that shortage of food for the larvae is one of the stimuli that drive increased forager recruitment.

For the rate of change of the infected hive bee population, we have

$$\frac{dH_I}{dt} = (\beta_{HH} H_I + \beta_{HF} F_I) H_S - d_H H_I. \tag{5}$$

Here, we assume that infected hive bees cannot be recruited to foraging duties because they are at risk of dying from the disease before they can be recruited. Thus, we denote $d_H$ as the death rate at which this happens.

Susceptible foragers are recruited from susceptible hive bees and may subsequently suffer natural death, at a rate $m$, or become infected. Their rate of change is therefore governed by the equation

$$\frac{dF_S}{dt} = RH_S - \beta_{FF} F_I F_S - mF_S. \tag{6}$$

Infected foragers are recruited from susceptible foragers that have become infected. If the death rate from the infection is assumed to be $d_F$ then their rate of change is governed by

$$\frac{dF_I}{dt} = \beta_{FF} F_I F_S - (m + d_F) F_I. \tag{7}$$

Note that the susceptible foragers can only be infected during foraging duties and from interacting with other infected foragers outside the hive.

The full dynamics of the bee colony are thus governed by the following equations to be solved simultaneously:

$$\frac{dB_S}{dt} = LS - \phi B_S - \beta_{BH} H_I B_S \tag{8}$$

$$\frac{dH_S}{dt} = \phi B_S - RH_S - (\beta_{HH} H_I + \beta_{HF} F_I) H_S \tag{9}$$

$$\frac{dH_I}{dt} = (\beta_{HH} H_I + \beta_{HF} F_I) H_S - d_H H_I \tag{10}$$

$$\frac{dF_S}{dt} = RH_S - \beta_{FF} F_I F_S - mF_S \tag{11}$$

$$\frac{dF_I}{dt} = \beta_{FF} F_I F_S - (m + d_F) F_I \tag{12}$$

The susceptible broods $B_S$, susceptible and infected hive bees, $H_S$ and $H_I$, live within the hive. New susceptible broods emerge from the eggs laid by the queen and are generated by surviving eggs through the survival function, $S$. New susceptible hive bees are generated by surviving broods through the survival function $\phi B_S$. New infected hive bees are generated through interactions of susceptible broods, susceptible hive bees, infected hive bees and infected foragers at rates $\beta_{HH}$ and $\beta_{HF}$. Hive bees are recruited to the survival foraging duties through the recruitment function $R$, which also allows for the reversal of duties, from foraging to hive duties. Foragers move into the infected compartment via interactions with infected foragers at rate $\beta_{FF}$. All infected bees die at rates $d_H$ or $d_F$, and foragers die naturally at rate $m$. A compartmental diagram of these dynamics is shown in Figure 1.

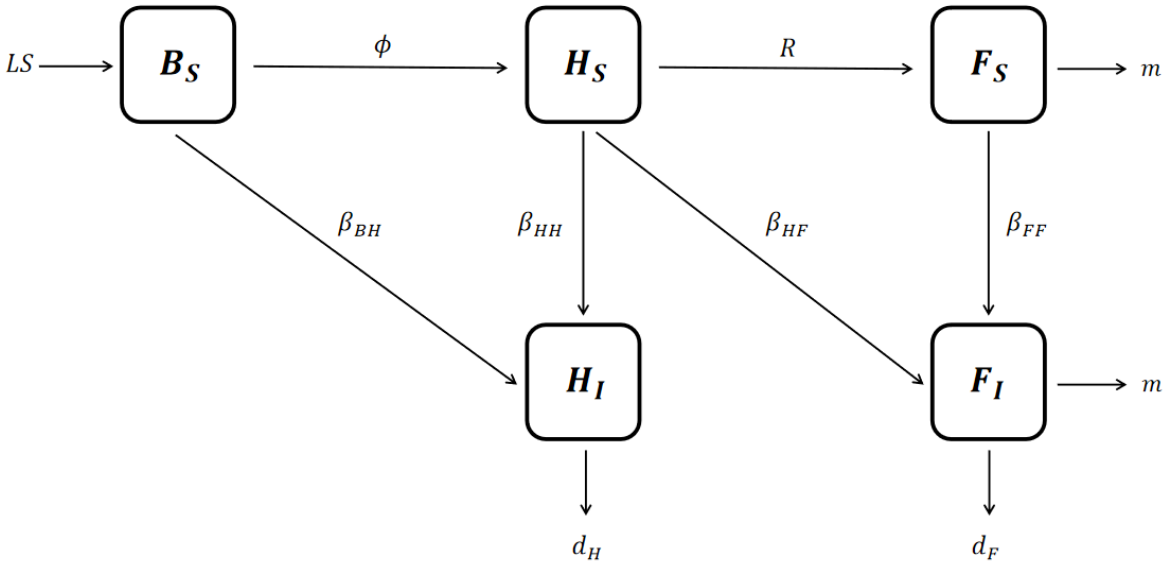


Figure 1: A compartmental diagram of the dynamics of the honey bee colony, including the broods, combined with the dynamics of an infectious disease.

Below is the summary of the variables and parameters used in constructing the model.

| Notation | Description |
|---|---|
| $\phi$ | - pupation rate |
| $L$ | - queen's egg laying rate |
| $R_b$ | - baseline recruitment rate |
| $\alpha_F$ | - effect of excess foragers on recruitment |
| $d_H$ | - death rate of hive bees due to infection |
| $d_F$ | - death rate of foragers due to infection |
| $m$ | - natural death rate of foragers |
| $w$ | - number of hive bees for 50% egg survival |
| $\beta_{BH}$ | - disease transmission rate: brood to hive bee |
| $\beta_{HH}$ | - disease transmission rate: hive bee to hive bee |
| $\beta_{HF}$ | - disease transmission rate: hive bee to forager |
| $\beta_{FF}$ | - disease transmission rate: forager to forager |

Table 1: List of variables and parameters.

# 4 Dynamical Properties of the Model

## 4.1 Positivity of Solutions

The model used describe the effects of *Nosema* infection to the population dynamics of bee colonies. It suffices to consider the case of strictly positive initial data. That is, it is assumed that the state variables are nonnegative. The following result establishes the positivity of the solution of the model.

**Theorem 4.1.** *The initial value problem of (8)-(12) with* $(B_S(0), H_S(0), H_I(0), F_S(0), F_I(0)) \in \omega$ *where*

$$\omega = \left\{(B_S, H_S, H_I, F_S, F_I) \in \mathbb{R}^5_+ \cup \{0\} : B_S(0), H_S(0), H_I(0), F_S(0), F_I(0) \geq 0\right\}$$

*possesses a nonnegative solution for all* $t > 0$*. That is,* $\omega$ *is positively invariant and attracts all solutions in* $\mathbb{R}^5 \cup \{0\}$*.*

## 4.2 Boundedness of the Solutions

The boundedness of solutions can be understood as a natural limitation for the population growth as a consequence of limited resources and the effect of the infection. The boundedness of the solution of the system (8)-(12) is presented as follows.

**Theorem 4.2.** *Let the system (8)-(12) be an epidemic model. Then the following hold:*

*(i) The number of broods* $B_S$ *is bounded above, i.e.,*

$$\limsup_{t\to+\infty} B_S(t) \leq \frac{L}{\phi + \beta_{BH}}.$$

*(ii) The number of hive bees* $H_S + H_I$ *is bounded above, i.e.,*

$$\limsup_{t\to+\infty} [H_S(t) + H_I(t)] \leq \frac{\phi L + \alpha_F(\phi + \beta_{BH})}{R_b(\phi + \beta_{BH})}.$$

*(iii) The number of forager bees $F_S + F_I$ is bounded above, i.e.,*

$$\limsup_{t\to+\infty}[F_S(t) + F_I(t)] \leq \frac{\phi L + \alpha_F(\phi + \beta_{BH})}{m(\phi + \beta_{BH})}.$$

*(iv) Let $M(t)$ be the total population of the bee colony. The system (8)-(12) is uniformly bounded, i.e.,*

$$\limsup_{t\to+\infty} M(t) \leq \frac{L}{\beta_{BH} + m + d}.$$

## 4.3 Local Asymptotic Stability

At this juncture, we analyse the behaviour of the dynamics governed by model (8)-(12) as its solutions approach the disease-free and endemic equilibria.

### 4.3.1 Disease-Free Equilibrium

The disease-free equilibrium (DFE) is defined as the point at which no disease is present in the population. In the absence of the infectious disease, the model (8)-(12) has a unique disease-free steady state

$$E_0 = (B_S^*, H_S^*, 0, F_S^*, 0).$$

Setting $\frac{dB_S}{dt}, \frac{dH_S}{dt}, \frac{dF_S}{dt} = 0$ and using elementary elimination and substitution, we have the disease-free equilibrium $E_0(B_S^*, H_S^*, 0, F_S^*, 0)$ where $2L + w\epsilon > w\sqrt{\epsilon^2 + 4mR_b}$ with

$$B_S^* = \frac{(2L + w\epsilon) - w\sqrt{\epsilon^2 + 4mR_b}}{2\phi} \tag{13}$$

$$H_S^* = \frac{(2L + w\epsilon) - w\sqrt{\epsilon^2 + 4mR_b}}{\sqrt{\epsilon^2 + 4mR_b} - \epsilon} \tag{14}$$

$$F_S^* = \frac{(2L + w\epsilon) - w\sqrt{\epsilon^2 + 4mR_b}}{2m} \tag{15}$$

where $\epsilon = m + \alpha_F - R_b$.

### 4.3.2 Basic Reproduction Number

One of the most important concerns about any infectious diseases is its ability to invade a population. Many epidemiological models have a DFE at which the population remains in the absence of disease. These models usually have a threshold parameter, known as the *basic reproduction number* $\mathcal{R}_0$ defined to be the expected number of secondary infection caused by a single infectious individual during its entire infectious lifetime. If $\mathcal{R}_0 < 1$, then the DFE is locally asymptotically stable, and the disease cannot invade the population. But if $\mathcal{R}_0 > 1$, then the DFE is unstable and invasion is always possible.

Following the process in calculating the basic reproduction number $\mathcal{R}_0$ presented in [5] using the Next Generation Matrix (NGM) method, the infected subsystem is given by

$$\frac{dH_I}{dt} = (\beta_{HH}H_I + \beta_{HF}F_I)H_S - d_H H_I \tag{16}$$

$$\frac{dF_I}{dt} = \beta_{FF}F_I F_S - (m + d_F)F_I \tag{17}$$

and is linearized using the DFE. The transmission matrix $\mathbf{T}$, which contains the entries corresponding to the transmission events, is given by

$$\mathbf{T} = \begin{bmatrix} \beta_{HH} H_S & \beta_{HF} H_S \\ 0 & \beta_{FF} F_S \end{bmatrix} \tag{18}$$

where $B_S, H_S$ and $F_S$ are the steady state solutions of the disease free system and the transition matrix $\mathbf{S}$, which contains the entries corresponding to all other changes of state (specifically death), is given by

$$\mathbf{S} = \begin{bmatrix} -d_H & 0 \\ 0 & -(m + d_F) \end{bmatrix}. \tag{19}$$

The $ij^{th}$ entry of $-\mathbf{S}^{-1}$ can be interpreted as the expected time that a bee who presently has an infected state $j$ will spend in infected state $i$ until it dies. Because the $ij^{th}$ entry of $\mathbf{T}$ is the rate at which a bee in infected state $j$ produces bees with infected state $i$, the $ij^{th}$ entry of $\mathbf{K} = -\mathbf{T}\mathbf{S}^{-1}$ is the expected number of infected offspring with state $i$ at infection produced throughout its entire future infected life by a bee presently in infected state $j$. Hence, the NGM denoted by $\mathbf{K}$, is given by

$$\mathbf{K} = -\mathbf{T}\mathbf{S}^{-1} = \begin{bmatrix} \frac{\beta_{HH} H_S}{d_H} & \frac{\beta_{HF} H_S}{m+d_F} \\ 0 & \frac{\beta_{FF} F_S}{m+d_F} \end{bmatrix}.$$

The basic reproduction number $\mathcal{R}_0$ represents the average number of secondary infections that occur when the infectious forager bees are introduced into a completely susceptible bee colony. It can be obtained by solving for the dominant eigenvalue of $\mathbf{K}$ and is given by

$$\mathcal{R}_0 = \frac{\beta_{FF} F_S}{m + d_F} = \frac{\beta_{FF}[(2L + w\epsilon) - w\sqrt{\epsilon^2 + 4mR_b}]}{2m(m + d_F)}.$$

### 4.3.3 Stability of the Disease-Free Steady State

In the absence of the infectious disease, the model (8)-(12) has a unique DFE $E_0(B_S^*, 0, H_S^*, 0, F_S^*, 0)$ with $B_S^*, H_S^*$ and $F_S^*$ given by (19)-(21). In the following theorem, the local stability of the disease-free steady state is established.

**Theorem 4.3.** *The disease-free steady state $E_0$ of the model (8)-(12) is locally asymptotically stable if $\mathcal{R}_0 < 1$ and unstable if $\mathcal{R}_0 > 1$.*

*Proof.* A necessary and sufficient condition for local asymptotic stability is for the real part of the eigenvalue to be in the negative half plane. The Jacobian of the model evaluated at the disease-free equilibrium point $E_0$ is obtained as

$$J_{E_0}^* = \begin{bmatrix} \frac{\partial B_S^*}{\partial B_S} & \frac{\partial B_S^*}{\partial H_S} & \frac{\partial B_S^*}{\partial H_I} & 0 & 0 \\ \frac{\partial H_S^*}{\partial B_S} & \frac{\partial H_S^*}{\partial H_S} & \frac{\partial H_S^*}{\partial H_I} & \frac{\partial H_S^*}{\partial F_S} & \frac{\partial H_S^*}{\partial F_I} \\ 0 & 0 & \frac{\partial H_I^*}{\partial H_I} & 0 & \frac{\partial H_I^*}{\partial F_I} \\ 0 & \frac{\partial F_S^*}{\partial H_S} & \frac{\partial F_S^*}{\partial H_I} & \frac{\partial F_S^*}{\partial F_S} & \frac{\partial F_S^*}{\partial F_I} \\ 0 & 0 & 0 & 0 & \frac{\partial F_I^*}{\partial F_I} \end{bmatrix}.$$

Thus, we need to show that $J^*_{E_0}$ has all its eigenvalues with negative real part. Now, the characteristic equation of $J^*_{E_0}$ is of the form

$$(\lambda + Q_1)(\lambda + Q_2)(\lambda + Q_3)(\lambda + Q_4)(\lambda + Q_5) = 0$$

where

$$\begin{aligned}
Q_1 &= \phi - \beta_{BH} \\
Q_2 &= R_b - \alpha_F \left(\frac{F_S}{H_S + F_S}\right)^2 \\
Q_3 &= -\beta_{HH} H_S + d_H \\
Q_4 &= \alpha_F \left(\frac{H_S}{H_S + F_S}\right)^2 + m \\
Q_5 &= -\beta_{FF} F_S + (m + d_F).
\end{aligned}$$

Further expansion of the characteristic equation gives

$$C_5\lambda^5 + C_4\lambda^4 + C_3\lambda^3 + C_2\lambda^2 + C_1\lambda + C_0 = 0 \tag{20}$$

where

$$\begin{aligned}
C_5 &= 1 \\
C_4 &= Q_1 + Q_2 + Q_3 + Q_4 + Q_5 \\
C_3 &= Q_1Q_2 + Q_1Q_3 + Q_1Q_4 + Q_1Q_5 + Q_2Q_3 + Q_2Q_4 + Q_2Q_5 + Q_3Q_4 + Q_3Q_5 + Q_4Q_5 \\
C_2 &= Q_1Q_2Q_3 + Q_1Q_2Q_4 + Q_1Q_2Q_5 + Q_1Q_4Q_5 + Q_1Q_3Q_5 + Q_1Q_3Q_4 + Q_2Q_3Q_5 \\
&\quad + Q_2Q_4Q_5 + Q_2Q_3Q_5 + Q_2Q_3Q_4 \\
C_1 &= Q_1Q_2Q_3Q_4 + Q_1Q_2Q_3Q_5 + Q_1Q_2Q_4Q_5 + Q_1Q_3Q_4Q_5 + Q_2Q_3Q_4Q_5 \\
C_0 &= Q_1Q_2Q_3Q_4Q_5.
\end{aligned}$$

Since $Q_5 = -\beta_{FF}F_S + (m + d_F)$ and $\mathcal{R}_0 = \dfrac{\beta_{FF}F_S}{m + d_F}$, we can write $Q_5$ as $Q_5 = (m + d_F)(1 - \mathcal{R}_0)$. Hence, it is easy to see that $C_0$ can be written in terms of $\mathcal{R}_0$ as

$$C_0 = Q_1Q_2Q_3Q_4(m + d_F)(1 - \mathcal{R}_0).$$

If $\mathcal{R}_0 < 1$, then $C_0 > 0$. Since the coefficients $C_i, i = 1, 2, 3, 4, 5$ and the Hurwitz matrices of the polynomial are positive and both parameters $m$ and $d_F$ are nonnegative, using Routh-Hurwitz criterion, all the eigenvalues of have negative real parts. Therefore, the disease-free equilibrium $E_0$ is stable. Otherwise, whenever $\mathcal{R}_0 > 1$, then $C_0 < 0$. By the Descartes' rule of signs, there exists one eigenvalue with positive real part. Hence, $E_0$ is unstable for $\mathcal{R}_0 > 1$. □

The epidemiological implication of the above result is that the infection governed by the model can be eliminated from the population whenever an influx by infectious individuals is small such that $\mathcal{R}_0 < 1$.

### 4.3.4 Stability of the Endemic Equilibrium

The steady-state solution of the model when all the state variables are positive is referred to as the endemic equilibrium point denoted by $E_e = (B^{**}_S, H^{**}_S, H^{**}_I, F^{**}_S, F^{**}_I)$. It is quite burdensome to obtain the explicit form of the endemic equilibrium point of the model. However, the existence and local stability of $E_e$ shall be explored using a center manifold theory of

bifurcation analysis described in Castillo-Chavez et. al. [2]. To this purpose, let the epidemic model be written in the vector form

$$\frac{dX}{dt} = F(X),$$

where $X = (x_1, x_2, x_3, x_4, x_5)^T$ and $F = (f_1, f_2, f_3, f_4, f_5)^T$ with $x_1 = B_S, x_2 = H_S, x_3 = H_I$, $x_4 = F_S$ and $x_5 = F_I$. Then (8)-(12) becomes

$$f_1 := \frac{dx_1}{dt} = LS - \phi x_1 - \beta_{BH} x_3 x_1 \tag{21}$$

$$f_2 := \frac{dx_2}{dt} = \phi x_1 - Rx_2 - (\beta_{HH} x_3 + \beta_{HF} x_5) x_2 \tag{22}$$

$$f_3 := \frac{dx_3}{dt} = (\beta_{HH} x_3 + \beta_{HF} x_5) x_2 - d_H x_3 \tag{23}$$

$$f_4 := \frac{dx_4}{dt} = Rx_2 - (\beta_{FH} x_3 + \beta_{FF} x_5) x_4 - m x_4 \tag{24}$$

$$f_5 := \frac{dx_5}{dt} = \beta_{FF} x_5 x_4 - (m + d_F) x_5 \tag{25}$$

with $S = \dfrac{x_2}{w + x_2}$ and $R = R_b - \alpha_F \left( \dfrac{x_4}{x_2 + x_4} \right)$.

**Theorem 4.4.** *The epidemic model exhibits a (supercritical) forward bifurcation at the threshold $\mathcal{R}_0 = 1$. Equivalently, there exists an endemic equilibrium $E_e$ which is locally asymptotically stable whenever $\mathcal{R}_0 > 1$ but near $\mathcal{R} = 1$.*

*Proof.* To investigate the type of bifurcation, we let

$$\beta^* = \frac{2m(m + d_F)}{2L + w\epsilon - w\sigma} \tag{26}$$

be the bifurcation parameter. The Jacobian matrix around the disease-free equilibrium $E_0$ and evaluated at the bifurcation parameter $\beta^* = \beta_{FF}$ is given by $J_{E_0,\beta^*}$. The eigenvalues of $J_{E_0,\beta^*}$ at $\mathcal{R}_0 = 1$ are the roots of the characteristic equation given by the polynomial $\mathcal{M}(\lambda) = 0$ of degree five whose all roots are negative except one simple zero eigenvalue. The left eigenvector corresponding to the simple zero eigenvalue $\lambda_5 = 0$ of $J_{E_0,\beta^*}$ is obtained from $\mathbf{v} J_{E_0,\beta^*} = 0$ and is given by $\mathbf{v} = (v_1, v_2, v_3, v_4, v_5)$ where $v_1 = 1, v_2 = 1, v_5 = 0$,

$$v_3 = \frac{m(m + d_F)(2\phi + \sigma - \epsilon)}{\phi[2m(m + d_F) - d_H(\sigma - \epsilon)]} \quad \text{and} \quad v_4 = \frac{4m\alpha_F}{4m\alpha_F + (2m + \sigma - \epsilon)^2}.$$

Further, the right eigenvector, $\mathbf{w} = (w_1, w_2, w_3, w_4, w_5, w_6)^T$, associated with this simple zero eigenvalue can be obtained from $J_{E_0,\beta^*}\mathbf{w} = 0$. As a result, we have $w_1 = 1, w_3 = 0, w_5 = 0$,

$$w_2 = \frac{4L\phi}{w(\sigma - \epsilon)} \quad \text{and} \quad w_4 = \frac{4L\phi[R_b(2m + \sigma - \epsilon)^2 - \alpha_F(\sigma - \epsilon)^2]}{w(\sigma - \epsilon)[4m^2\alpha_F + m(2m + \sigma - \epsilon)^2]}.$$

It should be noted that the components of $\mathbf{v}$ and $\mathbf{w}$ are obtained so that $\mathbf{v} \cdot \mathbf{w} = 1$ as required. All the second-order partial derivatives of $f_i, i = 1, 2, 3, 4, 5$ from the system are zero

at point $(E_0, \beta^*)$ except the following

$$\frac{\partial^2 f_1}{\partial x_1 \partial x_4} = \frac{\partial^2 f_1}{\partial x_4 \partial x_1} = -\beta_{BH} \qquad \frac{\partial^2 f_3}{\partial x_2 \partial x_5} = \frac{\partial^2 f_3}{\partial x_5 \partial x_2} = \beta_{HF}$$
$$\frac{\partial^2 f_2}{\partial x_2 \partial x_3} = \frac{\partial^2 f_2}{\partial x_3 \partial x_2} = -\beta_{HH} \qquad \frac{\partial^2 f_4}{\partial x_3 \partial x_4} = \frac{\partial^2 f_4}{\partial x_4 \partial x_3} = -\beta_{FF}$$
$$\frac{\partial^2 f_2}{\partial x_2 \partial x_5} = \frac{\partial^2 f_2}{\partial x_5 \partial x_2} = -\beta_{HF} \qquad \frac{\partial^2 f_4}{\partial x_4 \partial x_5} = \frac{\partial^2 f_4}{\partial x_5 \partial x_4} = -\beta_{FF}$$
$$\frac{\partial^2 f_2}{\partial x_2 \partial x_3} = \frac{\partial^2 f_3}{\partial x_3 \partial x_2} = \beta_{HH} \qquad \frac{\partial^2 f_5}{\partial x_4 \partial x_5} = \frac{\partial^2 f_5}{\partial x_5 \partial x_4} = \beta_{FF}$$
$$\frac{\partial^2 f_2}{\partial x_2 \partial x_4} = \frac{\partial^2 f_2}{\partial x_4 \partial x_2} = -\alpha_F \left[\frac{2x_2 x_4}{(x_2+x_4)^3}\right] \qquad \frac{\partial^2 f_4}{\partial x_2 \partial x_4} = \frac{\partial^2 f_4}{\partial x_4 \partial x_2} = \alpha_F \left[\frac{2x_2 x_4}{(x_2+x_4)^3}\right]$$

Following the works of Castillo-Chavez et. al [2] the direction of the bifurcation at $\mathcal{R}_0 = 1$ is determined by the signs of the bifurcation coefficients **a** and **b**, obtained from the above partial derivatives, given, respectively by

$$\mathbf{a} = \sum_{k,i,j=1}^{5} v_k w_i w_j \frac{\partial^2 f_k}{\partial x_i \partial x_j}(E_0, \beta^*) = -4\alpha_F \left[\frac{2x_2 x_4}{(x_2+x_4)^3}\right] w_2 w_4 \left[\frac{(2m+\sigma-\epsilon)^2}{4m+(2m+\sigma-\epsilon)^2}\right].$$

Note that $\alpha_F, x_2, x_4 > 0$ and by inspection, $w_2, w_4 > 0$. Consequently, $\mathbf{a} < 0$. Also,

$$\mathbf{b} = \sum_{k,i=1}^{5} v_k w_i \frac{\partial^2 f_k}{\partial x_i \partial \beta}(E_0, \beta^*) = v_3 w_2 (x_3 + x_5) - v_1 w_1 x_3$$

where $v_1 w_1 = 1$ and $v_3 w_2 (x_3 + x_5) > x_3$ since $v_3, w_2 > 0$ and $x_2, x_4 \geq 0$. This means that $\mathbf{b} > 0$.

Since $\mathbf{a} < 0$ and $\mathbf{b} > 0$, following from Theorem 4.1 (iv) [2], the endemic equilibrium $E_e$ changes its stability from stable to unstable. This scenario indicates that the system exhibits supercritical (forward) bifurcation at $\beta_{FF} = \beta^*$ and the endemic equilibrium $E_e$ is locally asymptotically stable. □

The implication of this above result is that a small inflow of infectious forager bees into a completely susceptible population will lead to the persistence of the disease in the colony whenever $\mathcal{R}_0 > 1$. Biologically, as long as the basic reproduction number $\mathcal{R}_0 < 1$, the infection can be eliminated from the population. If parameters change and result in $\mathcal{R}_0 > 1$, a small endemic state may occur.

## 4.4 Sensitivity Analysis

We perform a sensitivity analysis of the basic reproduction number $\mathcal{R}_0$ to determine several parameters that are most influencial on the prevalence and transmission of *Nosema* infection in the colony. If a small change in a parameter can cause a large change in the number of the basic reproduction number, then this parameter is called a *sensitive factor*, otherwise called an *insensitive factor*. Mimicking the techniques used in Obabiyi and Olaniyi [24, 25], the sensitivity

indices $\mathcal{R}_0$ with respect to the model parameters are computed below:

$$\begin{aligned}
\Upsilon_{\beta_{FF}}^{\mathcal{R}_0} &= \frac{\partial \mathcal{R}_0}{\partial \beta_{FF}} \times \frac{\beta_{FF}}{\mathcal{R}_0} = 1 \\
\Upsilon_{L}^{\mathcal{R}_0} &= \frac{\partial \mathcal{R}_0}{\partial L} \times \frac{L}{\mathcal{R}_0} = \frac{2L}{2L + w\epsilon - w\sigma} \\
\Upsilon_{w}^{\mathcal{R}_0} &= \frac{\partial \mathcal{R}_0}{\partial w} \times \frac{w}{\mathcal{R}_0} = \frac{-w(\sigma - \epsilon)}{2L + w\epsilon - w\sigma} \\
\Upsilon_{m}^{\mathcal{R}_0} &= \frac{\partial \mathcal{R}_0}{\partial m} \times \frac{m}{\mathcal{R}_0} = \frac{m(m + d_F)[w(\sigma - \epsilon) - 2R_b] + \sigma(2m + d_F)[w(\sigma - \epsilon) - 2L]}{\sigma(m + d_F)(2L + w\epsilon - w\sigma)} \\
\Upsilon_{\alpha_F}^{\mathcal{R}_0} &= \frac{\partial \mathcal{R}_0}{\partial \alpha_F} \times \frac{\alpha_F}{\mathcal{R}_0} = \frac{w\alpha_F(\sigma - \epsilon)}{\sigma(2L + w\epsilon - w\sigma)} \\
\Upsilon_{R_b}^{\mathcal{R}_0} &= \frac{\partial \mathcal{R}_0}{\partial R_b} \times \frac{R_b}{\mathcal{R}_0} = \frac{wR_b(\sigma - \epsilon - 2m)}{2L + w\epsilon - w\sigma} \\
\Upsilon_{d_F}^{\mathcal{R}_0} &= \frac{\partial \mathcal{R}_0}{\partial d_F} \times \frac{d_F}{\mathcal{R}_0} = d_F(m + d_F)\ln(m + d_F)
\end{aligned}$$

To compute the sensitivity indices of $\mathcal{R}_0$, we use the parameter values shown on the table below.

| **Notation** | **Parameter Values** | **Reference** |
|---|---|---|
| $\beta_{FF}$ | $5.0 \times 10^{-5}$ | [1] |
| $L$ | 2000 | [20] |
| $w$ | 5000 | [20] |
| $\alpha_F$ | 0.75 | [20] |
| $R_b$ | 0.25 | [9] |
| $d_F$ | 0.14 | [13] |
| $m$ | 0.14 | [7] |

Table 2: List of parameter values with references.

Observe that based on computing the sensitivity indices of the basic reproduction number $\mathcal{R}_0$ with respect to the parameters as stipulated in Table 3 below, parameters $\beta_{FF}, L$ and $\alpha_F$ generated positive indices implying that these parameters indicate direct relation to $\mathcal{R}_0$. In other words, increasing (or decreasing) the disease transmission rate $\beta_{FF}$ between foragers, the queen's egg-laying rate $L$, or the strength of social inhibition $\alpha_F$ by 10%, $\mathcal{R}_0$ increases (or decreases) by 10%, 11.45%, or 14.67%, respectively. Biologically, this means that an increase (or decrease) on $\beta_{FF}, L$, or $\alpha_F$ will lead to the persistence (or reduction) of the disease in the population.

| **Parameter** | **Sensitivity Indices** | **Relationship to $\mathcal{R}_0$** |
|---|---|---|
| $\beta_{FF}$ | +1.0000 | direct relation |
| $L$ | +1.1450 | direct relation |
| $w$ | −1.1450 | inverse relation |
| $\alpha_F$ | +0.1467 | direct relation |
| $R_b$ | −0.0862 | inverse relation |
| $d_F$ | −0.0499 | inverse relation |
| $m$ | −1.4727 | inverse relation |

Table 3: Sensitivity indices of $\mathcal{R}_0$ with respect to the parameters.

On the other hand, parameters $w, R_b, d_F$ and $m$ generated negative sensitivity indices. This implies that these parameters have inverse relationship to $\mathcal{R}_0$. That is, a 10% decrease (or increase) on $w, R_b, d_F$ or $m$ will result to 14.5%, 8.62%, 4.99%, or 14.73% increase (or decrease) of $\mathcal{R}_0$, respectively. This tells us that if we decrease the number of hive bees nurturing the eggs for 50% survival, the baseline recruitment rate, the death rate of the foragers due to infection or even the death rate of the foragers caused by some environmental factors, the infection will continue to propagate in the colony.

Moreover, the greater the absolute value of the sensitivity index, the more sensitive the parameter is to $\mathcal{R}_0$. Based on the computation of the sensitivity indices of $\mathcal{R}_0$, the most sensitive parameter to $\mathcal{R}_0$ is the natural death rate $m$ of the foragers bees. That is, a small change on the value of $m$ may have a significant impact to the persistence or reduction of the disease in the population. On the contrary, the death rate $d_F$ of the foragers due to the infection is the least sensitive parameter to $\mathcal{R}_0$. A sufficient change in the value of $d_F$ may or may not have a significant impact on the spread or reduction of the infection.

## 5 Results and Discussions

In this section, we present the results of the numerical simulations of the key scenarios that illustrate the main dynamics of the bee colony in the presence of the disease. In simulating the dynamics of the bee colony, we integrate the governing model equations numerically, with initial conditions $H_I(0) = F_I(0) = 0$ and $B_S(0), H_S(0), F_S(0)$ based on the steady state values for the disease-free equilibrium which was determined analytically.

### Scenario 1.

In this scenario, we illustrate the baseline demographic dynamics of the colony in the absence of the disease. Consider the epidemic model constructed (8)-(12) with the following parameters and their corresponding references.

| Parameter | Values | Reference | Parameter | Values | Reference |
|---|---|---|---|---|---|
| $L$ | 2000 | [20] | $\phi$ | 0.11 | [21] |
| $d_H$ | 0.14 | [13] | $\alpha_F$ | 0.75 | [20] |
| $d_F$ | 0.14 | [13] | $\beta_{BF}$ | $5.0 \times 10^{-5}$ | [1] |
| $m$ | 0.14 | [7] | $\beta_{HH}$ | $5.0 \times 10^{-5}$ | [1] |
| $R_b$ | 0.25 | [9] | $\beta_{HF}$ | $5.0 \times 10^{-5}$ | [1] |
| $w$ | 5000 | [20] | $\beta_{FF}$ | $5.0 \times 10^{-5}$ | [1] |

Table 4: List of parameter values used in the absence of the infection.

The disease-free equilibrium is given by $E_0 = (15879, 34467, 0, 12476, 0)$. We used an initial condition with components slightly greater than $E_0$. Based on the simulations, as the number of days $t$ increases, the population of the susceptible broods, hive bees and foragers get closer and closer to the equilibrium, respectively. This means that the disease-free steady state $E_0$ of the system is locally asymptotically stable. The same is true when we set the components of the initial condition slightly less than the components of $E_0$.

### Scenario 2.

In this scenario, we illustrate the population dynamics of the colony in the presence of the disease. Consider the epidemic model (8)-(12) with the following parameters shown in Table 4

and the corresponding references.

Let $E_e$ be the endemic equilibrium point. Using center manifold theory, we explored the stability of $E_e$ with the bifurcation parameter

$$\beta^* = \frac{2m(m+d_F)}{2L + w\epsilon - w\sigma} \approx 3.44 \times 10^{-5}$$

and the bifurcation coefficients $\mathbf{a}$ and $\mathbf{b}$ with

$$\mathbf{a} = -4\alpha_F \left[\frac{2x_2x_4}{(x_2+x_4)^3}\right] w_2w_4 \left[\frac{(2m+\sigma-\epsilon)^2}{4m+(2m+\sigma-\epsilon)^2}\right]$$
$$\mathbf{b} = v_3w_2(x_3+x_5) - v_1w_1x_3.$$

Checking numerically, $\mathbf{a} < 0$ and $\mathbf{b} > 0$. This means that by Theorem 4.1 (iv) [2], exhibits a supercritical (forward) bifurcation at $\beta^*$ and the endemic equilibrium $E_e$ is locally asymptotically stable. However, notice that in $\mathbf{a}$, all the factors are always positive, except for $w_4$ which may be negative if $\alpha_F$ is sufficiently large. Consequently, the direction of the bifurcation changes if $\alpha_F$ is sufficiently large. That is, if $\alpha_F$ is such that $\mathbf{a} > 0$, then there exists an unstable subthreshold endemic equilibrium near $E_e$. The analysis of the center manifold tells us that not only $E_e$ is unstable, but there is no nonzero stable equilibrium near $E_e$ and thus a small invasion of the infection to the colony will grow rapidly to significant proportions even for $\mathcal{R}_0$ near 1.

Since the local analysis of the center manifold yields a parameter $\mathbf{a} < 0$, there are stable subthreshold endemic equilibria near $E_e$. Thus reducing $\mathcal{R}_0$ through one lowers the incidence of the disease until it is eliminated as $\mathcal{R}_0$ passes below one. Notice that based on the simulation as shown in Figure 2, the population of the susceptible bees initially decreases significantly, then increases and decreases over time until it asymptotically stabilize near $E_e$. Moreover, the population of the infected bees initially increased on the first few days and eventually stabilize over time. This unstability of subthreshold endemic equilibrium is due to the effect of excess foragers on recruitment represented by the parameter $\alpha_F$. Note that if $\alpha_F$ is sufficiently large, there will be an overflow of foragers being recruited which increases the chance of being infected during foraging duties.

### Scenario 3.

In this scenario, we illustrate the population dynamics of the colony both in the absence and in the presence of the disease with $\mathcal{R}_0 = 1$. Using the same parameters in Table 4, we have $B_S \approx 7,129, H_S \approx 3,224$ and $F_S \approx 5,600$. Setting initial conditions slightly away from the values (approximate) of $B_S, H_S$ and $F_S$, as shown in Figures 3 and 4, the population of the susceptible broods $B_S$ and susceptible hive bees $H_S$ increase over time. Although the population of the forager bees $F_S$ decline on the first few days, it rebounds and increases over time. As expected, the number of bees in the colony increase over time in the absence of the infection.

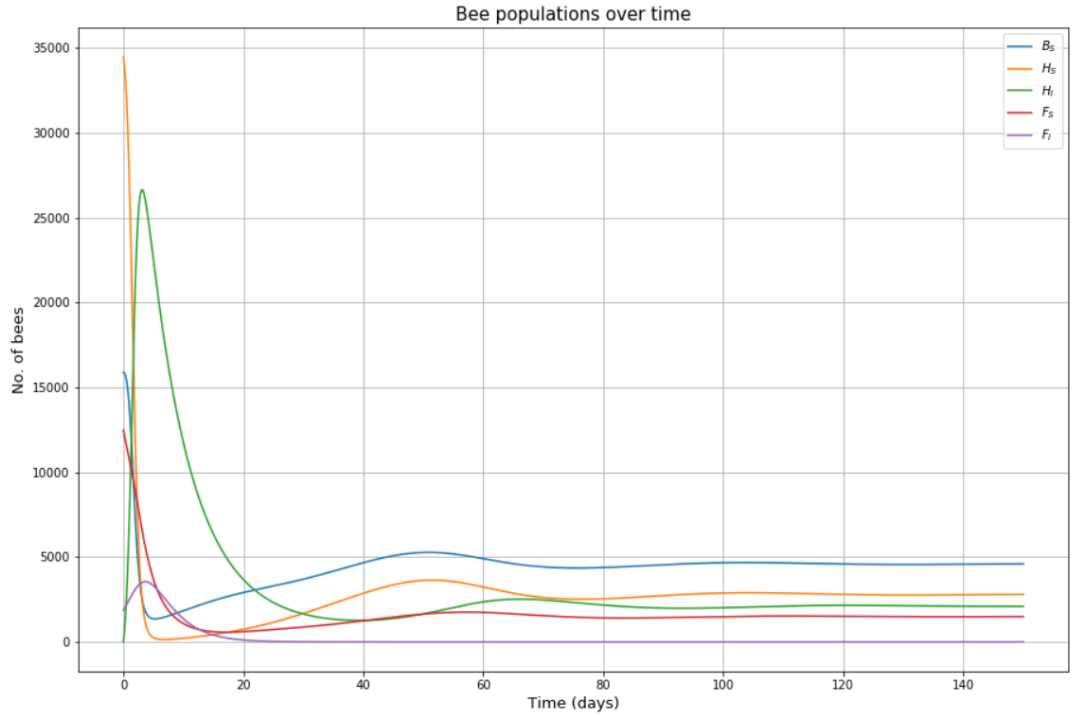


Figure 2: Population dynamics of the bee colony in the presence of the disease.

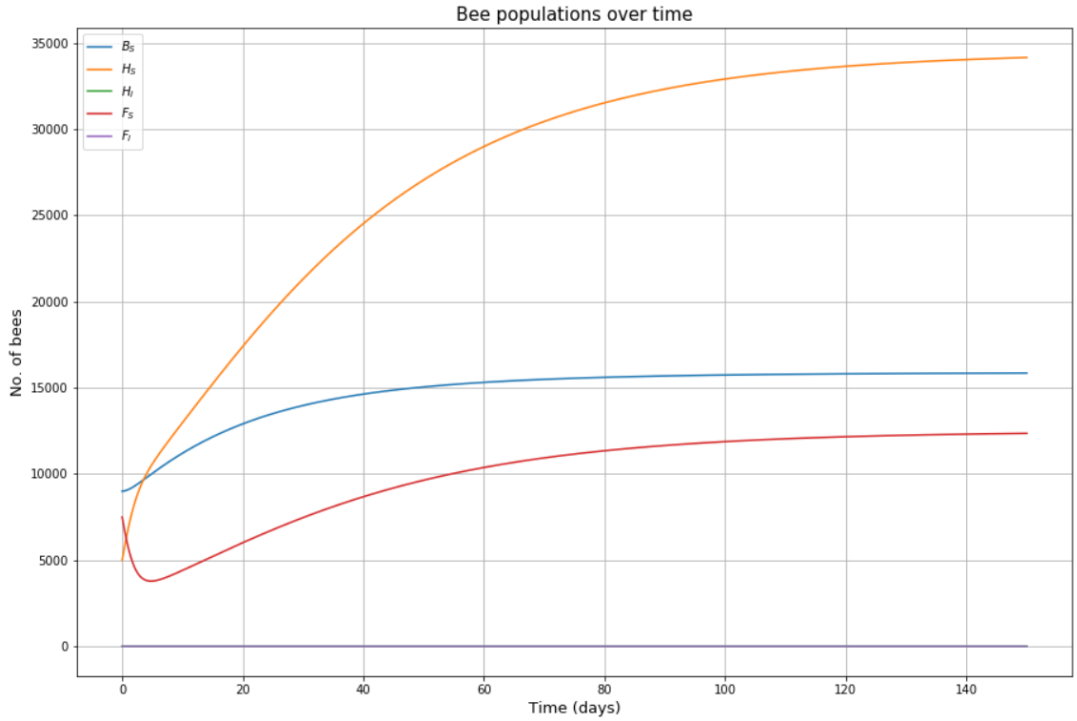


Figure 3: Population dynamics of the bee colony in the absence of the disease with $\mathcal{R}_0 = 1$ and initial condition $(9000, 5000, 0, 7500, 0)$.

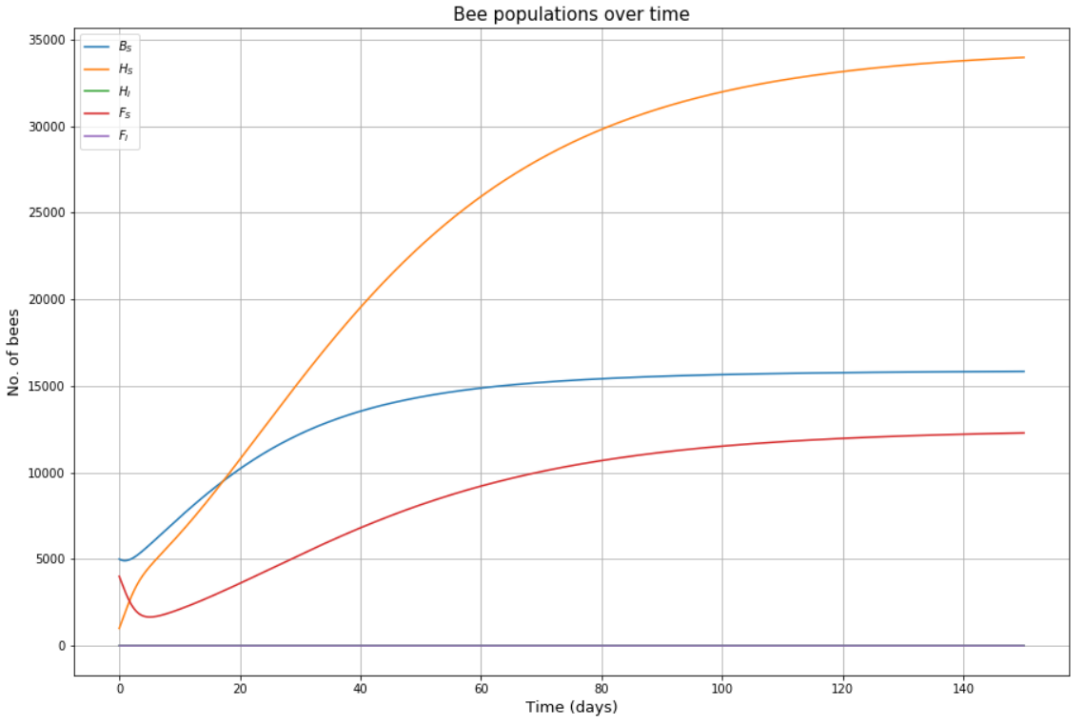


Figure 4: Population dynamics of the bee colony in the absence of the disease with $\mathcal{R}_0 = 1$ and initial condition $(5000, 1000, 0, 4000, 0)$.

Introducing the infected foragers into the system, we again used initial conditions away from what we have obtained when $\mathcal{R}_0 = 1$. Notice that in Figure 5, the population of the susceptible broods and the susceptible foragers suffer a drastic drop and the majority of the foragers become infected. The infection greatly reduces the overall population of the colony. However, a new equilibrium is reached and approximately half of the population sustained the infection.

Setting the initial condition to $(5000, 1000, 100, 4000, 400)$, result is shown in Figure 6. The population of the susceptible broods and susceptible hive bees initially increases on the first few days and drops drastically in some period of time. Still, majority of the foragers become infected and the number of susceptible foragers decline due to the natural death. It can also be observed that the number of hive bees, both susceptible and infected increases over time. Although the infection reduces the population of the colony, there are sufficient number of bees remain sustaining the disease.

### Scenario 4.

In this scenario, we investigated the effect of a more severe infection. We set the disease transmission rates unchanged but the mortality rates from the disease are increased to $d_H = d_F = 0.56$. As shown in Figure 7, the results show that after an initial drastic drop due to the severity of the infection, the population of the susceptible bees begins to recover approximately 8-10 days after the onset of the infection. This means that even the population faces a more severe infection in the sense that it kills faster, the colony can sustain the infection and survive.

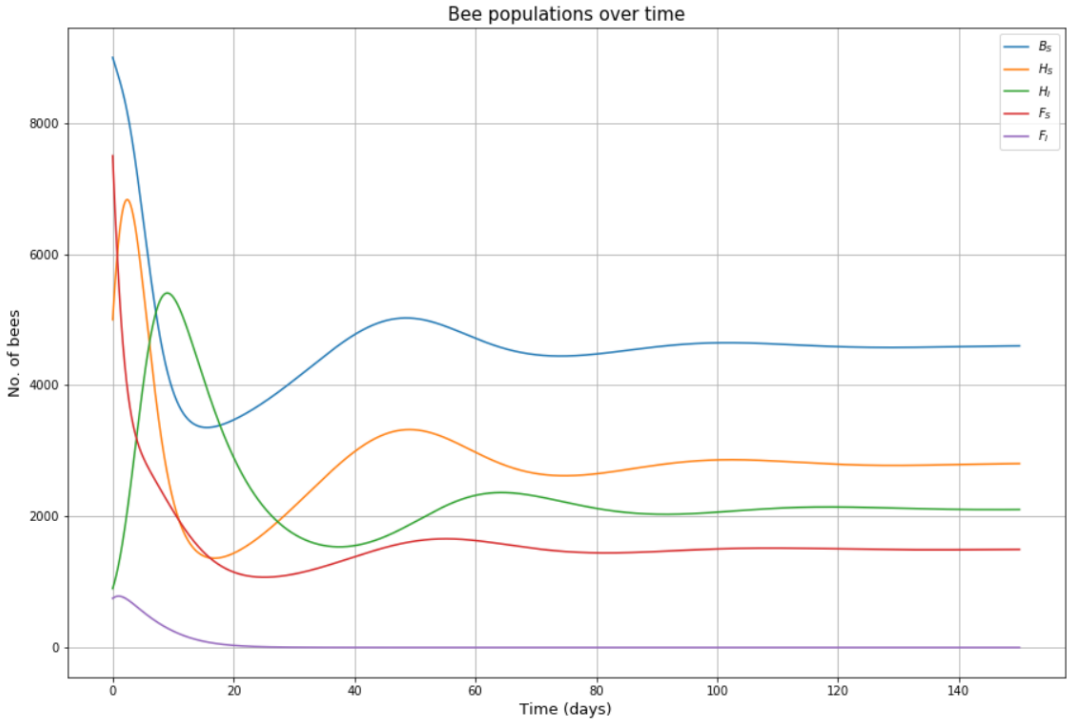


Figure 5: Population dynamics of the bee colony in the presence of the disease with $\mathcal{R}_0 = 1$ and initial condition $(9000, 5000, 500, 7500, 750)$.

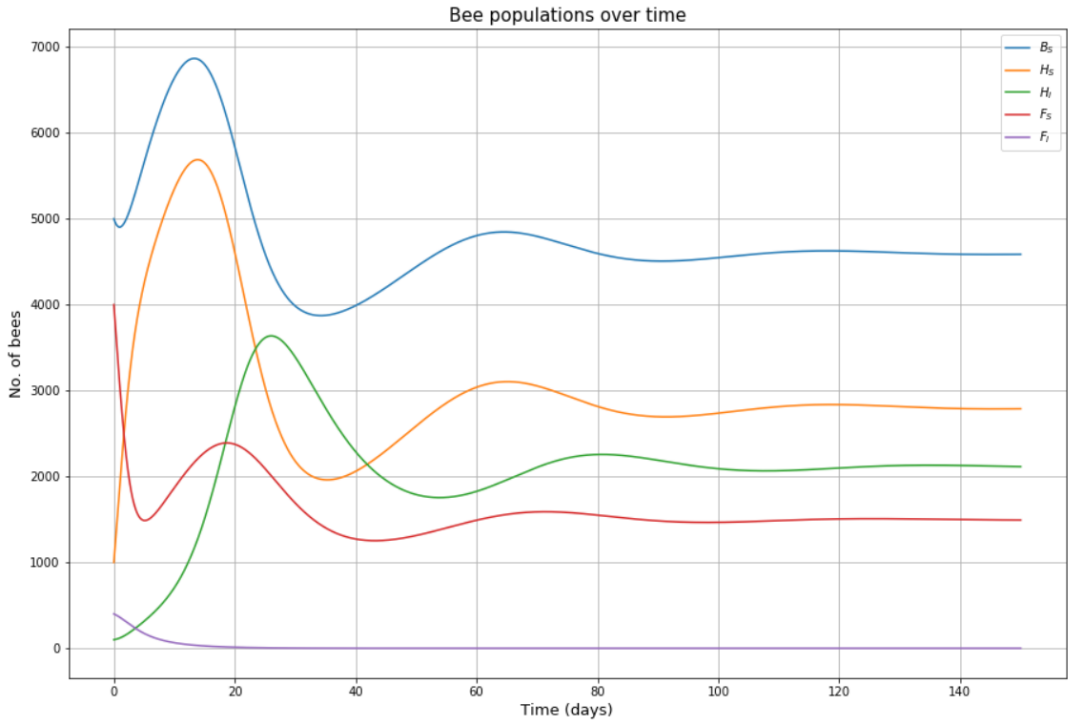


Figure 6: Population dynamics of the bee colony in the presence of the disease with $\mathcal{R}_0 = 1$ and initial condition $(5000, 1000, 100, 4000, 400)$.

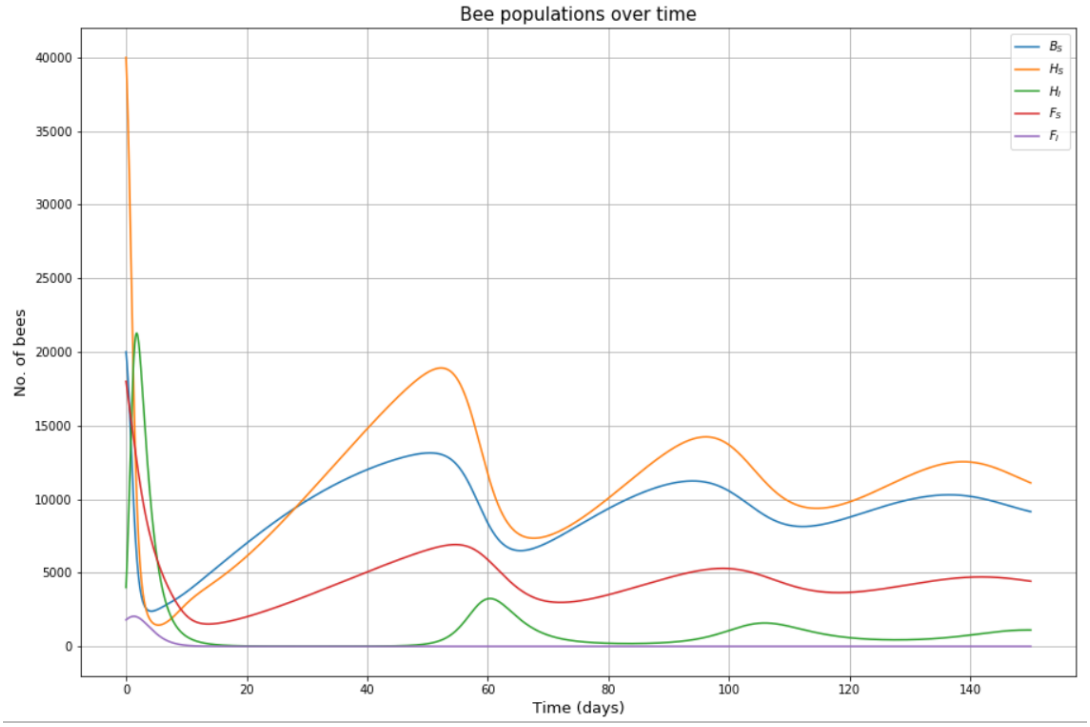


Figure 7: Population dynamics under a more severe infection represented by higher death rates ($d_H = d_F = 0.56$) due to the disease with unchanged transmission rates.

## 6 Summary and Conclusion

Extending the study of Betti et. al. [1] and following the preliminary concepts of Khoury et. al. [20, 21], the main objective of this paper was to construct an epidemic model for investigating the effects of infection in the dynamics of a honey bee colony including the brood population. The model is presented in term of a set of governing ordinary differential equations representing the interplay between the dynamics of the spread of the disease and the normal demographic dynamics of the bee colony. In illustrating the utility of the model, parameter values associated with *Nosema ceranae* were employed because its availability and has been experimentally well studied. The summarized list of the parameter values used in this model were tabulated in Table 4 with their corresponding references of experimental studies.

In basic dynamical properties of the model, the basic reproduction number $\mathcal{R}_0$ was derived using the Next Generation Matrix (NGM) method explicitly discussed by Diekmann et. al. in [5, 6]. The local asymptotic stability of the disease-free equilibrium (DFE) was proven using the analysis of the characteristics equation based on $\mathcal{R}_0$. The endemic equilibrium was not explicitly established but its local asymptotic stability at $\mathcal{R}_0$ was explored using the center manifold theory of bifurcation describe by Castillo-Chavez et. al. in [2].

Following the method of Obabiyi et. al. [24], the sensitivity analysis of the basic reproduction number $\mathcal{R}_0$ was explored to determine several parameters that are most influential on the prevalence and transmission of the infection. It was determined that the disease transmission rate $\beta_{FF}$ between foragers, the queen's egg laying rate $L$ and the strength of social inhibition $\alpha_F$ generated positive indices implying their direct relation to $\mathcal{R}_0$. That is, if there increase on $\beta_{FF}, L$ and $\alpha_F$, the basics reproduction number also increases. On the other hand, if the proportion of the hive bees $w$ needed for the eggs' survival, the baseline recruitment rate $R_b$, and the mortality rates $d_F$ and $m$ increase, then $\mathcal{R}_0$ decrease since these parameters yielded

negative indices and have inverse relation to $\mathcal{R}_0$. Lastly, the most sensitive parameter to $\mathcal{R}_0$ is the natural death rate $m$ of the foragers bees. That is, a small change on the value of $m$ may have a significant impact to the persistence or reduction of the disease in the population. On the contrary, the death rate $d_F$ of the foragers due to the infection is the least sensitive parameter to $\mathcal{R}_0$. A sufficient change in the value of $d_F$ may or may not have a significant impact on the spread or reduction of the infection.

Based on the numerical simulations performed, the model suggests that the key factors in the survival or collapse of a bee colony in the face of an infection are the disease rates of transmission $\beta_{BH}, \beta_{HH}, \beta_{HF}, \beta_{FF}$, the disease-induced death rates $d_H$ and $d_F$. An increase in the disease-induced death rates, which can be thought of as an increase in the severity of the disease, may actually help the colony overcome the disease and survive over time (Scenario 3). In contrary, an increase in the disease transmission rates, which means that bees are being infected at an earlier age, has a drastic deleterious effect which can lead to the collapse of the entire colony (Scenario 4).

Another important finding relates to the baseline recruitment rate $R_b$. It suggests that largely increasing $R_b$ even when the food storage is sufficient implies having an overwhelming excessive number of foragers and increases the chance of getting the disease during foraging duties which may lead to the annihilation of the colony over time.

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