A patchy model for Chikungunya-like diseases

Samuel Bowong
University of Douala
Faculty of Science
UMI 209 IRD/UPMC UMMISCO
GRIMCAPE project team
Douala, Cameroon
sbowong@gmail.com

Yves Dumont
CIRAD
Umr AMAP 34392
Montpellier, France
yves.dumont@cirad.fr

Jean Jules Tewa
National Advanced School of Engineering
University of Yaoundé I,
UMI 209 IRD/UPMC UMMISCO
GRIMCAPE project team
Yaoundé, Cameroon
tewajules@gmail.com

Received: 21 February 2013, accepted: 23 July 2013, published: 31 July 2013

Abstract—We consider a $n$-patches model, to study the impact of human population movements between cities (patches) in the spread of Chikungunya or even Dengue diseases. In previous works, it was showed that the basic reproduction number can vary from place to place, but this result was obtained without taking into account human movements. We provide a theoretical study of the patchy model, and derive $R_0^2$, the basic reproduction number, which may depend on Human movement rates between the patches and on local population sizes. We show that $R_0$ is bounded from above (below) by the maximum (minimum) of the values of the local basic reproduction numbers. We also show that there exists a disease-free equilibrium $E_{DF}$ that is locally asymptotically stable whenever $R_0^2 < 1$. Under suitable assumptions, $E_{DF}$ is even globally asymptotically stable. We emphasize that Human movements are of particular importance to evaluate the spreading or not of Chikungunya or Dengue diseases, and thus Human movement rates has to be estimated very accurately. We confirm also the importance to know where local basic reproduction numbers are large and show that local field interventions can help to control/reduce the spread of the disease. A full analytical study for the 2-patches model and several simulations are provided to illustrate that human movements can either increase or reduce the spreading of the disease.

Keywords—Patch; Chikungunya; Dengue; Movements; Disease free equilibrium; Basic reproduction number; Endemic equilibrium; Local and Global Stability.

AMS Classification: 92-08, 92D30, 37M05, 65L12, 92C60.

I. INTRODUCTION

Chikungunya is a vector-borne disease caused by Aedes albopictus. It is an uncommon and not well-known tropical disease whose dynamics and behaviour are yet to be fully understood [33]. A good understanding of its transmission dynamics and its ecology in emergent epidemic regions like Réunion Island can help to improve the control of future epidemics around the world. Mathematical models provide a quantitative and potentially valuable tool for this purpose. The ability to forecast, understand and control the spread of infectious diseases increasingly depends on the capacity to formulate and test mathematical models capturing key mechanisms. The present study builds on and extend previous works on the Chikungunya disease [18], [19], [23].

Chikungunya is endemic in Est Africa and in Asia. The main symptoms are fever, headache and arthritis, that can lead to severe clinical cases, and sometimes, deaths [31]. It appeared in developed countries, like Réunion Island, in 2005 and 2006, in Italy and India, in 2007, and recently in Congo-Brazzaville, in 2011. Two cases have also been reported in September 2010 in the South-East of France. The principal vector of the Chikungunya in Réunion Island and in Italy is Aedes albopictus (sometimes called the Asian tiger because it originated from Asia and it is an agressive mosquito), which is also a prospective vector for Dengue transmission. In a recent period, there has been a tremendous
progress in our knowledge about the vector and the
relationships between the virus and the vector (see, for
instance, [12], [13], [14], [15], [28], [35], [36]). One of
the first models for the Chikungunya epidemic of 2005-
2006 in Réunion Island was proposed in [18]. The focus
in [19] was on the study on chemical and mechanical
tools available to stop or to control an epidemic, where it
is shown that the combination of Deltamethrin, the only
authorized adulticide in the European Union, and me-
chanical control, which consists in reducing the breeding
sites, could have been useful to stop the huge epidemic
of 2006. Recently, a study has been done on the Sterile
Insect Technique as a potential vector control tool for
the Chikungunya Disease [23].

Another very important point is that two strains of
the virus were isolated in Réunion Island. The first one,
strain 05.115, was isolated in May 2005, during the first
outbreak, and the second one, strain 06.21, was isolated
later, mid November 2005 (in fact we don’t know exactly
when the mutation happened). Vazeille et al. proved that
strain 06.21 had a larger rate of transmission from human
to mosquito [35]. In [18], the authors were the first to
take into account this assumption. Their numerical simu-
lations showed that strain 06.21 was certainly responsible
of the explosive epidemic from 2006. Moreover strain
06.21 had a direct impact on the lifespan of infected
mosquitoes [28]. It is an unusual assumption (usually,
in vector-borne disease models, the mean mortality rate
of the mosquito, in the different epidemiological states,
is assumed to be constant) that makes the theoretical
analysis of the model more difficult [19], [23]. In this
paper, we will only consider one strain, to simplify the
analysis.

Since a couple of years, metapopulation models have
been studied a lot, in particular to understand the dynam-
ics of infectious diseases [7], [26]. In [26], the authors
have revisited how metapopulation processes operate at
various spatial scales (individual level, local, and
regional epidemics). They have illustrated the resultant
spatio-temporal dynamics by a series of case studies
which explore diseases metapopulation dynamics at the
interface of models and data. However, the mathematical
analysis of the model (existence and stability of equilib-
ria) has not been done in their studies. More recently,
metapopulation or patch models have been applied to
Malaria disease [8], [24] indicating clearly that human
population movement is an important component to
understand the time course of an epidemic.

In [18], [19], the studies only focused on local places,
in order to detect where the epidemiological risk is high,
in other words, where the basic reproduction number
is greater than one. In recent works [21], [22], [16],
[17], spatio-temporal models, using partial differential
equations, have been developed to study mosquito dis-
placements according to landscape elements. In [17],
the authors took into account environmental factors, like
temperature, to study different SIT control strategies,
taking into account periodic releases of sterile males.

The aim of this work is now to link the cities, taking
into account human movements, that could explain the
spread of the disease. For instance, in Réunion Island,
in 2005, the first Chikungunya case was referenced in
Saint-Pierre, the 22th of February 2005, the next in Saint-
Denis and, then in La Possession, and Le Port.... Using
temporal (and even spatio-temporal) data from the ARS
(Regional French Health agency) in Réunion Island, we
know about the time and spatial spread of the epidemic.
In Réunion Island, the car is the favorite transportation
and people travel a lot from place to place in the island
to go from Home to work and back or to visit family or
friends. Moreover, it is well known that due to arthritis
many infected and even recovered (not infectious) people
were not able to move [31]. Thus, in our model, we
intend to take into account limitation movements of
infected populations and show that it can have an impact
in the spread and the force of the epidemic. Of course,
a possible and nice extension of our model would be
to consider the different Islands in Indian Ocean, like
Mauritius, la Réunion, Comoros, and Madagascar as a
possible 4 patches model.

The outline of the paper is as follows: in section 2, we
present the migration model and the full epidemiological
model for n cities. In section 3, we compute the DFE,
and the general basic reproduction number, \( R_0 \), and
show that the DFE is locally asymptotically stable (LAS).
Then we show that the DFE can sometimes be globally
asymptotically stable. In section 4, we study the spread-
ing of the disease. Finally, we validate our theoretical
results with a two patches model.

A. The human migration model

Our study focus on four cities in Réunion Island, but
we present the migration model in a general setting, in
order to have a generic modeling. All cities are more or
less connected, and, in principle, people can move from
one town to another. We assume that the total population
is constant. In Figure 1 we present an example of a n-
patches model: each city is a patch. In this figure, the
solid line stand from one city to another which means
that there is a strong connection between the cities, while
the dotted line is from one city to another which means that the connection is weak.

Fig. 1. A general n-patches model for the transmission of the Chikungunya virus between n cities in Réunion Island.

Assuming that the total human population in each patch is denoted by \( N_{i,h} \), we have \( N_{i,h} = S_{i,h} + I_{i,h} + R_{i,h} \), for \( i = 1, ..., n \). Moreover, the total population \( N_{tot} \) verifies \( N_{tot} = N_{1,h} + \cdots + N_{i,h} + \cdots + N_{n,h} \).

For each epidemiological states, we consider the following migration model:

\[
\frac{dX_{i,h}}{dt} = \sum_{j=1 \atop j \neq i}^{n} m_{ij} X_{j,h} - \sum_{j=1}^{n} m_{ji} X_{i,h},
\]

where \( X_{i,h} \in \{S_{i,h}, I_{i,h}, R_{i,h}\} \). Now, setting \( X_h = (X_{1,h}, \ldots, X_{n,h})^T \), the migration model (1) becomes

\[
\frac{dX_h}{dt} = \mathcal{M}^X_h X_h,
\]

where \( \mathcal{M}^X_h = \begin{pmatrix} \mathcal{M}_{1,1} & \cdots & \mathcal{M}_{1,n} \\ \vdots & \ddots & \vdots \\ \mathcal{M}_{n,1} & \cdots & \mathcal{M}_{n,n} \end{pmatrix} \), with

\[
\mathcal{M}_{1,1} = \begin{pmatrix} - \sum_{j=2}^{n} m_{1,j} & m_{1,2} \\ m_{2,1} & - \sum_{j=2 \atop j \neq 2}^{n} m_{2,j} \end{pmatrix},
\]

\[
\mathcal{M}_{1,n} = \begin{pmatrix} m_{1,n-1} & m_{1,n} \\ m_{2,n-1} & m_{2,n} \end{pmatrix},
\]

\[
\mathcal{M}_{n,1} = \begin{pmatrix} m_{n-1,1} & m_{n-1,2} \\ m_{n,1} & m_{n,2} \end{pmatrix}.
\]

The coefficients \( m_{i,j}^X \) are chosen according to the movement of the epidemiological state \( X_h \) between the cities and with the constraints that a possible equilibrium of the total population \( N_{tot} \) corresponds to the inhabitants in each city.

We point out that \( \mathcal{M}^X_{h} \) is a Metzler matrix, i.e. a matrix with all off-diagonal terms nonnegative, and is irreducible [9]. Thus, if \( X_h(0) \in \mathbb{R}^n_+ \), then \( X_h(t) \in \mathbb{R}^n_+ \). Indeed, there exist a real \( s > 0 \) and a matrix \( B \geq 0 \) such that

\[
-\mathcal{M}^X_h = s I_{n \times n} - B,
\]

which implies that \( -\mathcal{M} \) is a singular irreducible \( n \times n \) matrix of order \( n \). Thus, following Theorem 4.16, page 156 in [9], there exists a positive vector \( L_h >> 0 \) such that

\[
\mathcal{M}^X_h L = 0 \quad \text{with} \quad \sum_{i=1}^{n} L_{i,h} = 1.
\]

From Perron-Froebenius theorem [9], we deduce that there exists a simple eigenvalue \( \lambda_{\text{max}} = \max_{\lambda \in \text{spec}(\mathcal{M}^X_h)} \Re(\lambda) \) and an eigenvector \( \omega_h >> 0 \) such that

\[
\mathcal{M}^X_h \omega_h = \lambda_{\text{max}} \omega_h.
\]

Now, using the fact that \( 1^T \mathcal{M}^X_h = 0 \), where \( 1^T = (1, 1, \ldots, 1) \), we have \( \lambda_{\text{max}} = 0 \), and thus all other eigenvalues have a negative real part. Thus, the result hold.

\textit{Proposition 1:} Equation (2) admits a unique positive equilibrium \( X_h^0 = X_{tot}^0 \) which is globally asymptotically stable on the hyperplane orthogonal to \( 1^T \).

\textbf{B. The full epidemiological model for n cities.}

For each city we have temporal data, and some of them have been studied independently in Refs. [13], [19]. Our aim is to consider an epidemiological model in each patch and to take into account human movement between the patches. In patch \( i \), we assume that the human population is constant and equal to \( N_{i,h} \), and is subdivided in three compartmental stages: the susceptible, \( S_{i,h} \), the infected \( I_{i,h} \), and the recovered, \( R_{i,h} \). \( \mu_{i,h} \) is the per capita death rate of humans in susceptible, infectious and recover stages in patch \( i \); this parameter
is also assumed to be the recruitment rate of humans in the susceptible compartment stage, proportionally to the human population. In a same manner, we assume that all patches have got mosquitoes and, we consider three stages for the mosquitoes: an aquatic stage, $A_{i,m}$, the susceptible, $S_{i,m}$, and the Infected $I_{i,m}$. $K_i$ is the carrying capacity of all breeding sites, and $\mu_{i,h}$ is the number of eggs layed per day and per (female) mosquito, in patch $i$. An infected mosquito in patch $i$ can only infect a susceptible human from patch $i$. In each patch, we assume that mosquitoes and humans are homogeneously distributed. The aquatic state includes the eggs, larvae and pupae. Both humans and mosquitoes are assumed to be born susceptible. $\eta_{i,h}$ is the recovering rate of infected human in patch $i$ such that an infected human is infectious during $\frac{1}{\eta_{i,h}}$ days, called the viremic period, and then becomes resistant or immune. The parameter $\alpha_i$ is related to the carrying capacity and represents the level of mechanical control in patch $i$: when $\alpha_i = 1$, there is no mechanical control; when $\alpha_i = 0.5$, it indicates that 50 percent of the breeding sites have been removed in patch $i$. $\mu_{i,A}$ is the per capita death rate of mosquitoes in aquatic stage in patch $i$; $\mu_{i,m}$ is the per capita death rate of mosquitoes in susceptible and infectious stages in patch $i$; $\eta_{i,A}$ is the rate of mosquitoes of patch $i$. An infected mosquito in patch $i$ can only infect a susceptible individual in patch $i$ when $\beta_{i,hm} = B_{i} p_{i,hm}$ is the contact rate between infectious mosquitoes and susceptible hosts. Similarly, $\beta_{i,hm} = B_{i} p_{i,hm}$ is the contact rate between infectious mosquitoes and susceptible hosts, where $p_{i,hm}$ is the probability that a bite on an infected individual will lead to vector infection.

In Réunion Island, 80% of the population being living at the sea level, we assume that the parameters in the human compartments are the same in each patch (at least from Saint-Denis to Saint-Pierre). In patch $i$, we assume that the average lifespan for susceptible and infected mosquitoes is $1/\mu_{i,m}$. Let us recall also, that in Réunion Island, it was proved that a mutation in the initial strain leads to a new strain that influences the lifespan of the infected mosquito: it is almost halved [28]. This uncommon result can influence the dynamics of the disease [19]. Here, for sake of simplicity in the analysis, we will only consider one strain (no mutation). For other vector-borne diseases it has never been observed that a mutation in the virus influences the lifespan of an infected mosquito. There is no evidence of vertical transmission [36]. We also assume that the mosquito parameters may change from patch to patch.

Note also that we don’t consider the “exposed” stage, like in [18], [19], for sake of simplicity. All together, for $i = 1, ..., n$, we have the following system for the mosquitoes population:

$$\begin{aligned}
\frac{dA_{i,m}}{dt} &= \mu_{i,b} \left( 1 - \frac{A_{i,m}}{\alpha_i K_i} \right) (S_{i,m} + I_{i,m}) \\
&\quad - (\eta_{i,A} + \mu_{i,A}) A_{i,m}, \\
\frac{dS_{i,m}}{dt} &= -\beta_{i,hm} \left( \frac{I_{i,h}}{N_{i,h}} \right) S_{i,m} - \mu_{i,m} S_{i,m} \\
&\quad + \eta_{i,A} A_{i,m}, \\
\frac{dI_{i,m}}{dt} &= \beta_{i,hm} \left( \frac{I_{i,h}}{N_{i,h}} \right) S_{i,m} - \mu_{i,m} I_{i,m},
\end{aligned}$$

and the following differential system for the human population:

$$\begin{aligned}
\frac{dS_{i,h}}{dt} &= \mu_{i,h} N_{i,h} - \beta_{i,mh} \frac{I_{i,m}}{N_{i,h}} S_{i,h} - \mu_{i,h} S_{i,h} \\
&\quad + \sum_{j=1}^{n} m_{ij} s_j S_{j,h} - \left( \sum_{j=1}^{n} m_{ij}^s \right) S_{i,h}, \\
\frac{dI_{i,h}}{dt} &= \beta_{i,mh} \frac{I_{i,m}}{N_{i,h}} S_{i,h} - (\mu_{i,h} + \eta_{i,h}) I_{i,h} \\
&\quad + \sum_{j=1}^{n} \gamma_{ij} m_{ij}^l I_{j,h} - \gamma_i \left( \sum_{j=1}^{n} m_{ij}^l \right) I_{i,h}, \\
\frac{dR_{i,h}}{dt} &= \eta_{i,h} I_{i,h} - \mu_{i,h} R_{i,h} + \sum_{j=1}^{n} m_{ij}^R R_{j,h} \\
&\quad - \left( \sum_{j=1}^{n} m_{ij}^R \right) R_{i,h},
\end{aligned}$$

where $S_{i,h} + I_{i,h} + R_{i,h} = N_{i,h}$. In addition, we have the following initial conditions in each patch: $(\alpha_i K_i; m_i N_{i,h}; 0; N_{i,h} - I_{i,h}^0; I_{i,h}^0; 0)$, where $I_{i,h}^0$ is the initial number of infected people in patch $i$, and $m_i$ a positive real number. In numerical simulations, we will consider that $K_i = k_i N_{i,h}$, where $k_i$ is a positive real number.

Setting $S_h = (S_{1,h}, ..., S_{n,h})$, $I_h = (I_{1,h}, ..., I_{n,h})$, $R_h = (R_{1,h}, ..., R_{n,h})$, $A_m = (A_{1,m}, ..., A_{n,m})$, $S_m = (S_{1,m}, ..., S_{n,m})$ and $I_m = (I_{1,m}, ..., I_{n,m})$. 


In the sequel, we will assume that the migration models for the epidemiological states \( S_h \) and \( R_h \) are the same, i.e. \( \mathcal{M}^{S_h} = \mathcal{M}^{R_h} = \mathcal{M} \). However, Chikungunya fever is general symmetric with joint pains that occur in wrists, elbows, fingers, knees,..., leading sometimes to arthritis [32] such that it can be very difficult to drive and thus, going from one city to another and cannot always be possible for infected people. Thus, we will assume that \( \mathcal{M}^{R_h} = \mathcal{M} \Gamma \), with \( \Gamma = \text{diag}(\gamma_i) \), where \( \gamma_i \in [0, 1] \); \( \gamma_i \) indicates the proportion of infected people that were able to move from patch \( i \) to the other patches. We could also assume that \( \mathcal{M}^{R_h} = \mathcal{M} \Gamma \) too, since this joint problems can persist several weeks or months after the people had become viremic, but this hypothesis do not change the rest of the paper mainly because recovered people do not become susceptible again. So for sake of simplicity, we keep \( \mathcal{M}^{R_h} = \mathcal{M} \) as assumed previously.

Remark 1: Our migration model doesn’t take into account the home of the individuals, which would imply a far more complex model. In our modelling, we don’t take into account people that moves daily, for instance from home to work and back. Indeed, a. albopictus is only activ early in the morning and late in the afternoon, thus more or less outside the office hours in Réunion island. Thus, we only consider people that stay more than one day, and thus have more or less the same probability than local people to be bitten. This is why we don’t make distinction in a patch between people coming from different patches.

Therefore, in terms of \( S_h, I_h, R_h, A_m, S_m \) and \( I_m \), the differential equations (3) and (4) can be rewritten in the following vectorial form:

\[
\begin{aligned}
\frac{dA_m}{dt} &= \text{diag}(\mu_b) \text{diag}(K)^{-1} \text{diag} \left( K - \frac{A_m}{\alpha} \right) S_m + \text{diag}(\mu_b) \text{diag}(K)^{-1} \text{diag} \left( K - \frac{A_m}{\alpha} \right) I_m \\
&\quad - \text{diag}(\eta_A + \mu_A) A_m, \\
\frac{dS_m}{dt} &= -\text{diag}(N_h)^{-1} \text{diag}(\beta_{hm} I_h) S_m - \text{diag}(\mu_m) S_m + \text{diag}(\eta_A) A_m, \\
\frac{dI_m}{dt} &= \text{diag}(N_h)^{-1} \text{diag}(\beta_{hm} I_h) S_m - \text{diag}(\mu_m) I_m,
\end{aligned}
\]

and

\[
\begin{aligned}
\frac{dS_h}{dt} &= \mu_h I_n (I_h + R_h) - \text{diag}(N_h)^{-1} \text{diag}(\beta_{mh} I_m) S_h + \mathcal{M} S_h, \\
\frac{dI_h}{dt} &= \text{diag}(N_h)^{-1} \text{diag}(\beta_{mh} I_m) S_h - (\eta_h + \mu_h) I_n I_h + \mathcal{M} I_h, \\
\frac{dR_h}{dt} &= \eta_h I_n I_h - \mu_h I_n R_h + \mathcal{M} R_h,
\end{aligned}
\]

where \( \mu_b = (\mu_{1b}, \cdots, \mu_{nb})^T, K = (K_1, \cdots, K_n)^T, \alpha = (\alpha_1, \cdots, \alpha_n)^T, \eta_A = (\eta_{1A}, \cdots, \eta_{nA})^T, \mu_A = (\mu_{1A}, \cdots, \mu_{nA})^T, \beta_{hm} = (\beta_{1hm}, \cdots, \beta_{nhm})^T, \mu_m = (\mu_{1m}, \cdots, \mu_{nm})^T \) and \( \text{diag}(Y) \) denotes the diagonal matrix of order \( n \) defined by the vector \( Y \) of \( n \).

Summing sub-systems (5), (6), and (7) gives

\[
\begin{aligned}
\frac{dN_h}{dt} &= \mathcal{M} S_h + \mathcal{M} \Gamma I_h + \mathcal{M} R_h = \mathcal{M} N_h - \mathcal{M} (I_d_n - \Gamma) I_h.
\end{aligned}
\]

Then, the coupled system (5)-(7) may be rewritten in the following compact form:

\[
\begin{aligned}
\frac{dV_m}{dt} &= \mathcal{A}(H,V_m) V_m, \\
\frac{dH}{dt} &= \mathcal{B}(H,V_m) H,
\end{aligned}
\]

where

\[
\begin{aligned}
H &= (S_{1h}, \cdots, S_{nh}, I_{1h}, \cdots, I_{nh}, R_{1h}, \cdots, R_{nh})^T, \\
V_m &= (A_{1m}, \cdots, A_{nm}, S_{1m}, \cdots, S_{nm}, I_{1m}, \cdots, I_{nm})^T,
\end{aligned}
\]

\[
\mathcal{B}(H,V_m) = \begin{pmatrix}
-b_{11} & \mu_h I_d_n & \mu_h I_d_n \\
-b_{21} & -b_{22} & 0 \\
0 & \eta_h I_d_n & (-\mu_h I_d_n - \mathcal{M})
\end{pmatrix},
\]

with \( b_{11} = (\text{diag}(N_h)^{-1} \text{diag}(\beta_{mh} I_m) - \mathcal{M}), b_{21} = (\text{diag}(N_h)^{-1} \text{diag}(\beta_{mh} I_m), b_{22} = ((\eta_h + \mu_h) I_d_n - \Gamma) \mathcal{M},
\]

\[
\mathcal{A}(H,V_m) = \begin{pmatrix}
-A_{11} & \text{diag}(\mu_b) & \text{diag}(\mu_b) \\
\text{diag}(\eta_A) & -A_{22} & 0 \\
0 & a_{32} & -\text{diag}(\mu_b)
\end{pmatrix},
\]

where \( I_d_n \) denote the identity matrix of \( n \), \( A_{11} = (\text{diag}(\mu_b) \text{diag}(\alpha K)^{-1} (S_m + I_m) + \text{diag}(\eta_A + \mu_A)) \) and \( A_{22} = (\text{diag}(N_h)^{-1} \text{diag}(\beta_{hm} I_h) + \text{diag}(\mu_m)), a_{32} = (\text{diag}(\beta_{hm}) \text{diag}(N_h)^{-1} \text{diag}(I_h).
\]

Note that \( \mathcal{A}(H,V_m) \) and \( \mathcal{B}(H,V_m) \) are Metzler matrices for all \( V_m \in \mathbb{R}^{3n} \) and all \( H \in \mathbb{R}^{3n} \). Thus, system (8) is positively invariant in \( \mathbb{R}^{3n} \times \mathbb{R}^{3n} \), which means
that any trajectory of the system starting from an initial state in the positive orthant \( \mathbb{R}^3_+ \times \mathbb{R}^3_+ \) remains forever in \( \mathbb{R}^3_+ \times \mathbb{R}^3_+ \). Note also that the right-hand side of system (8) being Lipschitz continuous so that there exists a unique maximal solution.

Now, let us show that the solutions are bounded. Using equation (7) and the fact that \( \Gamma \leq I \) gives

\[
\frac{dN_i}{dt} \leq \mathcal{M} N_i.
\]

Since \( \sum_{i=1}^n \mathcal{M}_{ij} = 0 \), for all \( j \), the total population \( N_{tot} = \sum_{i=1}^n N_{i,h} \) in the full system is bounded by the initial total population \( N^0_{tot} = N^0_{1,h} + \cdots + N^0_{n,h} \), which implies that \( N_{i,h} \), the total population in a given patch \( i \), is also bounded by \( N^0_{i,h} \). Thus, \( S_i, I_i \) and \( R_i \) are such that \( (S_i, I_i, R_i) \leq (I_{d,h}, I_{d,h}, I_{d,h})N^0_{i,h} \), where \( N^0_{i,h} = (N^0_{1,h}, \ldots, N^0_{3,h})^T \).

In each patch, the basic offspring number related to the mosquitoes population is defined by

\[
N_{i,m} = \frac{\mu_{i,b} \eta_{i,A}}{(\mu_{i,A} + \mu_{i,m})}. \tag{9}
\]

Without infectious mosquitoes and infectious humans, the mosquito dynamical system in each path \( i \) reduces to

\[
\begin{aligned}
\frac{dA_{i,m}}{dt} &= \mu_{i,b} \left(1 - \frac{A_{i,m}}{a_i K_i}\right) S_{i,m} - \left(\eta_{i,A} + \mu_{i,m}\right) A_{i,m}, \\
\frac{dS_{i,m}}{dt} &= -\mu_{i,m} S_{i,m} + \eta_{i,A} A_{i,m}.
\end{aligned} \tag{10}
\]

System (10) has two equilibria \( E_0 = (0,0) \) and, when \( N_{i,m} > 1 \), \( E^* = (A^*_0, S^*_0) \), with

\[
A^*_0 = \left(1 - \frac{1}{N_{i,m}}\right) a_i K_i, \quad S^*_0 = \frac{\eta_{i,A}}{\mu_{i,m}} \left(1 - \frac{1}{N_{i,m}}\right) a_i K_i.
\]

In fact, using (9), we can show the following

**Theorem 1:** Let \( A_{i,m}(0) \leq a_i K_i \), then the following results hold

1) System (10) defines a cooperative dissipative dynamical system on \( \mathbb{R}^2_+ \).

2) If \( N_{i,m} \leq 1 \), then the equilibrium \( E_0 \) is globally asymptotically stable on \( \mathbb{R}^2_+ \).

3) If \( N_{i,m} > 1 \), then system (10) has two equilibria \( E_0 \) and \( E^* \), where \( E_0 \) is unstable and \( E^* \) is stable with basin of attraction \( \mathbb{R}^2_+ \setminus \{E_0\} \).

**Proof:** It suffices to verify the assumptions of Theorem 5 given in Annex A (see also [5]) with \( a = E_0 \) and \( b = \left(a_i K_i, 2 \frac{\alpha_i \eta_{i,A}}{\mu_{i,m}} K_i\right) \). \( \square \)

Finally, in patch \( i \), from equations (5) and (6), we derive

\[
\begin{aligned}
\dot{A}_{i,m} &= \mu_{i,b} \left(1 - \frac{A_{i,m}}{a_i K_i}\right) S_{i,m} - \left(\eta_{i,A} + \mu_{i,m}\right) A_{i,m}, \\
\dot{S}_{i,m} &= \eta_{i,A} A_{i,m} - \mu_{i,m} (I_{i,m} + S_{i,m}).
\end{aligned}
\]

Then, straightforward computations show that in each patch \( i \), we have

\[
A_{i,m}(t) \leq \bar{A}_m = \max\left(A_{i,m}(0), \alpha_i K_i\right),
\]

\[
S_{i,m}(t) + I_{i,m}(t) \leq \max\left(S_{i,m}(0) + I_{i,m}(0), \eta_{i,A} A_{i,m}\right).
\]

Therefore, the following theorem hold.

**Theorem 2:** Setting \( H = (S_h, I_h, R_h) \) and \( V_m = (A_m, S_m, I_m) \). System (5) and (6) is invariant in \( B = \{ (V_m, H) \in \mathbb{R}^3_+ \times \mathbb{R}^3_+ | S_h + I_h + R_h = N_h, \ A_m \leq \bar{A}_m, \ S_m + I_m \leq \max\left(S_m(0) + I_m(0), \eta_{i,A} A_{i,m}\right) \} \)

II. DISEASE-FREE EQUILIBRIUM AND BASIC REPRODUCTION NUMBER

A. The disease-free equilibrium

We consider systems (5) and (6) together. At the disease-free equilibrium (DFE), \( I_h = 0 \) and \( I_0 = 0 \). Then, system (5) and (6) at the DFE gives

\[
\begin{aligned}
-\mu_h N^0_h &= (-\mu_h I_d + \mathcal{M}) S^0_h, \\
(-\mu_h I_d + \mathcal{M}) R^0_h &= 0, \\
\text{diag}(\mu_h) \text{diag}(K)^{-1} \text{diag} \left( K - \frac{A^0_m}{\alpha} \right) S^0_m &= \text{diag}(\eta_A + \mu_A) A^0_m, \\
\mu_h S^0_m &= \eta_A A^0_m.
\end{aligned} \tag{12}
\]

- Using Proposition 1 and choosing \( S^0_n = N^0_n \), Eq. (12) is verified.

- Equation (12) implies that \( R^0_h = 0 \). Also, from Eq. (12) one has

\[
\text{diag}(\mu_h) \text{diag}(K)^{-1} \text{diag} \left( K - \frac{A^0_m}{\alpha} \right) S^0_m = \text{diag}(\eta_A + \mu_A) A^0_m.
\]

Since \( \mu_i, S^0_{i,m} = \eta_{i,A} A^0_{i,m} \), for each \( i \), the above equation becomes

\[
\text{diag}(\mu_h) \text{diag}(K)^{-1} \text{diag} \left( K - \frac{A^0_m}{\alpha} \right) \text{diag}(\eta_A) \times (\text{diag}(\mu_h))^{-1} A^0_m = \text{diag}(\eta_A + \mu_A) A^0_m.
\]
Then, in each patch, there are two possibilities: $A^0_{i,m} = S^0_{i,m} = 0$, or $A^0_{i,m} > 0$ and $S^0_{i,m} > 0$, depending on the value taken by the basic offspring number $N_{i,m}$ (see the previous computations above).

Finally, we have the following result.

**Proposition 2:** Let consider the coupled system (3)-(4).

- There always exists an Equilibrium without disease, $E_{DF}$, depending on the threshold $N_{i,m}$ in each patch.
- When $N_{n,m} = (N_{i,m})_{i=1,...,n} > 1_n$, then we call DFE, the Disease Free Equilibrium, $(S^0_{n}, 0, A^0_{m}, S^0_{m}, 0)$, where $A^0_{m} > 0$ and $S^0_{m} > 0$.

### B. The basic reproduction number

Let us now compute a general expression related to an equilibrium without disease. We will consider system (5)-(6), without equation $dH_{i,m}/dt$, because the human population is constant. The expressions which coming from the other compartments due to the contamination are those in $dH_{i,m}/dt$ and $dI_{i,m}/dt$, that is

\[
\begin{align*}
\frac{dH_{i,m}}{dt} &= \text{diag}(\beta_{mh})\text{diag}(N_{h})^{-1}\text{diag}(I_{m})S_{h} - \text{diag}(\eta_{h} + \mu_{h})I_{h} + M\Gamma I_{h}, \\
\frac{dI_{i,m}}{dt} &= \text{diag}(\beta_{hm})\text{diag}(N_{h})^{-1}\text{diag}(I_{h})S_{m} - \text{diag}(\mu_{m})I_{m}.
\end{align*}
\]

The above equation can be rewritten as follows:

\[
\begin{align*}
\frac{dH_{i,m}}{dt} &= \text{diag}(\beta_{mh})\text{diag}(N_{h})^{-1}\text{diag}(S_{h})I_{m} - \text{diag}(\eta_{h} + \mu_{h})I_{h} + M\Gamma I_{h}, \\
\frac{dI_{i,m}}{dt} &= \text{diag}(\beta_{hm})\text{diag}(N_{h})^{-1}\text{diag}(S_{m})I_{h} - \text{diag}(\mu_{m})I_{m},
\end{align*}
\]

Here we consider a general equilibrium without disease such that $S_{m} \geq 0$, such that some components could be equal to zero. We compute the Jacobian of the system at a nonnegative equilibrium, without disease, $E_{DF} = (A^0_{m}, S^0_{m}, 0, N^0_{h}, 0, 0)$, which leads to

\[J_M = F - V,\]

and

\[V = \begin{pmatrix} (\eta_{h} + \mu_{h})I_{d_{n}} - M\Gamma & 0_{n \times n} \\ 0_{n \times n} & \text{diag}(\mu_{m}) \end{pmatrix},\]

which is invertible. Then, the next generation matrix is:

\[FV^{-1} = \begin{pmatrix} 0_{n \times n} & f_{12} \\ f_{21} & 0_{n \times n} \end{pmatrix},\]

where $f_{12} = \text{diag}(\beta_{mh})(\text{diag}(\mu_{m}))^{-1}$, $f_{21} = \text{diag}(\beta_{hm})\text{diag}(N^0_{h})^{-1}\text{diag}(S^0_{m})\times((\eta_{h} + \mu_{h})I_{d_{n}} - M\Gamma)^{-1}$.

The basic reproduction number related to $E_{DF}$ is the spectral radius of the next generation matrix, i.e. $R^2_0 = \rho(FV^{-1})$ (34). After a brief computation, we obtain

\[R^2_0 = \rho(\text{diag}(\beta_{mh}\beta_{hm})(\text{diag}(\mu_{m}))^{-1}\text{diag}(N^0_{h})^{-1}\times\text{diag}(S^0_{m})((\eta_{h} + \mu_{h})I_{d_{n}} - M\Gamma)^{-1}).\]  

(13)

$R^2_0$ is also the general basic reproduction number related to the whole system. Using (34), we have the following result.

**Theorem 3:** If $R^2_0 < 1$, then $E_{DF}$ is locally asymptotically stable. If $R^2_0 > 1$, then $E_{DF}$ is unstable.

**Remark 2:** If $N_{m} < 1_n$, then only the infection free equilibrium (IFE) $E_0 = (S^0_{h}, 0, 0, 0, 0, 0)$ exists and is globally asymptotically stable. If $N_{m} > 1_n$, the IFE still exists but is unstable and the DFE $E^*$ exists. This DFE as we have shown in Theorem 1 is stable. This means that at $N_{m} = 1_n$, we have a transcritical bifurcation.

**Remark 3:** When $\Gamma = 0$, then

\[R^2_0 = \max_i (R^2_{0,i}),\]

where $R^2_{0,i}$ is the basic reproduction number in patch $i$, and is defined as follows (23):

\[R^2_{0,i} = \frac{\beta_{i,mh}\beta_{i,hm}S^0_{i,m}}{\mu_{i,m}(\eta_{h} + \mu_{h})N^0_{i,h}}.\]  

(14)

From the previous computations, we are able to derive interesting results, in particular for the vector control. A first general and obvious result is that when the migration increases, the basic reproduction number also increases.

Indeed, $(\eta_{h} + \mu_{h})I_{d_{n}} - M\Gamma$ being an $M -$ matrix, its inverse $((\eta_{h} + \mu_{h})I_{d_{n}} - M\Gamma)^{-1}$ is a positive matrix.
Moreover
\[
\begin{align*}
\text{diag} \left( N^0_h \right)^{-1} \text{diag} \left( S^0_m \right) &= \begin{pmatrix}
\frac{S^0_{1,m}}{N^0_{1,h}} \\
\frac{S^0_{n,m}}{N^0_{n,h}} \\
\vdots
\end{pmatrix} \\
&\leq \max_{1 \leq i \leq n} \left( \frac{S^0_{i,m}}{N^0_{i,h}} \right) I_d_n.
\end{align*}
\]

Thus, we have
\[
\begin{align*}
\text{diag} \left( \frac{\beta_{m,h} \beta_{h,m}}{\mu_m} \right) \text{diag} \left( N^0_h \right)^{-1} \text{diag} \left( S^0_m \right) \\
\times \left( (\eta_h + \mu_h) I_d_n - \Lambda \Gamma \right)^{-1} \\
&\leq \max_{1 \leq i \leq n} \left( \frac{\beta_{i,mh} \beta_{h,mh} S^0_{i,m}}{\mu_i \mu_m N^0_{i,h}} \right) \left( (\eta_h + \mu_h) I_d_n - \Lambda \Gamma \right)^{-1}.
\end{align*}
\]

Then using a nice property of positive matrices, we have
\[
\rho \left( \text{diag} \left( \frac{\beta_{m,h} \beta_{h,m}}{\mu_m} \right) \text{diag} \left( N^0_h \right)^{-1} \text{diag} \left( S^0_m \right) \right) \times \left( (\eta_h + \mu_h) I_d_n - \Lambda \Gamma \right)^{-1} \\
\leq \max_{1 \leq i \leq n} \left( \frac{\beta_{i,mh} \beta_{h,mh} S^0_{i,m}}{\mu_i \mu_m N^0_{i,h}} \right) \rho \left( \left( (\eta_h + \mu_h) I_d_n - \Lambda \Gamma \right)^{-1} \right)
\]

But \((\eta_h + \mu_h) I_d_n - \Lambda \Gamma\) being a non singular \(M\)-matrix, since the stability modulus of \(M\) is \(\alpha(M) = 0\), where \(\alpha(M) = \max \{\text{Re}(\lambda): \lambda \text{ eigenvalue of } M\}\), we deduce \(9\)

\[
\rho \left( \left( (\eta_h + \mu_h) I_d_n - \Lambda \Gamma \right)^{-1} \right) = \frac{1}{\eta_h + \mu_h},
\]

which implies, using \(14\), that
\[
R^2_0 \leq \max_i \left( R^2_{0,i} \right).
\]

Using the same reasoning it is possible to show that
\[
\min_i \left( R^2_{0,i} \right) \leq R^2_0.
\]

Altogether, we summarize in the following proposition

**Proposition 3:** The Basic reproduction Number of the patch system verifies
\[
\min_i \left( R^2_{0,i} \right) \leq R^2_0 \leq \max_i \left( R^2_{0,i} \right).
\]

Thus, human movements can induce a spreading of the epidemiological risk in places where local basic reproduction numbers are low, when some places have large local reproduction number, i.e. greater than 1.

Let us now consider a particular case of \(n\) patches with the same population \(N^0_{i,h} = N_{1,h}\), the same capacity \(K_i = K_1\), and the same parameters values in each patch, for \(i = 2, \ldots, n\), such that \(N_{i,m} > 1\). Then we have the same equilibrium for the susceptible mosquito population, i.e. \(S^0_{i,m} = S^0_{1,m}\), for \(i = 2, \ldots, n\). Thus the basic reproduction number reduces to
\[
R^2_0 = \rho \left( \text{diag} \left( \frac{\beta_{m,h} \beta_{h,m}}{\mu_m} \right) \text{diag} \left( N^0_h \right)^{-1} \text{diag} \left( S^0_m \right) \right) \times \left( (\eta_h + \mu_h) I_d_n - \Lambda \Gamma \right)^{-1},
\]

with
\[
\rho \left( \left( (\eta_h + \mu_h) I_d_n - \Lambda \Gamma \right)^{-1} \right) = \frac{1}{\eta_h + \mu_h},
\]

which implies that
\[
R^2_0 = R^2_{0,1} = R^2_{0,i} = \frac{\beta_{1,hm} \beta_{1,mh} S^0_{1,m}}{\mu_{1,m} (\eta_h + \mu_h) N^0_{1,h}}.
\]

In this particular case, human movements has no impact on the basic reproduction number. Thus, for cities of equal size, and with the same biological parameters whatever the migration, the global and local risks are the same. This unexpected result is due to the fact that \((\eta_h + \mu_h) I_d_n - \Lambda \Gamma\) has always the same spectral radius, \(\frac{1}{\gamma_1}\), whatever the matrices \(\Gamma\) and \(M\).

Let us now consider a two-patches example, with \(N_{1,m} > 1\) and \(N_{m,2} < 1\). Thus, after straightforward computations, the basic reproduction number becomes
\[
R^2_0 = \frac{\beta_{1,hm} \beta_{1,mh}}{\mu_{m} (\eta_h + \mu_h) (\eta_h + \mu_h + \gamma (m_2 + m_{21}))} \frac{S^0_{1,m}}{N^0_{1,h}}
\]

which leads to
\[
R^2_0 = R^2_{0,1} \left( 1 - \frac{m_{21}}{(\eta_h + \mu_h + \gamma (m_2 + m_{21}))} \right)
\]

indicating that if the infected population is in one patch, with infectious mosquitoes, has back and forth movement with another patch which is free of mosquitoes, then the basic reproduction ratio will decrease....In particular if all infected people go out from the infected area, this will lower the epidemiological risk....

**C. Global asymptotic stability of the DFE**

In this section, we study the global asymptotic stability (GAS) of the DFE of coupled system \(5\)–\(6\). We assume that the population in each patch is constant, i.e. \(N_{i,h} = N^0_{i,h}\). Set
\[
R^2_{\text{GAS}} = \rho \left( \text{diag} (\beta_{m,h} \beta_{h,m}) (\text{diag} (\mu_m))^{-1} \text{diag} \left( N^0_h \right)^{-1} \times \text{diag} (S^0) \left( (\eta_h + \mu_h) I_d_n - \Lambda \Gamma \right)^{-1} \right),
\]

where
with
\[ S_{\text{max}} = \max \left( S_m(0), S_m^0 \right). \]

We have the following result

**Theorem 4:** The DFE of the coupled system [5]–[6] is globally asymptotically stable in the nonnegative orthant, if \( R^2_{GAS} < 1 \).

**Proof:** Let us consider Eqs. [5] and [6]. Using the fact that \( S_h \) is bounded, i.e., \( S_h \leq S_h^0 \), and \( S_m < S_{\text{max}} \), we obtain the following linear differential inequations system:
\[
\begin{pmatrix}
\frac{dI_m}{dt} \\
\frac{dI_h}{dt}
\end{pmatrix} \leq \begin{pmatrix}
-\text{diag}(\mu_m) & g_{21} \\
\text{diag}(\beta_{mh}) & -g_{22}
\end{pmatrix} \begin{pmatrix}
I_m \\
I_h
\end{pmatrix},
\]
where
\[ g_{21} = \text{diag}(\beta_{hm})\text{diag}(N_h^0)^{-1} \text{diag}(S_{\text{max}}), \]
\[ g_{22} = (\text{diag}(\eta_h + \mu_h) - M\Gamma). \]
Let
\[ G = \begin{pmatrix}
-\text{diag}(\mu_m) & g_{21} \\
\text{diag}(\beta_{mh}) & -g_{22}
\end{pmatrix}. \]
Note that \( G \) is a Metzler matrix, which admits a regular splitting [7] (close similar to the regular splitting obtained to compute the basic reproduction number \( R^2_0 \)), \( N + M \), with
\[ M = \begin{pmatrix}
0 & \text{diag}(\beta_{hm})\text{diag}(N_h^0)^{-1} \text{diag}(S_{\text{max}}) \\
\text{diag}(\beta_{mh}) & 0
\end{pmatrix}, \]
and
\[ N = \begin{pmatrix}
-\text{diag}(\mu_m) & 0 \\
0 & -\text{diag}(\eta_h + \mu_h) - M\Gamma
\end{pmatrix}. \]
Thus, using [7], \( G \) is Metzler stable if \( \rho \left( -N^{-1}M \right) < 1 \).
A simple computation gives
\[ -N^{-1}M = \begin{pmatrix}
0 & nm_{12} \\
0 & 0
\end{pmatrix}, \]
where
\[ nm_{12} = \text{diag}(\beta_{hm})\text{diag}(N_h^0)^{-1} \text{diag}(S_{\text{max}}) \times \text{diag}(\mu_m)^{-1}, \]
\[ nm_{21} = (\text{diag}(\eta_h + \mu_h) - M\Gamma)^{-1} \text{diag}(\beta_{mh}). \]
Then, \( \rho \left( -N^{-1}M \right) < 1 \) if and only if
\[ R^2_{\text{GAS}} = \rho(\text{diag} \left( \frac{\beta_{mh}\beta_{hm}}{\mu_m} \right) \text{diag}(N_h^0)^{-1} \times \text{diag}(S_{\text{max}}) \left( \text{diag}(\eta_h + \mu_h)I_d - M\Gamma \right)^{-1}) < 1. \]
Thus, using a comparison principle [27], we have
\[ \lim_{t \to +\infty} I_h = \lim_{t \to +\infty} I_m = 0. \]
Then, having \( -(\mu_h I_d - M) \leq 0 \), we deduce that \( \lim_{t \to +\infty} R_h = 0 \). Since the total population in each patch is constant, and using the fact that \( S_h + I_h + R_h = N_h \), we deduce that \( \lim_{t \to +\infty} S_h = N_h \).
Obviously we have \( R^2_0 \leq R^2_{\text{GAS}} \). Thus since \( R^2_{\text{GAS}} < 1 \), we have uniqueness of the EDF, which implies that \( \lim_{t \to +\infty} A_m = A_m^0 \) and \( \lim_{t \to +\infty} S_m = S_m^0 \). Then, one can conclude that the EDF is GAS when \( R^2_{\text{GAS}} < 1 \). This achieves the proof.

**Remark 4:** When \( S_{\text{max}} = S_m^0 \), we have \( R^2_{\text{GAS}} = R^2_0 \).

**Remark 5:** Our result generalized to a metapopulation the results obtained in [18], [19], [23].

**III. SPREADING OF THE DISEASE**

First of all, when \( M \neq 0 \), we show with a simple example that having an endemic equilibrium in some patches and a Disease Free equilibrium in other patches, is impossible.

Indeed, we consider a two-patches system assuming that in the first patch \( R_{0,1} < 1 \) and in the second ones, \( R_{0,2} > 1 \). Looking for an equilibrium leads to the following systems to solve

(Patch 1)
\[
\begin{align*}
\mu_h N_{1,h} - \beta_{1,mh} I_{1,m}^I S_{1,h} \\
-\mu_h S_{1,h} + m_{12}^I S_{2,h} - m_{21}^S S_{1,h} = 0, \\
\beta_{1,mh} \frac{I_{1,m}^I}{N_{1,h}} S_{1,h} - (\mu_h + \eta_h) I_{1,h} \\
+ \gamma_2 m_{12}^I I_{2,h} - \gamma_1 m_{21}^I I_{1,h} = 0,
\end{align*}
\]

(Patch 2)
\[
\begin{align*}
\mu_h N_{2,h} - \beta_{2,mh} I_{2,m}^I S_{2,h} - \mu_h S_{2,h} \\
+ m_{21}^I S_{1,h} - m_{21}^S S_{2,h} = 0, \\
\beta_{2,mh} \frac{I_{2,m}^I}{N_{2,h}} S_{2,h} - (\mu_h + \eta_h) I_{2,h} \\
+ \gamma_1 m_{21}^I I_{1,h} - \gamma_2 m_{12}^I I_{2,h} = 0.
\end{align*}
\]

From (Patch 1)1, we deduce
\[ \gamma_2 m_{12}^I I_{2,h} = (\mu_h + \eta_h + \gamma_1 m_{21}^I) I_{1,h} - \beta_{1,mh} \frac{I_{1,m}^I}{N_{1,h}} S_{1,h}. \]
(15)

We assume \( \gamma_1 > 0 \). Since \( R_{0,1} < 1 \) in the first patch, then one possible equilibrium verifies \( I_{1,h} = I_{1,m} = 0 \). Using (15)2, we have immediately \( I_{2,h} = 0 \) in the second patch. Then using the fact that
\[ \beta_{2,mh} \frac{I_{2,m}^I}{N_{2,h}} S_{2,m} = \mu_{2,m} I_{2,m}, \]
we deduce that \( I_{2,m} = 0 \). Thus, assuming movements in the infectious population implies automatically that
we cannot have co-existence of an endemic equilibrium in one patch and a disease free equilibrium in the other one. Therefore, an endemic equilibrium for the patchy system is an equilibrium without zero component.

Existence of an endemic equilibrium is not obvious to show in a metapopulation model. However, it is possible to have some insights in the dynamic of the disease, in particular regarding its spreading. Our preliminary results allows us to conjecture that a unique endemic equilibrium exists. Let us now state this result for one patch.

Proposition 4: Consider system (5)-(6).

1) When $\mathcal{M} = 0$, a unique endemic equilibrium exists, if $R_{0,i}^2 > 1$ in each patch $i$.

2) When $\mathcal{M} \neq 0$, the only equilibrium with one zero component is $E_{DF}$.

Proof: Let us first show the existence of a possible equilibrium. Then, using some of the computations derived in [19], we can derive explicit solution for the endemic equilibrium... Let us consider system (3)-(4) or system (5)-(6) at the endemic equilibrium. Then, using some of the computations derived in [20], we can derive explicit solution for the endemic equilibrium, using fix point argument. We will use Theorem 2.1 given in [25].

Thus using (16), we deduce

$$\text{diag}(\mu_h - M) R_h^2 = \eta_h I_h^*,$$

$$\left(\text{diag}(N_h^*)^{-1} \text{diag}(\beta_{mh} I_m^*) - M \right) S_h^* = \mu_h (I_h^* + R_h^*),$$

$$\text{diag}(\eta_h + \mu_h - M \Gamma) I_h^* = \text{diag}(N_h^*)^{-1} \text{diag}(\beta_{mh} I_m^*) S_h^*.$$ (16)

From Eq. (16), we deduce

$$\left(\text{diag}(N_h^*)^{-1} \text{diag}(\beta_{mh} I_m^*) + \mu_h \text{Id} - M \right) S_h^* = \mu_h N_h^*.$$ (17)

Thus using (16) and (17), we deduce

$$\left(\text{diag}(\eta_h + \mu_h) - M \Gamma \right) I_h^* = \mu_h \text{diag}\left(\frac{\beta_{mh} I_m^*}{N_h^*}\right),$$

$$\left(\text{diag}(N_h^*)^{-1} \text{diag}(\beta_{mh} I_m^*) + \mu_h \text{Id} - M \right)^{-1} N_h^*,$$

that is

$$I_h^* = \mu_h \left(\text{diag}(\eta_h + \mu_h) - M \Gamma\right)^{-1} \text{diag}\left(\frac{\beta_{mh} I_m^*}{N_h^*}\right)$$

$$\times \left(\text{diag}(N_h^*)^{-1} \text{diag}(\beta_{mh} I_m^*) + \mu_h \text{Id} - M \right)^{-1} N_h^*.$$ (18)

Now, we have two cases to consider: $\mathcal{M} = 0$ and $\mathcal{M} \neq 0$.

Suppose $\mathcal{M} = 0$. Then the patches are disjoint and in this case it suffices to follow the computations given in [19], [28]. Indeed, we have

$$I_{i,m}^* = \frac{\eta_{i,A} \beta_{i,mh} I_{i,h}^*}{\mu_{i,am} \left(\mu_{i,m} N_{i,h}^* + \beta_{i,mh} I_{i,h}^*\right)} \left(1 - \frac{1}{N_{i,m}}\right) \alpha_i K_i,$$

and from Eq. (16), we have

$$\frac{I_{i,h}^*}{N_{i,h}^*} = \frac{\mu_h}{\eta_h + \mu_h} \beta_{i,mh} I_{i,m}^* + \mu_h N_{i,h}^*.$$ (20)

From the two previous equation, we deduce:

$$I_{i,m}^* = \frac{\beta_{i,mh} \left(1 + \frac{\beta_{i,mh}}{\eta_h + \beta_{i,mh}}\right) (R_{0,i}^2 - 1) N_{i,h}^*}{\mu_{i,m} \left(\mu_{i,m} N_{i,h}^* + \beta_{i,mh} X_{i,h}\right)},$$ (21)

Note that Eq. (21) has a sense if and only if $R_{0,i}^2 > 1$, where $R_{0,i}^2$ is defined as in Eq. (14). Thus each patch admits an endemic equilibrium if $R_{0,i}^2 > 1$.

2) Using some of the previous computations, we now suppose that $\mathcal{M} \neq 0$. In that case, the computations are absolutely not obvious and we need to show the existence of the endemic equilibrium, using fix point argument. We will use Theorem 2.1 given in [25]. Using the previous computations we have to combine Eqs. (18) and (19), which lead to a fix point problem, $G(X) = X$, with

$$G(X) = \mu_h \left(\text{diag}(\eta_h + \mu_h) - M \Gamma\right)^{-1}$$

$$\times \text{diag}\left(\frac{\beta_{i,mh} X_{i,h}^*}{\mu_{i,m} \left(\mu_{i,m} N_{i,h}^* + \beta_{i,mh} X_{i,h}\right)}\right)$$

$$\times (u^* + \mu_h \text{Id}_n - M)^{-1} N_{i,h}^*,$$

where $u_{i,hm} = \frac{\eta_{i,A} \beta_{i,hm} X_{i,h}}{\mu_{i,m} \left(\mu_{i,m} N_{i,h}^* + \beta_{i,hm} X_{i,h}\right)}$ and $u^* = \frac{\eta_{i,A} \beta_{i,hm} X_{i,h}}{\mu_{i,m} \left(\mu_{i,m} N_{i,h}^* + \beta_{i,hm} X_{i,h}\right)}$.

Note carefully that the matrices

$$\left(\text{diag}(N_h^*)^{-1} \text{diag}(\beta_{mh} u_{i,hm}^* (1 - \frac{1}{N_{i,m}}) \alpha_i K_i)$

are M-Matrices and thus their inverses are positive.

We now verify the assumptions of Theorem 2.1 in [25]: it is obvious that $G$ is continuous, monotone nondecreasing, and strictly sublinear. $G$ is bounded and stay in the nonnegative orthant. Moreover $G(0) = 0$, and $G'(0)$ exists and is irreducible. Thus from Theorem 2.1
we can deduce that the only equilibrium with one zero component is the $E_{DF}$. It means that if at least one patch is infected, then all patches will become infected through population movements, unless $\Gamma = 0$ or $M = 0$. □

Remark 6: From the epidemiological point of view, the previous result states that if at least one patch is infected, then every patch will become infected, due to Human movements when $\gamma > 0$. This result seems obvious, but it clearly shows that if early vector control is not undertaken in places where infective people are recorded the disease will spread due to Human movements... and, of course, it will far more difficult to control the epidemic. □

Remark 7: In fact, using the same theorem we could go further, if we were able to show that $\rho(G'(0)) > 1$, then we could deduce the existence of a unique positive fixed point. In our case, we have

$$G'(0) = diag(\eta_h + \mu_h) (diag(\eta_h + \mu_h) - M\Gamma)^{-1} \times diag \left( \frac{1}{\eta_h + \mu_h} \eta_i A \beta_i, mh \beta_i, km \mu_i, m, N_{i,h}^* \right) \left( 1 - \frac{1}{N_{i,m}} \right) \alpha_i K_i$$

In the diagonal matrix, we recognize the local basic reproduction Number $R_{i,0}^2$. Thus

$$G'(0) = diag(\eta_h + \mu_h) (diag(\eta_h + \mu_h) - M\Gamma)^{-1} \times diag \left( R_{i,0}^2 \right) diag(\mu_h) (\mu_h Id_n - M)^{-1}.$$  

Although in the previous equality, all "ingredients" are there. In particular, we have

$$\rho \left( \left( diag(\eta_h + \mu_h) - M\Gamma \right)^{-1} \right) = diag \left( \frac{1}{\eta_h + \mu_h} \right)$$

$$\rho \left( \left( diag(\mu_h) - M \right)^{-1} \right) = diag \left( \frac{1}{\mu_h} \right).$$

Therefore, we can only conjecture that

$$\rho \left( G'(0) \right) \geq \max_i R_{i,0}^2.$$  

Then, $\rho(G'(0)) > 1$ if and only if there exists at least one patch $k$ where $R_{k,0}^2 > 1$.

Remark 8: When $M \neq 0$, we conjecture also that there exists a unique endemic equilibrium when $\max_i R_{i,0}^2 > 1$. This result is particularly useful: it shows that local and fast intervention when Chikungunya cases are suspected is the best way to stop the spreading of the disease. Fast and localized intervention is now the standard procedure in Réunion Island.

IV. Applications

We consider a two patch-model in order to illustrate the complexity of the results depending on the population size in each patch and the movement rates between patches. In particular, since the choices for the migration matrix are numerous, we would like to emphasize the fact that the construction of the migration model is highly sensitive. From these choices, the results may change drastically and then drive to wrong decisions either to decide or not about field interventions in order to control a disease or not.

The main advantage of this approach with two patches is that we are able to provide an analytical formula for $R_i^2$ and thus to discuss the impacts of different parameters. For simplification, we assume that $\mu_m = \mu_{am}$. Then, Equations (13) and (19) are equivalent to the following fix point problem

$$\left( \begin{array}{c} X \\ Y \end{array} \right) = \left( \begin{array}{c} F(Y) \\ G(X) \end{array} \right),$$

with

$$F(Y) = \mu_h \left( \left( diag(\eta_h + \mu_h) - M\Gamma \right)^{-1} diag \left( \frac{\beta mh Y}{N_h^*} \right) \times \left( \left( \frac{\beta mh Y}{N_h^*} \right) \left( \mu_i Id_2 - M \right)^{-1} \right) \right) \frac{\eta_i A \beta_i, hm X_i}{\mu_i, m N_{i,h}^* + \beta_i, hm X_i} \left( 1 - \frac{1}{N_{i,m}} \right) \alpha_i K_i,$$

Let us compute $G'(0)$ for a two patches system with a human population $(N_{1,h}, N_{2,h})$. The movement matrix is choosen such that $M N_{h}^* = 0$. For instance, $M$ could be choosen as follows:

$$M = \left( \begin{array}{c} -1 \frac{N_{1,h}^*}{N_{2,h}^*} \\ 1 - \frac{N_{1,h}^*}{N_{2,h}^*} \end{array} \right)$$

In particular, we deduce that $diag(\alpha) - M$ is an $M$-matrix when at least one $\alpha_i > 0$, which implies that

$$\left( \begin{array}{cc} \alpha_1 & 0 \\ 0 & \alpha_2 \end{array} \right) - M \left( \begin{array}{c} m_{12} \\ m_{21} \end{array} \right) = \left( \begin{array}{c} \alpha_2 + m_{12} \\ m_{21} \end{array} \right) \left( \frac{1}{\alpha_1 + m_{21}} \right) > 0.$$
Using the previous result, we compute
\[
\left( \text{diag} \left( \frac{\beta_{mh}}{N_h} \right) + \mu_h I_{d_2} - \mathcal{M} \right)^{-1}
\]
and
\[
\text{diag} \left( \frac{\beta_{mh} Y}{N_h} \right) \left( \text{diag} \left( \frac{\beta_{mh} Y}{N_h} \right) + \mu_h I_{d_2} - \mathcal{M} \right)^{-1} N_h^* = \left( \begin{array}{cc}
\frac{\beta_{1,mh} Y_1}{N_{1,h}} + \mu_h + m_{21} & \frac{\beta_{2,mh} Y_2}{N_{2,h}} + \mu_h + m_{12} - m_{21} m_{12} \\
\frac{\beta_{1,mh} Y_1}{N_{1,h}} + \mu_h + m_{12} & m_{21}
\end{array} \right)
\]
Moreover
\[
\mu_h \left( \text{diag}(\eta_h + \mu_h) - \mathcal{M} \Gamma \right)^{-1} = \frac{\mu_h}{(\eta_h + \mu_h)(\eta_h + \mu_h + \gamma_1 m_{21} + \gamma_2 m_{12})} \left( \begin{array}{c}
\eta_h + \mu_h + \gamma_2 m_{12} \\
\gamma_1 m_{21}
\end{array} \right) \left( \begin{array}{c}
\eta_h + \mu_h + \gamma_1 m_{21} \\
\gamma_2 m_{12}
\end{array} \right).
\]
Finally
\[
F_\gamma(Y) = \frac{1}{\Delta_1} \left( \begin{array}{cc}
F'_\gamma(11) & F'_\gamma(12) \\
F'_\gamma(21) & F'_\gamma(22)
\end{array} \right),
\]
where
\[
\Delta_1 = (\eta_h + \mu_h)(\eta_h + \mu_h + \gamma_1 m_{21} + \gamma_2 m_{12}),
\]
and
\[
F'_\gamma(11) = (\eta_h + \mu_h + \gamma_2 m_{12}) \beta_{1,mh} \frac{\mu_h + m_{21} + m_{21} N_{1,h}^*}{\mu_h + m_{12} + m_{21}},
\]
\[
F'_\gamma(12) = \gamma_2 m_{12} \beta_{2,mh} \frac{\mu_h + m_{21} + m_{21} N_{2,h}^*}{\mu_h + m_{12} + m_{21}},
\]
\[
F'_\gamma(21) = \gamma_1 m_{21} \beta_{1,mh} \frac{\mu_h + m_{21} + m_{21} N_{2,h}^*}{\mu_h + m_{12} + m_{21}},
\]
\[
F'_\gamma(22) = \gamma_1 m_{21} \beta_{1,mh} \frac{\mu_h + m_{21} + m_{21} N_{1,h}^*}{\mu_h + m_{12} + m_{21}}.
\]
Then using the fact that
\[
m_{21} N_{1,h}^* = m_{12} N_{2,h}^*,
\]
we deduce
\[
G'(0) = \left( \begin{array}{cc}
g_{1,hm} & 0 \\
0 & g_{2,hm}
\end{array} \right),
\]
where
\[
g_{1,hm}' = \frac{\eta_{1,A} \beta_{1,hm}}{\mu_{1,m} N_{1,h}^*} \left( 1 - \frac{1}{N_1} \right) \alpha_1 K^1
\]
\[
g_{2,hm}' = \frac{\eta_{2,A} \beta_{2,hm}}{\mu_{2,m} N_{2,h}^*} \left( 1 - \frac{1}{N_2} \right) \alpha_2 K^2
\]
and
\[
w_{1,hm} = \beta_{1,mh} Y_1 \left( \frac{\beta_{2,mh} Y_2}{N_{2,h}} + \mu_h + m_{12} + m_{21} N_{1,h}^* \right),
\]
\[
w_{2,hm} = \beta_{2,mh} Y_2 \left( \frac{\beta_{1,mh} Y_1}{N_{1,h}} + \mu_h + m_{21} + m_{21} N_{2,h}^* \right).
We derive the Jacobian of our system at 0

\[ J'(0) = \begin{pmatrix} 0 & 0 & F_1 & F_2 \\ 0 & 0 & F_3 & F_4 \\ g'_{1,hm} & 0 & 0 \\ 0 & g'_{2,hm} & 0 & 0 \end{pmatrix} \]

where

\[
F_1 = \frac{(\eta_h + \mu_h + \gamma_1 m_{12}) \beta_{1,mh}}{(\eta_h + \mu_h + \gamma_1 m_{21} + \gamma_2 m_{12})}, \\
F_2 = \frac{(\eta_h + \mu_h)(\eta_h + \mu_h + \gamma_1 m_{21} + \gamma_2 m_{12})}{\gamma_2 m_{12} \beta_{2,mh}}, \\
F_3 = \frac{(\eta_h + \mu_h)(\eta_h + \mu_h + \gamma_1 m_{21} + \gamma_2 m_{12})}{\gamma_1 m_{21} \beta_{1,mh}}, \\
F_4 = \frac{(\eta_h + \mu_h)(\eta_h + \mu_h + \gamma_1 m_{21} + \gamma_2 m_{12})}{(\eta_h + \mu_h + \gamma_1 m_{21} + \gamma_2 m_{12})}.
\]

The characteristic polynomial gives

\[
p(\lambda) = \lambda^4 - \frac{1}{\eta_h + \mu_h + \gamma_1 m_{21} + \gamma_2 m_{12}} \times ((\eta_h + \mu_h + \gamma_1 m_{21}) R_{0,2}^2 + (\eta_h + \mu_h + \gamma_2 m_{12}) R_{0,1}^2) \lambda^2
\]

\[
+ \left( \frac{1}{\eta_h + \mu_h + \gamma_1 m_{21} + \gamma_2 m_{12}} \right)^2 \times ((\eta_h + \mu_h + \gamma_2 m_{12}) R_{0,2}^2 R_{0,1} - \gamma_2 m_{21} \gamma_1 m_{12} R_{0,2}^2 R_{0,1}) \lambda
\]

Thus setting \( x = \lambda^2 \), we obtain the following a second order polynomial

\[
p(x) = x^2 - \frac{1}{\eta_h + \mu_h + \gamma_1 m_{21} + \gamma_2 m_{12}} \times ((\eta_h + \mu_h + \gamma_1 m_{21}) R_{0,2}^2 + (\eta_h + \mu_h + \gamma_2 m_{12}) R_{0,1}^2) x
\]

\[
+ \left( \frac{1}{\eta_h + \mu_h + \gamma_1 m_{21} + \gamma_2 m_{12}} \right)^2 \times ((\eta_h + \mu_h + \gamma_2 m_{12}) R_{0,2}^2 R_{0,1} - \gamma_2 m_{21} \gamma_1 m_{12} R_{0,2}^2 R_{0,1})
\]

Then

\[
p(x) = (x - \frac{1}{2} \eta_h + \mu_h + \gamma_1 m_{21} + \gamma_2 m_{12}) \times ((\eta_h + \mu_h + \gamma_1 m_{21}) R_{0,2}^2 + (\eta_h + \mu_h + \gamma_2 m_{12}) R_{0,1}^2) \times \left( \frac{1}{\eta_h + \mu_h + \gamma_1 m_{21} + \gamma_2 m_{12}} \right)^2
\]

Thus

\[
p(x) = (x - \frac{1}{2} \eta_h + \mu_h + \gamma_1 m_{21} + \gamma_2 m_{12}) \times \left( \frac{1}{k_{2h} R_{0,2}^2 + k_{1h} R_{0,1}^2 - \sqrt{Z(\gamma)}} \right)
\]

\[
\times (x - \frac{1}{2} \eta_h + \mu_h + \gamma_1 m_{21} + \gamma_2 m_{12}) \left( k_{2h} R_{0,2}^2 + k_{1h} R_{0,1}^2 + \sqrt{Z(\gamma)} \right)
\]

with

\[
Z(\gamma) = (k_{2h} R_{0,2}^2 - k_{1h} R_{0,1}^2)^2 + 4 \gamma_2 m_{12} \gamma_1 m_{21} R_{0,2}^2 R_{0,1}^2 > 0, \\
k_{1h} = (\eta_h + \mu_h + \gamma_2 m_{12}), \\
k_{2h} = (\eta_h + \mu_h + \gamma_1 m_{21}).
\]

Thus, we deduce the following exact formula for \( R_0^2 \):

\[
R_0^2 = \frac{1}{2} \left( R_{0,2}^2 + R_{0,1}^2 + |R_{0,2}^2 - R_{0,1}^2| \right),
\]

which is equivalent to \( R_0^2 = \max( R_{0,1}^2, R_{0,2}^2 ) \).

Moreover, if \( R_{0,1} = R_{0,2} \), then \( R_0^2 = R_{0,1} = R_{0,2} \) whatever the migration matrix is.

Then, we will consider various migration matrix between city 1 and city 2, to show how different can be the results according to the movement rates....

Example 1: The numerical simulations presented here have been obtained using nonstandard finite difference scheme (see [1], [2], [3], [10], [11] for an overview on nonstandard methods, and applications to biological systems [19], [20], [23]). We consider parameters (see table V in Appendix B) such that \( R_{0,1} > 1 \) and \( R_{0,2} < 1 \), i.e.

<table>
<thead>
<tr>
<th>( N_{1,h} )</th>
<th>( K_1 )</th>
<th>( R_{0,1} )</th>
<th>( N_{2,h} )</th>
<th>( K_2 )</th>
<th>( R_{0,2} )</th>
</tr>
</thead>
<tbody>
<tr>
<td>Case 1 8.10^4</td>
<td>3.10^4</td>
<td>1.3017</td>
<td>8.10^4</td>
<td>2.10^4</td>
<td>0.434</td>
</tr>
<tr>
<td>Case 2 2.10^4</td>
<td>3.10^4</td>
<td>1.3017</td>
<td>10^4</td>
<td>2.10^4</td>
<td>0.434</td>
</tr>
<tr>
<td>Case 3 10^4</td>
<td>3.10^4</td>
<td>1.3017</td>
<td>8.10^4</td>
<td>2.10^4</td>
<td>0.434</td>
</tr>
</tbody>
</table>

Thus, in order to keep the total population \( N_1 + N_2 \) constant, we have many choices for the migration matrix. Let us first consider simply

\[
M = M_c \begin{pmatrix} -1 & N_{1,h} \\ N_{2,h} & -1 \end{pmatrix}
\]
where $M_c$ is a positive constant. We would like to study the time evolution of the disease with respect to $\Gamma = \text{diag}(\gamma_1, \gamma_2)$, taking into account the movement rates and different values for $M_c$.

- Case 1. We consider $M_c = 1$ and cities with the same populations, except for the larvae capacity such that we obtain distinct basic reproduction numbers. In Fig. [2] we consider $\gamma_1 = \gamma_2$: in that case, when $\gamma > 0$, the epidemic occurs in both cities.

If we consider $\gamma_1 = 1$ and $\gamma_2 = 0.5$, then the dynamic change: if less infected people from city 2 are able to move to city 1, i.e. $\gamma_2$ small, then the general basic reproduction number decreases rapidly under 1. This is confirmed in Fig. [4] where we show $R_0$ with respect to $\gamma_1$ and $\gamma_2$: it clearly indicates that small values for $\gamma_2$ help to decrease the epidemiological risk. In fact, following Fig. [4] the best combination being $\Gamma = \text{diag}(1, 0)$. This is due to the fact that city 1 has the largest basic reproduction number: thus movement of infected individuals from city 1 to city 2, reduce the number of infected people in city 1, and thus the risk of propagation. The model behaves like a quarantine model.

- Case 2. We consider $M_c = 1$. Numerical simulations are presented in Fig. [5] for different values of $\gamma$. Of course without Human movements, the first city faces a huge epidemic while in the second nothing occurs. For $\gamma > 0$, then the dynamic changes drastically: in particular in city 1, we observe a decay in the Number of Infected people as soon as $\gamma >$, while in city 2, an outbreak appears. This is confirmed in Fig. [6] where $R_0$ has been computed with the exact formula (22): as long as $\gamma_{12} > 0$, $R_0 > 1$, which indicates that an epidemic will occur in all cities.

In this case, city 1, where $R_0^2 > 1$, has a large population compared to city 2. Thus the flow of infected people from city 1 to city 2, will increase the number of infected people in city 2, as long as the epidemic occurs in city 2. Thus disease control is necessary in city 1 in order to avoid the risk of disease spreading. In particular, reducing local and large $R_{0,i}$ could be helpful to lower the general basic reproduction number, but this may not be
not necessarily sufficient or, even, possible. In this case, city 1, with the largest population, has the strongest impact on \( R_0 \); compare also with Fig. 4 where both cities have the same population. Even if \( M_c \) decays this will not change the overall behavior: \( R_0 \) will stay greater than one. In fact decaying \( M_c \) is like decaying \( \gamma \) and it is obvious that for small values of \( \gamma \) human movements implies a spreading of the disease in all patches.

\[
\begin{array}{cccc}
\gamma_1=0, \gamma_2=0 & \gamma_1=0.1, \gamma_2=0.1 & \gamma_1=0.2, \gamma_2=0.2 \\
\gamma_1=0.5, \gamma_2=0.5 & \gamma_1=0.7, \gamma_2=0.7 & \gamma_1=1, \gamma_2=1
\end{array}
\]

Fig. 5. Simulation of the evolution of the infected population per week for different values of \( \gamma \).

\[
\begin{array}{cccc}
\gamma_1=0, \gamma_2=0 & \gamma_1=0.1, \gamma_2=0.1 & \gamma_1=0.2, \gamma_2=0.2 \\
\gamma_1=0.5, \gamma_2=0.5 & \gamma_1=0.7, \gamma_2=0.7 & \gamma_1=1, \gamma_2=1
\end{array}
\]

Fig. 7. Simulation of the evolution of the infected population per week for different values of \( \gamma \).

Fig. 6. Evolution of \( R_0 \) with respect to \( \gamma_1 \) and \( \gamma_2 \).

Case 3. We first consider \( M_c = 1 \). Numerical simulations are presented in Fig. 7. Contrary to case 1, city 2 has now the largest population with \( R_{0,2} < 1 \). Thus without migration, only city 1 is impacted by a huge epidemic, but, as soon as \( \gamma_1 > 0 \), the number of infected cases reduce drastically in city 1, while in city 2 a small outbreak appears. Finally, as \( \gamma_1 \) increases, the epidemic becomes a small outbreak for both cities (see Fig. 8). Thus, the city with the largest population and a local basic reproduction number less than one has a positive impact on the general basic reproduction number: compare Fig. 8 with Fig. 6.

Indeed, if we consider \( M_c = 0.1 \), for instance, then the dynamic is completely different and shows that the disease will spread in all patches (see Figs. 9) but as \( \gamma \) increases the force of the disease...
decays, which is confirmed by the computation of the basic reproduction number in Fig. 10. In fact this computation is equivalent to consider \( \gamma_i \in [0, 0.1] \) when \( M_c = 1 \).

In any case, it is clear that Human movements may have a benefit effect when the largest city has a basic reproduction number lower than 1.

Our example emphasizes the importance of the migration (movement) matrice and how important is the construction of such a matrice to understand or to well capture the whole dynamic. And this is only in a 2-patches model!

In fact we can go further with this example and for instance, consider another movement matrix:

\[
M = M_c \begin{pmatrix}
-\frac{N_{1,h}}{N_{2,h}} & 1 \\
-\frac{N_{2,h}}{N_{1,h}} & -1
\end{pmatrix}
\]

with \( M_c = 1 \). Let us first consider case 2: compare Figs. 11 and 6. \( R_0^2 \) is not the same, but, following Fig. 11, it seems that human movements have limited impacts on \( R_0^2 \). This example clearly shows that if locally the basic reproduction number is lower than 1 then the impact of the disease will be limited, even if there infected people moves from city 1.

In case 3, in contrary, the new movement matrix changes drastically the behavior of the basic reproduction number: compare Figs. 12 and 9. Here, the impact of the largest city (city 2) is really important. In that case, even if \( \gamma \) is small, \( R_0^2 \) decays rapidly and the disease dies out.

Thus depending on \( M \), \( R_0^2 \) can be very different, which may imply inappropriate control decisions. This clearly indicates the importance of the construction of the movement Matrices.

\[ R_0 \text{ computed with the analytical formula} \]

\[ R_0 \text{ computed with the analytical formula} \]

Thus, in case 3, at least, vector control can be necessary or not, depending on the movements matrice: when \( M_c = 1 \), the disease will disappear naturally... while, not when \( M_c = 0.1 \).

In any case, it is clear that Human movements may have a benefit effect when the largest city has a basic reproduction number lower than 1.

Our example emphasizes the importance of the migration (movement) matrice and how important is the construction of such a matrice to understand...
the whole population. Mathematically the model is not very easy to handle, but we have been able to show some interesting results. In particular, we showed the link between the general basic reproduction number and local ones, which is really important from a practical point of view. Indeed, our illustrative examples indicate that measuring or estimating local basic reproductive numbers is of major importance not only to map the epidemiological risk in order to take into account where the risk of an epidemic is high, but to be able to indicates priority to lower some local basic reproduction numbers.

Thus, among all cities where the risk is high, it seems important to make vector control in priority in cities where the Human population is large. In any case, each city has to make appropriate vector control campaign to lower the epidemiological risk. In the case where some cities, with the largest populations, may have for any reason large basic reproduction numbers, then the disease can spread quickly to the whole domain, even when $\Gamma$ is small, according to the network. In contrary when only small cities have large local basic reproduction numbers, human movements can have a benefit effect, i.e. the disease dies out.

Of course, the model can still be improved in different ways. It might be interesting to consider a variable total population and/or time-dependant parameters [17]. But, for a practical use in Réunion Island, a first step would be to build the right movements matrix between cities, using precise human movements data. Finally, this model could be adapted to link several islands (Mauritius, La Réunion, Comoros and Madagascar) located in the Indian Ocean.

**ACKNOWLEDGMENT**

This study was funded by the French Ministry of Health and the European Regional Development Fund (ERDF) within the “SIT feasibility programme” in Réunion Island. The authors would like to thank Guy Lemperiere (IRD, coordinator of the SIT project in Réunion island), Louis-Clément Gouagna (IRD), Jérémie Gilles and Clélia Oliva (AIEA, Seibersdorf, Austria), Sébastien Boyer (IRD), and Jean-Sebastien Dehecq (ARS, Réunion island), for stimulating discussions. YD is also grateful to Didier Fontenille (IRD, head of the SIT project) and Koussay Delligi (CRVOI, Réunion island) for their support.

The authors would like to thank the reviewers for their constructive comments that helped to improve the manuscript.

AMAP (Botany and Computational Plant Architecture) is a joint research unit which associates CIRAD (UMR51), CNRS (UMR5120), INRA (UMR931), IRD (2M123), and Montpellier 2 University (UM27); http://amap.cirad.fr/

**REFERENCES**


Y. Dumont, J.-M. Tchuenche, and M. Le Corre,
Modeling Mosquito distribution. Impact of the Vegetation.


http://dx.doi.org/10.1063/1.3659916


http://dx.doi.org/10.1007/s00285-011-0477-6


http://dx.doi.org/10.1137/T110850761


http://dx.doi.org/10.1016/0266-2592(85)90038-0


http://dx.doi.org/10.1186/1472-6785-10-8


http://dx.doi.org/10.1016/j.mbs.2010.10.008


http://dx.doi.org/10.1016/j.mbs.2008.08.010

Let \( \mathbf{a}, \mathbf{b} \in \mathbb{R}^n \) with \( \mathbf{a} \leq \mathbf{b} \), and
\[
[\mathbf{a}, \mathbf{b}] = \{ x \in \mathbb{R}^n : \mathbf{a} \leq x \leq \mathbf{b} \}.
\]

**Theorem 5 ([5]):** Let \( \mathbf{a}, \mathbf{b} \in D \) be such that \( \mathbf{a} < \mathbf{b} \), \( [\mathbf{a}, \mathbf{b}] \subseteq D \) and \( g(\mathbf{b}) \leq 0 \leq g(\mathbf{a}) \). Then (23) defines a (positive) dynamical system on \( [\mathbf{a}, \mathbf{b}] \). Moreover, if \( [\mathbf{a}, \mathbf{b}] \) contains a unique equilibrium \( p \) then \( p \) is globally asymptotically stable on \( [\mathbf{a}, \mathbf{b}] \).

**Appendix B: Biological Parameters**

<table>
<thead>
<tr>
<th>Parameters Description</th>
<th>Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>( \beta_{mh} ) (Transmission probability from ( I_m ) (per bite))</td>
<td>0.365</td>
</tr>
<tr>
<td>( \beta_{hm} ) (Transmission probability from ( I_h ) (per bite))</td>
<td>0.365</td>
</tr>
<tr>
<td>( 1/\mu_h ) (Average lifespan of humans (in days))</td>
<td>78 \times 365</td>
</tr>
<tr>
<td>( 1/\eta_m ) (Extrinsic incubation period (in days))</td>
<td>7</td>
</tr>
<tr>
<td>( 1/\mu_m ) (Average lifespan of female mosquitoes (in days))</td>
<td>3</td>
</tr>
<tr>
<td>( \mu_b ) (Nb of eggs at each deposit per capita (per day))</td>
<td>7</td>
</tr>
<tr>
<td>( \mu_A ) (Natural mortality of larvae (per day))</td>
<td>2</td>
</tr>
<tr>
<td>( \eta_A ) (Maturation rate from larvae to adult (per day))</td>
<td>( \approx 0.05 )</td>
</tr>
</tbody>
</table>