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On synergistic co-infection in crop diseases. The case of the Maize Lethal Necrosis Disease

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Abstract

Crops are often subject to intense attacks by pests and diseases. Among them, Maize Lethal Necrosis (MLN) is a serious disease that impact maize crops in many Southern countries. It results from the synergistic interaction of two plant viruses, transmitted by two vectors. In this paper, we develop a general crop-vector-borne disease deterministic model for synergistic co-infection, with a particular focus on MLN disease. A theoretical analysis shows that different thresholds exists that drive the dynamics of the system: the well known basic reproduction numbers and also invasion reproduction numbers. The latter are essential for the emergence or not of the MLN disease. After a global sensitivity analysis, we illustrate our results through numerical simulations and discuss potential control methods such as vector control and roguing.

Keywords: vector-borne plant disease, co-infection, synergistic interaction, invasion reproduction number, sensitivity analysis, numerical simulations

1. Introduction

Plants, wild and domestic, are subject to diseases. Understanding and controlling of plant diseases is of critical importance for reliable food production. There are wide ranging examples of devastating plant diseases preceding the earliest writings (see [1] and references there in). For example, the Bible and other early writings mention diseases such as rusts, mildews and blights. More recent disease outbreaks with far-reaching consequences include the late blight of potato in Ireland (1845 -1860), powdery mildew of grapes in France (1851), Southern corn leaf blight in Africa (1990 - present), and many others. In addition to climate change and lack of investment in farming, plant diseases cause major food insecurity throughout the world. The Food and Agriculture Organisation estimates that pests and diseases are responsible for about 25% of crop loss. These loses may result in hunger and starvation, especially in less-developed countries. However, disease control in crops is generally successful. Among other methods, disease control can be achieved by using disease resistant or non-susceptible plants, crop rotation, use of pathogen free seeds, control of moisture levels, pesticides, etc.

Plant diseases generally involve interaction between multiple pathogens and the complexities are not captured in single host-single disease systems. In particular, co-infection is the infection of a multiple pathogen species to a single host that may be the causative of distinct diseases or variants of the same parasite [2]. In humans/animal hosts, mixed virus infections are relatively infrequent and are generally associated with depression of the immune system. In plant hosts, interactions between viruses can result in synergism, antagonism, coexistence, mutualism, cooperation or a neutral interaction, see for example [3, 4]. In plant diseases, the level of damage on the plant will depend on the outcome of the interactions and the host response. Advances in the study of host-pathogen dynamics suggests co-infection can lead to several outcomes. These include, competitive exclusion where over time one pathogen outcompetes the other, mutualistic coexistence where both pathogens benefit from the interaction, or emergence of

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new recombined, and more damaging epidemic. Several examples of synergistic interaction are given in [3], but here we focus on the Maize Lethal Necrosis case.

Maize, rice and wheat are the three most widely grown crops around the world, in particular, in developing countries. Maize alone contributes to at least 30% of the food calories to more than 4.5 billion people in 94 developing countries and it plays a crucial role in the livelihoods of millions of small scale farmers [5]. For instance, in 2017, the area harvested in Africa was around 40 million hectare (ha) and produced around 8.04 million tons. In Southern Africa alone, the area harvested was around 2.9 million ha and produced around 1.07 million tons [6]. Although maize is the basis for food security in the majority of countries in Africa, the yield has drastically decreased over the years due to several factors, including high incidence of diseases, pests and weeds. Diseases that have threatened corn production in Sub-Saharan Africa include Maize Streak Virus (MSV), and Parasitic Weed Striga [5]. In 2011, a devastating disease of maize, the Maize Lethal Necrosis Disease (MLND), also called Corn Lethal Necrosis Disease (CLND), see for instance, [7, 8], was first reported on the African continent in Kenya. The disease affected almost all commercial varieties causing a loss ranging between 30-100% depending on the severity of the disease and the time of infection [9]. In 2012, just in Kenya alone, the MLND affected around 77 000 ha, translating into an estimated loss of US$52 million [10, 7].

MLND is caused by a synergistic interaction between Maize Chlorotic Mottle Virus (MCMV) and one of several viruses from the Potyviridae family [11]. MLND was first reported in Kansas (USA), as a synergism interaction of MCMV and either Maize Dwarf Mosaic Virus (MDMV) or Wheat Streak Mosaic Virus (WSMV), and later in Nebraska [12, 13]. In 2011, MLND was reported in China as a synergism interaction between MCMV and Sugarcane Mozaic Virus (SCMV) [14]. In Africa, the first outbreak of MLND was reported in Kenya [10], and was associated with potyvirus SCMV and later in Rwanda [15], Uganda, Tanzania [9] and Ethiopia [9]. There are other viruses in the family Potyviridae that cause MLND in co-infection with MCMV, including, MDMV and Johnsongrass Mosaic Virus (JGMV) [16]. Among these Potyvirus, SCMV is the most predominant [9]. MCMV is the primary virus that drives MLND, [7]. The natural host of MCMV is maize, however there is a broad range of plants serving as reservoir of MCMV including sugarcane and finger millet [9]. The above clearly shows the complex nature of MLND.

MCMV outbreak was reported from the Southern Rift Valley of Kenya in 2011 [10]. Maize infected with MCMV show an array of symptoms ranging from mild chlorosis mottling to severe mosaic and stunting, yellowing necrosis and premature plant death [7]. However, as reported in [11], it is not clear whether this symptoms are due to MCMV infection alone, or in co-infection with another virus, or stress. MCMV co-infection with potyvirus is a synergism, that is, the disease progression and symptoms are greater in maize infected with MCMV and potyvirus. For instance, in [17] it was reported that the concentration of MCMV were $1.9 - 11$ fold higher in maize infected with MCMV and potyvirus than in singly infected maize. Maize infected with SCMV shows symptoms similar to those by MCMV: mosaic, chlorosis and stunting in maize. Symptoms can be bright or muted depending on environment and time of infection [11, 7]. There are a number of vectors transmitting MCMV. For instance, beetles [18], flower thrips [19], and maize thrips [19, 7]. In this work, for the sake of simplicity, we focus on maize thrips (*Frankliniella Williamsi*) that transmit MCMV in a semi-persistent manner [19]. In Eastern Africa, thrips have been observed in high densities in fields affected by MLND and MCMV, even several years prior to the first report of MLN, [7, 10]. Although the range of vectors transmitting MCMV in Africa is not known, thrips have been observed in all fields where maize has been grown, including in MLND- and SCMV-affected regions [9], which suggest that they play a major role on MCMV transmission in Africa. MCMV can also be transmitted via seed from MCMV-infected maize [20] or soil from MCMV-affected fields [7]. In this work, we will not consider these transmission routes. However, seed transmission can be considered implicitly through the initial conditions, thus assuming that maize crop is initially infected by MCMV. Taking into account soil transmission would require additional compartments, see for example the work [21]

Aphids species are the vectors for maize-infecting members of the genus potyvirus. For instance, MDMV and SCMV are transmitted by a number of hosts including *Rhopalosiphum maidis*, *Rhopalosiphum padi*, *Myzus persicae*, *Schizaphis graminum*, in a non persistent manner, which means that the vectors acquires the virus within seconds of feeding on maize and not retained for more than a few hours with no report of latent period [22]. Aphids are widely distributed and seem to be abundant in regions where maize is grown, including East Africa. Apart from other potyvirus, WSMV is transmitted by the

virus *Wheat Curl Mite* in a semi persistent manner [23]. However, WSMV has not been reported in East Africa. SCMV can also be transmitted via seed [24] and soil [7].

Mathematical models of plant-virus and plant-vectors-virus have played an important role on guiding research direction and improving understanding across the characteristic spatio-temporal scales of plants virus epidemic and, in some cases, led to the direct application in disease control [25, 26, 1]. There are several mathematical models formulated to study co-infection in humans/animals, particularly, HIV/TB [27, 28], HIV/Malaria [29], and many others [30]. However, very few models have been proposed to study co-infection in plants/crops. For co-infection in plants/crops, we highlight the work [2], where the authors proposed a deterministic model to investigate some of the general principles of epidemiological plant diseases caused by helper dependent virus complex. In [3], the authors proposed a model in which transmission loss rates are due to the different viruses - including possibilities of co-infection. Also, in a very recent work, the authors in [31] studied a crop co-infection model with one vector.

The only MLND co-infection mathematical models we are familiar with were proposed in [32], where the spread of MLND within and between growing seasons of maize was modeled. The authors considered the local transmission through vectors, seed and infested sources of inoculum in the soil. However, in their model, the vectors dynamics is implicitly modeled, such that the vector borne transmissions within a field depends on the densities of infected and uninfected plants. In their work, control strategies such as clean seeds, insecticides, crop rotation and roguing were proposed. More recently, the authors in [33] proposed a general epidemiological model for one vector specie and one plant specie with co-infection in the host. Contrary to the models proposed in [32, 33], in this work we formulate a two vector species (aphids transmitting SCMV and thrips transmitting MCMV) and one host plant specie (maize) model that allows co-infection of the host.

The rest of the paper is organized as follows. In the following section we will formulate a generic synergistic co-infection vector-borne model, applied to the MLN system. The mathematical analysis of the model is given in Section 3. The Type/Invasion Reproduction numbers are derived in this section and we show permanence of the model. Parameter estimation, global sensitivity analysis and numerical simulations that support the theory are given in Section 4, where also we discuss the usefulness of different control strategies. The last section gives some concluding remarks.

2. Model formulation

Our model is intended to be as generic as possible so that it can be applied to different co-infection systems, like co-infections with MCMV and any other potyvirus [3]. However, for sake of clarity and taking into account the predominance of potyvirus, e.g., SCMV implicated in MLND in Africa, we consider the SCMV as one example of potyvirus. Therefore, motivated by the work [32], we model the dynamics of MCMV and SCMV within a single growing season and we focus only on the transmission through vectors. Potyvirus, and so is SCMV, are transmitted by aphids in a non-persistent manner and there are approximately 25 aphids species that vector SCMV [34, 9, 35]. There are several insects that vector MCMV and these include several species of beetles [18] and thrips [9] in a semi-persistent manner. However, according to [9], thrips have been observed in all fields where maize has grown including MCMV-affected field. A thrip (aphid) is infected by the MCMV (SCMV) virus after feeding on an infected maize plant. Therefore, we assume, for simplicity that the thrips are the vectors transmitting MCMV. For convenience, we refer to MCMV as virus a and SCMV as virus b. To make the model more comprehensible, the following assumptions are made:

1. All viruses are not vertically transmitted in both the vector and in plants. However, as alluded above, this assumption can be relaxed through initial conditions by assuming an initially infected maize crop.
2. Maize is planted and grows almost in the same way.
3. Each vector specie can only be inoculated by only a single virus.
4. The vector population (both thrips and aphids) is not impacted by the presents of the virus.

The maize plats are divided into four epidemiological states: the number of healthy plants H_p, the number of plants infected by MCMV I_{pa}, the number plants infected by SCMV I_{pb}, and the number of plants infected by both viruses I_{pm}. The total population of maize in the field is given by $N = H_p + I_{pa} + I_{pb} + I_{pm}$. For vector population, we consider two epidemiological states for each vector specie:

the susceptible S_a and S_b, and the infective, I_a and I_b, such that the total population of thrips (aphids) is given by $V_a = I_a + S_a$ ($V_b = S_b + I_a$). Since our study takes place in East and Central Africa with appropriate environmental conditions, such that maize is produced along the year, we also assume vital dynamics for both the maize crops and the pests.

We assume a net planting rate, Λ_p, in the healthy host compartment only, and, for sake of genericity, we consider a recovery rate, ω_{pi} of plants infected by virus $i = a, b$. Since the synergistic interaction leads to high MCMV concentrations in mix-infected plants compared with single-infected plants [36], and because MLND can cause total yield loss, we assume that MLN-infected maize cannot recover. Healthy crops are assumed to have a mortality rate μ_{ph} and plants infected with virus $i = a, b, m$ have mortality μ_{pi}. Unlike the work of [32], where the co-infection does not induce mortality, here these rates includes the natural mortality and the virus induced mortality. Thus, according to our literature review, we consider, in the rest of the paper, that $\mu_{pm} \geq \mu_{pi} \geq \mu_{ph}$, $i = a, b$. The net vector birth rate is given by Λ_i, δ is the recovery rate of thrips from the MCMV virus, γ is the recovery rate of aphids affected by the SCMV virus, μ_a is the death rate of the thrips and μ_b is the death rate of aphids.

The infection between the vectors and the plants is modelled by the mass action principle and we assume frequency dependent transmission. The vectors are assumed to have little to no damage on the plants. We consider the contact rate β_{pa} (β_{pb}) as the number of contacts per unit time (day) between thrips (aphids) and plants which would result in infection if the thrip (aphid) is infectious. Also, the contact rate β_{ap} (β_{bp}) represents the number of contacts per unit of time (day) that effectively transmit the infection to thrips (aphids) from an infectious plant. Furthermore, we consider additional contact rates, β_{am}, β_{ma}, β_{bm} and β_{mb}, related to the MLN disease transmission. However, so far, there is no specific knowledge (estimates) about these parameters. Details on the selection of parameters will be given in Section 4.

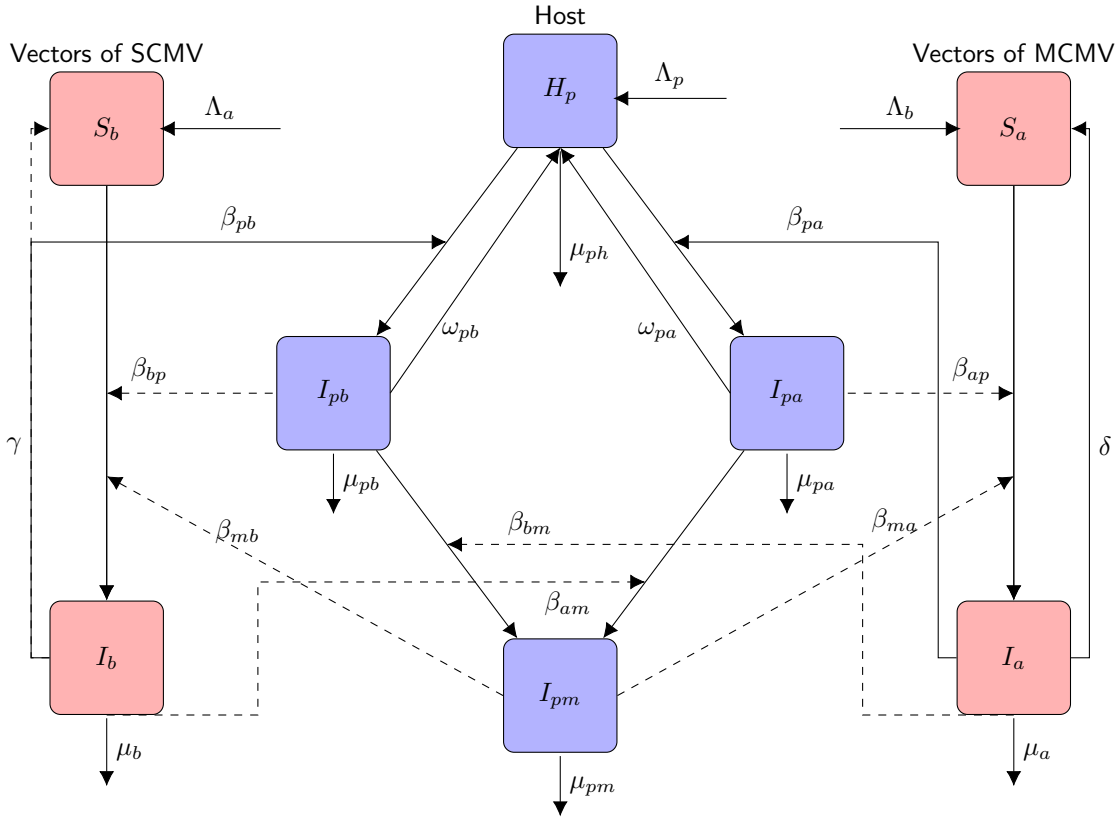

Figure 1: MLND co-infection model flow chart

All the model parameters are non-negative and we summarise them in Table 1, page 6. According to the compartmental diagram provided in Figure 1, page 4, the MLND mathematical model is as follows:

$$\begin{cases} \dfrac{dH_p}{dt} &= \Lambda_p - \beta_{pa}\dfrac{I_a}{V_a}H_p - \beta_{pb}\dfrac{I_b}{V_b}H_p + \omega_{pa}I_{pa} + \omega_{pb}I_{pb} - \mu_{ph}H_p \\ \dfrac{dI_{pa}}{dt} &= \beta_{pa}\dfrac{I_a}{V_a}H_p - \beta_{am}\dfrac{I_b}{V_b}I_{pa} - (\omega_{pa}+\mu_{pa})\,I_{pa} \\ \dfrac{dI_{pb}}{dt} &= \beta_{pb}\dfrac{I_b}{V_b}H_p - \beta_{bm}\dfrac{I_a}{V_a}I_{pb} - (\omega_{pb}+\mu_{pb})\,I_{pb} \\ \dfrac{dI_{pm}}{dt} &= \beta_{bm}\dfrac{I_a}{V_a}I_{pb} + \beta_{am}\dfrac{I_b}{V_b}I_{pa} - \mu_{pm}I_{pm}, \end{cases}, \tag{1}$$

with

$$\begin{cases} \dfrac{dS_a}{dt} &= \Lambda_a - \mu_a S_a - \beta_{ap}\dfrac{I_{pa}}{N}S_a - \beta_{ma}\dfrac{I_{pm}}{N}S_a + \delta I_a \\ \dfrac{dI_a}{dt} &= \beta_{ma}\dfrac{I_{pm}}{N}S_a + \beta_{ap}\dfrac{I_{pa}}{N}S_a - \delta I_a - \mu_a I_a \end{cases}, \tag{2}$$

and

$$\begin{cases} \dfrac{dS_b}{dt} &= \Lambda_b - \mu_b S_b - \beta_{bp}\dfrac{I_{pb}}{N}S_b - \beta_{mb}\dfrac{I_{pm}}{N}S_b + \gamma I_b \\ \dfrac{dI_b}{dt} &= \beta_{mb}\dfrac{I_{pm}}{N}S_b + \beta_{bp}\dfrac{I_{pb}}{N}S_b - \gamma I_b - \mu_b I_b, \end{cases}, \tag{3}$$

where $N = H_p + I_{pa} + I_{pb} + I_{pm}$, $V_a = S_a + I_a$ and $V_b = S_b + I_b$. The total populations take the following form

$$\begin{cases} \dfrac{dN}{dt} &= \Lambda_p - \mu_h H_p - \mu_{pa}I_{pa} - \mu_{pb}I_{pb} - \mu_{pm}I_{pm} \geq \Lambda_p - \mu_m N \\ \dfrac{dV_a}{dt} &= \Lambda_a - \mu_a V_a \\ \dfrac{dV_b}{dt} &= \Lambda_b - \mu_b V_b \end{cases}, \tag{4}$$

with $N(0) = N^0 \geq 0$, $V_a(0) = V_a^0 \geq 0$, $V_b = V_b^0 \geq 0$, where we have assumed $\mu_m = \max\{\mu_{ph}, \mu_{pa}, \mu_{pb}, \mu_{pm}\} > 0$.

From $(4)_2$ and $(4)_3$, we deduce $V_a \to \dfrac{\Lambda_a}{\mu_a}$, $V_b \to \dfrac{\Lambda_b}{\mu_b}$, respectively, when $t \to \infty$. We set $\tilde{V}_a = \dfrac{\Lambda_a}{\mu_a}$, $\tilde{V}_b = \dfrac{\Lambda_b}{\mu_b}$ and $\tilde{N}_p = \dfrac{\Lambda_p}{\mu_{ph}}$. Note also that since $V_a(0), V_b(0), N(0) \geq 0$, then, from (4), $V_a(t) > 0$, $V_b(t) > 0$, and $N(t) > 0$, for all $t > 0$. Thus, the solution of (4), for any initial condition in Ω_{N,V_a,V_b}, remains in Ω_{N,V_a,V_b}, where

$$\Omega_{N,V_a,V_b} = \left]0, \tilde{N}_p\right] \times \left]0, \tilde{V}_a\right[\times \left]0, \tilde{V}_b\right].$$

3. The mathematical analysis

First, through the next result, we show that the model (1)-(2)-(3) is mathematically and biologically well posed in

$$\Omega = \left\{(H_p, I_{pa}, I_{pb}, I_{pm}, S_a, I_a, S_b, I_b) \in [0, \tilde{N}_p]^4 \times [0, \tilde{V}_a]^2 \times [0, \tilde{V}_b]^2\right\}.$$

Theorem 3.1. *Assuming that all initial conditions lie in Ω, then system (1)-(2)-(3) has a unique solution that remains in Ω for all positive time t.*

Proof : The right hand sides of (1)-(2)-(3) are continuously differentiable map (C^1). Then, by the Cauchy-Lipschitz theorem [47], system (1)-(2)-(3) has a unique maximal solution. Rewriting the system (1)-(2)-(3) in the form

$$\frac{dx}{dt} = A(x)x + b,$$

Parameters	Description	Unit	Range	Baseline for simulations	Reference
β_{pa}	Rate of transmission of MCMV from Infected thrips to Susceptible plant	day^{-1}	$[0.04; 0.25]$	0.073	[19, 37]
β_{pb}	Rate of transmission of SCMV from Infected aphids to Susceptible plant	day^{-1}	$[0.04; 0.25]$	0.07	[38, 39, 40, 41]
β_{am}	Rate of transmission of MLND from SCMV Infected thrips to MCMV Infected plant	day^{-1}	$[0.08; 0.90]$	0.1	
β_{bm}	Rate of transmission of MLND from MCMV Infected aphids to SCMV Infected plant	day^{-1}	$[0.08; 0.90]$	0.2	
β_{ap}		day^{-1}	$[0.04; 0.25]$	0.073	
β_{bp}		day^{-1}	$[0.02; 0.72]$	0.07	
β_{ma}		day^{-1}	$[0.08; 0.90]$	0.1	
β_{mb}		day^{-1}	$[0.08; 0.90]$	0.2	
μ_a	Natural death rate for thrips.	day^{-1}	$[0.07; 0.14]$	0.092	[37, 42]
μ_b	Natural death rate for aphids.	day^{-1}	$[0.07; 0.1]$	0.079	[40, 41, 43]
μ_{ph}	Death/harvest rate for susceptible plant	day^{-1}	$[1/100, 1/40]$	1/60	
μ_{pa}	Death/harvest rate for plant infected with MCMV.	day^{-1}	$[1/100, 1/40]$	1/60	
μ_{pb}	Death/harvest rate for plant infected with SCMV.	day^{-1}	$[1/100, 1/40]$	1/60	
μ_{pm}	Death/harvest rate for MLND infected plant.	day^{-1}	$[1/100, 1/20]$	1/30	
ω_{pa}	Recovery rate for plant infected with MCMV.	day^{-1}	$[0, 1/30]$	0	
ω_{pb}	Recovery rate for plant infected with SCMV.	day^{-1}	$[0, 1/20]$	0	
Λ_p	Recruitment rate for plant.	Ind $\times$ day^{-1}	$[378, 890]$	600	[44]
Λ_a	Recruitment rate for thrips.	Ind $\times$ day^{-1}	$[32338; 88749]$	50535	[45]
Λ_b	Recruitment rate for aphids	Ind $\times$ day^{-1}	$[20132; 33238]$	25638	[45, 41]
δ	Recovery rate for thrips infected by MCMV	day^{-1}	$[0.13; 0.17]$	0.15	[19]
γ	Recovery rate for aphids infected by SCMV	day^{-1}	$[2, 12]$	3	[46]

Table 1: The parameters for the MLND model (1), (2) and (3). The parameters estimates are explained in section 4.

where $x = (H_p, I_{pa}, I_{pb}, I_{pm}, S_a, I_a, S_b, I_b)^T$, $b = (\Lambda_p, 0, 0, 0, \Lambda_a, 0, \Lambda_b, 0)^T \geq 0$ and

$$A = \begin{pmatrix} -A_{11} & \omega_{pa} & \omega_{pb} & 0 & 0 & 0 & 0 & 0 \\ \beta_{pa}\frac{I_a}{V_a} & -A_{22} & 0 & 0 & 0 & 0 & 0 & 0 \\ \beta_{pb}\frac{I_b}{V_b} & 0 & -A_{33} & 0 & 0 & 0 & 0 & 0 \\ 0 & \beta_{am}\frac{I_b}{V_b} & \beta_{bm}\frac{I_a}{V_a} & -\mu_{pm} & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & -A_{55} & \delta & 0 & 0 \\ 0 & 0 & 0 & 0 & \beta_{ap}\frac{I_{pa}}{N} + \beta_{ma}\frac{I_{pm}}{N} & -A_{66} & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & -A_{77} & \gamma \\ 0 & 0 & 0 & 0 & 0 & 0 & \beta_{bp}\frac{I_{pb}}{N} + \beta_{mb}\frac{I_{pm}}{N} & -A_{88} \end{pmatrix},$$

with $A_{11} = \beta_{pa}\frac{I_a}{V_a} + \beta_{pb}\frac{I_b}{V_b} + \mu_{ph}$, $A_{22} = \beta_{am}\frac{I_b}{V_b} + (\omega_{pa} + \mu_{pa})$, $A_{33} = \beta_{bm}\frac{I_a}{V_a} + (\omega_b + \mu_{pb})$, $A_{55} = \mu_a + \beta_{ap}\frac{I_{pa}}{N} + \beta_{ma}\frac{I_{pm}}{N}$, $A_{66} = \mu_a + \delta$, $A_{77} = \mu_b + \beta_{bp}\frac{I_{pb}}{N} + \beta_{mb}\frac{I_{pm}}{N}$ and $A_{88} = \gamma + \mu_b$. We notice that $A(x)$ is a Metzler Matrix, i.e., all off diagonal terms are non-negative, for $x \in \Omega$. Therefore, since $x(0) \geq 0$, then $x(t) \geq 0$, for all time $t > 0$. In addition since $(N, V_a, V_b) \in \Omega_{N,V_a,V_b}$, we deduce that $x(t) \in \Omega$, for all $t \geq 0$.

□

3.1. About the Disease Free Equilibrium

Without the disease, it is easy to see that $\mathcal{P}_0 = \{\tilde{N}_p, 0, 0, 0, \tilde{V}_a, 0, \tilde{V}_b, 0\}$ is the disease-free equilibrium (DFE) of system (1)-(2)-(3). Just like in models for animal/human diseases, we derive the basic reproduction number, $\mathcal{R}_0$, using the *Next Generation Matrix* (NGM) approach, see [48]. The basic reproduction number is defined as the average number of new cases of an infection caused by one typical infected individual, in a population consisting of susceptible individuals only, [49]. The next generation matrix is a matrix that relates the numbers of newly infected individuals in the various categories in consecutive generations.

To apply the next generation approach, we notice that the variables associated with strain a and strain b are $I_{pa}, I_{pb}, I_{pm}, I_a$ and I_b. Thus, we define $y_I = (I_{pa}, I_{pb}, I_{pm}, I_a, I_b)$, to denote the disease compartments, and $y_S = (H_p, S_a, S_b)$ to denote the susceptible compartments. Rewriting the system as the difference of a new-infection terms (inflow) and outflow terms, we have

$$\frac{dy_I}{dt} = \mathcal{F}(y_S, y_I) - \mathcal{V}(y_S, y_I) \tag{5}$$

$$= \begin{pmatrix} \beta_{pa}\frac{I_a}{V_a}H_p \\ \beta_{pb}\frac{I_b}{V_b}H_p \\ 0 \\ \left(\beta_{ap}\frac{I_{pa}}{N} + \beta_{ma}\frac{I_{pm}}{N}\right)S_a \\ \left(\beta_{bp}\frac{I_{pb}}{N} + \beta_{mb}\frac{I_{pm}}{N}\right)S_b \end{pmatrix} - \begin{pmatrix} \left(\beta_{am}\frac{I_b}{V_b} + (\omega_{pa} + \mu_{pa})\right)I_{pa} \\ \left(\beta_{bm}\frac{I_a}{V_a} + (\omega_b + \mu_{pb})\right)I_{pb} \\ -\beta_{am}\frac{I_b}{V_b}I_{pa} - \beta_{bm}\frac{I_a}{V_a}I_{pb} + \mu_{pm}I_{pm} \\ (\delta + \mu_a)I_a \\ (\gamma + \mu_b)I_b \end{pmatrix}. \tag{6}$$

Then, computing the Jacobians of $\mathcal{F}$ and $\mathcal{V}$ at DFE, i.e., F and V respectively, we derive

$$\text{NGM} = FV^{-1}$$

$$= \begin{pmatrix} 0 & 0 & 0 & \beta_{pa}\frac{\tilde{N}_p}{\tilde{V}_a} & 0 \\ 0 & 0 & 0 & 0 & \beta_{pb}\frac{\tilde{N}_p}{\tilde{V}_b} \\ 0 & 0 & 0 & 0 & 0 \\ \beta_{ap}\frac{\tilde{V}_a}{\tilde{N}_p} & 0 & \beta_{ma}\frac{\tilde{V}_a}{\tilde{N}_p} & 0 & 0 \\ 0 & \beta_{bp}\frac{\tilde{V}_b}{\tilde{N}_p} & \beta_{mb}\frac{\tilde{V}_b}{\tilde{N}_p} & 0 & 0 \end{pmatrix} \begin{pmatrix} \frac{1}{\omega_{pa}+\mu_{pa}} & 0 & 0 & 0 & 0 \\ 0 & \frac{1}{\omega_{pb}+\mu_{pb}} & 0 & 0 & 0 \\ 0 & 0 & \frac{1}{\mu_{pm}} & 0 & 0 \\ 0 & 0 & 0 & \frac{1}{\delta+\mu_a} & 0 \\ 0 & 0 & 0 & 0 & \frac{1}{\gamma+\mu_b} \end{pmatrix}$$

$$= \begin{pmatrix} 0 & 0 & 0 & \beta_{pa}\frac{\tilde{N}_p}{\tilde{V}_a(\delta+\mu_a)} & 0 \\ 0 & 0 & 0 & 0 & \beta_{pb}\frac{\tilde{N}_p}{\tilde{V}_b(\gamma+\mu_b)} \\ 0 & 0 & 0 & 0 & 0 \\ \frac{\beta_{ap}\tilde{V}_a}{\tilde{N}_p(\omega_{pa}+\mu_{pa})} & 0 & \beta_{ma}\frac{\tilde{V}_a}{\tilde{N}_p\mu_{pm}} & 0 & 0 \\ 0 & \frac{\beta_{bp}\tilde{V}_b}{\tilde{N}_p(\omega_{pb}+\mu_{pb})} & \beta_{mb}\frac{\tilde{V}_b}{\tilde{N}_p\mu_{pm}} & 0 & 0 \end{pmatrix}.$$

Then, according to Van den Driessche & Watmough, [48], the basic reproduction number, $\mathcal{R}_0$, is defined as the spectral radius of NGM at DFE. After some computations, we derive that the characteristics polynomial is defined as follows

$$p(\lambda) = -\lambda\left(\lambda^2 - \frac{\beta_{pa}\beta_{ap}}{(\omega_{pa}+\mu_{pa})(\delta+\mu_a)}\right)\left(\lambda^2 - \frac{\beta_{pb}\beta_{bp}}{(\omega_{pb}+\mu_{pb})(\gamma+\mu_b)}\right)$$

such that

$$\mathcal{R}_0 = \max\{\mathcal{R}_{0,a}, \mathcal{R}_{0,b}\},$$

where $\mathcal{R}_{0,a} = \sqrt{\frac{\beta_{pa}\beta_{ap}}{(\omega_{pa}+\mu_{pa})(\delta+\mu_a)}}$ and $\mathcal{R}_{0,b} = \sqrt{\frac{\beta_{pb}\beta_{bp}}{(\omega_{pb}+\mu_{pb})(\gamma+\mu_b)}}$ are the basic reproduction numbers for the MCMV and SCMV diseases, respectively. According to Van den Driessche & Watmough [48], we deduce

Theorem 3.2. *The DFE point, $\mathcal{P}_0$, is locally asymptotically stable when $\mathcal{R}_0 < 1$, and unstable when $\mathcal{R}_0 > 1$.*

Remark 3.1. *The term $\mathcal{R}_{ap} = \frac{\beta_{ap}}{\delta+\mu_a}$ in the square root represents the disease $\mathcal{R}_0$ from MCMV infected thrips to susceptible Maize as the product between the rate of transmission β_{ap} and the thrips life span $\frac{1}{\delta+\mu_a}$. The remaining term in the square root $\mathcal{R}_{pa} = \frac{\beta_{pa}}{\omega_{pa}+\mu_{pa}}$ gives $\mathcal{R}_{0a}$ from Maize to thrips, which is the product between the rate of transmission β_{pa} and the maize viremic period $\frac{1}{\omega_a}$. Likewise, the term $\mathcal{R}_{bp} = \frac{\beta_{bp}}{\gamma+\mu_b}$ in the square root represents the disease $\mathcal{R}_{0b}$ from aphid infected with SCMV to Maize as the product between the rate of transmission β_{bp} and the aphid life span $\frac{1}{\gamma+\mu_b}$. The remaining term in the square root $\mathcal{R}_{pb} = \frac{\beta_{pb}}{\omega_{pb}+\mu_{pb}}$ gives $\mathcal{R}_0$ from Maize to aphid, which is the product between the rate of transmission β_{pb} and the maize viremic period $\frac{1}{\omega_{pb}+\mu_{pb}}$.*

Remark 3.2. *It might be surprising for the reader that parameters related to the MLN disease do not appear in $\mathcal{R}_0$. In fact, this is not, as $\mathcal{R}_0$ is only related to new infectious individuals. To become infected by MLN, a maize plant needs first to be infected by the MCMV or the SCMV virus. To get a better insight into the control and transmission of MLN, in the Section 3.3, page 9 we consider the invasion reproduction numbers.*

3.2. Existence of boundary and endemic equilibria

Other than the DFE, in this subsection we investigate the existence and local stability of other remaining boundary equilibria.

Proposition 3.1. *System (1)-(2)-(3) has the following boundary equilibria.*

1. *When $\mathcal{R}_{0,a} > 1$, an MCMV equilibrium, E^*_{MCMV}, exists where*

$$E^*_{MCMV} = \left(H^*_{pa}, I^*_{pa}, 0, 0, S^*_a, I^*_a, \tilde{V}_b, 0\right)$$

with

$$I^*_{pa} = \frac{\left(\mathcal{R}^2_{0,a}-1\right)}{\frac{\beta_{ap}}{(\delta+\mu_a)} + 1 + \frac{\mu_{pa}}{\mu_{ph}}\left(\mathcal{R}^2_{0,a}-1\right)}\tilde{N}_p,$$

$$I^*_a = \frac{\beta_{ap}\frac{I^*_{pa}}{\tilde{N}_p - \frac{\mu_{pa}-\mu_{ph}}{\mu_{ph}}I^*_{pa}}}{\beta_{ap}\frac{I^*_{pa}}{\tilde{N}_p - \frac{\mu_{pa}-\mu_{ph}}{\mu_{ph}}I^*_{pa}} + \delta + \mu_a}\tilde{V}_a,$$

and $H^*_{pa} + \dfrac{\mu_{pa}}{\mu_{ph}} I^*_{pa} = \tilde{N}_p$ *and* $S^*_a + I^*_a = \tilde{V}_A$.

2. *When* $\mathcal{R}_{0,b} > 1$, *an SCMV equilibrium,* E^*_{SCMV}, *exists where*

$$E^*_{SCMV} = \left(H^*_{pb}, 0, I^*_{pb}, 0, \tilde{V}_a, 0, S^*_b, I^*_b\right)$$

with

$$I^*_{pb} = \frac{\left(\mathcal{R}^2_{0,b} - 1\right)}{\dfrac{\beta_{bp}}{(\gamma+\mu_b)} + 1 + \dfrac{\mu_{pb}}{\mu_{ph}}\left(\mathcal{R}^2_{0,b} - 1\right)} \tilde{N}_p,$$

$$I^*_b = \frac{\beta_{bp} \dfrac{I^*_{pb}}{\tilde{N}_p - \dfrac{\mu_{pb}-\mu_{ph}}{\mu_{ph}} I^*_{pb}}}{\beta_{bp} \dfrac{I^*_{pb}}{\tilde{N}_p - \dfrac{\mu_{pb}-\mu_{ph}}{\mu_{ph}} I^*_{pb}} + \gamma + \mu_b} \tilde{V}_b$$

and $H^*_{pb} + \dfrac{\mu_{pb}}{\mu_{ph}} I^*_{pb} = \tilde{N}_p$ *and* $S^*_b + I^*_b = \tilde{V}_B$

Proof : See Appendix A, page 28.

□

Remark 3.3. *It is impossible to find an analytical expression of the MLN equilibrium. However, we will provide some insight into the existence of the MLN equilibrium. See Section 3.5, page 12.*

Each virus, taken one by one, leads to a standard vector-borne disease model for which standard tools can be used to study their asymptotic behavior. Here, we need to go further and consider the interaction of both viruses and in particular, to find conditions such that both viruses can co-exist and thus induce the MLN disease.

3.3. About the invasion of the SCMV (MCMV) virus

The MLN disease only occurs when MCMV and SCMV viruses are both circulating. In the field, in general, one virus first invades the crops followed by others. In that case, the basic reproduction ratios computed previously are not really useful as they are supposed to indicate if one virus can invade a fully susceptible population. That is why it is necessary to consider specifically the case when SCMV and MCMV viruses co-exist and lead to MLND.

Assume that the system is at the MCMV equilibrium, E^*_{MCMV}. We would like to know if it can be invaded by the SCMV virus. To answer this question, we can estimate the invasion reproduction number, $\mathcal{R}^{SCMV}_{0,inv}$. We consider the subsystem with the variables $y = (I_{pb}, I_{pm}, I_b)$, with H_p, I_{pa}, I_a at equilibrium, such that

$$\begin{aligned}
\frac{dy}{dt} &= \mathcal{F}_{inv}(y, E^*_{\text{MCMV}}) - \mathcal{V}_{inv}(y, E^*_{\text{MCMV}}) \\
&= \begin{pmatrix} \beta_{pb} \dfrac{I_b}{V_b} H^*_{pa} \\ \beta_{bm} \dfrac{I^*_a}{\tilde{V}_a} I_{pb} + \beta_{am} \dfrac{I_b}{\tilde{V}_b} I^*_{pa} \\ \beta_{bp} \dfrac{I_{pb}}{N} S_b + \beta_{mb} \dfrac{I_{pm}}{N} S_b \end{pmatrix} - \begin{pmatrix} \beta_{bm} \dfrac{I^*_a}{\tilde{V}_a} I_{pb} + (\omega_{pb} + \mu_{pb}) I_{pb} \\ \mu_{pm} I_{pm} \\ (\gamma + \mu_b) I_b \end{pmatrix}.
\end{aligned}$$

We compute the Jacobian F and V of $\mathcal{F}_{inv}$ and $\mathcal{V}_{inv}$ respectively, at $(I_{pb}, I_{pm}, I_b) = (0, 0, 0)$. The Invasion reproduction number is defined as $R^{SCMV}_{inv} = \rho(FV^{-1})$. Thus, we compute

$$F = \begin{pmatrix} 0 & 0 & \beta_{pb} \dfrac{H^*_{pa}}{\tilde{V}_b} \\ \beta_{bm} \dfrac{I^*_a}{\tilde{V}_a} & 0 & \beta_{am} \dfrac{I^*_{pa}}{\tilde{V}_b} \\ \beta_{bp} \dfrac{\tilde{V}_b}{\tilde{N}_p} & \beta_{mb} \dfrac{\tilde{V}_b}{\tilde{N}_p} & 0 \end{pmatrix} \quad \text{and} \quad V = \begin{pmatrix} \beta_{bm} \dfrac{I^*_a}{\tilde{V}_a} + \omega_{pb} + \mu_{pb} & 0 & 0 \\ 0 & \mu_{pm} & 0 \\ 0 & 0 & \gamma + \mu_b \end{pmatrix}. \quad (7)$$

from which we deduce

$$INV_{NGM} = FV^{-1} = \begin{pmatrix} 0 & 0 & \beta_{pb}\dfrac{H^*_{pa}}{\tilde{V}_b} \\ \beta_{bm} d \dfrac{I^*_a}{\tilde{V}_a} & 0 & \beta_{am}\dfrac{I^*_{pa}}{\tilde{V}_b} \\ \beta_{bp}\dfrac{\tilde{V}_b}{\tilde{N}_p} & \beta_{mb}\dfrac{\tilde{V}_b}{\tilde{N}_p} & 0 \end{pmatrix} \begin{pmatrix} \dfrac{1}{\beta_{bm}\dfrac{I^*_a}{\tilde{V}_a} + \omega_{pb} + \mu_{pb}} & 0 & 0 \\ 0 & \dfrac{1}{\mu_{pm}} & 0 \\ 0 & 0 & \dfrac{1}{\gamma + \mu_b} \end{pmatrix}.$$

That is,

$$INV^{NGM}_{SCMV} = \begin{pmatrix} 0 & 0 & \beta_{pb}\dfrac{H^*_{pa}}{(\gamma+\mu_b)\,\tilde{V}_b} \\ \dfrac{\beta_{bm} I^*_a}{\left(\beta_{bm}\dfrac{I^*_a}{\tilde{V}_a} + \omega_{pb} + \mu_{pb}\right)\tilde{V}_a} & 0 & \beta_{am}\dfrac{I^*_{pa}}{(\gamma+\mu_b)\,\tilde{V}_b} \\ \dfrac{\beta_{bp}}{\beta_{bm}\dfrac{I^*_a}{\tilde{V}_a} + \omega_{pb} + \mu_{pb}}\dfrac{\tilde{V}_b}{\tilde{N}_p} & \dfrac{\beta_{mb}}{\mu_{pm}}\dfrac{\tilde{V}_b}{\tilde{N}_p} & 0 \end{pmatrix}.$$

Direct and straightforward computations lead to the following characteristic polynomial:

$$\begin{aligned} p_{SCMV}(\lambda) = \; & -\lambda^3 + \lambda\left(\frac{\beta_{am}\beta_{mb}}{\mu_{pm}}\frac{I^*_{pa}}{(\gamma+\mu_b)\,\tilde{N}_p} + \frac{\beta_{bp}}{\beta_{bm}\dfrac{I^*_a}{\tilde{V}_a} + \omega_{pb} + \mu_{pb}}\frac{\beta_{pb}}{(\gamma+\mu_b)}\frac{H^*_{pa}}{\tilde{N}_p}\right) \\ & + \frac{\beta_{pb}}{(\gamma+\mu_b)}\frac{\beta_{bm} I^*_a}{\left(\beta_{bm}\dfrac{I^*_a}{\tilde{V}_a} + \omega_{pb} + \mu_{pb}\right)\tilde{V}_a}\frac{\beta_{mb}}{\mu_{pm}}\frac{H^*_{pa}}{\tilde{N}_p} \end{aligned}$$

or, equivalently,

$$p_{SCMV}(\lambda) = \lambda^3 - (\mathcal{R}_{0,1} + \mathcal{R}_{0,2})\,\lambda - \mathcal{R}_{0,3},$$

with

$$\mathcal{R}_{0,1} := \frac{\beta_{am}\beta_{mb}}{\mu_{pm}\,(\gamma+\mu_b)}\frac{I^*_{pa}}{\tilde{N}_p},$$

$$\mathcal{R}_{0,2} := \frac{\beta_{bp}}{\beta_{bm}\dfrac{I^*_a}{\tilde{V}_a} + \omega_{pb} + \mu_{pb}}\frac{\beta_{pb}}{(\gamma+\mu_b)}\frac{H^*_{pa}}{\tilde{N}_p}$$

and

$$\mathcal{R}_{0,3} := \frac{\beta_{pb}}{(\gamma+\mu_b)}\frac{\beta_{bm} I^*_a}{\left(\omega_{pb} + \mu_{pb} + \beta_{bm}\dfrac{I^*_a}{\tilde{V}_a}\right)\tilde{V}_a}\frac{\beta_{mb}}{\mu_{pm}}\frac{H^*_{pa}}{\tilde{N}_p}.$$

The roots of the reduced third order equation p_{SCMV} can be obtained using Cardano's formula [50]. Let A be a cubic root of

$$\frac{1}{2}\mathcal{R}_{b3} + \sqrt{\frac{1}{4}\mathcal{R}^2_{0,3} - \frac{1}{27}\left(\mathcal{R}_{0,1} + \mathcal{R}_{0,2}\right)^3}$$

and B the unique cubic root of

$$\frac{1}{2}\mathcal{R}_{b3} - \sqrt{\frac{1}{4}\mathcal{R}^2_{0,3} - \frac{1}{27}\left(\mathcal{R}_{0,1} + \mathcal{R}_{0,2}\right)^3}$$

satisfying $M \cdot N = \dfrac{1}{3}\mathcal{R}_{0,3}$. Then, the three roots of p_{SCMV} are given by

$$\lambda_1 = A + B, \quad \lambda_2 = \xi A + \xi^2 B \;\text{ and }\; \lambda_3 = \xi^2 A + \xi B, \tag{8}$$

where $\xi = -\frac{1}{2} + \frac{\sqrt{3}}{2}i$. Hence, the SCMV invasion reproduction number, $\mathcal{R}_{0,inv}^{SCMV}$ is defined as the largest root in modulus, i.e.

$$\mathcal{R}_{0,inv}^{SCMV} = \max\left\{|\lambda_1|, |\lambda_2|, |\lambda_3|\right\}. \tag{9}$$

Remark 3.4. *However, the previous formula, while being the right threshold number, does not necessarily provide a useful analytical expression of the SCMV Invasion Reproduction number. It would be more convenient to find an invasion threshold that would be easier to manipulate. In fact, setting* $\hat{\mathcal{R}}_{0,inv}^{SCMV} = \mathcal{R}_{0,1} + \mathcal{R}_{0,2} + \mathcal{R}_{0,3}$*, that is*

$$\begin{aligned}\hat{\mathcal{R}}_{0,inv}^{SCMV} &= \frac{\beta_{am}\beta_{mb}}{\mu_{pm}(\gamma+\mu_b)}\frac{I_{pa}^*}{\tilde{N}_p} + \frac{\beta_{bp}}{\beta_{bm}\dfrac{I_a^*}{\tilde{V}_a} + \omega_{pb} + \mu_{pb}}\frac{\beta_{pb}}{(\gamma+\mu_b)}\frac{H_{pa}^*}{\tilde{N}_p} + \\ &+ \frac{\beta_{pb}}{(\gamma+\mu_b)}\frac{\beta_{bm}I_a^*}{\left(\omega_{pb}+\mu_{pb}+\beta_{bm}\dfrac{I_a^*}{\tilde{V}_a}\right)\tilde{V}_a}\frac{\beta_{mb}}{\mu_{pm}}\frac{H_{pa}^*}{\tilde{N}_p},\end{aligned} \tag{10}$$

it is interesting to notice that $p_{SCMV}(1) = 1 - \hat{\mathcal{R}}_{0,inv}^{SCMV}$*, such* $p_{SCMV}(\lambda) = 0$ *is equivalent to* $\hat{\mathcal{R}}_{0,inv}^{SCMV} = 1$*. Thust, we have* $\hat{\mathcal{R}}_{0,inv}^{SCMV} = 1$ *if and only if* $\mathcal{R}_{0,inv}^{SCMV} = 1$*. Thus* $\hat{\mathcal{R}}_{0,inv}^{SCMV}$ *is equivalent to* $\mathcal{R}_{0,inv}^{SCMV}$ *to know if SCMV can invade or not. Formula (10) is much simpler to use and provides an analytical formula related to the model's parameters that will be useful later to discuss control methods.*

We need to have in mind that $\mathcal{R}_{0,inv}^{SCMV}$ or $\hat{\mathcal{R}}_{0,inv}^{SCMV}$ represents the number of new cases of I_{pm}, I_{pb} or I_b after the introduction of one infected plant (infected by SCMV or MLN) or one infected vector by the SCMV virus. To this end, we now have to give a "biological interpretation" of $\mathcal{R}_{0,1}$, $\mathcal{R}_{0,2}$, and $\mathcal{R}_{0,3}$.

- The term $\mathcal{R}_{0,3}$ can be decomposed as follows: $\dfrac{\beta_{pb}}{\gamma+\mu_b}\dfrac{H_{pa}^*}{\tilde{N}_p}$ represents the number of secondary SCMV plant infections caused by one infectious aphid when SCMV-healthy plants are at MCMV equilibrium; $\dfrac{\beta_{mb}}{\mu_{pm}}$ represents the number of secondary SCMV aphid infections caused by one MLN-infectious plant; $\dfrac{\beta_{bm}}{\left(\omega_{pb}+\mu_{pb}+\beta_{bm}\dfrac{I_a^*}{\tilde{V}_a}\right)}\dfrac{I_a^*}{\tilde{V}_a}$ represents the number of secondary MLN infections by one SCMV-infected plant when MCMV-infected thrips are at equilibrium. Thus $\mathcal{R}_{0,3}$ is the MLN basic reproduction number from healthy plants to MLN-infected plant through SCMV-infected plants and MCMV-vectors.

- Similarly for $\mathcal{R}_{0,1}$: the term $\dfrac{\beta_{am}}{\mu_{pm}}$ represents the number of secondary MCMV thrip infections caused by one MLN-infectious plant; $\dfrac{\beta_{mb}}{(\gamma+\mu_b)}\dfrac{I_{pa}^*}{\tilde{N}_p}$ represents the number of secondary MLN plant infections caused by one SCMV infectious aphid on MCMV plants. Thus altogether, $\mathcal{R}_{0,1}$ represents the MLN-$\mathcal{R}_0$ from MLN-infected plants by SCMV infectious aphids on MCMV infectious plants.

- $\mathcal{R}_{0,2}$ represents the SCMV basic reproduction number at $MCMV$ equilibrium. When $\beta_{bm} = 0$ (no infection by MLN), we recover $\mathcal{R}_{0,b}^2$.

To summarize: a successful invasion of the SCMV virus occurs when $\mathcal{R}_{0,inv}^{SCMV} > 1$ or $\hat{\mathcal{R}}_{0,inv}^{SCMV} > 1$. In contrary, when $\mathcal{R}_{0,inv}^{SCMV} < 1$ or $\hat{\mathcal{R}}_{0,inv}^{SCMV} < 1$, SCMV cannot invade the system. It is also important to emphasize that even if $\mathcal{R}_{0,a}$ and $\mathcal{R}_{0,b}$ are both greater than 1, this does not necessarily imply the MLN disease can exist. This is directly linked to the fact that $\mathcal{R}_{0,inv}^{SCMV} > 1$ ($\mathcal{R}_{0,inv}^{MCMV} > 1$) or not. This will be illustrated with numerical simulations. The same reasoning holds for an MCMV invasion. We derive

the following Invasion Matrix

$$INV_{MCMV}^{NGM} = \begin{pmatrix} 0 & 0 & \beta_{pa}\dfrac{H_{pb}^*}{(\delta+\mu_a)\,\tilde{V}_a} \\ \dfrac{\beta_{am}I_b^*}{\left(\omega_{pa}+\mu_{pa}+\beta_{am}\dfrac{I_b^*}{\tilde{V}_b}\right)\tilde{V}_b} & 0 & \beta_{bm}\dfrac{I_{pb}^*}{(\delta+\mu_a)\,\tilde{V}_a} \\ \dfrac{\beta_{ap}}{\omega_{pa}+\mu_{pa}+\beta_{am}\dfrac{I_b^*}{\tilde{V}_b}}\dfrac{\tilde{V}_b}{\tilde{N}_p} & \dfrac{\beta_{ma}}{\mu_{pm}}\dfrac{\tilde{V}_a}{\tilde{N}_p} & 0 \end{pmatrix},$$

from which we deduce the following characteristic polynomial

$$p_{MCMV}(\lambda) = -\lambda^3 + \lambda\left(\frac{\beta_{ma}\beta_{bm}}{\mu_{pm}}\frac{I_{pb}^*}{(\delta+\mu_a)\,\tilde{N}_p} + \frac{\beta_{pa}}{(\delta+\mu_a)}\frac{\beta_{ap}}{\omega_{pa}+\mu_{pa}+\beta_{am}\dfrac{I_b^*}{\tilde{V}_b}}\frac{H_{pb}^*}{\tilde{N}_p}\right) + \frac{\beta_{pa}}{(\delta+\mu_a)}\frac{\beta_{am}I_b^*}{\left(\omega_{pa}+\mu_{pa}+\beta_{am}\dfrac{I_b^*}{\tilde{V}_b}\right)\tilde{V}_b}\frac{\beta_{ma}}{\mu_{pm}}\frac{H_{pb}^*}{\tilde{N}_p},$$

and $\mathcal{R}_{0,inv}^{MCMV}$ is the positive root of p_{MCMV}. However, like before, we will consider the following threshold:

$$\begin{aligned}\hat{\mathcal{R}}_{0,inv}^{MCMV} = \; & \frac{\beta_{ma}\beta_{bm}}{\mu_{pm}}\frac{I_{pb}^*}{(\delta+\mu_a)\,\tilde{N}_p} + \frac{\beta_{pa}}{(\delta+\mu_a)}\frac{\beta_{ap}}{\omega_{pa}+\mu_{pa}+\beta_{am}\dfrac{I_b^*}{\tilde{V}_b}}\frac{H_{pb}^*}{\tilde{N}_p} \\ & + \frac{\beta_{pa}}{(\delta+\mu_a)}\frac{\beta_{am}I_b^*}{\left(\omega_{pa}+\mu_{pa}+\beta_{am}\dfrac{I_b^*}{\tilde{V}_b}\right)\tilde{V}_b}\frac{\beta_{ma}}{\mu_{pm}}\frac{H_{pb}^*}{\tilde{N}_p}\end{aligned} \tag{11}$$

Thus, clearly, if one of the previous sub-thresholds is greater than one, then MCMV invasion can occur and thus MLN can appear.

3.4. About the Stability of the boundary equilibria, E_{MCMV}^* and E_{SCMV}^*

After long and tedious computations, it is possible to show that the boundary equilibria, E_{MCMV}^* and E_{SCMV}^* are locally asymptotically stable. This is summarised in the following result.

Proposition 3.2.
- *When $\mathcal{R}_{0,a} > 1$ and $\mathcal{R}_{0,inv}^{SCMV} < 1$ or $\hat{\mathcal{R}}_{0,inv}^{SCMV} < 1$, then E_{MCMV}^* is locally asymptotically stable.*
- *When $\mathcal{R}_{0,b} > 1$ and $\mathcal{R}_{0,inv}^{MCMV} < 1$ or $\hat{\mathcal{R}}_{0,inv}^{MCMV} < 1$, then E_{SCMV}^* is locally asymptotically stable.*

Proof : See Appendix B.

□

Remark 3.5. *Certainly, the boundary equilibria E_{MCMV}^* and E_{SCMV}^* are GAS under the same previous conditions.*

3.5. Uniform persistence of system (1)-(2)-(3)

Thanks to the high non-linearities in the model, a direct study of the existence of an endemic (positive) MLN equilibrium is not really possible. We circumvent this by studying the uniform persistence. We will now study the cases when it occurs.

Consider the subset of Ω

$$\Omega_0 = \{(H_{pa}, I_{pa}, I_{pb}, I_{pm}, S_a, I_a, S_b, I_b) \in \Omega : (I_a > 0 \text{ or } I_{pa} > 0) \text{ and } (I_b > 0 \text{ or } I_{pb} > 0) \text{ or } I_{pm} > 0\},$$

such that

$$\partial\Omega_0 = \Omega\backslash\Omega_0 = \{(H_{pa}, I_{pa}, I_{pb}, I_{pm}, S_a, I_a, S_b, I_b) \in \Omega : (I_{pa} = 0 \text{ and } I_a = 0) \text{ or } (I_{pb} = 0 \text{ and } I_b = 0), I_{pm} = 0\}.$$

Both Ω_0 and $\partial\Omega_0$ are positively invariant, and $\partial\Omega_0$ is relatively closed in Ω. All solutions are bounded and system (1)-(2)-(3) is a point dissipative system.

We denote $\phi_t(x_0)$ the flow corresponding to system (), such that the solution of the system starting at x_0. Let $M_\partial = \{x \in \partial\Omega_0/\phi_t(x) \in \partial\Omega_0 \text{ for } t \geq 0\}$. Then, we have $M_\partial = \partial\Omega_0$. The boundary equilibria DFE, E_a, E_b are in M_∂. Let $W^S(DFE)$, $W^S(E^*_{MCMV})$, and $W^S(E^*_{SCMV})$ be the stable manifold of DFE, E^*_{MCMV} and E^*_{SCMV} respectively.

We have to show that $W^S(DFE) \cap \Omega_0 = \emptyset$, $W^S(E^*_{MCMV}) \cap \Omega_0 = \emptyset$ and $W^S(E^*_{SCMV}) \cap \Omega_0 = \emptyset$ hold when the previous conditions hold.

Let us first show that $W^S(DFE) \cap \Omega_0 = \emptyset$. Since $\mathcal{R}^2_{0,a} > 1$, and $\mathcal{R}^2_{0,b} > 1$, there is an $\epsilon_{a,b} > 0$, such that for all $\epsilon \in [0, \epsilon_{ab}]$, we have

$$\frac{\beta_{ap}\beta_{pa}}{\left(\frac{\beta_{am}}{\tilde{V}_a}\epsilon + \omega_{pa} + \mu_{pa}\right)(\delta + \mu_a)}\left(1 - \frac{K_a\epsilon}{\tilde{V}_a}\right)\left(1 - \frac{K\epsilon}{N_p}\right) > 1,$$

and

$$\frac{\beta_{bp}\beta_{pb}}{\left(\frac{\beta_{bm}}{\tilde{V}_b}\epsilon + \omega_{pb} + \mu_{pb}\right)(\gamma + \mu_b)}\left(1 - \frac{K_b\epsilon}{\tilde{V}_b}\right)\left(1 - \frac{K\epsilon}{N_p}\right) > 1.$$

For all $x_0 \in \Omega_0$, we claim that $\limsup_{t\to+\infty} \|\phi_t(x_0) - DFE\| > \eta_0$. Suppose that this is not true. Thus, there exists $T > 0$ such that for $t > T$, we have

$$H_p \geq N_p - K\epsilon, \quad S_a \geq \tilde{V}_a - K_a\epsilon, \quad S_b \geq \tilde{V}_b - K_b\epsilon, \quad I_a \leq \epsilon, \quad I_b \leq \epsilon$$

which implies

$$\begin{cases} \frac{dI_{pa}}{dt} \geq \beta_{pa}\frac{I_a}{\tilde{V}_a}(N_p - K\epsilon) - \left(\frac{\beta_{am}}{\tilde{V}_a}\epsilon + \omega_{pa} + \mu_{pa}\right)I_{pa} \\ \frac{dI_a}{dt} \geq \beta_{ap}\frac{I_{pa}}{N_p}\left(\tilde{V}_a - K_a\epsilon\right) - (\delta + \mu_a)I_a \end{cases}$$

$$\begin{cases} \frac{dI_{pb}}{dt} \geq \beta_{pb}\frac{I_b}{\tilde{V}_b}(N_p - K\epsilon) - \left(\frac{\beta_{bm}}{\tilde{V}_b}\epsilon + \omega_{pb} + \mu_{pb}\right)I_{pb} \\ \frac{dI_b}{dt} \geq \beta_{bp}\frac{I_{pb}}{N_p}\left(\tilde{V}_b - K_b\epsilon\right) - (\gamma + \mu_b)I_b, \end{cases}$$

leading to the following linear systems

$$\begin{pmatrix} \dot{u}_a \\ \dot{v}_a \end{pmatrix} = \begin{pmatrix} -\left(\frac{\beta_{am}}{\tilde{V}_a}\epsilon + \omega_{pa} + \mu_{pa}\right) & \frac{\beta_{pa}}{\tilde{V}_a}(N_p - K\epsilon) \\ \frac{\beta_{ap}}{N_p}\left(\tilde{V}_a - K_a\epsilon\right) & -(\delta + \mu_a) \end{pmatrix}\begin{pmatrix} u_a \\ v_a \end{pmatrix} = A\begin{pmatrix} u_a \\ v_a \end{pmatrix},$$

and

$$\begin{pmatrix} \dot{u}_b \\ \dot{v}_b \end{pmatrix} = \begin{pmatrix} -\left(\frac{\beta_{bm}}{\tilde{V}_b}\epsilon + \omega_{pb} + \mu_{pb}\right) & \frac{\beta_{pb}}{\tilde{V}_b}(N_p - K\epsilon) \\ \frac{\beta_{bp}}{N_p}\left(\tilde{V}_b - K_b\epsilon\right) & -(\gamma + \mu_b) \end{pmatrix}\begin{pmatrix} u_b \\ v_b \end{pmatrix} = B\begin{pmatrix} u_b \\ v_b \end{pmatrix}.$$

computing $\det(A)$ and $\det(B)$, we derive

$$det\,(A) = \left(\frac{\beta_{am}}{\tilde{V}_a}\epsilon + \omega_{pa} + \mu_{pa}\right)(\delta + \mu_a) - \beta_{ap}\beta_{pa}\left(1 - \frac{K_a\epsilon}{\tilde{V}_a}\right)\left(1 - \frac{K\epsilon}{N_p}\right) < 0,$$

and

$$\det(B) = \left(\frac{\beta_{bm}}{\tilde{V}_b}\epsilon + \omega_{pb} + \mu_{pb}\right)(\gamma + \mu_b) - \beta_{bp}\beta_{pb}\left(1 - \frac{K_b\epsilon}{\tilde{V}_b}\right)\left(1 - \frac{K\epsilon}{N_p}\right) < 0$$

which implies that the characteristic equations of both linear systems have a positive real root, which means the positive solutions of () are unbounded, which implies the unboundedness of I_{pa}, I_{a}., I_{pb}, I_b . A contradiction. Thus $W^S(DFE) \cap \Omega_0 = \emptyset$.

Assume $\mathcal{R}_{0,a} > 1$ and $\mathcal{R}_{0,inv}^{SCMV} > 1$. Now we show that $W^S(E^*_{MCMV}) \cap \Omega_0 = \emptyset$. For all $x_0 \in \Omega_0$, we claim that $\limsup_{t\to+\infty} \|\phi_t(x_0) - E_{MCMV}\| > \epsilon$. Suppose that this is not true. Thus, there exists $T > 0$ such that for $t > T$, we have

$$H^*_{pa} - \epsilon \leq H_p \leq H^*_{pa} + \epsilon, \quad I^*_{pa} - \epsilon \leq I_{pa} \leq I^*_{pa} + \epsilon, \quad I^*_a - \epsilon \leq I_a \leq I^*_a + \epsilon,$$

$$S_b \geq \tilde{V}_b - K_b\epsilon, \quad I_{pb} \leq \epsilon, \quad I_b \leq \epsilon,$$

such that at the limit, we derive

$$\begin{cases} \dfrac{dI_{pb}}{dt} & \geq \beta_{pb}\frac{I_b}{V_b}\left(H^*_{pa} - \epsilon\right) - \beta_{bm}\dfrac{I^*_a + \epsilon}{V_a} I_{pb} - \left(\omega_{pb} + \mu_{pb}\right) I_{pb} \\ \dfrac{dI_{pm}}{dt} & \geq \beta_{bm}\frac{I^*_a - \epsilon}{V_a} I_{pb} + \beta_{am}\dfrac{I_b}{V_b}\left(I^*_{pa} - \epsilon\right) - \mu_{pm} I_{pm}, \\ \dfrac{dI_b}{dt} & \geq \beta_{mb}\frac{I_{pm}}{N_p}\left(\tilde{V}_b - K_b\epsilon\right) + \beta_{bp}\dfrac{I_{pb}}{N_p}\left(\tilde{V}_b - K_b\epsilon\right) - \left(\gamma + \mu_b\right) I_b, \end{cases}$$

from which we derive the following linear system

$$\begin{pmatrix} \dot{u} \\ \dot{v} \\ \dot{w} \end{pmatrix} = C_\epsilon \begin{pmatrix} u \\ v \\ w \end{pmatrix},$$

with

$$C_\epsilon = \begin{pmatrix} -\left(\beta_{bm}\dfrac{I^*_a + \epsilon}{V_a} + \omega_{pb} + \mu_{pb}\right) & 0 & \frac{\beta_{pb}}{V_b}\left(H^*_{pa} - \epsilon\right) \\ \beta_{bm}\frac{I^*_a - \epsilon}{V_a} & -\mu_{pm} & \frac{\beta_{am}}{V_b}\left(I^*_{pa} - \epsilon\right) \\ \frac{\beta_{bp}}{N_p}\left(\tilde{V}_b - K_b\epsilon\right) & \frac{\beta_{mb}}{N_p}\left(\tilde{V}_b - K_b\epsilon\right) & -\left(\gamma + \mu_b\right) \end{pmatrix}$$

$$C_\epsilon = F - V - \epsilon \begin{pmatrix} \frac{\beta_{bm}}{V_a} & 0 & \frac{\beta_{pb}}{V_b} \\ \frac{\beta_{bm}}{V_a} & 0 & \frac{\beta_{am}}{V_b} \\ \frac{K_b\beta_{bp}}{N_p} & \frac{K_b\beta_{bp}}{N_p} & 0 \end{pmatrix} = F - V - \epsilon M,$$

where F and V are defined in 7, page 9. Since $\sigma(F - V) > 0$ iff $\mathcal{R}^{inv}_{0,SCMV} > 1$, there exists $\epsilon_a > 0$, such that $\sigma(C_\epsilon) = \sigma\left(F - V - \epsilon M\right) > 0$ for $\epsilon \in [0, \epsilon_a]$. This implies that C_ϵ has a real positive eigenvalue with a positive eigenvector, meaning that the solutions are unbounded and so are I_{pb}, I_b, and I_{pm}. A contradiction.
Using a similar reasoning, we show that when $\mathcal{R}_{0,b} > 1$ and $\mathcal{R}^{MCMV}_{0,inv} > 1$, then $W^S(E^*_{SCMV}) \cap \Omega_0 = \emptyset$. Altogether, we have $W^S(DFE) = \{DFE\}$, $W^S(E^*_{MCMV}) = \{(H_{pa}, I_{pa}, I_{pb}, I_{pm}, S_a, I_a, S_b, I_b) \in \Omega : I_a > 0,$ $, I_{pa} > 0, I_b = 0, I_{pb} = 0$ and $I_{pm} = 0\}$, $W^S(E^*_{SCMV}) = \{(H_{pa}, I_{pa}, I_{pb}, I_{pm}, S_a, I_a, S_b, I_b) \in \Omega : I_b > 0,$ $I_{pb} > 0, I_a = 0, I_{pa} = 0$ and $I_{pm} = 0\}$ such that $M_\partial = W^S(DFE) \cup W^S(E_1) \cup W^S(E_2)$. In addition each equilibrium is isolated and acyclic in M_∂. We can now apply Theorem 4.6 in [51] and deduce that system (1)-(2)-(3) is uniformly persistent with respect to $(\Omega_0, \partial\Omega_0)$, under suitable conditions on the reproduction numbers. Last, using the invariance of Ω, the dissipativity of system (1)-(2)-(3), and its uniform persistence, Theorem 2.6 in [52] or Theorem D.3 in [53], we can deduce the existence of a MLN equilibrium.

We summarize as follows:

Theorem 3.3. *Under appropriate initial conditions, assuming that one of the following conditions hold true*

- $\mathcal{R}_{0,a} > 1$, $\mathcal{R}_{0,b} > 1$, $\mathcal{R}^{MCMV}_{0,inv} > 1$, *and* $\mathcal{R}^{SCMV}_{0,inv} > 1$,
- $\mathcal{R}_{0,a} > 1$, *and* $\mathcal{R}_{0,b} < 1$, *and* $\mathcal{R}^{SCMV}_{0,inv} > 1$,
- $\mathcal{R}_{0,b} > 1$, *and* $\mathcal{R}_{0,a} < 1$, *and* $\mathcal{R}^{MCMV}_{0,inv} > 1$,

then system (1)-(2)-(3) *is uniformly persistent. Moreover, a MLN equilibrium exists.*

3.6. Global stability of the disease-free equilibrium

First, we consider the following theorem that is helpful to reduce the stability analysis to a smaller system:

Theorem 3.4 ([54], Theorem 3.1). . *Consider the following C^1 system :*

$$\begin{cases} \dot{x} = f(x) \\ \dot{y} = g(x,y) \end{cases} \quad \forall x \in \mathbb{R}^m, \forall y \in \mathbb{R}^n \tag{12}$$

with an equilibrium point, (x^*, y^*), i.e.,$f(x^*) = 0$ and $g(x^*, y^*) = 0$. If x^* is globally asymptotically stable (GAS) in $\mathbb{R}^m$ for the system $\dot{x} = f(x)$, and if y^* is GAS in $\mathbb{R}^n$, for the system $\dot{y} = g(x^*, y)$, then (x^*, y^*) is (locally) asymptotically stable for (12). Moreover, if all the trajectories of (12) are forward bounded, then (x^*, y^*) is GAS for (12).

Using $(4)_2$ and $(4)_3$, applying Theorem 3.4, we see that the stability analysis of system (1)-(2)-(3) is equivalent to the stability analysis of the following system:

$$\begin{cases} \dfrac{dH_p}{dt} = \Lambda_p - \beta_{pa}\dfrac{I_a}{\tilde{V}_a}H_p - \beta_{pb}\dfrac{I_b}{\tilde{V}_b}H_p + \omega_{pa}I_{pa} + \omega_{pb}I_{pb} - \mu_{ph}H_p \\ \dfrac{dI_{pa}}{dt} = \beta_{pa}\dfrac{I_a}{\tilde{V}_a}H_p - \beta_{am}\dfrac{I_b}{\tilde{V}_b}I_{pa} - (\omega_{pa} + \mu_{pa})\, I_{pa} \\ \dfrac{dI_{pb}}{dt} = \beta_{pb}\dfrac{I_b}{\tilde{V}_b}H_p - \beta_{bm}\dfrac{I_a}{\tilde{V}_a}I_{pb} - (\omega_{pb} + \mu_{pb})\, I_{pb} \\ \dfrac{dI_{pm}}{dt} = \beta_{bm}\dfrac{I_a}{\tilde{V}_a}I_{pb} + \beta_{am}\dfrac{I_b}{\tilde{V}_b}I_{pa} - \mu_{pm}I_{pm}, \\ \dfrac{dI_a}{dt} = \left(\beta_{ma}\dfrac{I_{pm}}{\bar{N}} + \beta_{ap}\dfrac{I_{pa}}{\bar{N}}\right)\left(\tilde{V}_a - I_a\right) - (\delta - \mu_a)\, I_a, \\ \dfrac{dI_b}{dt} = \left(\beta_{mb}\dfrac{I_{pm}}{\bar{N}} + \beta_{bp}\dfrac{I_{pb}}{\bar{N}}\right)\left(\tilde{V}_b - I_b\right) - (\gamma - \mu_b)\, I_b. \end{cases} \tag{13}$$

Last, but not least, from $(4)_1$, asymptotically, we have $\bar{N} \geq \dfrac{\Lambda}{\mu_m} = \dfrac{\mu_{ph}}{\mu_m}\tilde{N}_p$, where $\mu_m = \max\{\mu_{ph}, \mu_{pa}, \mu_{pb}, \mu_{pm}\}$. This will be useful in the sequel. Of course, it is clear that all solutions of (13) are forward bounded.

To show GAS, we consider the following Lyapunov functional

$$L(I_{pa}, I_{pb}, I_{pm}, I_a, I_b) = c_{1a}I_{pa} + c_{1b}I_{pb} + KI_{pm} + c_{2a}I_a + c_{3b}I_b,$$

where $L(0,0,0,0,0) = 0$ and $L > 0$ otherwise. We compute

$$\begin{aligned} \frac{dL}{dt} &= c_{1a}\left(\beta_{pa}\frac{I_a}{\tilde{V}_a}H_p - \beta_{am}\frac{I_b}{\tilde{V}_b}I_{pa} - (\omega_{pa} + \mu_{pa})\, I_{pa}\right) + c_{1b}\left(\beta_{pb}\frac{I_b}{\tilde{V}_b}H_p - \beta_{bm}\frac{I_a}{\tilde{V}_a}I_{pb} - (\omega_{pb} + \mu_{pb})\, I_{pb}\right) + \\ &+c_{2a}\left(\beta_{ma}\frac{I_{pm}}{\bar{N}}\left(\tilde{V}_a - I_a\right) + \beta_{ap}\frac{I_{pa}}{\bar{N}}S_a - (\delta + \mu_a)\, I_a\right) + c_{3b}\left(\beta_{mb}\frac{I_{pm}}{\bar{N}}\left(\tilde{V}_b - I_b\right) + \beta_{bp}\frac{I_{pb}}{\bar{N}}S_b - (\gamma + \mu_b)\, I_b\right) + \\ &+K\left(\beta_{bm}\frac{I_a}{V_a}I_{pb} + \beta_{am}\frac{I_b}{V_b}I_{pa} - \mu_{pm}I_{pm}\right) \\ &\leq (K - c_{ia})\,\beta_{am}\frac{I_b}{V_b}I_{pa} + (K - c_{1b})\beta_{bm}\frac{I_a}{\tilde{V}_a}I_{pb} + \left(c_{1a}\beta_{pa}\frac{H_p}{\tilde{V}_a} - (\delta + \mu_a)\right)I_a + \\ &\left(c_{2a}\beta_{ap}\frac{S_a}{N} - c_{1a}\,(\omega_{pa} + \mu_{pa})\right)I_{pa} + \left(c_{2b}\beta_{bp}\frac{S_b}{N} - c_{1b}\,(\omega_{pb} + \mu_{pb})\right)I_{pb} + \\ &+\left(c_{2a}\beta_{ma}\frac{\tilde{V}_a}{\bar{N}} + c_{3b}\beta_{mb}\frac{\tilde{V}_b}{\bar{N}} - K\mu_{pm}\right)I_m \\ &\leq (K - c_{ia})\,\beta_{am}\frac{I_b}{\tilde{V}_b}I_{pa} + (K - c_{1b})\beta_{bm}\frac{I_a}{\tilde{V}_a}I_{pb} + \left(c_{1a}\beta_{pa}\frac{N_h}{\tilde{V}_a} - c_{2a}\,(\delta + \mu_a)\right)I_a + \end{aligned}$$

$$+\left(c_{1b}\beta_{pb}\frac{N_h}{\tilde{V}_b}-c_{3b}\left(\gamma+\mu_b\right)\right)I_b$$

$$\left(c_{2a}\beta_{ap}\frac{\tilde{V}_a}{\tilde{N}}-c_{1a}\left(\omega_{pa}+\mu_{pa}\right)\right)I_{pa}+\left(c_{3b}\beta_{bp}\frac{\tilde{V}_b}{\tilde{N}}-c_{1b}\left(\omega_{pb}+\mu_{pb}\right)\right)I_{pb}+$$

$$+\left(c_{2a}\beta_{ma}\frac{\tilde{V}_a}{\tilde{N}}+c_{3b}\beta_{mb}\frac{\tilde{V}_b}{\tilde{N}}-K\mu_{pm}\right)I_{pm}.$$

choosing $K=c_{1a}=c_{1b}=\dfrac{1}{\tilde{N}_p}$ leads to

$$\frac{dL}{dt}\leq\left(\beta_{pa}\frac{1}{\tilde{V}_a}-c_{2a}\left(\delta+\mu_a\right)\right)I_a+\left(c_{1b}\beta_{pb}\frac{1}{\tilde{V}_b}-c_{3b}\left(\gamma+\mu_b\right)\right)I_b+$$

$$\left(c_{2a}\beta_{ap}\frac{\tilde{V}_a}{\tilde{N}}-\frac{1}{N_h}\left(\omega_{pa}+\mu_{pa}\right)\right)I_{pa}+\left(c_{3b}\beta_{bp}\frac{\tilde{V}_b}{\tilde{N}}-\frac{1}{N_h}\left(\omega_{pb}+\mu_{pb}\right)\right)I_{pb}+$$

$$+\left(c_{2a}\beta_{ma}\frac{\tilde{V}_a}{\tilde{N}}+c_{3b}\beta_{mb}\frac{\tilde{V}_b}{\tilde{N}}-\frac{1}{N_h}\mu_{pm}\right)I_{pm}.$$

Then $c_{2a}=\dfrac{\beta_{pa}}{\left(\delta+\mu_a\right)\tilde{V}_a}$ and $c_{3b}=\dfrac{\beta_{pb}}{\tilde{V}_b\left(\gamma+\mu_b\right)}$ lead to

$$\frac{dL}{dt}\leq\frac{1}{\tilde{N}_p}\left(\omega_{pa}+\mu_{pa}\right)\left(\frac{\tilde{N}_p}{\tilde{N}}\mathcal{R}_{0,a}^2-1\right)I_{pa}+\frac{1}{\tilde{N}_p}\left(\omega_{pb}+\mu_{pb}\right)\left(\frac{\tilde{N}_p}{\tilde{N}}\mathcal{R}_{0,b}^2-1\right)I_{pb}$$

$$+$$

$$+\frac{1}{\tilde{N}_p}\mu_{pm}\left(\left(\frac{\beta_{ma}\beta_{pa}}{\mu_{pm}\left(\delta+\mu_a\right)\tilde{V}_a}+\frac{\beta_{pb}\beta_{mb}}{\mu_{pm}\left(\gamma+\mu_b\right)}\right)\frac{\mu_m}{\mu_{ph}}-1\right)I_{pm},$$

or equivalently

$$\frac{dL}{dt}\leq\frac{1}{\tilde{N}_p}\left(\omega_{pa}+\mu_{pa}\right)\left(\frac{\mu_m}{\mu_{ph}}\mathcal{R}_{0,a}^2-1\right)I_{pa}+\frac{1}{\tilde{N}_p}\left(\omega_{pb}+\mu_{pb}\right)\left(\frac{\mu_m}{\mu_{ph}}\mathcal{R}_{0,b}^2-1\right)I_{pb}$$

$$+$$

$$+\frac{1}{\tilde{N}_p}\mu_{pm}\left(\left(\frac{\beta_{ma}\beta_{pa}}{\mu_{pm}\left(\delta+\mu_a\right)\tilde{V}_a}+\frac{\beta_{pb}\beta_{mb}}{\mu_{pm}\left(\gamma+\mu_b\right)}\right)\frac{\mu_m}{\mu_{ph}}-1\right)I_{pm}.$$

Thus assuming

$$\frac{\mu_m}{\mu_{ph}}\mathcal{R}_{0,a}^2<1,\qquad\frac{\mu_m}{\mu_{ph}}\mathcal{R}_{0,b}^2<1,$$

and

$$\left(\frac{\beta_{ma}\beta_{pa}}{\mu_{pm}\left(\delta+\mu_a\right)}+\frac{\beta_{pb}\beta_{mb}}{\mu_{pm}\left(\gamma+\mu_b\right)}\right)\frac{\mu_m}{\mu_{ph}}<1$$

leads to $\dfrac{dL}{dt}\leq 0.$, for all $(I_{ap},I_{bp},I_{pm},I_a,I_b)\in\times[0,\tilde{N}_p]^3\times[0,\tilde{V}_a]\times[0,\tilde{V}_b]$. The largest invariant set Ω_s is $\left\{(I_{pa},I_{pb},I_{pm},I_a,I_b)/\dfrac{dL}{dt}=0\right\}$ is reduced to $(0,0,0,0,0)$. Thus according to Krasovskii-LaSalle Theorem and Theorem 3.4, the DFE is GAS.

Of course, when no additional mortality exists, i.e. $\mu_m=\mu_{ph}$, then the previous conditions relax and we recover $\mathcal{R}_0^2<1$ with

$$\mathcal{R}_{0,m}^2=\frac{\beta_{ma}\beta_{pa}}{\mu_{pm}\left(\delta+\mu_a\right)}+\frac{\beta_{pb}\beta_{mb}}{\mu_{pm}\left(\gamma+\mu_b\right)}<1. \tag{14}$$

Thus, we can conclude

Theorem 3.5. *When* $\frac{\mu_m}{\mu_{ph}}\mathcal{R}_0^2 < 1$ *and* $\frac{\mu_m}{\mu_{ph}}\mathcal{R}_{0,m}^2 < 1$ *then DFE is globally asymptotically stable in* Ω.

Remark 3.6. *Condition* (14) *is not a surprise since MLN-infectious plants can infect healthy plants through infectious thrips and aphids, such that we have additional basic reproduction numbers related to MLN-infectious plants. Indeed,* $\frac{\beta_{ma}}{\mu_{pm}}$ *represents the number of secondary MCMV thrip infections caused by one infectious MLN crop host;* $\frac{\beta_{pa}}{\delta+\mu_a}$ *represents the number of secondary MCMV crop infectious cause by one infectious thrip. Thus, altogether,* $\frac{\beta_{ma}}{\mu_{pm}}\frac{\beta_{pa}}{\delta+\mu_a}$ *represents the MCMV-*$\mathcal{R}_0$ *from an MLN infectious plant to a Healthy plant. Similarly* $\frac{\beta_{mb}}{\mu_{pm}}\frac{\beta_{pb}}{\gamma+\mu_b}$ *represents the SCMV-*$\mathcal{R}_0$ *from a MLN infectious plant to an Healthy plant.*

Remark 3.7. *In terms of control strategy, Theorem 3.5 shows that, at anytime, whatever the epidemiological state, it is possible to reduce the epidemiological risk of MCMV and SCMV diseases (and thus MLN disease) if, by suitable control strategies, we are able to reduce* $\frac{\mu_m}{\mu_{ph}}\mathcal{R}_0^2$ *and* $\frac{\mu_m}{\mu_{ph}}\mathcal{R}_{0,m}^2$ *below* 1. *However, as showed earlier other control strategies can be used to reduce the risk of MLN disease by decaying one (or both) invasion reproduction number(s) below* 1.

Thanks to all previous results, we now summarize all possible dynamics in Table 2, page 17.

Table 2: Dynamics of the equilibrium

Case	$\mathcal{R}_0$	$\mathcal{R}_{0,inv}^{SCMV}$ / $\mathcal{R}_{0,inv}^{MCMV}$	Long term Dynamics
1	$\frac{\mu_m}{\mu_{ph}}\mathcal{R}_0^2 < 1$, $\frac{\mu_m}{\mu_{ph}}\mathcal{R}_{0,m}^2 < 1$	–	E^*_{DFE} is a global attractor
2	$\mathcal{R}_{0,a} > 1$, $\mathcal{R}_{0,b} < 1$	$\mathcal{R}_{0,inv}^{SCMV} < 1$	E^*_{MCMV} is a global attractor
3	$\mathcal{R}_{0,a} < 1$, $\mathcal{R}_{0,b} > 1$	$\mathcal{R}_{0,inv}^{MCMV} < 1$	E^*_{SCMV} is a global attractor
4	$\mathcal{R}_{0,a} > 1$, $\mathcal{R}_{0,b} > 1$	$\mathcal{R}_{0,inv}^{MCMV} < 1$, $\mathcal{R}_{0,inv}^{SCMV} > 1$	E^*_{SCMV} is a global attractor
5	$\mathcal{R}_{0,a} > 1$, $\mathcal{R}_{0,b} > 1$	$\mathcal{R}_{0,inv}^{MCMV} > 1$, $\mathcal{R}_{0,inv}^{SCMV} < 1$	E^*_{MCMV} is a global attractor
6	$\mathcal{R}_{0,b} > 1$, $\mathcal{R}_{0,a} > 1$	$\mathcal{R}_{0,inv}^{MCMV} < 1$, $\mathcal{R}_{0,inv}^{SCMV} < 1$	either E^*_{MCMV} or E^*_{SCMV}
7	$\mathcal{R}_{0,a} > 1$, $\mathcal{R}_{0,b} < 1$	$\mathcal{R}_{0,inv}^{MCMV} < 1$ $\mathcal{R}_{0,inv}^{SCMV} > 1$	E^*_{MLN} is a global attractor
8	$\mathcal{R}_{0,a} < 1$, $\mathcal{R}_{0,b} > 1$	$\mathcal{R}_{0,inv}^{MCMV} > 1$ $\mathcal{R}_{0,inv}^{SCMV} < 1$	E^*_{MLN} is a global attractor
9	$\mathcal{R}_{0,a} > 1$, $\mathcal{R}_{0,b} > 1$	$\mathcal{R}_{0,inv}^{MCMV} > 1$, $\mathcal{R}_{0,inv}^{SCMV} > 1$	E^*_{MLN} is a global attractor

More than the basic reproduction numbers, the previous table shows the importance of the invasion reproduction numbers, in particular for MLN control. Indeed, when $\mathcal{R}_0 > 1$, we have to deal with different subcases related to the invasion reproduction numbers: from case 4 to case 9. Indeed four long term dynamics can occur: either the system converges to one of the boundary equilibria, E^*_{MCMV} or E^*_{SCMV}, or to the full equilibrium, E^*_{MLN}. However, when both invasion reproduction numbers are less than 1, then the system converges either to E^*_{MCMV} or to E^*_{SCMV}, depending on the initial conditions. We will illustrate some of these cases in the section devoted to numerical simulations. However, we first derive a global sensitivity analysis in order to discuss the usefulness of some feasible control strategies.

4. Parameter estimate and sensitivity analysis

Before starting the global sensitivity analysis, we detail the parameters values given in Table 1, page 6. Most often, parameters estimation is always a critical issue: despite a deep review of the literature, some important parameters were difficult to find or to estimate.

4.1. SCMV-Aphids

For the estimates for parameters of aphids we mainly focus on *Rhopalosiphum maids* as the main vectors for SCMV. The authors in [40], studied the life history of *Rhopalosiphum Maids* under different temperature condition on corn leaves, *Zea mays;* they obtained longevity of adults *Rhopalosiphum Maids* under 25^oc of 12.0 ± 0.9days. In [41], *Rhopalosiphum Maids* were reared on six grown maize hybrids ($K3640/3 \times MO17$, Simon, SC704, EXP1, VRE26×K18 and VRE27×K18) carried under 25 ± 1^oC condition; they estimated a longevity of adult *Rhopalosiphum maids* of 11.85 ± 2.35 days depending on the maize hybrids. Therefore, we consider the maximum interval $[9.5, 14.2]$, meaning that the interval for the death rate is $[0.07, 0.1]$ under 24 ± 1^oC (mean temperature of Kenya from 1901 to 2016 [43]). The highest titre from *Rhopalosiphum maids* in maize at 38%; was reported by [39]. Here we assume the range from 14% to 38%. It is known that aphids have alternative host [9], such that, they may feed on alternative plants along its life. It is also well know that they can bite several times on the same plant. Therefore, we assume from 1 to 4 visits along its life, and also between 1 and 3 bites per visit, so that we obtain the range values of $\left[\frac{1}{14.2} \times 0.14, \frac{6 \times 3}{9.5} \times 0.38\right] = [0.01, 0.72]$ for β_{pb}. We also assume that the presence of MCMV in maize does not interfere in the transmission of SCMV, therefore, we take the same range of values from infectious aphids to plant already infected with MCMV. The authors in [25], reported the mean infectious period of 6 hours in nonpersistently transmitted mode. Here, we assume an infectious period with range, $[2, 8]$ hours such that we obtain the recovery rate in the range $[2,]$. The recommended spacing and planting maize population vary depending on the weather conditions and the moisture status of the soil, for instance, in the highland and medium areas where the soils are well-drained sandy-loam soils, the densities of maize per hectare recommended vary between 37.850 to 53.333 which corresponds to spacing of 90×30cm and 75×25cm, respectively; while in dry and coastal low land areas where the soil is dry and/or sandy the density of maize plant recommended per hectare is 44.444, [44]. We assume the range of the density of maize per hectare of $[37.850, 53.333]$.

4.2. MCMV-Thrips

Althought the thrips *Frankliniella Occidentalis* is not implicated in the MCMV transmission in Kenya, [9], it has been widely studied in the literature. Thus, like many other works, we assume that most of the parameters fit for both species. The authors in [37] investigated the longevity of *F. Occidentalis* on *Cabbage, Cucumber, Bean* and *Tomato*. They estimated the range of female longevity at 13.32 ± 5.02 days and the range of male longevity at 7.32 ± 1.05 days. Considering that we have male-female sex-ratio of 0.4 (see for instance [37]), we derive a mean life span of the population to be 10.72 ± 3.43 days. Thus, we estimate the death rate of $[0.07, 0.14]$. In [19] the authors reported transmission of MCMV by the thrips, *Frankliniella Williams*, ranging from 30% to 45% with a mean of 37%. Similar to aphids, we assume from 1 to 4 visits along thrips life, so that we obtain the range values of $\left[\frac{1}{14.15} \times 0.3, \frac{4}{7.29} \times 0.45\right] = [0.02, 0.25]$ for β_{pa}. We also assume that the presence of SCMV in maize does not interfere in the transmission of MCMV, therefore, we also take the same range values of $[0.04, 0.12]$ from infectious thrips to plant already infected with SCMV. In [19], it was reported that the thrips, *Frankliniella williams*, loose the ability to transmit the virus 6 days post acquisition. We assume that the necessary period to recover from disease range from 6 to 8 days. Thus, we estimate that the recovery rate, δ, lies in the range $[0.13, 0.17]$.

4.3. MLN-parameters

Unfortunately, parameters values related to MLND are not really available in the literature. Since, it is well known that plant viruses are able to manipulate their hosts or their vectors in order to enhance the transmission of viruses, it seems obvious to consider that the transmission rates are larger than the values estimated for MCMV- or SCMV-transmission rates. Thus for β_{m*} and β_{*m}, where $*$ stands for a or b, we will consider a range of values of $[0.08, 0.9]$. Within this range, we will be able to illustrate all cases given in Table 2, page 17.

4.4. Global Sensitivity Analysis

It is also important to check the impact of parameters changes on the dynamics of the system, and, in particular, what are the most sensitive parameters, for which the system may drastically change its behavior. That is why we derive a LHS-PRCC sensitivity analysis, where LHS stands for Latin Hypercube Sampling and PRCC for Partial rank correlation coefficient. The LHS-PRCC method provides mainly information about how the outputs are impacted if we increase (or decrease) the inputs of a specific parameter. The analysis is done on the time interval $[100, 500]$. The results are ordered from the most negative to the most positive ones.

In Figs. 2 and 3, we show the results for the crop's variables. No surprises, for the healthy plant, the dominant parameters are Λ_p and μ_{ph}. For crops infected by the viruses, the most sensitive parameters are those related either to the vectors or to the host transmission, and the mortality rates. The MLN variable, I_{pm} is sensitive to γ that is the parameter related to the non permanent transmission process. In Fig 4, γ and δ, the vectors recovery rates, are sensitive parameters: this makes sense since they are related to the duration vectors are infectious or not. The recovery rates ω_{pa} and ω_{pb} are sensitive parameters too. Only the death rate μ_a seems to play a role in the dynamics as well as the transmission parameters. Clearly, all of these parameters need to be estimated efficiently in order to capture the right dynamics of the system.

Next, we derive the LHS-PRCC sensitivity analysis for both invasion reproduction numbers: see Fig. 5, page 23. It is interesting to notice that both numbers are not sensitive to the same parameters, except μ_{pb}, γ, and μ_{ph} but with different impact. It seems also that $\hat{\mathcal{R}}_{0,inv}^{SCMV}$ is sensitive to most of the contact rates. Last $\hat{\mathcal{R}}_{0,inv}^{SCMV}$ is sensitive to infectious plants death rates, such that increasing these death rates using roguing seems to be a good control strategy to lower this invasion reproduction number. However, and surprisingly, increasing μ_{pb} can also increase the $\hat{\mathcal{R}}_{0,inv}^{MCMV}$ and, thus, eventually, favor the invasion of MCMV disease.

In terms of control, based on Table 2, page 17, and the previous sensitivity analysis, several control strategies seem feasible. Indeed, MLN control is possible through a combination of cultural practices, pesticides, and host tolerance. However, there is a gap in terms of pest management between export-oriented farmers and smallholders. The first ones can develop all these practices, while the second cannot and thus rely on conventional control methods. Theoretically, combination of control methods from vector control to host control are feasible: see for instance Fig. 3 in [7], showing the time evolution of MLN incidence when roguing and a combination of pesticides are used. This example occured in a experimental station. Practically, this is another story. In our model, some parameters, like the recovering parameters δ and γ cannot be modified. Like for vector-borne diseases, vector-control could be interesting and they are vector control methods in the field. However, standard vector control against aphids, i.e., increase μ_a with insecticide, is not efficient because they are infectious during a very short time. A possible (biological) control would be to use this non-persistent transmission property to attract the infected aphids to non-susceptible hosts. This require to mix maize crop with other non-susceptible crops. This would be possible for smallholders, but only if these other crops are also cash-crops. For thrips, it seems that insecticide is still the main control tool: see the recent study [55], which, according to our sensitivity analysis (see Fig 2b, page 20), seems meaningful. However, our global sensitivity analysis shows that the main effective controls should be on infected maize, either by reducing the contact rates and/or by removing the infected plants. Practically, reducing the contact rates is not so easy: using nets, like it is done in East-Africa to protect vegetable crops, seems feasible but not for maize. To the contrary, roguing, which consists of removing infected plants and thus in increasing the infected plants mortality rates, μ_{pa}, μ_{pb}, and μ_{pm}, seems to be the most effective control, impacting all infected compartments and also the invasion reproduction numbers. Note also that roguing is a standard control practice against diseases affecting crops in Africa, like cassava [1]. It is particularly useful for smallholders that cannot consider other control strategies against MLND, like crop rotation.

5. Numerical Simulation

In this section, we start providing some numerical simulations to illustrate the previous theoretical results and also discuss the results. We use the initial values for the parameters as listed in Table 3 and modify them to derive the different cases highlighted above in Table 2, page 17. For each case, we

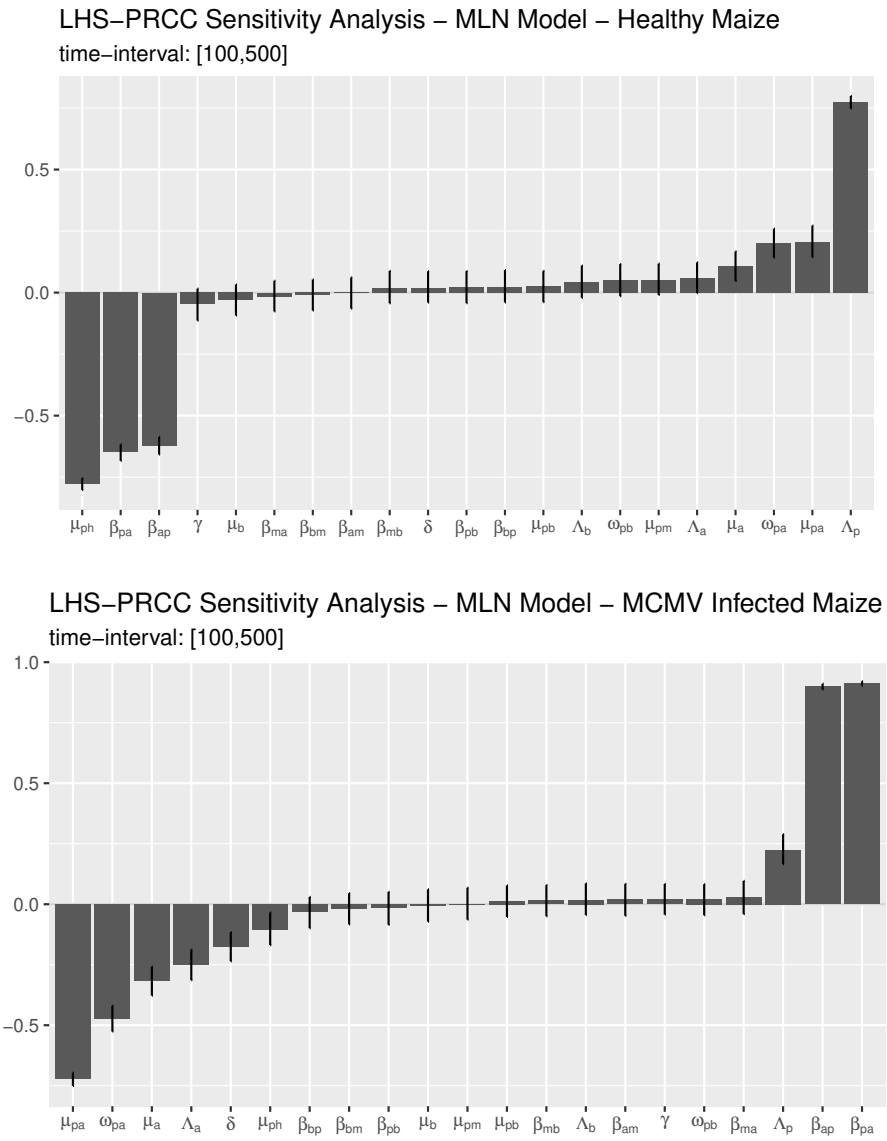

Figure 2: LHS-PRCC Sensitivity analysis - Maize crop

estimate the basic reproduction numbers and also the invasion reproduction numbers, $\mathcal{R}^*_{0,inv}$ and $\hat{\mathcal{R}}^*_{0,inv}$ (see formulae (10), page 11, and (11), page 12).

Case 2 Assuming $\gamma = 4.0$, then $\mathcal{R}^2_{0,a} > 1$, $\mathcal{R}^2_{0,b} < 1$, and $\hat{\mathcal{R}}^{SCMV}_{0,inv} < 1$ or $\mathcal{R}^{SCMV}_{0,inv} < 1$. Thus, the system converges to E^*_{SCMV}. Here, this case shows that with vector control on thrips it might be possible to avoid MLN disease using only the basic reproduction numbers. In fact this is due to the fact that MCMV is transmitted in a semi-permanent way by thrips. Since SCMV is transmitted in a non-persistent manner, it might be more difficult to control SCMV by aphids control, using for instance insecticide. See Fig. 6, page 24.

Case 5 Considering the previous table, we derive the following values for the Reproduction numbers

In this case, we have $\mathcal{R}^2_{0,a} > 1$ and $\mathcal{R}^2_{0,b} > 1$, but $\mathcal{R}^{SCMV}_{0,inv} < 1$. Thus the system converges to E^*_{MCMV}. This case illustrates that controlling aphids might be useful. Indeed, we don't need to lower $\mathcal{R}^2_{0,b}$ below 1. What is important is to lower $\mathcal{R}^{SCMV}_{0,inv} < 1$. See Fig 7, page 25: we have chosen random initial points to show that whatever the initial conditions, the system converges to

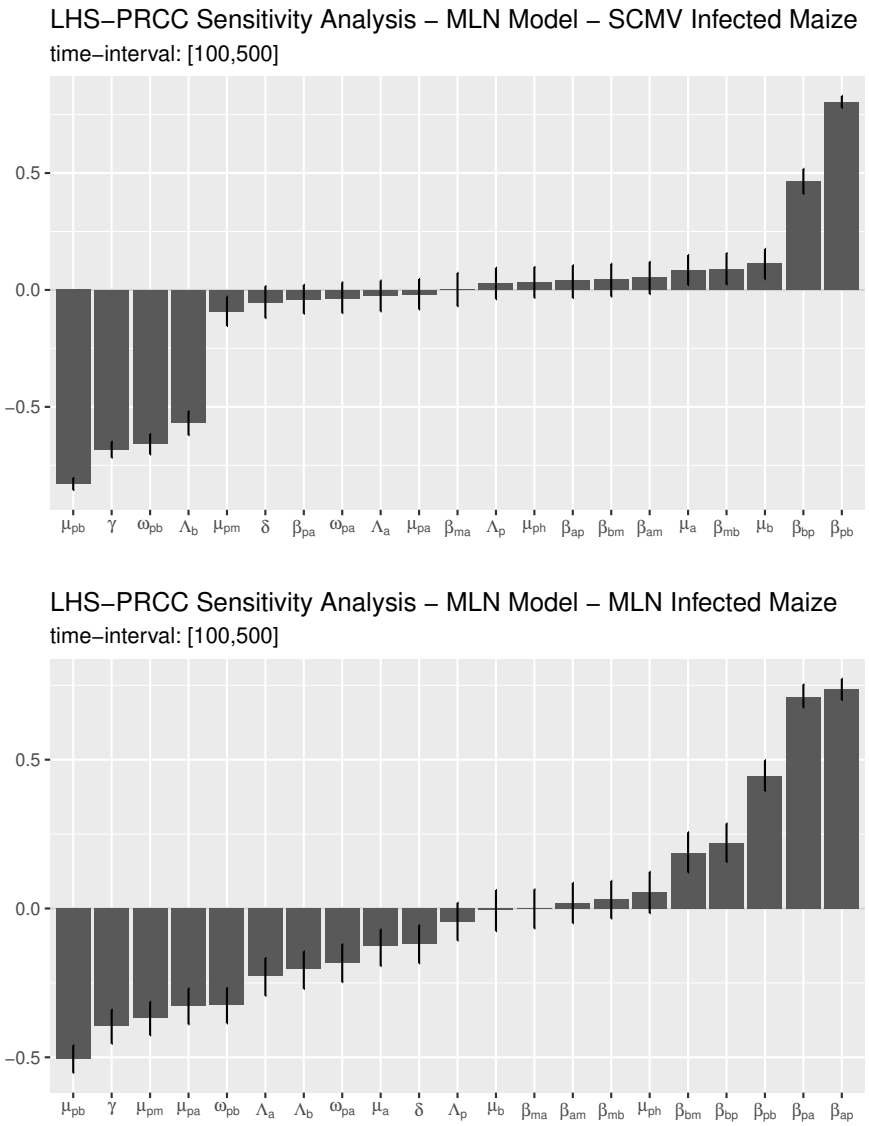

Figure 3: LHS-PRCC Sensitivity analysis - Maize crop

E^*_{MCMV}.

Case 6 We now consider the case: $\mathcal{R}^2_{0,a} > 1$ and $\mathcal{R}^2_{0,b} > 1$, with $\mathcal{R}^{SCMV}_{0,inv} < 1$ and $\mathcal{R}^{MCMV}_{0,inv} < 1$. In that case, the system converges to either E^*_{MCMV} or E^*_{SCMV} depending on the initial conditions. We choose $\mu_a = 0.125$, $\beta_{ma} = \beta_{mb} = 0.02$ in Table 3. See Fig. 8, page 25. This case is very interesting as it shows that even if both basic reproduction numbers are greater than one, if, through appropriate control strategies, we are able to set the invasion reproduction numbers less than one, then MLN Disease cannot occur.

Case 7 We consider $\gamma = 4$, $\beta_{mb} = 0.6$, $\beta_{am} = 0.5$, $\beta_{ma} = 0.5$ in Table 3. The values obtained for the reproduction numbers are summarized in Table 7, page 26. The system reaches the MLN equilibrium $E^*_{\text{MLN}} = \left(\frac{H^*_p}{N_p}, \frac{I^*_{pa}}{N_p}, \frac{I^*_{pb}}{N_p}, \frac{I^*_{pm}}{N_p}, \frac{S^*_a}{V_a}, \frac{I^*_a}{V_a}, \frac{S^*_b}{V_b}, \frac{I^*_b}{V_b}\right) = (0.3178, 0.2355, 0.0715, 0.1876, 0.6392, 0.3608, 0.9622, 0.0378)$: see Fig. 9a, page 26.

Case 8 Compared to case 7, we consider $\gamma = 3$, $\mu_a = 0.14$ and $\delta = 0.2$ in Table 3. The values obtained for the reproduction numbers are summarized in Table 8, page 26. The system reaches the MLN equi-

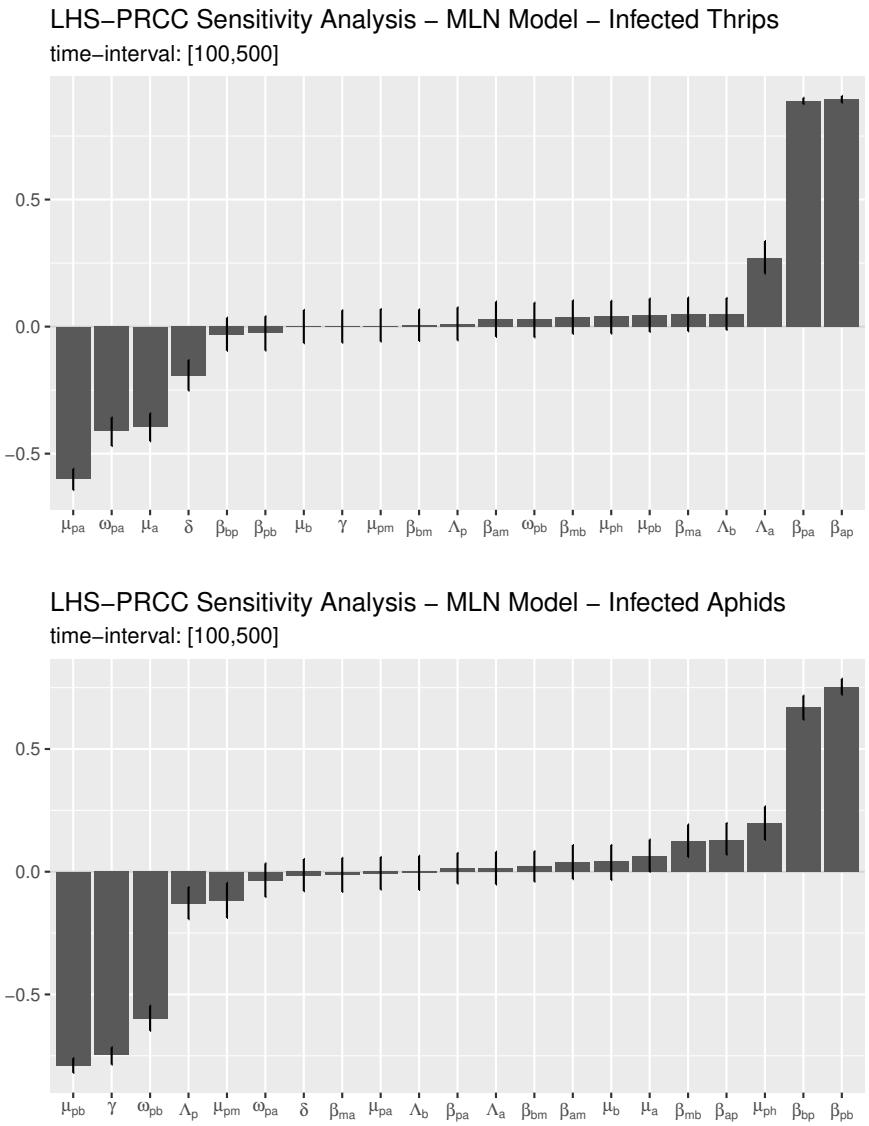

Figure 4: LHS-PRCC Sensitivity analysis - Vectors

librium $E^*_{\text{MLN}} = \left(\frac{H^*_p}{\tilde{N}_p}, \frac{I^*_{pa}}{\tilde{N}_p}, \frac{I^*_{pb}}{\tilde{N}_p}, \frac{I^*_{pm}}{\tilde{N}_p}, \frac{S^*_a}{\tilde{V}_a}, \frac{I^*_a}{\tilde{V}_a}, \frac{S^*_b}{\tilde{V}_b}, \frac{I^*_b}{\tilde{V}_b}\right) = (0.3136, 0.1485, 0.1226, 0.2077, 0.7014, 0.2986, 0.9413, 0.0587)$: see Fig. 9b, page 26.

Case 9 Compared to case 7, we just change the parameter γ, i.e. $\gamma = 3.0$. The values obtained for the reproduction numbers are summarized in Table 9, page 26. The system reaches the MLN equilibrium $E^*_{\text{MLN}} = \left(\frac{H^*_p}{\tilde{N}_p}, \frac{I^*_{pa}}{\tilde{N}_p}, \frac{I^*_{pb}}{\tilde{N}_p}, \frac{I^*_{pm}}{\tilde{N}_p}, \frac{S^*_a}{\tilde{V}_a}, \frac{I^*_a}{\tilde{V}_a}, \frac{S^*_b}{\tilde{V}_b}, \frac{I^*_b}{\tilde{V}_b}\right) = (0.2581, 0.1571, 0.0950, 0.2449, 0.5771, 0.4229, 0.9319, 0.0681)$: : see Fig. 10b, page 27.

Case 10 A last case, where we consider roguing which consists of removing infected plants. Here, since the symptoms of MCMV, SCMV or MLN are easy to check it is straightforward to select the infected plants. In Fig. 11, page 27, we show that depending on the roguing rate, the system can recover, i.e., converges to DFE. In particular, it is obvious to emphasize that the roguing effort is much more important when all Reproduction numbers are greater than 1 (case 9). Thus, clearly, when the risk of MLN in the area is high, it is better to react immediately once the symptoms of MCMV or SCMV are found.

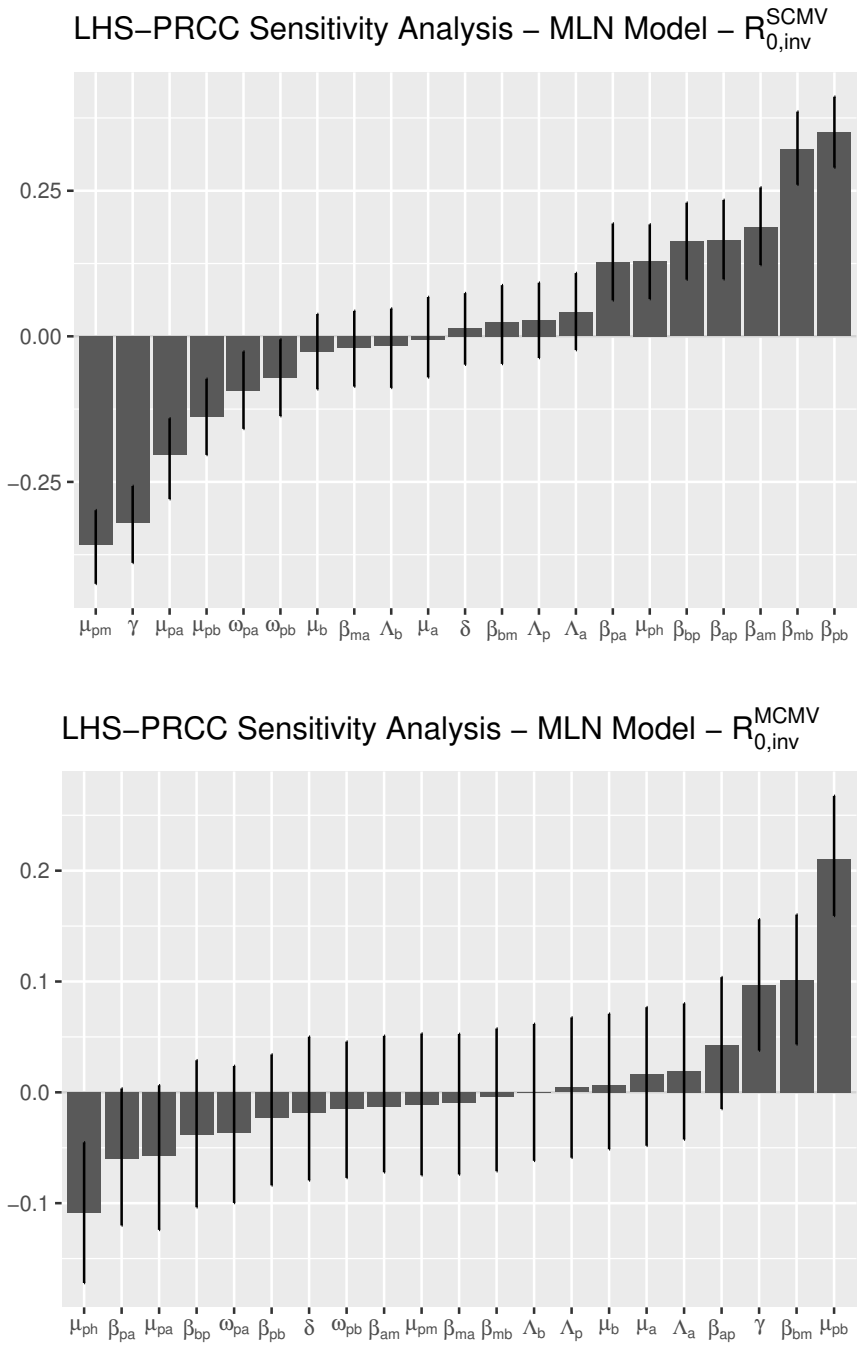

Figure 5: LHS-PRCC Sensitivity analysis - Invasive Reproduction Numbers

The previous simulations highlight the different dynamics of the MLND system. Clearly, more than the standard basic reproduction numbers $\mathcal{R}_{0,a}$ and $\mathcal{R}_{0,b}$, the invasion reproduction numbers $\mathcal{R}_{0,inv}^{MCMV}$ and $\mathcal{R}_{0,inv}^{SCMV}$ are important to estimate as they will drive the emergence of the MLN disease or not, even when one basic reproduction number is less than one. However, even when both numbers are less than 1, case 6 shows that E^*_{MCMV} and E^*_{SCMV} can be simultaneously locally asymptotically stable, such that the convergence of the system to one of the boundary equilibria may depend on the initial conditions.

In the field, MLN disease is difficult to control, especially because the SCMV virus is transmitted in

Table 3: Base parameters and numerical values

β_{ap}	β_{pa}	β_{am}	β_{ma}	δ	ω_{pa}	μ_a	μ_{ph}	μ_{pa}	μ_{pb}	μ_{pm}
0.073	0.073	0.075	0.1	0.15	0	0.092	1/60	1/60	1/60	1/30

Λ_p	Λ_a	Λ_b	β_{bp}	β_{pb}	β_{bm}	β_{mb}	γ	μ_b	ω_{pb}
$40000\mu_{ph}$	50535	25638	0.25	0.25	0.07	0.1	3	0.079	0

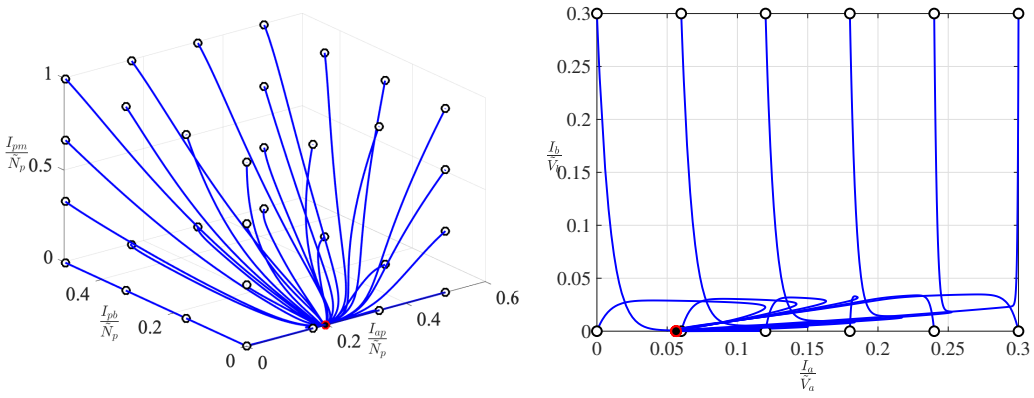

Figure 6: Case 2: $\mathcal{R}_{0,a} > 1$, $\mathcal{R}_{0,b} < 1$, convergence to E^*_{MCMV}.

Table 4: Case 2 Reproduction numbers

$\mathcal{R}^2_{0,a=MCMV}$	$\mathcal{R}^{MCMV}_{0,inv}$	$\hat{\mathcal{R}}^{MCMV}_{0,inv}$	$\mathcal{R}^2_{0,b=SCMV}$	$\mathcal{R}^{SCMV}_{0,inv}$	$\hat{\mathcal{R}}^{SCMV}_{0,inv}$
1.321	1.188	1.369	0.9193	0.8015	0.801

Table 5: Case 5 Reproduction numbers

$\mathcal{R}^2_{0,a=MCMV}$	$\mathcal{R}^{MCMV}_{0,inv}$	$\hat{\mathcal{R}}^{MCMV}_{0,inv}$	$\mathcal{R}^2_{0,b=SCMV}$	$\mathcal{R}^{SCMV}_{0,inv}$	$\hat{\mathcal{R}}^{SCMV}_{0,inv}$
1.321	1.105	1.225	1.218	0.919	0.842

Table 6: Case 6 - Reproduction numbers

$\mathcal{R}^2_{0,a=MCMV}$	$\mathcal{R}^{MCMV}_{0,inv}$	$\hat{\mathcal{R}}^{MCMV}_{0,inv}$	$\mathcal{R}^2_{0,b=SCMV}$	$\mathcal{R}^{SCMV}_{0,inv}$	$\hat{\mathcal{R}}^{SCMV}_{0,inv}$
1.163	0.973	0.946	1.218	0.983	0.967

a non-permanent way such that aphids control can be difficult. However, thrips control seems feasible, such that the MCMV disease can be controlled. In addition, roguing, i.e., removal of infected plants, can be an additional way to reduce the impact of the diseases and eventually to remove the disease (see Fig. 11, page 27). The figure shows the effect of roguing on the number of healthy plants. Here we assume roguing is introduced in the host compartments affected by MCMV, SCMV and MLN diseases since the infected plants are easy to identify.

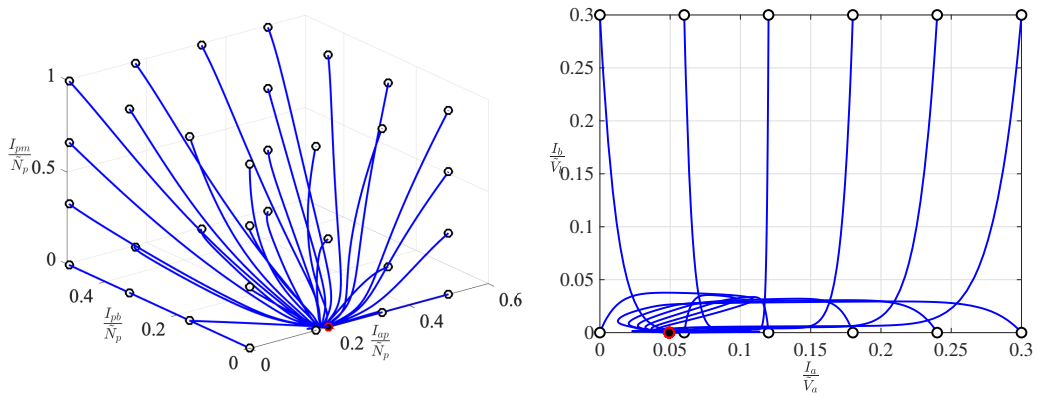

Figure 7: Case 5: $\mathcal{R}^2_{0,a} > 1$, $\mathcal{R}^{MCMV}_{0,inv} > 1$, $\mathcal{R}^2_{0,b} > 1$, and $\mathcal{R}^{SCMV}_{0,inv} < 1$ - Convergence to E^*_{MCMV}

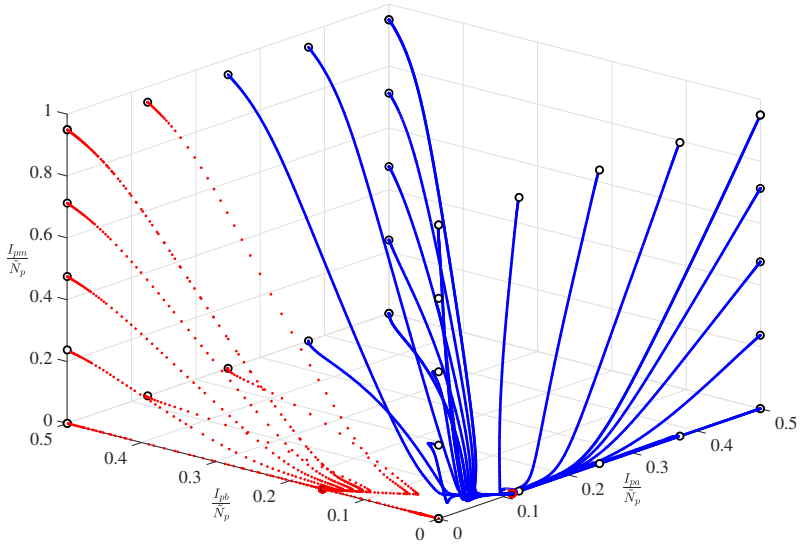

Figure 8: Case 6: $\mathcal{R}_{0,b} > \mathcal{R}_{0,a} > 1$, and $\mathcal{R}^{MCMV}_{0,inv} < 1$, $\mathcal{R}^{SCMV}_{0,inv} < 1$: bi-stable case. Convergence towards one boundary equilibrium depends on the initial conditions: the red-dotted trajectories converge to E_{SCMV} while the blue ones converge to E_{MCMV}.

6. Conclusion

For centuries, and with the expansion of mankind around the world, many crops have been transferred from their original area to new areas. Maize is one of the best example: it was imported back into Europe in 1493 by Christopher Colombus, and then spread throughout the world. Simultaneously, due to these movements, diseases and pests traveled too and also new diseases appeared. Since then, these dynamics have accelerated such that the impact of diseases has become even worse, thanks to local environmental changes and also by improving communication channels between countries and continents. Crops are simultaneously and strongly impacted by various diseases and also pests. While co-infection is relatively common in crops, synergistic interactions are not so common: since all mechanisms that drive synergism are not well known, it is very difficult to study in the field and also to consider control strategies. Modeling can be a way to help field researchers to test hypothesis before field experiments or/and to focus on specific experiments and in protocol building. Last, but not least, mathematical modeling and analysis can help to design control strategies or combination of strategies. In this work, we highlight the importance of

Table 7: Case 7 Reproduction numbers.

$\mathcal{R}^2_{0,a=MCMV}$	$\mathcal{R}^2_{0,b=SCMV}$	$\mathcal{R}^{SCMV}_{0,inv}$	$\hat{\mathcal{R}}^{SCMV}_{0,inv}$
1.3212	0.9193	1.0901	1.2023

Table 8: Case 8 Reproduction numbers.

$\mathcal{R}^2_{0,b=SCMV}$	$\mathcal{R}^2_{0,a=MCMV}$	$\mathcal{R}^{MCMV}_{0,inv}$	$\hat{\mathcal{R}}^{MCMV}_{0,inv}$
1.2179	0.9404	1.2929	1.8459

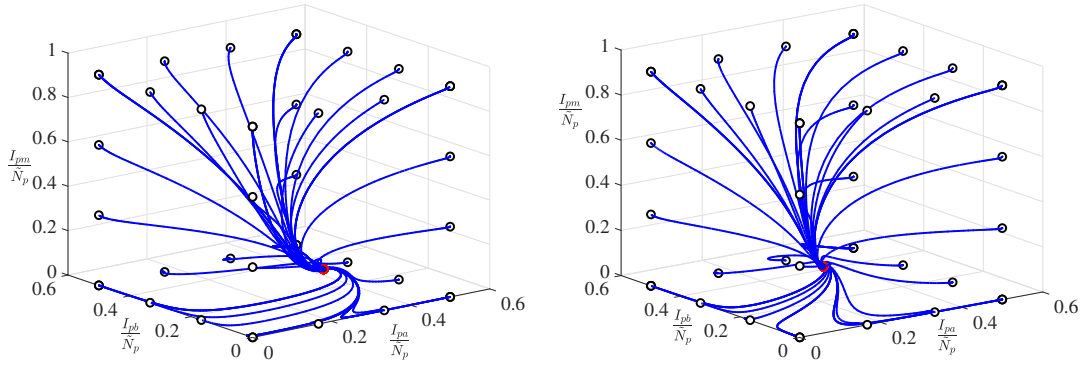

Figure 9: Case 7 & 8: Convergence to the MLN equilibrium, E^*_{MLN}, when one basic reproduction number is less than one while the related invasive reproduction number is greater than one.

Table 9: Case 9 Reproduction numbers.

$\mathcal{R}^2_{0,a=MCMV}$	$\mathcal{R}^{MCMV}_{0,inv}$	$\hat{\mathcal{R}}^{MCMV}_{0,inv}$	$\mathcal{R}^2_{0,b=SCMV}$	$\mathcal{R}^{SCMV}_{0,inv}$	$\hat{\mathcal{R}}^{SCMV}_{0,inv}$
1.3212	1.4951	2.5934	1.2179	1.2445	1.5928

estimating the basic and invasion reproduction numbers because they summarized the whole dynamics of the system. We also highlight that even if a basic reproduction number is less than one, MLN disease can occur. So far roguing, i.e., removal of infected plants, seem to be the best strategy but it needs to be set-up immediately as soon as the MCMV- or SCMV-symptoms are detected.

Like the works done in [33, 32], we do hope that our theoretical work can provide new insights in the MLND control, and also other (synergistic) co-infection issues. In particular, our study also reveals a certain lack in the knowledge related to the interactions between the virus, the vectors, and the host, and, thus, in the parameter estimates.

Further improvements in the models can be made, like taking into account plant growth in the different epidemiological states. In general this is never studied as observed in [1] because this would require new and difficult field observations. However, this would help to consider the impact of the MLN disease at different stages of the growth process taking into account the role of managing factors (cultivar choice, irrigation, etc). Last, since MLN-resistant Maize varieties exist, it would be interesting to estimate the optimal ratio of resistant plant to mix with standard healthy plants within a crop, like in [56, 46], to dilute the MLN-risk. Another option, more interesting for smallholder farmers, would be to consider Maize intercropping with non-susceptible host plants.

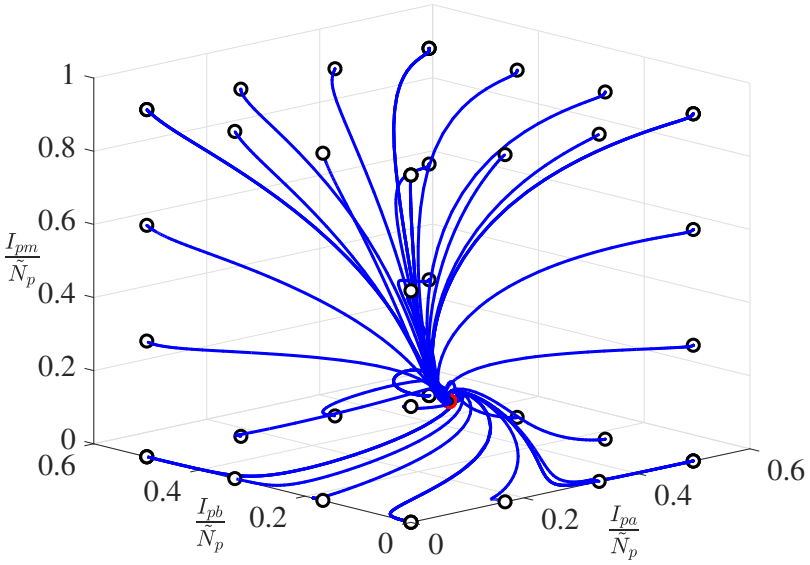

Figure 10: Case 9: $\mathcal{R}^2_{0,a} > 1$, $\mathcal{R}^2_{0,b} > 1$, $\mathcal{R}^{MCMV}_{0,inv} > 1$ and $\mathcal{R}^{SCMV}_{0,inv} > 1$ - Convergence to a MLN equilibrium, E^*_{MLN}.

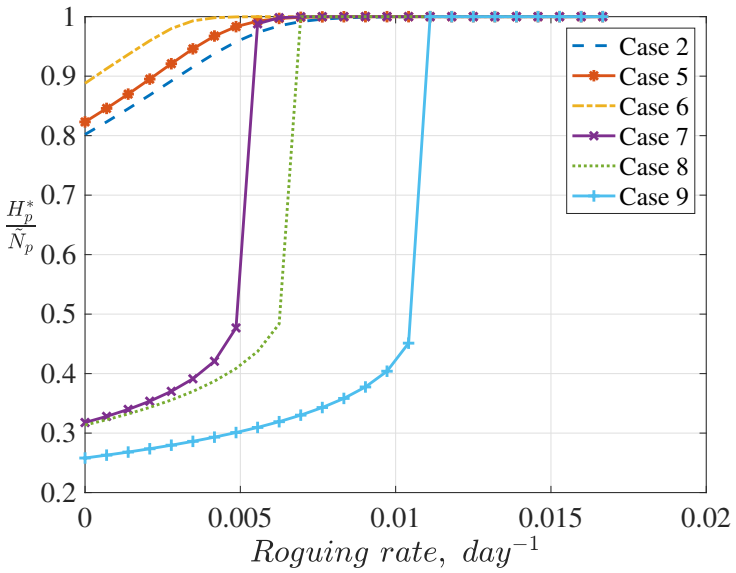

Figure 11: Impact of the roguing rate on the dynamics of the disease.

Acknowledgments

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Appendix A The boundary equilibria: E_{MCMV} and E_{SCMV}

We detail the calculations for the MCMV infection only. We have to solve the following system

$$\begin{cases} \Lambda_p - \beta_{pa}\dfrac{I_a^*}{V_a}H_p^* + \omega_{pa}I_{pa}^* - \mu_{ph}H_p^* &= 0 \\ \beta_{pa}\dfrac{I_a^*}{V_a}H_p^* - (\omega_{pa}+\mu_{pa})\,I_{pa}^* &= 0 \\ \Lambda_a - \mu_a S_a^* - \beta_{ap}\dfrac{I_{pa}^*}{N^*}S_a^* + \delta I_a^* &= 0 \\ \beta_{ap}\dfrac{I_{pa}^*}{N^*}S_a^* - \delta I_a^* - \mu_a I_a^* &= 0 \end{cases}. \tag{15}$$

Summing the first two equations, leads to the relationship

$$\mu_{ph}H_p^* + \mu_{pa}I_{pa}^* = \Lambda_p.$$

That is

$$H_p^* = \tilde{N}_p - \frac{\mu_{pa}}{\mu_{ph}}I_{pa}^*,$$

with $\mu_{pa} \geq \mu_{ph}$. Then

$$H_p^* + I_{ap}^* = N^* = \tilde{N}_p - \frac{\mu_{pa}-\mu_{ph}}{\mu_{ph}}I_{pa}^*.$$

From equation $(15)_2$, we have

$$\beta_{pa}\frac{I_a^*}{\tilde{V}_a}\left(\tilde{N}_p - \frac{\mu_{pa}}{\mu_{ph}}I_{pa}^*\right) = (\omega_{pa}+\mu_{pa})\,I_{pa}^*. \tag{16}$$

Summing the last two equations leads to $S_a^* + I_a^* = \tilde{V}_a$ and from the last equation we derive

$$\beta_{ap}\frac{I_{pa}^*}{N^*}S_a^* = \beta_{ap}\frac{I_{pa}^*}{\tilde{N}_p - \dfrac{\mu_{pa}-\mu_{ph}}{\mu_{ph}}I_{pa}^*}\left(\tilde{V}_a - I_a^*\right) = (\delta+\mu_a)\,I_a^*,$$

that is

$$\frac{I_a^*}{\tilde{V}_a} = \frac{\beta_{ap}\dfrac{I_{pa}^*}{\tilde{N}_p - \dfrac{\mu_{pa}-\mu_{ph}}{\mu_{ph}}I_{pa}^*}}{\beta_{ap}\dfrac{I_{pa}^*}{\tilde{N}_p - \dfrac{\mu_{pa}-\mu_{ph}}{\mu_{ph}}I_{pa}^*} + \delta + \mu_a} = \frac{\beta_{ap}I_{pa}^*}{\beta_{ap}I_{pa}^* + (\delta+\mu_a)\left(\tilde{N}_p - \dfrac{\mu_{pa}-\mu_{ph}}{\mu_{ph}}I_{pa}^*\right)}.$$

Replacing $\dfrac{I_a^*}{\tilde{V}_a}$ in (16) with the above expression, with $I_{pa} \neq 0$, we obtain

$$\beta_{pa}\beta_{ap}\left(\tilde{N}_p - \frac{\mu_{pa}}{\mu_{ph}}I_{pa}^*\right) = (\omega_{pa}+\mu_{pa})\left(\beta_{ap}I_{pa}^* + (\delta+\mu_a)\left(\tilde{N}_p - \frac{\mu_{pa}-\mu_{ph}}{\mu_{ph}}I_{pa}^*\right)\right)$$

$$\left(\beta_{pa}\beta_{ap} - (\omega_{pa}+\mu_{pa})(\delta+\mu_a)\right)\tilde{N}_p = (\omega_{pa}+\mu_{pa})\left(\frac{\beta_{pa}\beta_{ap}}{\omega_{pa}+\mu_{pa}}\frac{\mu_{pa}}{\mu_{ph}} + \beta_{ap} - (\delta+\mu_a)\frac{\mu_{pa}-\mu_{ph}}{\mu_{ph}}\right)I_{pa}^*$$

$$(\delta+\mu_a)\left(\mathcal{R}_{0,a}^2 - 1\right)\tilde{N}_p = \left(\beta_{ap} + (\delta+\mu_a)\left(1 + \frac{\mu_{pa}}{\mu_{ph}}\left(\mathcal{R}_{0,a}^2 - 1\right)\right)\right)I_{pa}^*$$

that is

$$I_{pa}^* = \frac{\left(\mathcal{R}_{0,a}^2 - 1\right)}{1 + \dfrac{\beta_{ap}}{\delta+\mu_a} + \dfrac{\mu_{pa}}{\mu_{ph}}\left(\mathcal{R}_{0,a}^2 - 1\right)}\tilde{N}_p$$

Thus, when $\mu_{pa} \geq \mu_{ph}$, I_{pa}^* exists iff $\mathcal{R}_{0,a} > 1$. We also deduce that E_{MCMV}^* exists iff $\mathcal{R}_{0,a} > 1$.

We obtain the same result for the SCMV virus: E_{SCMV}^* exists iff $\mathcal{R}_{0,b} > 1$.

Appendix B Local Asymptotic Stability of the boundary equilibria

Let us first assume that $\mu_{ph} = \mu_{pa} = \mu_{pb} = \mu_{pm}$. Then $H_p = \tilde{N} - I_{pa} - I_{pb} - I_{pm}$, $S_a = \tilde{V}_a - I_a$, and $S_b = \tilde{V}_b - I_b$. Thus, system (1)-(2)-(3) becomes

$$\frac{dy}{dt} = \begin{pmatrix} \beta_{pa}\frac{I_a}{\tilde{V}_a}\left(\tilde{N} - I_{pa} - I_{pb} - I_{pm}\right) - \beta_{am}\frac{I_{pa}I_b}{\tilde{V}_b} - (\omega_{pa} + \mu_{pa})I_{pa} \\ \left(\beta_{ap}\frac{I_{pa}}{\tilde{N}_p} + \beta_{ma}\frac{I_{pm}}{\tilde{N}_p}\right)\left(\tilde{V}_a - I_a\right) - (\delta + \mu_a)I_a \\ \beta_{pb}\frac{I_b}{\tilde{V}_b}\left(\tilde{N} - I_{pa} - I_{pb} - I_{pm}\right) - \left(\beta_{bm}\frac{I_a}{V_a} + (\omega_{pb} + \mu_{pb})\right)I_{pb} \\ \beta_{am}\frac{I_b}{V_b}I_{pa} + \beta_{bm}\frac{I_a}{V_a}I_{pb} - \mu_{pm}I_{pm} \\ \left(\beta_{bp}\frac{I_{pb}}{\tilde{N}_p} + \beta_{mb}\frac{I_{pm}}{\tilde{N}_p}\right)\left(\tilde{V}_b - I_b\right) - (\gamma + \mu_b)I_b \end{pmatrix}$$

with $y = (I_{pa}, I_a, I_{pb}, I_{pm}, I_b)$. Then, we compute the Jacobian $J(X)$ is given by

$$\begin{pmatrix} J_{1,1} & J_{1,2} & -\beta_{pa}\frac{I_a}{\tilde{V}_a} & -\beta_{pa}\frac{I_a}{\tilde{V}_a} & -\beta_{am}\frac{I_{pa}}{V_b} \\ \beta_{ap}\frac{\left(\tilde{V}_a - I_a\right)}{\tilde{N}_p} & J_{2,2} & 0 & \beta_{ma}\frac{\tilde{V}_a - I_a}{\tilde{N}_p} & 0 \\ -\beta_{pb}\frac{I_b}{\tilde{V}_b} & -\beta_{pb}\frac{I_b}{\tilde{V}_b} - \beta_{bm}\frac{I_{pb}}{V_a} & J_{3,3} & -\beta_{pb}\frac{I_b}{\tilde{V}_b} & \beta_{pb}\frac{\tilde{N}_p - I_{pa} - I_{pb} - I_{pm}}{\tilde{V}_b} \\ \beta_{am}\frac{I_b}{V_b} & \beta_{bm}\frac{I_{pb}}{V_a} & \beta_{bm}\frac{I_a}{V_a} & -\mu_{pm} & \beta_{am}\frac{I_{pa}}{V_b} \\ & & \beta_{bp}\frac{\tilde{V}_b}{\tilde{N}_p} - \beta_{mb}\frac{I_b}{\tilde{N}_p} & \beta_{mb}\frac{\tilde{V}_b}{\tilde{N}_p} - \beta_{mb}\frac{I_b}{\tilde{N}_p} & J_{5,5} \end{pmatrix},$$

with

$$\begin{aligned} J_{1,1} &= -\left(\beta_{pa}\frac{I_a}{\tilde{V}_a} + \beta_{am}\frac{I_b}{\tilde{V}_b} + (\omega_{pa} + \mu_{pa})\right), \\ J_{1,2} &= \beta_{pa}\frac{\left(\tilde{N}_p - I_{pa} - I_{pb} - I_{pm}\right)}{V_a}, \\ J_{2,2} &= -\left(\beta_{ap}\frac{I_{pa}}{\tilde{N}_p} + \beta_{ma}\frac{I_{pm}}{\tilde{N}_p}\right) - (\delta + \mu_a) \\ J_{3,3} &= \beta_{bm}\frac{I_a}{V_a} + (\omega_{pb} + \mu_{pb}), \\ J_{5,5} &= -\left(\beta_{bp}\frac{I_{pb}}{\tilde{N}_p} + \beta_{mb}\frac{I_{pm}}{\tilde{N}_p}\right) - (\gamma + \mu_b). \end{aligned}$$

We deduce $J(E^*_{\text{MCMV}})$ as follows

$$\begin{pmatrix} -\left(\omega_{pa} + \mu_{pa} + \beta_{pa}\frac{I^*_a}{\tilde{V}_a}\right) & \beta_{pa}\frac{\tilde{N}_p - I^*_{pa}}{\tilde{V}_a} & -\beta_{pa}\frac{I^*_a}{\tilde{V}_a} & 0 & \beta_{am}\frac{I^*_{pa}}{V_b} \\ \beta_{ap}\frac{S^*_a}{\tilde{N}_p} & -\left(\beta_{ap}\frac{I^*_{pa}}{\tilde{N}_p}\right) - (\delta + \mu_a) & 0 & \beta_{ma}\frac{S^*_a}{\tilde{N}_p} & 0 \\ 0 & 0 & -\left(\beta_{bm}\frac{I^*_a}{V_a} + (\omega_{pb} + \mu_{pb})\right) & 0 & \beta_{pb}\frac{H^*_p}{\tilde{V}_b} \\ 0 & 0 & \beta_{bm}\frac{I^*_a}{\tilde{V}_a} & -\mu_{pm} & \beta_{am}\frac{I^*_{pa}}{\tilde{V}_b} \\ 0 & 0 & \beta_{bp}\frac{\tilde{V}_b}{\tilde{N}_p} & \beta_{mb}\frac{\tilde{V}_b}{\tilde{N}_p} & -(\gamma + \mu_b) \end{pmatrix}$$

The Jacobian $J(E^*_{\text{MCMV}})$ can be written as a triangular upper-block matrix $\begin{pmatrix} M_{11} & M_{12} \\ 0 & M_{22} \end{pmatrix}$, such that it suffices to study M_{ii}, for $i = 1, 2$. We easily check that $tr(M_{11}) < 0$ and

$$det(M_{11}) = \left(\beta_{pa}\frac{I^*_a}{V_a} + \omega_{pa} + \mu_{pa}\right)\left(\beta_{ap}\frac{I^*_{pa}}{N} + \delta + \mu_a\right) - \beta_{ap}\left(1 - \frac{I^*_a}{\tilde{V}_a}\right)\beta_{pa}\left(1 - \frac{I^*_{pa}}{\tilde{N}_p}\right)$$

$$det(M_{11}) = \left(\beta_{pa}\frac{I^*_a}{\tilde{V}_a} + (\omega_{pa} + \mu_{pa})\right)\left(\beta_{ap}\frac{I^*_{pa}}{\tilde{N}_p} + (\delta + \mu_a)\right) - \beta_{ap}\beta_{pa}\left(1 - \frac{I^*_a}{\tilde{V}_a}\right)\left(1 - \frac{I^*_{pa}}{\tilde{N}_p}\right) =$$

$$= (\omega_{pa} + \mu_{pa})(\delta + \mu_a) - \beta_{ap}\beta_{pa} + \beta_{pa}\frac{I^*_a}{\tilde{V}_a}((\delta + \mu_a) + \beta_{ap}) + \beta_{ap}\frac{I^*_{pa}}{\tilde{N}_p}((\omega_{pa} + \mu_{pa}) + \beta_{pa})$$

$$= (\omega_{pa} + \mu_{pa})(\delta + \mu_a)\left(1 - \mathcal{R}^2_{0,a} + \frac{I^*_a}{\tilde{V}_a}\left(\frac{\beta_{pa}}{\omega_{pa} + \mu_{pa}} + \mathcal{R}^2_{0,a}\right) + \frac{I^*_{pa}}{\tilde{N}_p}\left(\frac{\beta_{ap}}{\delta + \mu_a} + \mathcal{R}^2_{0,a}\right)\right)$$

Since

$$I^*_{pa} = \frac{(\mathcal{R}^2_{0,a} - 1)}{\frac{\beta_{ap}}{(\delta + \mu_a)} + \mathcal{R}^2_{0,a}}\tilde{N}_p,$$

we deduce

$$det(M_{11}) = (\omega_{pa} + \mu_{pa})(\delta + \mu_a)\left(1 - \mathcal{R}^2_{0,a} + \frac{I^*_a}{\tilde{V}_a}\left(\frac{\beta_{pa}}{\omega_{pa} + \mu_{pa}} + \mathcal{R}^2_{0,a}\right) + \frac{(\mathcal{R}^2_{0,a} - 1)}{\frac{\beta_{ap}}{(\delta + \mu_a)} + \mathcal{R}^2_{0,a}}\left(\frac{\beta_{ap}}{\delta + \mu_a} + \mathcal{R}^2_{0,a}\right)\right)$$

$$= (\omega_{pa} + \mu_{pa})(\delta + \mu_a)\left(\frac{\beta_{pa}}{\omega_{pa} + \mu_{pa}} + \mathcal{R}^2_{0,a}\right)\frac{I^*_a}{\tilde{V}_a} > 0.$$

Thus M_{11} is a stable Matrix. Then, we consider

$$M_{22} = \begin{pmatrix} -\left(\beta_{bm}\frac{I^*_a}{V_a} + (\omega_{pb} + \mu_{pb})\right) & 0 & \beta_{pb}\frac{H^*_p}{\tilde{V}_b} \\ \beta_{bm}\frac{I^*_a}{\tilde{V}_a} & -\mu_{pm} & \beta_{am}\frac{I^*_{pa}}{\tilde{V}_b} \\ \beta_{bp}\frac{\tilde{V}_b}{\tilde{N}_p} & \beta_{mb}\frac{\tilde{V}_b}{\tilde{N}_p} & -(\gamma + \mu_b) \end{pmatrix}.$$

In fact matrix M_{22} is a Metzler matrix with the following regular splitting

$$M + N = \begin{pmatrix} 0 & 0 & \beta_{pb}\frac{H^*_p}{\tilde{V}_b} \\ \beta_{bm}\frac{I^*_a}{\tilde{V}_a} & 0 & \beta_{am}\frac{I^*_{pa}}{\tilde{V}_b} \\ \beta_{bp}\frac{\tilde{V}_b}{\tilde{N}_p} & \beta_{mb}\frac{\tilde{V}_b}{\tilde{N}_p} & 0 \end{pmatrix} + \begin{pmatrix} -\left(\beta_{bm}\frac{I^*_a}{V_a} + (\omega_{pb} + \mu_{pb})\right) & 0 & 0 \\ 0 & -\mu_{pm} & 0 \\ 0 & 0 & -(\gamma + \mu_b) \end{pmatrix},$$

where M is a non negative matrix, and N is a stable Metzler matrix. It is well known that $M + N$ is stable if and only if $\rho(-N^{-1}M) < 1$. However, we have $M = F$ and $N = -V$ where F and V are the matrices defined in subsection 3.3. We infer that the stability of M_{22} is thus related to the invasion reproduction number, $\mathcal{R}^{SCMV}_{inv}$. Thus it is easy to conclude that if $\mathcal{R}^{SCMV}_{inv} < 1$ and $\mathcal{R}^2_{0,a} > 1$, then E^*_{MCMV} is asymptotically stable.

If we assume that $\mu_{pm}, \mu_{pb}, \mu_{pa} \geq \mu_{ph}$, then the proof is a bit more tricky because we have to consider the system with $(H_p, I_{pa}, I_a, I_{pb}, I_b, I_{pm})$, such that the Jacobian matrix at E^*_{MCMV} is a 6-order upper-block triangular matrice $J(E^*_{\text{MCMV}}) = \begin{pmatrix} J_{11} & J_{12} \\ 0 & M_{22} \end{pmatrix}$, where M_{22} is the same invasion matrix than in

the previous proof, and

$$J_{11}=\begin{pmatrix} -\mu_{ph}-\beta_{pa}\dfrac{I_a^*}{\tilde{V}_a} & \omega_{pa} & -\beta_{pa}\dfrac{H_p^*}{\tilde{V}_a} \\ \beta_{pa}\dfrac{I_a^*}{\tilde{V}_a} & -(\omega_{pa}+\mu_{pa}) & \beta_{pa}\dfrac{H_p^*}{\tilde{V}_a} \\ -\beta_{ap}I_{pa}^*\dfrac{\left(\tilde{V}_a-I_a^*\right)}{\left(N_p^*\right)^2} & \beta_{ap}\dfrac{\left(\tilde{V}_a-I_a^*\right)}{\left(N_p^*\right)^2}H_p^* & -\beta_{ap}\dfrac{I_{pa}^*}{N_p^*}-(\delta+\mu_a) \end{pmatrix}.$$

To show that J_{11} is a stable matrix, we will use the well known-result [57]: let A be a 3×3 real matrix. If $tr(A)$, $det(A)$ and $det(A^{[2]})$ are all negative, then all of the eigenvalues of A have negative real part. Of course $tr(J_{11})<0$. Then, we compute

$$\begin{aligned} det(J_{11}) = & \begin{vmatrix} -\mu_p-\beta_{pa}\dfrac{I_a}{\tilde{V}_a} & \omega_{pa} & -\beta_{pa}\dfrac{H_p}{\tilde{V}_a} \\ \beta_{pa}\dfrac{I_a}{\tilde{V}_a} & -(\omega_{pa}+\mu_{pa}) & \beta_{pa}\dfrac{H_p}{\tilde{V}_a} \\ -(\beta_{ap}I_{pa})\dfrac{\left(\tilde{V}_a-I_a\right)}{\left(N_p^*\right)^2} & \beta_{ap}\dfrac{\left(\tilde{V}_a-I_a\right)}{\left(N_p^*\right)^2}H_p^* & -\left(\beta_{ap}\dfrac{I_{pa}}{N_p^*}\right)-(\delta+\mu_a) \end{vmatrix} = \\ & -\left(\mu_{ph}+\beta_{pa}\frac{I_a}{\tilde{V}_a}\right)\begin{vmatrix} -(\omega_{pa}+\mu_{pa}) & \beta_{pa}\dfrac{H_p}{\tilde{V}_a} \\ \beta_{ap}\dfrac{\left(\tilde{V}_a-I_a\right)}{\left(N_p^*\right)^2}H_p^* & -\left(\beta_{ap}\dfrac{I_{pa}^*}{N_p^*}\right)-(\delta+\mu_a) \end{vmatrix} \\ & +\beta_{pa}\frac{I_a^*}{\tilde{V}_a}\begin{vmatrix} \beta_{ap}\dfrac{\left(\tilde{V}_a-I_a^*\right)}{\left(N_p^*\right)^2}H_p^* & -\left(\beta_{ap}\dfrac{I_{pa}^*}{N_p^*}\right)-(\delta+\mu_a) \\ \omega_{pa} & -\beta_{pa}\dfrac{H_p}{\tilde{V}_a} \end{vmatrix}+\mu_{pa}\beta_{pa}\beta_{ap}\frac{I_{pa}}{N_p^*}\frac{H_p}{N_p^*}\left(1-\frac{I_a}{\tilde{V}_a}\right) \end{aligned}$$

$$= \left(\mu_{ph}+\beta_{pa}\frac{I_a}{\tilde{V}_a}\right)\left(\beta_{pa}\beta_{ap}\frac{H_p}{N_p^*}\frac{H_p}{N_p^*}\left(1-\frac{I_a}{\tilde{V}_a}\right)-(\omega_{pa}+\mu_{pa})\left(\left(\beta_{ap}\frac{I_{pa}^*}{N_p^*}\right)+(\delta+\mu_a)\right)\right)+$$

$$+\beta_{pa}\frac{I_a}{\tilde{V}_a}\left(\omega_{pa}\left(\left(\beta_{ap}\frac{I_{pa}^*}{N_p^*}\right)+(\delta+\mu_a)\right)-\beta_{pa}\beta_{ap}\frac{H_p}{N_p^*}\frac{H_p}{N_p^*}\left(1-\frac{I_a}{\tilde{V}_a}\right)\right)+\mu_{pa}\beta_{pa}\beta_{ap}\frac{I_{pa}}{N_p^*}\frac{H_p}{N_p^*}\left(1-\frac{I_a}{\tilde{V}_a}\right)=$$

$$=\mu_{ph}\left(\beta_{pa}\beta_{ap}\frac{H_p}{N_p^*}\frac{H_p}{N_p^*}\left(1-\frac{I_a}{\tilde{V}_a}\right)-(\omega_{pa}+\mu_{pa})\left(\beta_{ap}\frac{I_{pa}^*}{N_p^*}+(\delta+\mu_a)\right)\right)-\beta_{pa}\frac{I_a}{\tilde{V}_a}(\omega_{pa}+\mu_{pa})\left(\beta_{ap}\frac{I_{pa}^*}{N_p^*}+(\delta+\mu_a)\right)+$$

$$+\beta_{pa}\frac{I_a}{\tilde{V}_a}\left(\omega_{pa}\left(\left(\beta_{ap}\frac{I_{pa}^*}{N_p^*}\right)+(\delta+\mu_a)\right)\right)+\mu_{pa}\beta_{pa}\beta_{ap}\frac{I_{pa}}{N_p^*}\frac{H_p}{N_p^*}\left(1-\frac{I_a}{\tilde{V}_a}\right)=$$

$$=\mu_{ph}\left(\beta_{pa}\beta_{ap}\frac{H_p}{N_p^*}\left(1-\frac{I_a}{\tilde{V}_a}\right)-(\omega_{pa}+\mu_{pa})\left(\left(\beta_{ap}\frac{I_{pa}^*}{N_p^*}\right)+(\delta+\mu_a)\right)\right)-\beta_{pa}\frac{I_a}{\tilde{V}_a}\mu_{pa}\left(\left(\beta_{ap}\frac{I_{pa}^*}{N_p^*}\right)+(\delta+\mu_a)\right)$$

$$=\mu_{ph}\left(\beta_{pa}\beta_{ap}\frac{{H_p}^*}{N_p^*}\left(1-\frac{I_a^*}{\tilde{V}_a}\right)-\left(\omega_{pa}+\mu_{pa}+\beta_{pa}\frac{I_a}{\tilde{V}_a}\frac{\mu_{pa}}{\mu_{ph}}\right)\left(\beta_{ap}\frac{I_{pa}^*}{N_p^*}+(\delta+\mu_a)\right)\right)$$

Using the fact that

$$I_{pa}^*=\frac{\left(\mathcal{R}_{0,a}^2-1\right)}{\dfrac{\beta_{ap}}{(\delta+\mu_a)}+1+\dfrac{\mu_{pa}}{\mu_{hp}}\left(\mathcal{R}_{0,a}^2-1\right)}\tilde{N}_p$$

$$\frac{I_a}{\tilde{V}_a}=\frac{\beta_{ap}I_{pa}^*}{\beta_{ap}I_{pa}^*+(\delta+\mu_a)\left(\tilde{N}_p-\dfrac{\mu_{pa}-\mu_{ph}}{\mu_{ph}}I_{pa}^*\right)}$$

$$1-\frac{I_a}{\tilde{V}_a}=\frac{(\delta+\mu_a)\left(\tilde{N}_p-\frac{\mu_{pa}-\mu_{ph}}{\mu_{ph}}I^*_{pa}\right)}{\beta_{ap}I^*_{pa}+(\delta+\mu_a)\tilde{N}_p}=\frac{(\delta+\mu_a)N^*_p}{\beta_{ap}I^*_{pa}+(\delta+\mu_a)\tilde{N}_p}$$

$$H_p=\tilde{N}_p-\frac{\mu_{pa}}{\mu_{hp}}I_{pa}=\tilde{N}_p-\frac{\mu_{pa}}{\mu_{hp}}\frac{\left(\mathcal{R}^2_{0,a}-1\right)}{\frac{\beta_{ap}}{(\delta+\mu_a)}+1+\frac{\mu_{pa}}{\mu_{hp}}\left(\mathcal{R}^2_{0,a}-1\right)}=\frac{\frac{\beta_{ap}}{(\delta+\mu_a)}+1}{\frac{\beta_{ap}}{(\delta+\mu_a)}+1+\frac{\mu_{pa}}{\mu_{hp}}\left(\mathcal{R}^2_{0,a}-1\right)}\tilde{N}_p,$$

we deduce

$$\beta_{pa}\beta_{ap}\frac{H^*_p}{N^*_p}\left(1-\frac{I^*_a}{\tilde{V}_a}\right)=\beta_{pa}\beta_{ap}\frac{\frac{\beta_{ap}}{(\delta+\mu_a)}+1}{\frac{\beta_{ap}}{(\delta+\mu_a)}+1+\frac{\mu_{pa}}{\mu_{hp}}\left(\mathcal{R}^2_{0,a}-1\right)}\frac{(\delta+\mu_a)}{\beta_{ap}I^*_{pa}+(\delta+\mu_a)\tilde{N}_p}\tilde{N}_p=$$

$$=\beta_{pa}\beta_{ap}\left(\frac{\beta_{ap}+(\delta+\mu_a)}{\beta_{ap}+(\delta+\mu_a)\left(1+\frac{\mu_{pa}}{\mu_{hp}}\left(\mathcal{R}^2_{0,a}-1\right)\right)}\right)\frac{(\delta+\mu_a)}{\beta_{ap}I^*_{pa}+(\delta+\mu_a)\tilde{N}_p}\tilde{N}_p.$$

However

$$\begin{aligned}\frac{(\delta+\mu_a)\tilde{N}_p}{\beta_{ap}I^*_{pa}+(\delta+\mu_a)\tilde{N}_p}&=\frac{(\delta+\mu_a)}{\beta_{ap}\frac{\left(\mathcal{R}^2_{0,a}-1\right)}{\frac{\beta_{ap}}{(\delta+\mu_a)}+1+\frac{\mu_{pa}}{\mu_{hp}}\left(\mathcal{R}^2_{0,a}-1\right)}+(\delta+\mu_a)}\\&=\frac{1}{\beta_{ap}\frac{\left(\mathcal{R}^2_{0,a}-1\right)}{\beta_{ap}+(\delta+\mu_a)\left(1+\frac{\mu_{pa}}{\mu_{hp}}\left(\mathcal{R}^2_{0,a}-1\right)\right)}+1}\\&=\frac{\beta_{ap}+(\delta+\mu_a)\left(1+\frac{\mu_{pa}}{\mu_{hp}}\left(\mathcal{R}^2_{0,a}-1\right)\right)}{\beta_{ap}\mathcal{R}^2_{0,a}+(\delta+\mu_a)\left(1+\frac{\mu_{pa}}{\mu_{hp}}\left(\mathcal{R}^2_{0,a}-1\right)\right)},\end{aligned}$$

such that

$$\begin{aligned}\beta_{pa}\beta_{ap}\frac{H^*_p}{N^*_p}\left(1-\frac{I^*_a}{\tilde{V}_a}\right)&=\beta_{pa}\beta_{ap}\left(\frac{\beta_{ap}+(\delta+\mu_a)}{\beta_{ap}\mathcal{R}^2_{0,a}+(\delta+\mu_a)\left(1+\frac{\mu_{pa}}{\mu_{hp}}\left(\mathcal{R}^2_{0,a}-1\right)\right)}\right)\frac{(\delta+\mu_a)\tilde{N}_p}{\beta_{ap}I^*_{pa}+(\delta+\mu_a)\tilde{N}_p}\\&\leq\frac{\beta_{pa}\beta_{ap}}{\mathcal{R}^2_{0,a}}=(\omega_{pa}+\mu_{pa})(\delta+\mu_a)\end{aligned}$$

Thus, immediately, we deduce

$$det(J)<0.$$

Let's consider the second compound matrice of J_{11} (see [58]) $J^{[2]}_{11}=$

$$\begin{pmatrix}-\mu_{ph}-\beta_{pa}\frac{I^*_a}{\tilde{V}_a}-(\omega_{pa}+\mu_{pa}) & \beta_{pa}\frac{H^*_p}{\tilde{V}_a} & \beta_{pa}\frac{H^*_p}{\tilde{V}_a}\\ \beta_{ap}\frac{\left(\tilde{V}_a-I^*_a\right)}{\left(N^*_p\right)^2}H^*_p & -\mu_{ph}-\beta_{pa}\frac{I^*_a}{\tilde{V}_a}-\left(\beta_{ap}\frac{I^*_{pa}}{N^*_p}\right)-(\delta+\mu_a) & \omega_{pa}\\ (\beta_{ap}I_{pa})\frac{\left(\tilde{V}_a-I^*_a\right)}{\left(N^*_p\right)^2} & \beta_{pa}\frac{I^*_a}{\tilde{V}_a} & -(\omega_{pa}+\mu_{pa})-\left(\beta_{ap}\frac{I^*_{pa}}{N^*_p}\right)-(\delta+\mu_a)\end{pmatrix}.$$

We have $det\left(J^{[2]}\right)=$

$$-\left(\mu_{ph}+\beta_{pa}\frac{I^*_a}{\tilde{V}_a}+(\omega_{pa}+\mu_{pa})\right)\begin{vmatrix}-\mu_{ph}-\beta_{pa}\frac{I^*_a}{\tilde{V}_a}-\left(\beta_{ap}\frac{I^*_{pa}}{N^*_p}\right)-(\delta+\mu_a) & \omega_{pa}\\ \beta_{pa}\frac{I^*_a}{\tilde{V}_a} & -(\omega_{pa}+\mu_{pa})-\left(\beta_{ap}\frac{I^*_{pa}}{N^*_p}\right)-(\delta+\mu_a)\end{vmatrix}+$$

$$+\beta_{ap}\frac{\left(\tilde{V}_a-I_a^*\right)}{\left(N_p^*\right)^2}H_p^*\begin{vmatrix}\beta_{pa}\frac{I_a^*}{\tilde{V}_a} & -(\omega_{pa}+\mu_{pa})-\left(\beta_{ap}\frac{I_{pa}^*}{N_p^*}\right)-(\delta+\mu_a)\\ \beta_{pa}\frac{H_p^*}{\tilde{V}_a} & \beta_{pa}\frac{H_p^*}{\tilde{V}_a}\end{vmatrix}+$$

$$(\beta_{ap}I_{pa})\frac{\left(\tilde{V}_a-I_a^*\right)}{\tilde{N}_p^2}\begin{vmatrix}\beta_{pa}\frac{H_p^*}{\tilde{V}_a} & \beta_{pa}\frac{H_p^*}{\tilde{V}_a}\\ -\mu_{ph}-\beta_{pa}\frac{I_a^*}{\tilde{V}_a}-\left(\beta_{ap}\frac{I_{pa}^*}{N_p^*}\right)-(\delta+\mu_a) & \omega_{pa}\end{vmatrix}$$

$$\begin{aligned}
&= -\left(\mu_{ph}+\beta_{pa}\frac{I_a^*}{\tilde{V}_a}+\omega_{pa}+\mu_{pa}\right)\left(\left(\mu_{ph}+\beta_{pa}\frac{I_a^*}{\tilde{V}_a}+\beta_{ap}\frac{I_{pa}^*}{N_p^*}+\delta+\mu_a\right)\left(\omega_{pa}+\mu_{pa}+\beta_{ap}\frac{I_{pa}^*}{N_p^*}+\delta+\mu_a\right)-\omega_{pa}\beta_{pa}\frac{I_a^*}{\tilde{V}_a}\right)+\\
&\quad+\beta_{ap}\frac{\left(\tilde{V}_a-I_a^*\right)}{\left(N_p^*\right)^2}H_p^*\left(\beta_{pa}\frac{H_p^*}{\tilde{V}_a}\left(\beta_{pa}\frac{I_a^*}{\tilde{V}_a}+\left((\omega_{pa}+\mu_{pa})+\left(\beta_{ap}\frac{I_{pa}^*}{N_p^*}\right)+(\delta+\mu_a)\right)\right)\right)+\\
&\quad+\left(\beta_{ap}{I_{pa}}^*\right)\frac{\left(\tilde{V}_a-I_a^*\right)}{\left(N_p^*\right)^2}\left(\beta_{pa}\frac{H_p^*}{\tilde{V}_a}\left(\omega_{pa}+\mu_{ph}+\beta_{pa}\frac{I_a^*}{\tilde{V}_a}+\left(\beta_{ap}\frac{I_{pa}^*}{N_p^*}\right)+(\delta+\mu_a)\right)\right)\\
&= -\left(\mu_{ph}+\beta_{pa}\frac{I_a^*}{\tilde{V}_a}+\omega_{pa}+\mu_{pa}\right)\left(\mu_{ph}+\beta_{ap}\frac{I_{pa}^*}{N_p^*}+\delta+\mu_a\right)\left(\omega_{pa}+\mu_{pa}+\beta_{ap}\frac{I_{pa}^*}{N_p^*}+\delta+\mu_a\right)+\\
&\quad-\omega_{pa}\left(\mu_{ph}+\beta_{pa}\frac{I_a^*}{\tilde{V}_a}+\omega_{pa}+\mu_{pa}\right)\left(\mu_{ph}+\beta_{ap}\frac{I_{pa}^*}{\overline{N}_p}+\delta+\mu_a\right)+\\
&\quad+\beta_{ap}\beta_{pa}\frac{H_p}{N_p^*}\left(1-\frac{I_a^*}{\tilde{V}_a}\right)\left(\beta_{pa}\frac{I_a}{\tilde{V}_a}+\left((\omega_{pa}+\mu_{pa})+\left(\beta_{ap}\frac{I_{pa}^*}{N_p^*}\right)+(\delta+\mu_a)\right)\right)=\\
&= \left(\beta_{ap}\beta_{pa}\frac{H_p^*}{N_p^*}\left(1-\frac{I_a^*}{\tilde{V}_a}\right)-\left(\mu_{ph}+\beta_{pa}\frac{I_a^*}{\tilde{V}_a}+\omega_{pa}+\mu_{pa}\right)\left(\mu_{ph}+\beta_{ap}\frac{I_{pa}^*}{N_p^*}+\delta+\mu_a\right)\right)\left(\omega_{pa}+\mu_{pa}+\beta_{ap}\frac{I_{pa}^*}{N_p^*}+\delta+\mu_a\right)+\\
&\quad-\omega_{pa}\left(\mu_{ph}+\beta_{pa}\frac{I_a^*}{\tilde{V}_a}+\omega_{pa}+\mu_{pa}\right)\left(\mu_{ph}+\beta_{ap}\frac{I_{pa}^*}{N_p^*}+\delta+\mu_a\right)+\beta_{ap}\beta_{pa}\frac{H_p^*}{N_p^*}\left(1-\frac{I_a^*}{\tilde{V}_a}\right)\beta_{pa}\frac{I_a^*}{\tilde{V}_a}.
\end{aligned}$$

We showed above that

$$\beta_{pa}\beta_{ap}\frac{H_p^*}{N_p^*}\left(1-\frac{I_a^*}{\tilde{V}_a}\right)\leq(\omega_{pa}+\mu_{pa})(\delta+\mu_a),$$

such that we have

$$\begin{aligned}
\det\left(J^{[2]}\right)\leq\ & ((\omega_{pa}+\mu_{pa})(\delta+\mu_a)\\
&-\left(\mu_{ph}+\beta_{pa}\frac{I_a^*}{\tilde{V}_a}+\omega_{pa}+\mu_{pa}\right)\left(\mu_{ph}+\beta_{ap}\frac{I_{pa}^*}{\overline{N}_p}+\delta+\mu_a\right)\Big)\left(\omega_{pa}+\mu_{pa}+\beta_{ap}\frac{I_{pa}^*}{\overline{N}_p}+\delta+\mu_a\right)\\
&-\omega_{pa}\left(\mu_{ph}+\beta_{pa}\frac{I_a^*}{\tilde{V}_a}+\omega_{pa}+\mu_{pa}\right)\left(\mu_{ph}+\beta_{ap}\frac{I_{pa}^*}{\overline{N}_p}+\delta+\mu_a\right)+(\omega_{pa}+\mu_{pa})(\delta+\mu_a)\beta_{pa}\frac{I_a^*}{\tilde{V}_a}.
\end{aligned}$$

Finally, we deduce

$$\begin{aligned}
\det\left(J^{[2]}\right)\leq\ & -\Bigg(\left(\mu_{ph}+\beta_{pa}\frac{I_a^*}{\tilde{V}_a}\right)\left(\mu_{ph}+\beta_{ap}\frac{I_{pa}^*}{\overline{N}_p}+\delta+\mu_a\right.\\
&\left.+(\omega_{pa}+\mu_{pa})\left(\mu_{ph}+\beta_{ap}\frac{I_{pa}^*}{\overline{N}_p}\right)\right)\Bigg)\left(\omega_{pa}+\mu_{pa}+\beta_{ap}\frac{I_{pa}^*}{\overline{N}_p}+\delta+\mu_a\right)\\
&-\omega_{pa}\left(\mu_{ph}+\beta_{pa}\frac{I_a^*}{\tilde{V}_a}+\omega_{pa}+\mu_{pa}\right)\left(\mu_{ph}+\beta_{ap}\frac{I_{pa}^*}{\overline{N}_p}+\delta+\mu_a\right)+(\omega_{pa}+\mu_{pa})(\delta+\mu_a)\beta_{pa}\frac{I_a^*}{\tilde{V}_a}\\
&<0.
\end{aligned}$$

Altogether, when $\mathcal{R}_{0,a}^2>1$ and $\mathcal{R}_{0,inv}^{SCMV}<1$, then E_{MCMV}^* exists and is LAS.

Of course, using the same reasoning, we deduce that if $\mathcal{R}_{0,b}>1$ and $\mathcal{R}_{0,inv}^{MCMV}<1$, then E_{SCMV}^* exists and is LAS.

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