Continuous and discrete dynamical systems for the declines of honeybee colonies

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Continuous and discrete dynamical systems for the declines of honeybee colonies

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Abstract
This work deals with two mathematical models on the declines of honeybee colonies. Starting from the model proposed by Khoury, Meyerscough and Barron - KMB model [25], we kept the eclosion function and changed the recruitment function to design a new model for social parasitism in honeybees. The KMB model is characterized by the fact that there exists a critical value of the foragers’ death rate above which there is colony collapse disorder (CCD) in the sense that the “trivial” equilibrium point is globally asymptotically stable (GAS). We design a nonstandard finite difference (NSFD) scheme that preserves this property. It is established that in the social parasitic (SP) model the colony decays exponentially to zero irrespective of the value of foragers’ death rate. A NSFD scheme is constructed for the SP model. The faster decline in the SP setting is demonstrated theoretically for the NSFD scheme. Numerical simulations are provided to confirm that the colony declines faster in the SP setting than in the KMB model.

Keywords: Colony collapse disorder, Capensis calamity, Dynamical systems, Global stability, Nonstandard finite difference method

1 Introduction
Honeybees are essential to man-kind and his environment through the direct and indirect services they render. These include pollination of key crops and hive products as outlined below.

Honeybees fertilize flowers through pollination as they forage. Pollination is important in agriculture since it assists in the production of food. Honeybees contribute to approximately 9.5% of the total global agriculture among other pollinators [22]. Insects, including honeybees, pollinate about 80% of 300 crops [3] and this value is worth about USD 194 billion per year in agriculture [27]. Both wild and managed honeybees pollinate about 33% of crops [27].

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Besides pollination, humans have partially domesticated honeybees mostly because of their by-products such as honey, wax, propolis and royal jelly. Humans use of honey include: medication such as cough drops, wound salves [41], and food nutrients as it provides a good source of energy [35]. It is therefore not surprising for the demand of honey to keep increasing globally [24]. In 2016, the total value of natural honey exported world wide was USD 2.2 billion [51]. While honeybees use the wax they secret to build comb cells where their brood (young ones) are raised, humans originally used beeswax to make church candles [15]. Nowadays, beeswax is used in various productions such as in the pharmaceutical industries for coating pills, production of cosmetics including baby products and fragrances [35]. Honeybee workers collect sticky resin from trees and other botanical sources and mix it with beeswax and utilize it as propolis [43]. Honeybees, use propolis to protect the colony from rain and colds, prevent parasites from entering the hive and also inhibit fungal and bacterial growth in the colony [43]. Humans use propolis for medication purposes such as in the cure of dental cavities [35] and to prevent diseases including cancer, heart ailments and inflammation [8]. A milky-white substance, royal jelly, which honeybee workers secrete and use as a nutrient to feed larvae in the hive, is mostly used in anti-aging and wrinkle prevention creams by humans [35]. The long term consumption of royal jelly by humans improve mental health and formation of red blood cells [34] and it is said to boost fertility [1].

Honeybees are crucial to the sustenance of our food, and our future as a race would be miserable without them [48]. There are clear evidences that key pollinators including managed honeybees are fast declining world wide [25,30]. In the past decades, beekeepers have witnessed an increase in colony losses through phenomena such as colony collapse disorder (CCD) and others [25, 47]. Losses credited to CCD are in the range of 1.8 to 85% of total managed honeybee colonies [37]. Apart from the CCD whose cause is not known [47], pests, diseases, and improper beekeeping management practices are other factors that hinder international trade of honeybees [37].

South Africa was not an exemption [9] on the 2006 honeybee colony failures that have been reported globally [25]. In this regards, we mention the so-called Capensis Calamity (CC) [2], that caused and is still causing great losses (up to 80%) in managed colonies of the Savannah honeybee (Apis mellifera scutellata) [38]. This was due to moving bees from the Cape region into the Northern part of South Africa for pollination where a clonal lineage of cape bee became a facultative social parasite [16]. Without pollinators in South Africa, it is estimated that about USD 700 billion per year of fruit export industries will negatively be affected, while in seed production, between USD 5-USD 6 million per year [9] will decline.

The purpose of this paper is to gain some insight on the honeybee colony declines. Among mathematical models that deal with the broader question of honeybee population dynamics, we can mention the following two categories:

- The works that model the colony failure as a contagion by a virus, which is transmitted by parasitic varroa mites [21,28,39];
- The works that explore the interactions of strategies (such as availability of food, climate change) and foragers mortality on colony fate [11,18,19, 26, 40].

Since this study focuses on colony declines, our point of departure is the mathematical model proposed by Khoury, Meyerscough and Barron - KMB model [25]. Being relatively
similar to its extension in [40] and [11], the KMB model offers the opportunity to express colony failure in a much simpler way. By keeping the eclosion function as in the KMB model, we develop a model that captures the CCD phenomenon in a social parasite, SP, scenario, such as the social parasitism by the Cape honeybees into the region of the Savannah honeybees [4]. We prove that in the SP setting, the colony collapses faster than in the KMB framework. More precisely, a colony decays exponentially to zero. We design nonstandard finite difference (NSFD) schemes for the KMB and SP models which are dynamically consistent with the general and faster decline property.

The rest of the paper is organized as below. In the next section, we summarize the KMB model and its dynamics. A new model, for social parasitism (SP), is proposed and analyzed in section 3. Two NSFD schemes that replicate the dynamics of the KMB and SP models are constructed and studied in section 4. Section 5, that is devoted to numerical simulations is followed by section 6 where concluding remarks and possible extensions are provided.

2 Preliminaries

In this section, we recall the mathematical model proposed by Khoury, Meyerscough and Barron-KMB model [25] and summarize the results which are needed in this paper.

The total population, \( N \), of honeybees being divided into two compartments, \( H \) of hive bees and \( F \) of foragers, the KMB model reads

\[
\begin{align*}
\frac{dH}{dt} &= \frac{L(H + F)}{H + F + \omega} - \left(\alpha - \sigma \frac{F}{H + F}\right)H \\
\frac{dF}{dt} &= \left(\alpha - \sigma \frac{F}{H + F}\right)H - mF,
\end{align*}
\]

(1)

where:

- \( E(H, F) = \frac{L(H + F)}{H + F + \omega} = \frac{LN}{N + \omega} \) is the eclosion function in which \( L \) represents the queens’ laying rate and \( \omega \) is the rate at which eclosion approaches \( L \) as \( N = H + F \) increases. (Note that \( \omega \) is also the number of the bees needed for emergence rate to reach \( L/2 \) [28]);

- \( R(H, F) = \left(\alpha - \sigma \frac{F}{H + F}\right)H \) is the recruitment function, in which \( \alpha \) is the rate at which hive bees are recruited to foragers class and \( \sigma \) is the rate at which social inhibition occurs;

- \( m \) is the death rate of foragers.

One key assumption that transpires from [25] is that a critical number of the total individuals consists of foragers. The specific choice, made in [25] is \( \alpha = 0.25 \) and \( \sigma = 0.75 \) so that the number of foragers is less than or equals to \( \frac{N}{3} \). We extend this assumption by assuming that

\[
\alpha < \frac{\sigma}{2}.
\]
Setting
\[ R(H,F) = \frac{R(H,F)}{H} = \alpha - \sigma \frac{F}{H+F}, \]
we will often be interested in the following two extreme situations:
\[ \Gamma^+ := \inf_{H>0,F>0} R(H,F) > 0, \]
which reflects the normal situation when social inhibition occurs, and
\[ \Gamma^- := \sup_{H>0,F>0} R(H,F) < 0, \]
which implies that there is no social inhibition.

The defining equations of an equilibrium point \((H,F)\) of the system (1) are
\[
\begin{cases}
\frac{L(H+F)}{H+F+\omega} - \left(\alpha - \sigma \frac{F}{H+F}\right)H = 0 \\
\left(\alpha - \sigma \frac{F}{H+F}\right)H - mF = 0.
\end{cases}
\]

It is clear that the origin \((0,0)\) is an equilibrium point of the system. It is equally clear that the system does not have boundary equilibrium points \((H^B,0)\) and \((0,F^B)\) for \(H^B > 0\) and \(F^B > 0\). For a potential interior equilibrium point \((H^*,F^*)\), we follow [11,25] and set
\[ H^* = \frac{1}{J} F^*. \]

Simple algebraic manipulations show that
\[ H^* = \frac{L}{Jm} - \frac{\omega}{1+J} \]
and
\[ F^* = \frac{L}{m} - \frac{\omega J}{1+J}, \]
where
\[ J = \frac{1}{2} \left[ \left(\frac{\alpha}{m} - \frac{\sigma}{m} - 1\right) + \sqrt{\left(\frac{\alpha}{m} - \frac{\sigma}{m} - 1\right)^2 + 4\frac{\alpha}{m}} \right] \]
is the unique positive root of the quadratic equation
\[ J^2 - \left(\frac{\alpha}{m} - \frac{\sigma}{m} - 1\right)J - \frac{\alpha}{m} = 0. \]

Due to the condition (2), we have
\[ J < 1. \]

Indeed, from (9), we have
\[ 2J = \left(\frac{\alpha}{m} - \frac{\sigma}{m} - 1\right) + \sqrt{\left(\frac{\alpha}{m} - \frac{\sigma}{m} - 1\right)^2 + 4\frac{\alpha}{m}} < 2. \]
if, and only if
\[
\sqrt{\left(\frac{\alpha}{m} - \frac{\sigma}{m} - 1\right)^2 + 4 \frac{\alpha}{m}} < \left(1 + \frac{\sigma - \alpha}{m}\right) + 2
\]
if, and only if
\[
\left(1 + \frac{\sigma - \alpha}{m}\right)^2 + 4 \frac{\alpha}{m} < \left(1 + \frac{\sigma - \alpha}{m}\right) + 4 \left(1 + \frac{\sigma - \alpha}{m}\right) + 4
\]
if, and only if
\[
\frac{2\alpha}{m} < \frac{\sigma}{m} + 2,
\]
which is true because
\[
\frac{2\alpha}{m} < \frac{\sigma}{m} \quad \text{by (2)}.
\]
Throughout this work, the following further assumption is made
\[
\alpha - \frac{L}{\omega} > 0. \tag{12}
\]
The qualitative properties of the KMB model are summarized in the following result:

**Theorem 2.1.**

1. Under the condition (3), the KMB model is a dynamical system in the biological feasible region:
\[
\Omega = \left\{ (H,F) \in \mathbb{R}_+^2 : H \leq \frac{L}{F^\beta} \quad \text{and} \quad F \leq \frac{\alpha L}{m F^\beta} \right\}.
\]

2. If
\[
m < \frac{L}{2\omega} \left(\frac{(\alpha + \sigma) + \sqrt{(\alpha - \sigma)^2 + 4 \frac{L}{\omega}}}{\alpha - \frac{L}{\omega}}\right), \tag{13}
\]
the KMB model (1) has a unique interior equilibrium point, which is globally asymptotically stable (GAS).

3. If the condition (13) is not satisfied, then (0,0) is globally asymptotically stable.

Parts 2 and 3 of Theorem 2.1 are announced in [25]. The proof will be provided in Appendix 1 in the following four main steps: Positivity of $F^*$ in (8), Local asymptotic stability of the equilibrium point, The system (1) has no periodic solutions, The global asymptotic stability of the equilibrium point.

Under the condition (4), the second equation in (1) leads to a decay inequality in $F$. Using this fact and Gronwall inequality, we obtain the next result.

**Theorem 2.2.** Under the condition (4), we have, for $t$ large enough
\[
H(t) \leq \frac{L}{\alpha} \quad \text{if} \quad H_0 \leq \frac{L}{\alpha} \quad \text{and, for all} \quad t, \quad F(t) \leq F_0 \exp\{-mt\} \quad \text{for any} \quad F_0.
\]
3 Social parasitic model

In this section, we propose a mathematical model for a honeybee colony invaded by social parasites. We show that the colony declines faster, namely in an exponential manner. The study is motivated by the so-called “Capensis Calamity”, CC, [2–4,6], in South Africa for which we give some background.

In social insects, “social parasitism” means benefiting from brood care or resources of the host colony. It has been shown that honeybee workers can function as social parasites in colonies of other bees [20].

There are two different sub-species of honeybees in South Africa, namely Savannah honeybees, \( Apis mellifera scutellata \), which are found in the northern part of the country and the Cape honeybees, \( Apis mellifera capensis \), that are found in the western region. These two sub-species are separated by a region of introgression or a buffer zone where hybrids exist [16]. One main difference between the Cape honeybees and Savannah honeybees, is that: The Capensis workers lay unfertilized eggs which develop into diploid (female) offspring through a process called thelytokous parthenogenesis [16], while Savannah workers only follow the normal arrhenotokous pathway where workers lay unfertilized haploid eggs that gives rise to males. Capensis workers fuse their eggs pronuclei with one of their polar bodies, which in the absence of meiosis produce female offsprings [23]. These clones of Capensis workers are the basis of the social parasitism in honeybees. If they (clones) enter the colony of other honeybee races, they mimic the pheromone of the host queen and start to lay eggs that develop into females [17]. The actively reproductive pseudo queens may suppress the developments of their clones which will result in dominance hierarchies [23]. Despite the fact that these clones are not reproductive, they do not contribute towards other activities of the colony such as foraging, since pollen combs and pollen baskets on their hind legs are suppressed and they receive royal treatments from the host workers [23].

There has been historically reported cases of colonies taken over by the Cape honeybees when moved into the regions of the Savannah honeybees and other honeybee races. The most recent invasion began in the 1990s when beekeepers moved some Cape honeybees into the region of the Savannah honeybees for the purpose of pollination. This migratory beekeeping resulted in the establishment of a clonal parasitic lineage of the Cape honeybees that caused and is still causing death of many Savannah honeybee colonies [6]. The social parasitism by the Cape honeybees is the biological basis for the CC due to subsequent re-invasion. CC causes serious problems for endemic honeybee populations and threatens biodiversity as honeybee colonies are lost [4].

For the formulation of the Social Parasitic (SP) model, we consider, as in the KMB model, the hive and foragers compartments with the same eclosion function. However, we add the following assumptions to capture the absence of social inhibition and the presence of the social parasites in the form of clone 1, clone 2 and clone 3 as shown in the flow diagram (Figure 1):

**Assumption 1** Brood diseases on clones and death rate of clones are negligible.

This assumption is motivated by the parasitic nature of clones which are to take over the colony with time. This makes them not to display workers’ characteristics such as grooming and trophallaxis that are known for vertical and horizontal disease transmission within the colony [14].
Assumption 2. Host foragers die at the rate $m$.

Assumption 3. The clones leave the hive compartment at the rate $\tilde{m}$.

Assumption 4. The host workers transit from hive class to foragers class at the rate $\alpha$ and there is no social inhibition.

Figure 1: The flow diagram of the host (Savannah honeybees) in the presence of social parasites (Cape honeybees). Clone 1 = clone brood, clone 2 = hive bees that are hatched from clone brood and clone 3 = effect of clones on foragers.

The assumptions and flow diagram lead to the following model:

$$
\begin{align*}
\frac{dH}{dt} &= \frac{L(H + F)}{H + F + \omega} - (\alpha + \tilde{m})H \\
\frac{dF}{dt} &= \alpha H - mF.
\end{align*}
$$

(14)

Remark 3.1. It should be noted that the impact of the ability of social parasite workers to mimic the queen's pheromone [17] is that the rate $\omega$, at which the maximum eclosion is approached, is large. On the other hand, the number of clones who do not contribute to foraging but leave the hive class at the rate $\tilde{m}$ after benefiting abusively from the food produced by the foragers and the number of host workers who are recruited to foragers class are large. It has been shown biologically that capensis parasites lay multiple eggs resulting in an increase in parasitic offspring [31, 36] and rapid spread in an apiary [12]. Also capensis workers need more (at least five) scutellata workers to tender their needs within the colony [42]. These facts are mathematically reflected by the following condition:

$$
\omega > \frac{L}{\min\{m, \tilde{m}\}}.
$$

(15)

Theorem 3.2.

1. The model (14) is a dynamical system on the biologically feasible region

$$
\Omega = \left\{(H, F) \in \mathbb{R}_+^2 : H \leq \frac{L}{\alpha + \tilde{m}} \quad \text{and} \quad F \leq \frac{\alpha L}{m(\alpha + \tilde{m})} \right\}.
$$

2. Under the condition (15), the colony decays exponentially to zero.

Proof. It is easy to show by contradiction and intermediate value theorem that all solutions of (14) corresponding to nonnegative initial conditions are nonnegative at all times.
1. From the first equation of (14), we have

\[ \frac{dH}{dt} \leq L - (\alpha + \tilde{m})H. \]

By Gronwall inequality, we obtain

\[ H(t) \leq \frac{L}{\alpha + \tilde{m}} \quad \text{if} \quad H_0 \leq \frac{L}{\alpha + \tilde{m}}. \]  

(16)

Using (16) in the second equation in (14), we have

\[ \frac{dF}{dt} \leq \frac{\alpha L}{\alpha + \tilde{m}} - mF. \]

Once again, Gronwall inequality gives

\[ F(t) \leq \frac{\alpha L}{m(\alpha + \tilde{m})} \quad \text{if} \quad F_0 \leq \frac{\alpha L}{m(\alpha + \tilde{m})}. \]  

(17)

This, combined with the obvious existence of a unique local solution, proves the first part of the theorem.

2. Regarding the second part of the theorem, it follows by adding the equations in (14) that

\[ \frac{dN}{dt} = LN + \omega - \tilde{m}H - mF \]

\[ \leq \left( \frac{L}{\omega} - \min\{\tilde{m}, m\} \right) N. \]

This implies that

\[ N(t) \leq N_0 e^{\left( \frac{L}{\omega} - \min\{m, \tilde{m}\} \right) t}, \]

which in view of (15), shows that the total population decays exponentially to zero as time \( t \to \infty \).

\[ \square \]

4 Nonstandard finite difference schemes

In this section, we propose reliable numerical methods which are dynamically consistent with the continuous, KMB and SP, models considered in sections 2 and 3, respectively. We use the nonstandard finite difference approach [32].

The time \( t \geq 0 \) is discretized into a sequence of \((t_n)_{n \geq 0}\) where \( t_n := n \Delta t \), and \( \Delta t \) is the step size. We denote by \( H_n \) and \( F_n \) approximations of \( H(t) \) and \( F(t) \) at \( t = t_n \).

We start with the KMB model. Mickens’ method of sub-equations applies here [7,32]. This means that the full equation is divided into simpler equations for which exact schemes are known or available useful qualitative information can be incorporated into the derived scheme.
In the absence of hive bees, the foragers are governed by the decay equation
\[
\frac{dF}{dt} = -mF,
\]
which has the exact scheme (see for instance \cite{32})
\[
\frac{F_{n+1} - F_n}{(1 - e^{-m\Delta t})/m} = -mF_n \quad \text{or} \quad \frac{F_{n+1} - F_n}{(e^{-m\Delta t} - 1)/m} = -mF_{n+1}.
\]
In the absence of foragers, hive bees are governed by the equation
\[
\frac{dH}{dt} = \frac{LH}{H + \omega} - \alpha H,
\]
which has both decay equation and Michaelis-Menten, M-M, equation.

In \cite{13} and \cite{33}, the term \(\frac{LH}{H + F}\) in (20) is approximated by using the Lambert omega function in order to obtain the exact scheme of the M-M equation. Further approximations used in \cite{13} are related to the forward and backward Euler methods. Inspired by these works and in order to preserve unconditional positivity, we propose the following NSFD scheme for Eq (20):
\[
\frac{H_{n+1} - H_n}{\phi} = \frac{LH_n}{H_n + \omega} - \alpha H_{n+1}.
\]
Combining the partial schemes in equations (19) and (21), we propose the following NSFD scheme for the KMB model:
\[
\begin{align*}
\frac{H_{n+1} - H_n}{\phi} &= \frac{L(H_n + F_n)}{H_n + F_n + \omega} - \alpha H_{n+1} + \frac{F_nH_{n+1}}{H_{n+1} + F_n} \\
\frac{F_{n+1} - F_n}{\phi} &= \alpha H_{n+1} - \sigma \frac{F_nH_{n+1}}{H_{n+1} + F_n} - mF_{n+1}.
\end{align*}
\]
Here and after
\[
\phi = \frac{1 - e^{-(\Delta t)Q}}{Q}
\]
is a complex denominator function where the number \(Q > 0\) to be determined shortly in different situations is supposed to capture the features of the model. Note that \(\phi\) satisfies the asymptotic relation
\[
\phi(\Delta t) = \Delta t + O([\Delta t]^2)
\]
in agreement with Mickens’ first rule for construction of NSFD schemes. Furthermore, Mickens’ second rule is applied, as the nonlinear term \(\frac{HF}{H + F}\) is approximated in a nonlocal manner. For the formal definition of the NSFD schemes, see \cite{7}.

The first equation in (22) is quadratic in \(H_{n+1}\). That is,
\[
AH_{n+1}^2 + B_nH_{n+1} - C_n = 0,
\]
where
\[
\begin{align*}
A &= L(1 + \sigma/m) \\
B_n &= L(1 + \sigma/m) - \alpha \\
C_n &= \alpha - L(1 + \sigma/m).
\end{align*}
\]
where
\[ A = 1 + \phi \alpha > 0 \]
\[ B_n = (1 + \phi \alpha - \phi \sigma) F_n - H_n - \phi \frac{LN_n}{N_n + \omega} \]
\[ C_n = H_n F_n + \phi \frac{LN_n F_n}{N_n + \omega} \]

Assuming that \( H_n \) and \( F_n \) are nonnegative, so that \( C_n > 0 \), there is a unique nonnegative root of (25), given by
\[ H_{n+1} = \frac{-B_n + \sqrt{B_n^2 + 4AC_n}}{2A}. \]  
(26)

Once \( H_{n+1} \) has been computed from (26), we continue in the Gauss-Seidel type process to compute \( F_{n+1} \) as follows:
\[ F_{n+1} = \left[ \phi \alpha H_{n+1} + F_n \left( 1 - \phi \sigma \frac{H_{n+1}}{F_n + H_{n+1}} \right) \right] \left( 1 + m \phi \right). \]  
(27)

In order for \( F_{n+1} \) in (27) to be positive, we assume that
\[ Q \geq \sigma \quad \text{so that} \quad 0 < \phi < \frac{1}{\sigma}. \]  
(28)

The root in (26) can also be written in the following implicit equivalent form:
\[ H_{n+1} = \frac{H_n + \phi \frac{L(H_n + F_n)}{(H_n + F_n + \omega)} \left( 1 + \phi \alpha - \phi \sigma \frac{F_n}{H_{n+1} + F_n} \right)}{1 + \phi \alpha} \]  
(29)

In view of (28) and in order to incorporate essential parameters \( m \) and \( \alpha \) that appear in the sub-equations (18)-(19) and (20)-(21), we choose the denominator function \( \phi \) in (23) such that
\[ Q \geq m + \alpha + \sigma. \]  
(30)

**Theorem 4.1.**

1. The NSFD scheme (22) is dynamically consistent with respect to positivity.
2. If \( \alpha - \sigma \frac{F}{H + F} \geq \Gamma^+ > 0 \), the NSFD scheme (22) is a dynamical system on the same biologically feasible region \( \Omega \) as for the continuous system (see Theorem 2.1).
3. If \( \alpha - \sigma \frac{F}{H+F} \leq \Gamma^- < 0 \) then, for \( n \) large,

\[
H_{n+1} \leq \frac{L}{\alpha} \quad \text{if} \quad H_n \leq \frac{L}{\alpha} \quad \text{and, for all} \quad n, \quad F_{n+1} \leq \frac{F_n}{1 + \phi_m}.
\]

Proof.

1. The positivity is obtained by construction of the NSFD scheme (22).

2. From the first equation of (22), we have

\[
\frac{H_{n+1} - H_n}{\phi} \leq L - \Gamma^+ H_{n+1}.
\]

Solving for \( H_{n+1} \) and assuming that \( H_n \leq \frac{L}{\Gamma^+} \), we get

\[
H_{n+1} \leq \frac{L}{\Gamma^+}.
\] (31)

The second equation of (22) and equation (31) yield

\[
\frac{F_{n+1} - F_n}{\phi} \leq \alpha H_{n+1} - mF_{n+1} \leq \frac{\alpha L}{\Gamma^+} - mF_{n+1},
\]

which implies that

\[
F_{n+1} \leq \frac{\alpha L}{m \Gamma^+} \quad \text{if} \quad F_n \leq \frac{\alpha L}{m \Gamma^+}.
\] (32)

Therefore the NSFD scheme (22) is a dynamical system on \( \Omega \).

3. From the second equation in (22), we have

\[-\sigma F_n - mF_{n+1} \leq \frac{F_{n+1} - F_n}{\phi} \leq -mF_{n+1},\]

and thus

\[
\left( \frac{1 - \phi \sigma}{1 + \phi m} \right) F_n \leq F_{n+1} \leq \left( \frac{1}{1 + \phi m} \right) F_n.
\] (33)

From (33), we notice that \( F_{n+1} \to 0 \) as \( n \to \infty \). Therefore, for \( n \) large, the first equation in (22) gives

\[
\frac{H_{n+1} - H_n}{\phi} \leq L - \alpha H_{n+1}.
\]

so that

\[
H_{n+1} \leq \frac{L}{\alpha} \quad \text{if} \quad H_n \leq \frac{L}{\alpha}.
\] (34)

Theorem 4.2.

1. The fixed-points of the NSFD scheme (22) are the equilibrium points of the continuous model (1). Furthermore, they have the same stability properties whenever for \( J_c \), representing the Jacobian matrix associated with KMB model (1) at the interior equilibrium point, \( Q \) is chosen such that \( Q \geq ||J_c||^2/|\text{trace}J_c| \) apart from satisfying (30). In other words, there exists a unique interior fixed point, which is GAS while the “trivial” fixed-point \((0,0)\) is not attractive under the condition (13).
2. If the condition (13) is not satisfied, then the fixed point \((0, 0)\) is globally asymptotically stable.

Proof. We prove the theorem in different steps.

Step 1 A point \((H^*, F^*)\) is a fixed point of the NSFD scheme (22) if, and only, if it satisfies the defining equations (5) of the equilibria of the continuous KMB model (1).

Step 2 We prove the local asymptotic stability of the interior fixed-point. Putting \(U = H - H^*\) and \(V = F - F^*\),

the linearized continuous equation (A6) about the equilibrium point \((H^*, F^*)\) in terms of the eclosion function \(E(H, F)\) and the recruitment function \(R(H, F)\) can be re-written as

\[
\begin{pmatrix}
\frac{dU}{dt} \\
\frac{dV}{dt}
\end{pmatrix} = J_c \begin{pmatrix} U \\ V \end{pmatrix}
\]

where

\[
J_c = \begin{pmatrix}
E_H - R_H & E_F - R_F \\
R_H & R_F - m
\end{pmatrix} = \begin{pmatrix}
E_H & E_F - R_F \\
0 & R_F
\end{pmatrix} - \begin{pmatrix}
E_H & 0 \\
-R_H & m
\end{pmatrix}
\] (35)

is the Jacobian matrix at the interior equilibrium point \((H^*, F^*)\). Here and after, \(E_H, R_H, E_F\) and \(R_F\) denote the partial derivatives of the functions \(E\) and \(R\) at the point \((H^*, F^*)\).

The NSFD scheme (22), can be re-written in the form

\[
\begin{aligned}
\frac{H_{n+1} - H_n}{\phi} &= E(H_n, F_n) - R(H_{n+1}, F_n) \\
\frac{F_{n+1} - F_n}{\phi} &= R(H_{n+1}, F_n) - mF_{n+1}.
\end{aligned}
\] (36)

The linearized equation about the interior fixed-point \((H^*, F^*)\) is

\[
\begin{aligned}
(1 + \phi R_H)U_{n+1} &= (1 + \phi E_H)U_n + \phi(E_F - R_F)V_n \\
\phi R_H U_{n+1} + (1 + \phi m)V_{n+1} &= (1 + \phi R_F)V_n.
\end{aligned}
\] (37)

Solving the algebraic system (37) in \(U_{n+1}, V_{n+1}\), the linearized NSFD scheme reads

\[
\begin{pmatrix}
U_{n+1} \\
V_{n+1}
\end{pmatrix} = J_d \begin{pmatrix} U_n \\ V_n \end{pmatrix}
\] (38)
where
\[
J_d = \begin{pmatrix} 1 + \phi R_H & 0 \\ -\phi R_H & 1 + \phi m \end{pmatrix}^{-1} \begin{pmatrix} 1 + \phi E_H & \phi(E_F - R_F) \\ 0 & 1 + \phi R_F \end{pmatrix}
\]
\[
= T \begin{pmatrix} 1 + \phi E_H & \phi(E_F - R_F) \\ 0 & 1 + \phi R_F \end{pmatrix}; \quad T = \begin{pmatrix} \frac{1}{1 + \phi R_H} & 0 \\ \phi R_H & \frac{1}{(1 + \phi R_H)(1 + \phi m)} \end{pmatrix}.
\]

From the expression of \( J_c \) in (35) we have
\[
J_d = I + \phi T J_c. \quad (39)
\]

With \( R_H(H^*, F^*) \) being positive, the spectral radius of the matrix \((T - I)\) is less than 1. Therefore, the sequence \((T - I)^n\) of matrices converges to the null matrix. Consequently, the difference system (38) has the same qualitative behavior as the system
\[
\begin{pmatrix} \tilde{U}_{n+1} \\ \tilde{V}_{n+1} \end{pmatrix} = \tilde{J}_d \begin{pmatrix} \tilde{U}_n \\ \tilde{V}_n \end{pmatrix} \quad (40)
\]
where
\[
\tilde{J}_d = I + \phi J_c. \quad (41)
\]

It is easy to check that \( \mu \) is an eigenvalue of the matrix \( \tilde{J}_d \) if, and only, if \( \lambda = \frac{\mu - 1}{\phi} \) is an eigenvalue of \( J_c \). That is
\[
\mu = \phi \lambda + 1 = (1 + \phi \lambda_1) + \phi \lambda_2.
\]

Furthermore,
\[
|\mu|^2 = \begin{cases} 
1 - 2\phi|\lambda_1| + \phi^2|\lambda|^2 & \text{if } \lambda_1 < 0 \\
1 + 2\phi|\lambda_1| + \phi^2|\lambda|^2 & \text{if } \lambda_1 > 0.
\end{cases}
\]

Thus \(|\mu| > 1 \) if \( \lambda_1 > 0 \), whereas \(|\mu| < 1 \) if \( \lambda_1 < 0 \) whenever
\[
\phi < \frac{2|\lambda_1|}{|\lambda|^2}. \quad (42)
\]

The function \( \phi \) in (23) with \( Q \) as specified in the statement of Theorem 4.2 satisfies this condition (42) because \( \phi < \frac{1}{Q} \) and \( Q \geq \frac{|\lambda|^2}{2|\lambda_1|} \) for each eigenvalue of \( J_c \), given that \(|\lambda| \geq ||J_c||\) and \( \lambda_1 = \text{trace}J_c \) for the 2x2 matrix \( J_c \). This proves the local asymptotic stability of the fixed-point \((H^*, F^*)\) whenever the interior equilibrium point is locally asymptotically stable.
Step 3 We prove that the interior fixed-point \( X^* = (H^*, F^*) \) is globally asymptotically stable. We denote \((H, F)\) by \(X\). The fixed-point \( X^* \) being locally asymptotically stable, there exists \( \delta > 0 \), such that
\[
||X_0 - X^*|| < \delta \implies \lim_{n \to \infty} ||X_n - X^*|| = 0. \tag{43}
\]
Since the sequence \((X_n)\) is bounded in \( \mathbb{R}^2 \), it follows from Bolzano-Weierstrass theorem that there exists a subsequence \((X_{n_k})\) of \((X_n)\) that is convergent and thus
\[
\lim_{k \to \infty} ||X_{n_k} - X^*|| = 0, \tag{44}
\]
by the uniqueness of the limit. There exists an integer \( K > 0 \) such that
\[
||X_{n_K} - X^*|| < \delta. \tag{45}
\]
For any initial guess \( X_0 \), we have
\[
\lim_{n \to \infty} ||X_n - X^*|| = \lim_{n > n_K} ||X_n - X^*|| = 0 \quad \text{by (43)}.
\]

Step 4 We deal with the trivial fixed-point \((0,0)\).

The case when \( \Gamma = \Gamma^- < 0 \), as given by (4), is straightforward from the second equation in (22): the solutions tend to \((0,0)\) irrespective of the condition (13). For \( \Gamma = \Gamma^+ > 0 \), as given by (3), we proceed directly as follows:

Adding the two equations in (22), we obtain the discrete conservation law
\[
\frac{N_{n+1} - N_n}{\phi} = \frac{LN_n}{N_n + \omega} - mF_{n+1}. \tag{46}
\]
Under the condition (13), and using the definition of \( \Omega \), we have
\[
\frac{N_{n+1} - N_n}{\phi} \geq \frac{LN_n}{2\alpha L + \omega} - \frac{m \alpha L}{m \Gamma^+}.
\]
Hence
\[
N_{n+1} \geq \left( 1 + \phi \frac{L}{2\alpha L + \omega} \right) N_n - \frac{m \alpha L}{m \Gamma^+}.
\]
By Gronwall inequality, the sequence \((N_n)\) cannot tend to zero.

Since the NSFD scheme (22) is convergent, the discrete conversation law (46) has the same behaviour as the difference equation
\[
N_{n+1} - N_n = \phi \frac{L(N_n)}{N_n + \omega} - \phi m F_n
\]
for \( n \) large enough. Consequently, when condition (13) is violated it follows from the Lyapunov argument used in the continuous setting (see Appendix 1) that
\[
N_{n+1} - N_n < 0 \quad \text{for} \quad 0 < H_n \leq r \quad \text{and} \quad 0 < F_n \leq r.
\]
This shows that the fixed point \((0,0)\) is locally asymptotically stable [49]. The global asymptotic stability of the fixed-point \((0,0)\) is proved as in the step 3 above.
We now construct a NSFD scheme for the SP model (14). Using the method of sub-equations that led to the scheme (22), we propose, for the SP model, the NSFD scheme

\[
\begin{align*}
H_{n+1} - H_n \phi &= \frac{L(H_n + F_n)}{H_n + F_n + \omega} - (\alpha + \tilde{m})H_{n+1} \\
F_{n+1} - F_n \phi &= \alpha H_{n+1} - mF_{n+1},
\end{align*}
\]

(47)

which is equivalent to

\[
\begin{align*}
H_{n+1} &= \frac{(\alpha L(H_n + F_n) + F_n)}{H_n + F_n + \omega} \\
F_{n+1} &= \frac{(\alpha \phi H_{n+1} + F_n)}{1 + \phi m},
\end{align*}
\]

(48)

where \( \phi \) is defined in (23) with \( Q = \alpha + \tilde{m} + m \). In a similar manner to the proof of Theorem 3.2, we obtain the result below.

**Theorem 4.3.** The NSFD scheme (47) is a dynamical system in the biological feasible region \( \Omega \) defined in Theorem 3.2.

Adding the equations in (47), we have the conservation law

\[
\frac{N_{n+1} - N_n}{\phi} = \frac{LN_n}{N_n + \omega} - \tilde{m}H_{n+1} - mF_{n+1},
\]

from which it follows that

\[
-(m + \tilde{m})N_{n+1} \leq \frac{N_{n+1} - N_n}{\phi} \leq \frac{LN_n}{\omega} - \min\{m, \tilde{m}\}N_{n+1}.
\]

(49)

Thus

\[
\left(1 + \frac{1}{\phi(m + \tilde{m})} \right) N_n \leq N_{n+1} \leq \left(1 + \frac{\phi L}{\omega \min\{m, \tilde{m}\}} \right) N_n.
\]

(50)

We, therefore, have established the following result, which shows the GAS of the “trivial” fixed point in the more specific way below.

**Theorem 4.4.** Under the condition (15), the colony declines to zero in a contractive manner.

5 Numerical simulations

For numerical simulations for the discrete KMB-NSFD scheme (22), we use the parameters in Table 1 below, which are taken from [25].

<table>
<thead>
<tr>
<th>Parameter</th>
<th>L</th>
<th>( \alpha )</th>
<th>( \sigma )</th>
<th>( \omega )</th>
</tr>
</thead>
<tbody>
<tr>
<td>Value</td>
<td>2000</td>
<td>0.25</td>
<td>0.75</td>
<td>27000</td>
</tr>
</tbody>
</table>
The choice of the values $\alpha = 0.25$ and $\sigma = 0.75$ is motivated by the fact that, in the absence of foragers, new workers become foragers in a minimum of four days, and there is reversion / social inhibition from foragers class to hive class if more than one third of the total population are foragers [25]. To Table 1, we add the critical value, $m_c$, of the foragers death rate obtained from Equation (13) as $m_c = 0.355$. We will therefore use the quantities below and above the critical value $m_c$ namely $m = 0.24$ and $m = 0.4$, as in [25]. Finally, we take $\Delta t = 2$, a large value of step size which shows the power of the nonstandard approach as this is not permissible in classical numerical schemes [32].

In the KMB setting, Figure 2 illustrates through both the phase plane and the population-time axes the GAS of the interior equilibrium or fixed point when $m = 0.24$ in accordance with Theorem 2.1(2) and Theorem 4.2(1). Furthermore, when $m = 0.4$, the CCD phenomenon or the GAS of the trivial equilibrium or fixed point occurs according to Theorem 2.1(3) and Theorem 4.2(2), as displayed in Figure 3.

Regarding the SP setting, we still use the values in Table 1 because the laying rate of the queen remains the same. The rate, $\tilde{m}$, at which parasites leave the hive class is, for simplicity, taken to be equal to the death rate $m = 0.24$ of foragers. As predicted by Theorem 3.2(2) and Theorem 4.4, Figure 4 exhibits the decay to zero of the total population in the following specific manner, which justifies the terminology “capensis calamity” [4]:

- The colony collapses in 100 days for the SP model when $m = 0.24$ (Figure 4b), while there is no CCD for the KMB model in this case (see Figure 2);
- The colony collapses in 400 days for the KMB model when $m = 0.4$ (Figure 3b).

![Graphs](image-url)

Figure 2: GAS of the interior fixed-point for the KMB-NSFD scheme (22).
6 Conclusion and discussion

The alarming declines of the population of honeybees constitute a serious threat to ecosystem and honeybee products. The situation is worsen nowadays by an increase in colony losses world-wide through a phenomenon known as colony collapse disorder (CCD) and particularly the Capensis Calamity (CC) in South Africa. Both phenomenon are characterised by imminent losses of honeybee colonies, thus resulting in the decline of pollinators.

This work is motivated by the paper [25], in which a mathematical model consisting of the hive compartment, $H$, and the foragers compartment, $F$, with variable recruitment rate, $R = R(H, F)$, is proposed. We have constructed a nonstandard finite difference (NSFD) scheme for this model, KMB, that is dynamically consistent with the decline property. More precisely, we have theoretically established and numerically illustrated the result below. There exists a critical value $m_c$ of the parameter $m$, the foragers death rate, which is a transcritical bifurcation. That is: for $m > m_c$, the trivial fixed point $(0, 0)$ is globally asymptotically stable (GAS), which means that the CCD phenomenon occurs. For $m < m_c$, there appears an interior fixed point which is GAS. When recruitment is negative, we proved that the CCD phenomenon arises irrespective of the value of $m$. 

Figure 3: CCD in the KMB-NSFD scheme (22).

Figure 4: Faster decline of the colony for SP-NSFD scheme (47)
Apart from the model in [25], we formulated a model relevant to South Africa (social parasitic, SP, model). This model is motivated by the CC that arose when some Cape honeybees (Apis mellifera capensis), parasites, were moved into the region of the Savannah honeybees (Apis mellifera scutellata), hosts. We have described the parasitic scenario by a low positive and constant recruitment rate due to the presence of parasites who do not contribute to foraging but leave the hive compartment at a constant rate after benefiting abusively from the food produced by the hosts. We have constructed a NSFD scheme for the SP model and proved theoretically and illustrated numerically the CC phenomenon. In the SP model, we observed a rapid decline in the total population of the host colony (Figure 4). This means that the SP model indeed represent abandoned brood as a diagnostic of CCD colonies [25]. In our simulations, it takes about 100 days for the Savannah honeybee population to reach zero, regardless of the foragers death rate, after the Capensis clones have entered their colony (Figure 4), whereas it takes about 400 days for a normal honeybee population, as in the KMB model, to reach zero when the death rate of foragers is above a certain critical point (Figure 3).

Along the lines of this paper, there arises the following natural question on which we are working; which measures and strategies are needed to avoid or at least to reduce honeybee colony declines? Since the model proposed in [25], suggests that the colony growth is attained when the death rate, $m$, of the foragers is low, a possible strategy is to make the critical value $m_c$ of $m$ small. Given the fact that $m_c$ is a function of the parameters $L, \alpha, \sigma,$ and $\omega$, it can be seen from (4) how $m_c$ can be reduced by appropriate choice of some of these parameters. From the biological point of view, growth of colonies can be achieved by providing enough resources, either through planting many different flowering plants (bee plants) closer to the colonies as this would prevent foragers from undertaking long and risky travel [46]. Enactment of legislation that will reduce or ban indiscriminate use of pesticides in agriculture could also help in this regard [44]. Regarding the model with social parasites, a strategy is to target the parasites and reduce their invading activity [5]. Measures that could be taken to reduce the spread of the social parasites in honeybees include, not moving honeybees of the different sub-species between regions [45] in South Africa as enshrined by the law. This is important since the clone relies heavily on anthropogenic factors to move from one region. Obeying enacted laws that prevent migratory beekeeping and enforcement of same would help in stopping the spread. In the event that migration is unavoidable, hives should be inspected properly after migration for the presence of the parasites and infested hives placed in quarantine and destroyed accordingly.

The study can be extended by considering in the model numerous factors responsible for colony declines. These include: death rate of hive bees and brood [11, 19], pests and diseases [18] and climate change [19]. It is of interest to investigate further the complex relation that exists between the number of eggs reared in a colony and the number of bees in the hive by considering other types of Holing functions. Additional compartments will be needed to model colony failure as contagious by viruses and other pathogens [21,28,39], and to investigate the interactions of suitable strategies (e.g food) and forager mortality on colony fate [26,40].

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7 Appendix 1

Proof of Theorem 2.1

Step 1 We prove that the KMB model (1) is a dynamical system in $\Omega$, in the sense that for an initial condition $(H_0, F_0) \in \Omega$, there exists a unique global solution $(H(t), F(t)) \in \Omega$ for all $t \geq 0$.

This is a consequence of the following:

a. The existence of unique local solution, which is trivial.

b. The positivity of any solution corresponding to positive initial conditions, which follows from the intermediate value theorem.

c. The boundedness of any solution, which is established by Gronwall inequality.

Step 2 We prove that the positivity of the forager component of the equilibrium point given in Equation (8) is equivalent to condition (see [11] and [29] for more details):

$$m < \frac{L}{2\omega} \left( (\alpha + \sigma) + \sqrt{(\alpha - \sigma)^2 + \frac{4L\omega}{\omega}} \right) := m_c,$$

(taking into account (12)).

The condition

$$F^* = \frac{L}{m} \frac{\omega J}{1 + J} > 0$$

(A2)

is, in view of (9), equivalent to

$$m < \frac{L}{\omega} \left[ \left( \frac{\alpha}{m} - \frac{\sigma}{m} - 1 \right) + \sqrt{\left( \frac{\alpha}{m} - \frac{\sigma}{m} - 1 \right)^2 + \frac{4\alpha}{m}} \right].$$

(A3)

Rationalising the denominator, we get

$$m < \frac{L}{2\omega} \left[ \left( \frac{\alpha}{m} + \frac{\sigma}{m} + 1 \right) + \sqrt{\left( \frac{\alpha}{m} - \frac{\sigma}{m} - 1 \right)^2 + \frac{4\alpha}{m}} \right],$$

(A4)

which is equivalent to

$$m\alpha - \frac{L}{2\omega} (\alpha + \sigma + m) < \frac{Lm}{2\omega} \sqrt{\left( \frac{\alpha}{m} - \frac{\sigma}{m} - 1 \right)^2 + \frac{4\alpha}{m}}$$

(A5)
From (A5), we deal with the cases when 
\( m\alpha - \frac{L}{\omega}(\alpha + \sigma + m) > 0 \) and 
\( m\alpha - \frac{L}{\omega}(\alpha + \sigma + m) \leq 0 \) separately. In both cases, algebraic manipulation lead to the condition (A1).

Step 3 We prove the local asymptotic stability of the interior equilibrium point. We use Hartman-Grobman [10] linearization process. The Jacobian matrix \( J_c \) of the right hand side of the model (1) is

\[
J_c = \begin{pmatrix}
\frac{\omega L}{(H + F + \omega)^2} - \alpha + \frac{\sigma F}{(H + F)^2} & \frac{\omega L}{(H + F + \omega)^2} + \frac{\sigma}{(H + F)^2} \\
\alpha - \frac{F}{(H + F)^2} & -\frac{m}{(H + F)^2} - \sigma
\end{pmatrix}
\]  

(A6)

At the interior equilibrium point \((H^*, F^*)\) given by (7)-(8), the Jacobian matrix \( J_c \) is

\[
J_c = \begin{pmatrix}
\frac{\omega m^2 J^2}{L(1 + J)^2} - \alpha + \frac{\sigma J^2}{(1 + J)^2} & \frac{\omega m^2 J^2}{L(1 + J)^2} + \frac{\sigma}{(1 + J)^2} \\
\alpha - \frac{\sigma J^2}{(1 + J)^2} & -\frac{\sigma}{(1 + J)^2} - m
\end{pmatrix}
\]  

(A7)

which has trace

\[
tr J_c = m \left( \frac{\omega m J^2}{L(1 + J)^2} - 1 \right) - \alpha + \frac{J - 1}{1 + J}
\]  

(A8)

and determinant

\[
det J_c = \frac{\omega m^3 J^2}{L(1 + J)^2} (J - 1) - \frac{\omega m^3 J^2}{L(1 + J)^2} + \alpha m - \frac{\sigma m J^2}{(1 + J)^2} - \frac{\omega m^2 J^2}{L(1 + J)^2}.
\]  

(A9)

We show that \( tr J_c < 0 \). Since \( J < 1 \) as shown in (11), it suffices to show that

\[
\frac{\omega m J^2}{L(1 + J)^2} - 1 < 0.
\]

Now,

\[
\left( \frac{\omega J^2 m}{L(1 + J)^2} - 1 \right) < \left( \frac{\omega J^2 (1 + J)}{\omega J (1 + J)^2} - 1 \right) \quad \text{using (A2) i.e.} \quad m \leq \frac{L(1 + J)}{\omega J}
\]

\[
= \left( \frac{J}{1 + J} - 1 \right)
\]

\[
< 0.
\]

Next, we show that \( det J_c > 0 \). From the defining equation of equilibrium points, (5) and (6), we have

\[
\sigma = \left( \alpha - m J \right) \frac{1 + J}{J}.
\]  

(A10)
With this, the expression (A9) of $\det J_c$ simplifies to

$$
\det J_c = \alpha m \left[ 1 - \frac{\omega m J}{L(1 + J)^2} - \frac{J}{1 + J} \right] + m^2 J^2 \left[ 1 - \frac{\omega m J}{L(1 + J)} \right].
$$

Thus,

$$
\det J_c > \alpha m \left[ 1 - \frac{1}{(1 + J)} - \frac{J}{(1 + J)} \right] + m^2 J^2 \left[ 1 - 1 \right] \text{ using } m < \frac{L(1 + J)}{\omega J}
$$

$$
= 0.
$$

**Step 4** The local qualitative analysis of the trivial equilibrium (0,0) is studied as follows: For $\Gamma := \Gamma^- < 0$, it is straightforward from the second equation of (5) that (0,0) is globally attractive regardless of the condition (A1). When $\Gamma := \Gamma^+ > 0$, we consider two cases:

1. The case when condition (A1) is violated so that there is no interior equilibrium point.

   Here we use a Lyapunov argument as in [21], based on the Lyapunov function

   $$
   V \equiv V(H, F) := H + F.
   $$

   The derivative $V'$ along the trajectories is

   $$
   V' = \frac{L(H + F)}{H + F + \omega} - mF,
   $$

   which is the right hand side of the following conservation law obtained by adding the equations in (1):

   $$
   \frac{dN}{dt} = LN - mF.
   $$

   Observe that

   $$
   \frac{\partial V'}{\partial F}(H, 0) = \frac{L\omega}{(H + \omega)^2}
   $$

   $$
   \leq \frac{L}{\omega} - m
   $$

   $$
   < 0 \text{ by (12) since the death rate is large, i.e } m > \alpha.
   $$

   Thus, there exists $r > 0$ such that $V'(H, F) < 0$ for $0 < H \leq r$ and $0 < F \leq r$. This implies that (0,0) is locally asymptotically stable [49,50].

2. The case when condition (A1) is satisfied.

   We show that (0,0) is not attractive. Using the fact that $m < m_c$ (see (A1)), $N \leq \frac{2\alpha L}{m \Gamma^+}$ and $F \leq \frac{\alpha L}{m \Gamma^+}$ (see definition of $\Omega$) we have from (A12)

   $$
   \frac{LN}{2\alpha L + \omega} - m_c \frac{\alpha L}{m \Gamma^+} \leq \frac{dN}{dt}.
   $$
By comparison theorem (Gronwall inequality)
\[ N(t) \geq N_0 e^{at} + \frac{b}{a}(1 - e^{at}) \quad \text{where} \quad a = \frac{L}{2\alpha L m \Gamma^+ + \omega}, \quad b = \beta \frac{aL}{m \Gamma^+}. \]

It follows that \( N(t) \) cannot tend to zero.

**Step 5** We prove that the system (1) has no periodic solutions. This follows from Dulac criterion [10] using the function
\[ \beta(H, F) = \frac{1}{HF}. \]

**Step 6** Combining step 1 - step 3, it follows from Poincaré-Bendixon theorem [10] that the interior equilibrium point is globally asymptotically stable when it exists; otherwise the trivial equilibrium point is globally asymptotically stable.

References


