Received: 11 October 2018 | Revised: 27 June 2019 | Accepted: 9 July 2019

DOI: 10.1002/cmm4.1059

RESEARCH ARTICLE

WILEY

A model for yellow fever with migration

Lourdes Esteva[1] | **Cristobal Vargas**[2] | **Hyun Mo Yang**[3]

[1]Departamento de Matemáticas, Facultad de Ciencias, Universidad Nacional Autónoma de México, Mexico City, Mexico

[2]Departamento de Control Automático, CINVESTAV-IPN, Mexico City, Mexico

[3]Departamento de Matemática Aplicada, Universidade Estadual de Campinas, Campinas, Brazil

Correspondence
Lourdes Esteva, Departamento de Matemáticas, Facultad de Ciencias, Universidad Nacional Autónoma de México, 04510 Mexico City, Mexico.
Email: lesteva@ciencias.unam.mx

In this work, we formulate a mathematical model to evaluate the risk of the introduction of yellow fever (YF) in towns by infected humans arriving from forest areas. We obtain the basic reproduction numbers associated with the forest and urban regions. We present numerical simulations to evaluate the risk of YF spread for different migration rates.

KEYWORDS

basic reproduction number, equilibria, migration, stability, yellow fever

1 | INTRODUCTION

Yellow fever (YF) is an infection of variable severity caused by Flavivirus and transmitted by the bite of mosquitoes. This disease occurs mainly in Sub-Saharan Africa as well as Central and South America. YF causes fever and headaches in most cases; however, roughly 15% of cases progress to a more severe form of the disease that is characterized by high fever, jaundice, bleeding, and eventually shock. Liver damage results in yellowing of the skin, hence the name "yellow fever."[1,2]

According to the World Health Organization,[3] there are approximately 200 000 cases of YF worldwide each year, and 30 000 deaths, with about 90% of all the cases occurring in Africa. At the present, there is a vaccine against YF with life immunity although it can be scarce. Other preventive measures, eg, use of insect repellent and mosquito netting are recommended. However, despite the vaccine and protective measures since 1980s, the number of YF cases has increased.[1]

In the Americas, YF virus presents two cycles: jungle and urban. The jungle or sylvatic cycle involves transmission of the virus mainly between nonhuman primates (eg, monkeys) and mosquito species found in the forest (*Haemagogus spp.* mosquitoes). The virus is transmitted by mosquitoes to humans that are visiting or working in the jungle.

The urban cycle involves transmission of the virus between humans and urban mosquitoes, primarily *Aedes aegypti*. The virus can be brought to the urban area by humans who were infected in the jungle. In Africa, in addition to the jungle and urban cycles, there is an intermediate (savannah) cycle that involves transmission of virus from mosquitoes to humans living or working in jungle border areas. In this cycle, the virus can be transmitted from monkey to human or from human to human via mosquitoes.[4]

It is worth to mention that the largest outbreak of YF in Brazil in the last years, with more than 2000 confirmed cases and 676 deaths from December 2016 to March 2018, was originated in nonhuman primates according to genetic and poblational studies.[5,6] Analysis of YF cases combined with genomes generated locally revealed an early phase of sylvatic transmission and posterior spatial expansion toward areas free of YF, followed by a rise in viral transmission to humans in urban areas in late 2016. The time series of confirmed cases in humans was compared with time series of nonhuman primates, finding that human cases appeared four days later than cases in nonhuman primates. It was also found that the risk of YF was higher for people who lived or worked in forested areas, where mosquitoes that usually feed on primates can bite and transmit the virus to humans. Furthermore, they found that at the origins of the outbreak, 85% of the cases were in men, who travel more than women to remote areas of the jungle.

Mathematical models have been developed in order to evaluate the risk of YF, as well as efficacy of vaccination programs. Raimundo et al[7] considered a model with vaccination and derived a threshold vaccination rate, above which the disease would be eradicated from the human population. Estimations of the proportion of vaccinated against YF taking into account the risk of serious adverse events associated with the vaccine are done in the works of Codeço et al[8] and Massad et al.[9] Codeço et al[10] also estimated the probability that infective individuals arriving from YF endemic areas can trigger an epidemic in a disease-free area. They found that the risk of urban YF emergence may reach values as high as 29% during the epizootic periods but they found that the precision of the estimate is low. Massad et al[11] designed a method to calculate the density of *Aedes aegypti* mosquito females using data of dengue disease incidence in Rio de Janeiro neighborhoods and verified that their estimates agreed with those obtained by means of traps. Using these data, they assessed the risk that the introduction of an infectious individual would lead to an epidemic outbreak of YF in Rio de Janeiro. Johansson et al[12] formulated a stochastic metapopulation model to simulate the global spread of YF from a single urban outbreak by infective airline travelers. They found that local incidence, travel rates, and basic transmission parameters are enough to estimate a probability of introduction of YF to urban areas.

Our purpose in this work is to assess the risk of acquiring YF by migrants in the forest areas, as well as the potential introduction of this disease into urban areas. We will only address the two-cycle case, given that we are particularly interested in the spread of YF in the Americas. For this, we propose a mathematical model assuming that no vaccinated humans enter to the forest to carry out specific works like cutting trees and constructing roads or for leisure like camping. These humans that we call migrants are under risk of acquiring YF due to exposition to forest epidemics and to bring the disease to urban settlements. The model is a variation of the Ross-MacDonald model in which we included migration from the urban to the wild area, and it is based on our previous works on vector borne transmission.[13,14]

We found necessary conditions for the introduction of YF to the urban area due to the movement of people returning from forest regions. These conditions are in terms of the basic reproductive numbers of urban, migrant, and wild region. In this model, we do not have restrictions about the inflow and outflow of healthy and infected persons, since we are not assuming a particular number of infected people going into the urban area or quantity of people that is traveling to the forest area.

This paper is structured as follows. In Section 2, a model is formulated to describe the YF transmission in forest an urban area. In Section 3, existence and stability of the model equilibria is investigated in order to assess the introduction of YF in urban areas. In Section 4, the risk of YF among migrants is evaluated, and numerical simulations are presented in Section 5. Finally, conclusions are given in Section 6.

2 | FORMULATION OF THE MODEL

We denote by N_w the wild zoonotic population that we assume composed by monkeys that inhabit the wild areas, by N_u the population that stays in the urban area, and by N_m the population that moves to the forest area, with $N_h = N_u + N_m$ the total human population. Furthermore, V_u and V_w are the populations of mosquito transmitters of YF in the wild area (*Haemagogus spp.*) and urban area (*A*edes aegypti), respectively. We assume that all the populations involved, N_w, N_h, V_w, and V_u, are constant, that is, their respective birth and mortality rates are the same, and we denote the mortality rates by μ_w, μ_u, ν_w, and ν_u, respectively. The zoonotic population is divided into susceptibles, S_w, infected, I_w, and recovered, Z_w, with $N_w = S_w + I_w + Z_w$. The urban population N_u is divided in S_u, I_u, and Z_u, with $N_u = S_u + I_u + Z_u$. Further, by S_m, it is denoted the susceptible humans that move to the forest region, and by I_m the humans that get infected there. In this work, we will assume that infected humans do not enter to the forest, and we will not consider vaccination and mortality due to the disease. The susceptible and infected mosquitoes in the wild area are M_w, and Y_w, while in the urban area are denoted by M_u and Y_u. Since the life span of the mosquitoes is very short, they do not recover from the infection, and therefore, their total population, V_i, is equal to $M_i + Y_i$, $i = u, w$, which implies that susceptible mosquitoes are obtained by the relation $M_i = V_i - I_i$, $i = u, w$, and it is enough to consider only the infective populations of both vectors. The average number of vectors per zoonotic and per urban populations are given by $V_w/(N_w + N_m)$ and $V_u/(N_u - N_m)$, respectively. Therefore, a particular individual of each population receives on average $b_w V_w/(N_w + N_m)$ and $b_u V_u/(N_u - N_m)$ bites, respectively, where b_i, $i = w, u$, are the biting rates of wild and urban mosquitoes. A proportion Y_i/V_i, $i = w, u$, of these bites comes from infected mosquitoes, and therefore, wild and urban susceptibles are infected by infectious mosquitoes at rates $b_w\bar{\beta}_w Y_w/(N_w + N_m)$ and $b_u\bar{\beta}_u Y_u/(N_u - N_m)$, respectively, where $\bar{\beta}_i$, $i = w, u$, denote the probabilities that a bite from an infective mosquito Y_i, $i = w, u$, gives rise to a infective case in the wild and urban populations. The susceptible population S_m that enters to the wild region get infected at a rate $b_w\bar{\beta}_m Y_w/(N_w + N_m)$, where $\bar{\beta}_m$ is the probability that a

bite from an infected *Haemagogus* gives rise to an infection in a migrant. Analogously, the infection rates from the forest and urban population to mosquitoes are given by $b_w\bar{\alpha}_w/(N_w+N_m)$ and $b_u\bar{\alpha}_u/(N_u-N_m)$, respectively, and $b_w\bar{\alpha}_m/(N_w+N_m)$ is the contribution of infectious migrants, where $\bar{\alpha}_i$, $i=u,m,w$, denote the probabilities that a mosquito becomes infected by I_u, I_m, or I_w populations, respectively. Finally, we assume that monkey and human populations recover at rates γ_w and γ_u, respectively.

We denote by δ the percentage of humans that travel to the forest region per unit of time, and by ϵ the percentage of humans that return to the urban area from the forest region per unit of time. When YF is endemic in the forest region, susceptible migrant humans (δS_u) are under YF risk during the periods of time spent in forest region due to infected mosquitoes Y_w. According to the above assumptions we obtain, the following systems of ODE to describe the forest and urban epidemic cycles of YF.

The forest epidemic cycle is given by

$$\begin{aligned}
\frac{dS_w}{dt} &= \mu_w N_w - \frac{b_w\bar{\beta}_w}{N_w+N_m}S_wY_w - \mu_w S_w\\
\frac{dI_w}{dt} &= \frac{b_w\bar{\beta}_w}{N_w+N_m}S_wY_w - \gamma_w I_w - \mu_w I_w\\
\frac{dZ_w}{dt} &= \gamma_w I_w - \mu_w Z_w\\
\frac{dY_w}{dt} &= \frac{b_w\bar{\alpha}_w}{N_w+N_m}(V_w-Y_w)I_w + \frac{b_w\bar{\alpha}_m}{N_w+N_m}(V_w-Y_w)I_m - \nu_w Y_w.
\end{aligned} \tag{1}$$

The epidemic cycle among humans in forest region is given by

$$\begin{aligned}
\frac{dS_m}{dt} &= \delta S_u - \frac{b_w\bar{\beta}_m}{N_w+N_m}S_mY_w - \epsilon S_m - \mu_u S_m\\
\frac{dI_m}{dt} &= \frac{b_w\bar{\beta}_m}{N_w+N_m}S_mY_w - \gamma_u I_m - \epsilon I_m - \mu_u I_m\\
\frac{dZ_m}{dt} &= \delta_1 Z_u + \gamma_u I_m - \epsilon Z_m - \mu_u Z_m,
\end{aligned} \tag{2}$$

and the urban YF epidemics sustained by infectious migrant humans is given by

$$\begin{aligned}
\frac{dS_u}{dt} &= \mu_u N_u - \frac{b_u\bar{\beta}_u}{N_u-N_m}S_uY_u - \mu_u S_u - \delta S_u + \epsilon S_m\\
\frac{dI_u}{dt} &= \frac{b_u\bar{\beta}_u}{N_u-N_m}S_uY_u - \gamma_u I_u - \mu_u I_u + \epsilon I_m\\
\frac{dZ_u}{dt} &= \gamma_u I_u - \delta_1 Z_u - \mu_u Z_u + \epsilon Z_m\\
\frac{dY_u}{dt} &= \frac{b_u\bar{\alpha}_u}{N_u-N_m}(V_u-Y_u)I_u - \nu_u Y_u,
\end{aligned} \tag{3}$$

where δ_1 is the migration rate of recovered individuals (in this work, we shall assume $\delta_1=\delta$). Adding up the equations corresponding to system (1), (2), and (3), we obtain the equations $dN_w/dt=0$ and $dN_h/dt=0$. Because the equations for recovered individuals Z_w, Z_m, and Z_u are decoupled, they can be removed from the system and retrieved by $Z_w = N_w-S_w-I_w$, $Z_m=N_m-S_m-I_m$, and $Z_u=N_u-S_u-I_u-(S_m+I_m+Z_m)$. Due to the fact that human and vector populations are constant, it is convenient to write the above equations in terms of the proportions

$$s_w=\frac{S_w}{N_w}, i_w=\frac{I_w}{N_w}, z_w=\frac{Z_w}{N_w}, s_u=\frac{S_u}{N_h}, i_u=\frac{I_u}{N_h}, z_u=\frac{Z_h}{N_h}, s_m=\frac{S_m}{N_h}, i_m=\frac{I_m}{N_m}, y_w=\frac{Y_w}{V_w}, y_u=\frac{Y_u}{V_u}.$$

Therefore, from Equations (1), (2), and (3), we obtain the following systems:

$$\begin{aligned}\frac{ds_w}{dt} &= \mu_w - \beta_w s_w y_w - \mu_w s_w\\ \frac{di_w}{dt} &= \beta_w s_w y_w - \gamma_w i_w - \mu_w i_w\\ \frac{dy_w}{dt} &= \alpha_w(1-y_w)i_w + \alpha_m(1-y_w)i_m - \nu_w y_w,\end{aligned} \tag{4}$$

for the forest epidemic cycle (with $z_w = 1 - s_w - i_w$),

$$\begin{aligned}\frac{ds_m}{dt} &= \delta s_u - \beta_m s_m y_w - \epsilon s_m - \mu_u s_m\\ \frac{di_m}{dt} &= \beta_m s_m y_w - \gamma_u i_m - \epsilon i_m - \mu_u i_m,\end{aligned} \tag{5}$$

for the epidemic cycle among humans in forest region (with $z_m = 1 - s_m - i_m - N_u/N_h$), and

$$\begin{aligned}\frac{ds_u}{dt} &= \mu_u - \beta_u s_u y_u - \mu_u s_u - \delta s_u + \epsilon s_m\\ \frac{di_u}{dt} &= \beta_u s_u y_u - \gamma_u i_u - \mu_u i_u + \epsilon i_m\\ \frac{dy_u}{dt} &= \alpha_u(1-y_u)i_u - \nu_u y_u,\end{aligned} \tag{6}$$

for the urban YF epidemics sustained by infectious migrant humans (with $z_u = 1 - s_u - i_u - N_m/N_h$).

To simplify the notation, in (4), (5), and (6), the infection coefficients are given by

$$\begin{aligned}\beta_w &= b_w\bar{\beta}_w\frac{V_w}{N_w+N_m} \qquad \alpha_w = b_w\bar{\alpha}_w\frac{N_w}{N_w+N_m}\\ \beta_m &= b_w\bar{\beta}_m\frac{V_w}{N_w+N_m} \qquad \alpha_m = b_w\bar{\alpha}_m\frac{N_u}{N_w+N_m}\\ \beta_u &= b_u\bar{\beta}_u\frac{V_u}{N_u-N_m} \qquad \alpha_u = b_u\bar{\alpha}_{u,}\frac{N_u}{N_u-N_m}.\end{aligned} \tag{7}$$

A summary of model parameters and their values is presented in Table 1.

3 | MATHEMATICAL ANALYSIS OF THE MODEL

3.1 | Disease-free equilibrium and the basic reproductive number

The *disease-free equilibrium* of Equations (4), (5), (6) is given by

$$\bar{P}_0 = \left(1, 0, 0, \frac{\delta}{\epsilon+\mu_u+\delta}, 0, 1-\frac{\delta}{\epsilon+\mu_u+\delta}, 0, 0\right)$$

and represents the state where the population is infection free. The *basic reproductive number*, denoted by R_0, represents the average number of secondary cases that one infective generates over the course of its infectious period in a whole susceptible population. It is a threshold condition that determines whether an epidemic can occur or a disease remains endemic, and thus, if $R_0 < 1$, the disease dyes out, while for $R_0 > 1$, it persists. Diekmann and Heesterbeek[21] define mathematically the basic reproductive number of a disease as the spectral ratio of the *next-generator operator* associated

to the disease-free equilibrium, which is given by the product of two matrices: the nonnegative matrix of the infection terms, K, and the inverse of the matrix of the transmission terms, T. For our model, these matrices are

$$K = \begin{pmatrix} K_1 & 0_{3\times 2} \\ K_3 & K_2 \end{pmatrix} \text{ and } T = \begin{pmatrix} \gamma_w + \mu_w & 0 & 0 & 0 & 0 \\ 0 & \nu_w & 0 & 0 & 0 \\ 0 & 0 & \gamma_u + \epsilon + \mu_u & 0 & 0 \\ 0 & 0 & 0 & \gamma_u + \mu_u & 0 \\ 0 & 0 & 0 & 0 & \nu_w \end{pmatrix}, \tag{8}$$

where

$$K_1 = \begin{pmatrix} 0 & \beta_w & 0 \\ \alpha_w & 0 & \alpha_m \\ 0 & \beta_m \frac{\delta}{\epsilon+\mu_u+\delta} & 0 \end{pmatrix}, \quad K_2 = \begin{pmatrix} 0 & \beta_u \left(1 - \frac{\delta}{\epsilon+\mu_u+\delta}\right) \\ \alpha_u & 0 \end{pmatrix}, \quad K_3 = \begin{pmatrix} 0 & 0 & \epsilon \\ 0 & 0 & 0 \end{pmatrix}. \tag{9}$$

The next-generation operator associated with the disease-free equilibrium $\bar{P}_0$ is given by

$$KT^{-1} = \begin{pmatrix} KT_1^{-1} & 0_{3\times 2} \\ KT_3^{-1} & KT_2^{-1} \end{pmatrix}, \tag{10}$$

with

$$KT_1^{-1} = \begin{pmatrix} 0 & \frac{\beta_w}{\nu_w} & 0 \\ \frac{\alpha_w}{\gamma_w+\mu_w} & 0 & \frac{\alpha_m}{\gamma_u+\epsilon+\mu_u} \\ 0 & \frac{\beta_m}{\nu_w}\frac{\delta}{\epsilon+\mu_u+\delta} & 0 \end{pmatrix}, \quad KT_2^{-1} = \begin{pmatrix} 0 & \frac{\beta_u}{\nu_u}\left(1 - \frac{\delta}{\epsilon+\mu_u+\delta}\right) \\ \frac{\alpha_u}{\gamma_u+\mu_u} & 0 \end{pmatrix}, \tag{11}$$

and KT_3^{-1} a 2×3 matrix, with null elements except $(KT_3^{-1})_{13} = \epsilon/(\gamma_u + \epsilon + \mu_u)$. The characteristic equation corresponding to KT^{-1} is

$$\left[\lambda^3 - \left(R_0^w + R_0^m\right)\lambda\right] \times \left(\lambda^2 - R_0^u\right) = 0, \tag{12}$$

with

$$\begin{aligned} R_0^w &= \frac{\alpha_w}{\gamma_w + \mu_w} \times \frac{\beta_w}{\nu_w} \\ R_0^m &= \frac{\alpha_m}{\gamma_u + \epsilon + \mu_u} \times \frac{\beta_m}{\nu_w} \times \frac{\delta}{\epsilon + \mu_u + \delta} \\ R_0^u &= \frac{\alpha_u}{\gamma_u + \mu_u} \times \frac{\beta_u}{\nu_u} \times \left(1 - \frac{\delta}{\epsilon + \mu_u + \delta}\right). \end{aligned} \tag{13}$$

R_0^u is the basic reproductive number in the urban area, while $R_0^s = R_0^m + R_0^w$ denotes the basic reproduction number in the wilderness. We define the basic reproductive number associated to the disease-free equilibrium $\bar{P}_0$ as

$$R_0 = \max\left(R_0^s, R_0^u\right). \tag{14}$$

TABLE 1 Summary of the model parameters (*allowed to vary)

Symbol	Meaning	Value	Reference
μ_w	Mortality rate of monkeys (alouattas)	0.0048 month^{-1}	Guessed
γ_w	Recovery rate in monkeys	3 months^{-1}	Moreno et al[15]
ν_w	Mortality rate of *Haemagogus*	0.46 month^{-1}	Raimundo et al[7]
b_w	Biting rate of *Haemagogus*	6 month^{-1}	Chadee et al[16]
$\bar{\beta}_w$	Trans. coefficient from *Haemagogus* to forest animals	0.4	Moreno et al[15]
$\bar{\beta}_m$	Trans. coefficient from *Haemagogus* to migrant humans	0.25	Guessed
$\bar{\alpha}_w$	Trans. coefficient from monkeys to *Haemagogus*	0.4	Moreno et al[15]
$\bar{\alpha}_m$	Trans. coefficient from migrant humans to *Haemagogus*	0.4	Guessed
μ_u	Mortality rate of humans	0.0012 month^{-1}	Guessed
γ_u	Humans recovery rate	4 month^{-1}	PAHO[17]
ϵ	Migration rate of humans from forest area	1.0* month^{-1}	Guessed
δ	Migration rate of humans to forest area (month^{-1})	0.02* month^{-1}	Guessed
ν_u	Mortality rate of *Aedes*	0.913 month^{-1}	Dengue Virus Net[18]
b_u	Biting rate of *Aedes*	6 month^{-1}	Seawright et al[19]
$\bar{\beta}_u$	Transmission coefficient from *Aedes* to humans	0.2	Guessed
$\bar{\alpha}_u$	Transmission coefficient from humans to *Aedes*	0.25	Johnson et al[20]

From Theorem 2 in the work of van den Driessche and Watmough,[22] the next result is established.

Theorem 1. *The equilibrium $\bar{P}_0$ is locally asymptotically stable if $R_0 < 1$ and unstable if $R_0 > 1$.*

Furthermore, following the work of Shuai and van den Driessche,[23] global stability of the disease-free equilibrium for $R_0 < 1$ can be established using a suitable Lyapunov function. This implies that, if the basic reproductive number is less than one, the infection cannot be maintained whatever the initial number of infectious.

Next, we shall study the situation where the reproduction number is bigger than one. We have to consider three cases.

I. YF circulating in the wild region: $R_0^s > 0$, $R_0^u < 0$.
II. YF circulating in the urban area: $R_0^s < 0$, $R_0^u > 0$.
III. YF circulating in both regions $R_0^s > 0$, $R_0^u > 0$.

Case I. This case is the most important one, since it implies that YF could be introduced from the forest to the urban areas. The basic reproduction numbers satisfy $R_0^s > 1$ and $R_0^u < 1$, and Equations (4), (5), (6) have an equilibrium $\bar{P}_w = (\bar{s}_w, \bar{\zeta}_w, \bar{y}_w, \bar{s}_m, \bar{\zeta}_m, \bar{s}_u, \bar{\zeta}_u, \bar{y}_u)$ with

$$\begin{aligned}
\bar{s}_w &= \frac{\mu_w}{\beta_w \bar{y}_w + \mu_w} \\
\bar{\zeta}_w &= \frac{\beta_w \mu_w \bar{y}_w}{(\beta_w \bar{y}_w + \mu_w)(\gamma_w + \mu_w)} \\
\bar{s}_m &= \frac{\delta \mu_u}{(\delta + \mu_u)\beta_m \bar{y}_w + \mu_u(\delta + \epsilon + \mu_u)} \\
\bar{\zeta}_m &= \frac{\delta \mu_u \beta_m \bar{y}_w}{[\delta + \mu_u)\beta_m \bar{y}_w + \mu_u(\delta + \epsilon + \mu_u)](\gamma_u + \epsilon + \mu_u)}, \\
\bar{s}_u &= \frac{\epsilon \bar{s}_m + \mu_u}{\delta + \mu_u} \\
\bar{\zeta}_u &= \frac{\epsilon \bar{\zeta}_m}{\gamma_u + \theta_u} \\
\bar{y}_u &= \frac{\alpha_u \bar{\zeta}_u}{\bar{\zeta}_u + \nu_u}
\end{aligned} \tag{15}$$

and $\bar{y}_w$ satisfies

$$c_1 \bar{y}_w^2 + c_2 \bar{y}_w + c_3 = 0, \tag{16}$$

$$\begin{aligned}
c_1 &= -\left[\beta_w \mu_w (\delta + \mu_u) R_0^w + \beta_w (\delta + \epsilon + \mu_u) R_0^m\right], \\
c_2 &= \mu_w \beta_w (\delta + \mu_u)\left(R_0^w - 1\right) + (\delta + \epsilon + \mu_u)\beta_w \left(R_0^m - 1\right) - \mu_u \mu_w (\delta + \epsilon + \mu_u) R_0^w, \\
c_3 &= \mu_u \mu_w (\delta + \epsilon + \mu_u)\left(R_0^m + R_0^w - 1\right).
\end{aligned} \tag{17}$$

If $R_0^s = R_0^w + R_0^m < 1$, the constants c_i, $i = 1, 2, 3$, are all negative, and thus, the quadratic equation does not have positive solutions. If $R_0^s > 1$, $c_3 > 0$, then by Descartes' rule, the equation has a unique positive root $\bar{\zeta}_w$. In the case $R_0^s = 1$, $c_3 = 0$, and the quadratic equation has a negative root and a zero root. Substituting $\bar{y}_w$ in (15), we shall obtain the coordinates of $\bar{P}_w$. In the following,we will prove that solutions of systems (4), (5), (6) with positive initial condition evolve to the endemic equilibrium $\bar{P}_w$. For this end, we will use a comparison theorem and a standard Lyapunov function for the variables $s_w, i_w, 1 - y_w, y_w, s_m$, and i_m.

Since $0 \leq s(t) \leq 1$, it follows from (6) the differential inequalities

$$\begin{aligned} \frac{di_u}{dt} &\leq \beta_u y_u - (\gamma_u + \mu_u) i_u + \epsilon i_m \\ \frac{dy_u}{dt} &\leq \alpha_u y_u - \nu_u y_u. \end{aligned} \tag{18}$$

Consider now the linear nonhomogeneous ODE system

$$\begin{aligned} \frac{d\tilde{\zeta}_u(t)}{dt} &= \beta_u \tilde{y_u} - (\gamma_u + \mu_u)\tilde{\zeta}_u + \epsilon i_m \\ \frac{d\tilde{y}_u(t)}{dt} &= \alpha_u \tilde{y_u} - \nu_u \tilde{y_u}. \end{aligned} \tag{19}$$

Since $R_0^u < 1$, the eigenvalues of the homogeneous system associated to (19), given by

$$\begin{aligned} \frac{d\tilde{\zeta}_u(t)}{dt} &= \beta_u \tilde{y_u} - (\gamma_u + \mu_u)\tilde{\zeta}_u \\ \frac{d\tilde{y}_u(t)}{dt} &= \alpha_u \tilde{y_u} - \nu_u \tilde{y_u}, \end{aligned} \tag{20}$$

have negative real part, which implies $\tilde{\zeta}_u(t)$ and $\tilde{y}_u(t)$ go to zero as $t \to \infty$. By a comparison principle,[24] it follows that $i_u(t)$ and $y_u(t)$ solutions of

$$\begin{aligned} \frac{di_u}{dt} &= \beta_u s_u y_u - (\gamma_u + \mu_u) i_u \\ \frac{dy_u}{dt} &= \alpha_u s_u y_u - \nu_u y_u \end{aligned}$$

also approach zero when $t \to \infty$. Therefore, the right-hand side of the equation for i_u in system (6) becomes $\epsilon\bar{\zeta}_m$, which implies that $i_u(t) \to f(\bar{\zeta}_m)$. Since there is only a nontrivial equilibrium when $R_0^u < 1$ and $R_0^s > 1$, then $f(\bar{\zeta}_m) = \bar{\zeta}_u$. It follows that $s_u \to \bar{s}_u$, and $y_u \to \bar{y}_u$.

In the following, we will assume that the variables s_u, i_u, y_u are already in the steady state. Global stability of $\bar{P}_w$ is proved using the following Lyapunov function W in the variables $s_w, i_w, v_w = 1 - y_w, s_m$, and y_w

$$\begin{aligned} W = {} & b_1 \left(s_w - \bar{s}_w - \bar{s}_w \ln \frac{s_w}{\bar{s}_w} \right) + b_2 \left(i_w - \bar{\zeta}_w - \bar{\zeta}_w \ln \frac{i_w}{\bar{\zeta}_w} \right) \\ & + b_3 \left(v_w - \bar{v}_w - \bar{v}_w \ln \frac{v_w}{\bar{v}_w} \right) + b_4 \left(y_w - \bar{y}_w - \bar{y}_w \ln \frac{y_w}{\bar{y}_w} \right) \\ & + b_5 \left(s_m - \bar{s}_m - \bar{s}_m \ln \frac{s_m}{\bar{s}_m} \right) + b_6 \left(i_m - \bar{\zeta}_m - \bar{\zeta}_m \ln \frac{i_m}{\bar{\zeta}_m} \right), \end{aligned} \tag{21}$$

where

$$b_1 = b_2 = 1, \quad b_3 = b_4 = \frac{\beta_w \bar{s}_w \bar{y}_w}{\alpha_w \bar{v}_w \bar{\zeta}_w}, \quad b_5 = b_6 = \frac{\alpha_m \bar{v}_w \bar{\zeta}_m}{\beta_m \bar{s}_m \bar{y}_w} b_3, \tag{22}$$

and v_w is the proportion of susceptible *Haemagogus*. The orbital derivative of W is given by

$$\begin{aligned}\dot{W} = & b_1\left(1-\frac{\bar{s}_w}{s_w}\right)(\mu_w - \beta_w s_w y_w - \mu_w s_w)\\ & + b_2\left(1-\frac{\bar{\zeta}_w}{i_w}\right)(\beta_w s_w y_w - (\gamma_w+\mu_w) i_w)\\ & + b_3\left(1-\frac{\bar{v}_w}{v_w}\right)(\nu_w - \alpha_w v_w i_w - \alpha_m v_w i_m - \nu_w v_w)\\ & + b_4\left(1-\frac{\bar{y}_w}{y_w}\right)(\alpha_w v_w i_w - \alpha_m v_w i_m - \nu_w y_w)\\ & + b_5\left(1-\frac{\bar{s}_m}{s_m}\right)(\delta \bar{s}_u - \beta_m s_m y_w - (\epsilon s_m + \mu_u) s_m)\\ & + b_6\left(1-\frac{\bar{\zeta}_m}{i_m}\right)(\beta_m s_m y_w - (\gamma_u + \epsilon + \mu_u) i_m)\,.\end{aligned} \tag{23}$$

From systems (4) and (5) at equilibrium, we obtain the following relations:

$$\begin{aligned}\mu_w &= \beta_w \bar{s}_w \bar{y}_w + \mu_w \bar{s}_w\\ \gamma_w + \mu_w &= \frac{\beta_w \bar{s}_w \bar{y}_w}{\bar{\zeta}_w}\\ \nu_w &= \alpha_w \bar{v}_w \bar{\zeta}_w + \alpha_m \bar{v}_w \bar{\zeta}_m + \nu_w \bar{v}_w\\ \nu_w &= \frac{\alpha_w \bar{v}_w \bar{\zeta}_w}{\bar{y}_w} + \frac{\alpha_m \bar{v}_w \bar{\zeta}_m}{\bar{y}_w}\\ \delta \bar{s}_u &= \beta_m \bar{s}_m \bar{y}_w + (\epsilon + \mu_u)\bar{s}_m.\end{aligned} \tag{24}$$

Substituting the constants b_i, and the parameters μ_w up to $\delta \bar{s}_u$ in the Lyapunov derivative (23), we obtain, after several calculations and simplifications,

$$\begin{aligned}\dot{W} = & -b_1 \mu_w \frac{(s_w - \bar{s}_w)^2}{s_w} - b_3 \nu_w \frac{(v_w - \bar{v}_w)^2}{s_m} - b_5\left[\epsilon + \mu_w \frac{(v_w - \bar{s}_m)^2}{v_w}\right]\\ & - A_1\left(\frac{\bar{s}_w}{s_w} + \frac{\bar{s}_v}{v_w} + \frac{s_w y_w \bar{\zeta}_w}{\bar{s}_w \bar{y}_w i_w} + \frac{v_w i_w \bar{y}_w}{\bar{s}_v \bar{\zeta}_w y_w} - 4\right)\\ & - A_2\left(\frac{\bar{s}_v}{v_w} + \frac{\bar{s}_m}{s_m} + \frac{v_w i_m \bar{y}_w}{\bar{s}_v \bar{\zeta}_m y_w} - 4\right),\end{aligned} \tag{25}$$

where $A_1 = \beta_w \bar{s}_w \bar{y}_w$ and $A_2 = \alpha_m \beta_w \bar{s}_w \bar{\zeta}_m / \left(\alpha_w \beta_m \bar{s}_m \bar{\zeta}_w\right)$.

Using the fact that the geometric mean is less or equal than the arithmetic mean, it can be proved that the expressions inside the parenthesis of the last two terms in (25) are greater or equal to zero, and they are zero if and only if $s_w = \bar{s}_w$, $i_w = \bar{\zeta}_w$, $v_w = \bar{v}_w$, $y_w = \bar{y}_w$, $s_m = \bar{s}_m$, and $i_m = \bar{\zeta}_m$. Therefore, $\dot{W} \leq 0$, and $\dot{W} = 0$ only for $\bar{s}_w$, $\bar{\zeta}_w$, $\bar{s}_v$, $\bar{y}_w$, $\bar{s}_m$, and $\bar{\zeta}_m$. This implies that all trajectories with positive initial conditions approach $\bar{P}_w$ as $t \to \infty$.

From the above results, we have the following.

Theorem 2. *If $R_0^s > 1$, the system given by (4) and (5) has a unique endemic equilibrium $\bar{P}_w$, which is globally asymptotically stable.*

The urban equilibrium is found by substituting $\bar{s}_m$ and $\bar{\zeta}_m$ in system (4) and solving the resulting steady state equations. Since $R_0^s > 1$, regardless of the value of R_0^u, there is always a positive number of infected individuals in the urban area due to the infective migrant population that depends on the migration rates.

Case II. We assume YF is circulating only in the urban area, that is, $R_0^s < 1$, $R_0^u > 1$. In this case, we have the endemic equilibrium

$$\bar{P}_u = (s_w^*, i_w^*, y_w^*, s_m^*, i_m^*, s_u^*, i_u^*, y_u^*) \tag{26}$$

with coordinates given by $s_w^* = 1$, $i_w^* = 0$, $y_w^* = 0$, $s_m^* = \frac{\delta s_u^*}{\epsilon + \mu_u}$, $i_m^* = 0$, and

$$s_u^* = \frac{\mu_u}{\beta_u y_u^* + \frac{\mu_u(\epsilon+\delta+\mu_u)}{\epsilon+\mu}}, \quad i_u^* = \frac{\beta_u s_u^* y_u^*}{(\gamma_u + \mu_u)}, \quad y_u^* = \frac{\mu_u(\epsilon + \delta + \mu_u)\left(R_0^u - 1\right)}{\beta_u(\epsilon + \mu_u) + \mu_u(\epsilon + \delta + \mu)R_0^u}. \tag{27}$$

Using the function

$$Q(x) = \frac{\alpha_w}{\nu_w}\frac{1}{\gamma_w + \mu_w}\frac{1}{\eta} i_w + \frac{1}{\nu_w} y_w + \frac{\alpha_m}{\nu_w}\frac{1}{\gamma_u + \epsilon + \mu_u}\frac{1}{\eta} i_m, \tag{28}$$

with $\eta = \sqrt{R_0^s}$, which is indeed a Lyapunov function, it can be proved that

$$\lim_{t\to\infty}(s_w(t), i_w(t), y_w(t), s_m(t), i_m(t)) = (1, 0, 0, s_m^*, 0)\,.$$

Assuming that systems (4) and (5) are already in the asymptotic form, system (6) becomes

$$\begin{aligned}
\frac{ds_u}{dt} &= \mu_u\left(1 - \frac{\delta s_m^*}{\epsilon + \mu_u}\right) - \beta_u s_u y_u - \mu_u s_u \\
\frac{di_u}{dt} &= \beta_u s_u y_u - \gamma_u i_u - \mu_u i_u \\
\frac{dy_u}{dt} &= \alpha_u(1 - i_v) i_u - \nu_u y_u,
\end{aligned}$$

substituting $s_m(t)$ and $i_m(t)$ by s_m^* and i_m^*, respectively.

As in Case 1, it can be proved that positive solutions $(s_u(t), i_u(t), y_u(t))$ approach (s_u^*, i_u^*, y_u^*) when $t \to \infty$ using the Lyapunov function

$$\begin{aligned}
V = a_1\left(s_u - s_u^* - s_u^* \ln\frac{s_u}{s_u^*}\right) + a_2\left(i_u - i_u - i_u^* \ln\frac{i_u}{i_u^*}\right) \\
+ a_3\left(v_u - v_u^* - v_u^* \ln\frac{v_u}{v_u^*}\right) + a_4\left(y_u - y_u - y_u^* \ln\frac{y_u}{y_u^*}\right),
\end{aligned} \tag{29}$$

where

$$v_u = 1 - y_u \quad a_1 = a_2 = \frac{1}{\beta_u s_u^*}, \quad a_3 = a_4 = \frac{1}{\alpha_u s_v^* i_u^*}.$$

Case III. YF in urban and wilderness is well established. We assume that the number of migrants infected in the forest that return to the city is very small compared to the number of urban YF cases, which allow us to take $\delta \ll 1$. In this case, we obtain two decoupled systems for the urban and wild cycles and the following result is proved.

Theorem 3. *If $R_0^u \le 1$, all trajectories of system (4) with initial conditions $(s_u(0), i_u(0), y_u(0))$ in $\Omega = \{(s_u, i_u, y_u) | 0 \le s_u, 0 \le i_u, s_u + i_u \le 1, 0 \le y_u \le 1\}$ approach the disease-free equilibrium. If $R_0^u > 1$, the disease free equilibrium becomes unstable, and all trajectories with initial conditions $(s_u(0), i_u(0), y_u(0))$ in Ω with $i_0 > 0$ or $y_0 > 0$ approach a unique endemic equilibrium. (See the work of Esteva and Vargas[13] for a proof).*

Interchanging u by w, the above result is also valid for the forest system given by (4).

4 | RISK OF YF

In this section, we will find an expression to determine the risk of YF among migrants in the forest regions, assuming that this disease is endemic there and it is not circulating in the urban area. For this, let S_m be the number of migrant humans, and we assume $R_0^s = R_0^w + R_0^m > 1$. On average, an *Haemagogus* bites an individual (monkey or human) at a rate $b_w/(N_w + N_m)$, and the probability that this bite is given to a susceptible migrant human is $N_m/(N_w + N_m)$. Therefore, one *Haemagogus* bites a susceptible migrant human at a rate

$$\frac{b_w}{N_w + N_m} \times \frac{S_m}{N_w + N_m}. \tag{30}$$

On the other hand, a susceptible migrant human can be bitten by Y_w infectious *Haemagogus*, then the total rate of potentially infectious bites p_{in} is

$$p_{in} = \frac{b_w}{N_w + N_m} \times \frac{S_m}{N_w + N_m} \times Y_w, \tag{31}$$

and during the period of time staying in the forest region $(1/\epsilon)$, a migrant susceptible human receives an average of p_{in}/ϵ bites from all infectious *Haemagogus*.

Remembering that $\bar{\beta}_m$ is the probability of migrant humans to be infected by YF in the forest region and replacing this parameter by β_m given in (7) the risk, r_m, of susceptible migrant humans to be infected is

$$r_m = \frac{1}{\epsilon}(\bar{\beta}_m \times p_{in}) = \frac{1}{\epsilon}\left(\beta_m \frac{S_m}{N_w + N_m}\right) y_w,$$

where $y_w = \frac{Y_w}{V_w}$.

If all migrants are susceptible $(S_m = N_m)$ and forest YF is at the endemic steady state, we call r_m *the potential risk of YF*, and it becomes

$$r_m = \frac{\beta_m \frac{N_m}{N_w + N_m}}{\epsilon} \bar{y}_w, \tag{32}$$

where $\bar{y}_w$ is given by the positive solution of Equation (16).

5 | NUMERICAL SIMULATIONS

In the numerical simulations presented in this section, we show the temporal course of the dynamical variables for two values of the migrant rate δ, assuming that migrants stay one month in the forest $(\epsilon = 1)$. We assume that the total populations satisfy $N_m = \delta N_u/(\epsilon + \mu)$, $N_w = 0.5N_u$, $V_w = 3N_w$, and $V_u = 2N_u$. At the initial time, we assume that the urban area is free of YF, and the forest region is already at endemic equilibrium. Taking the values given in Table 1, we obtain $R_0^u = 0.9$, $R_0^m = 0$, and $R_0^w = R_0^s = 12$. For these values, all populations remain at equilibrium, the risk of a human migrant to contract YF is zero, and the incidence of YF in the forest region is high. In Figure 1, we assume that $\delta = 0.02$ for $t > 0$, that is, 2% of the urban population enters to the forest area per month. The numerical simulations show an initial epidemic outbreak in the urban area that decreases to an endemic proportion below to 2×10^{-4} corresponding to the proportion of infected migrants (see i_u graph in Figure 1).

Figure 2 illustrates the time course of the infective proportions when the migrant proportion increases to $\delta = 0.35$. In this case, the risk of contracting the infection elevates to $p_m = 0.004$, and the urban basic reproductive number R_0^u increases to 1.5 given rise to big YF urban outbreaks (see i_u graph in Figure 2). The disease can remain endemic in the urban area without the presence of infected migrants since $R_0^u > 1$. The basic reproductive number R_0^m increases to 0.57, and R_0^w decreases to 4.3, which indicates that the infection is moving through the migrants from the forest area to the city.

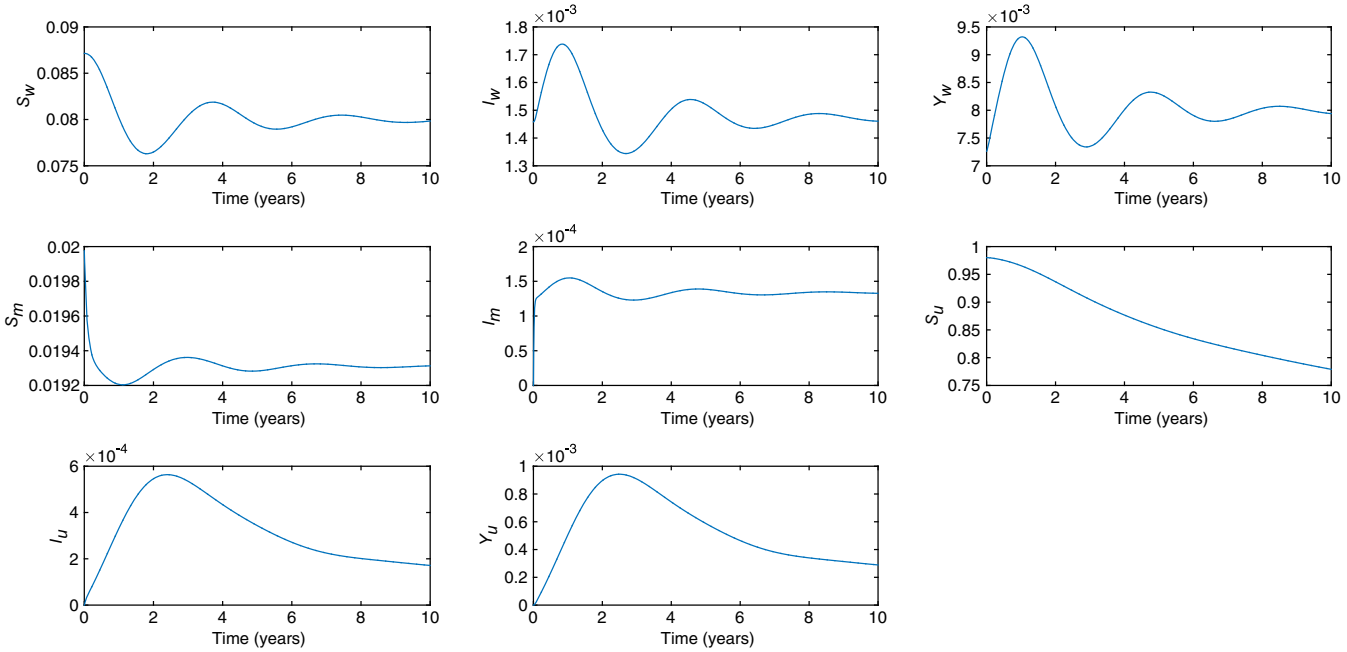

FIGURE 1 Numerical simulations of Equations (4), (5), and (6) showing the temporal evolution of the proportions of susceptible and infective populations of wild, urban, and migrant populations. In this case, $\delta = 0.02$, $\epsilon = 1$, $R_0^u = 1.0$, $R_0^m = 0.08$, $R_0^w = 11.5$, $r_m = 0.001$, and the rest of the parameters are as in Table 1.

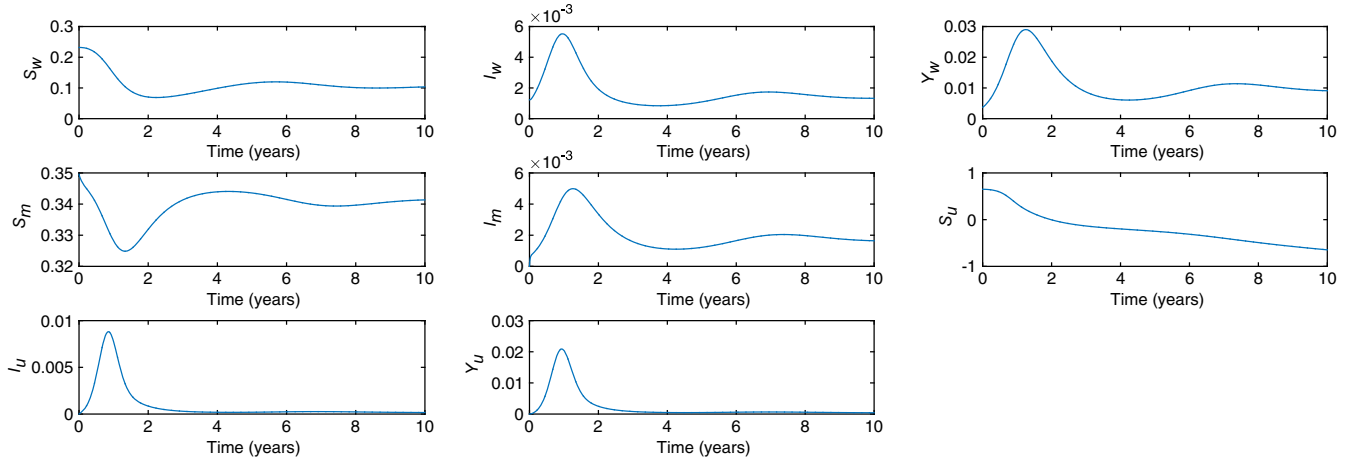

FIGURE 2 Numerical simulations of Equations (4), (5), and (6) showing the temporal evolution of the proportions of susceptible and infective populations of wild, urban, and migrant populations. In this case, $\delta = 0.35$, $\epsilon = 1$, $R_0^u = 1.4$, $R_0^m = 0.57$, $R_0^w = 4.3$, and $r_m = 0.004$

6 | CONCLUSIONS

One of the main factors behind the distribution and persistence of the diseases is the human migration. Migration allows the pathogens to invade places where the disease could disappear if they were isolated. Currently, diseases travel much faster due to large human movements, and there is a need to take rapid measures to control their invasion to virgin territories. In particular, vector-borne diseases have expanded geographically due to the migration of humans, animals, and insects. One of these diseases is YF, which can be taken to the cities due to migrants that travel to regions where this disease is mainly maintained by a cycle between wild animals and vectors.

In order to evaluate the risk of introduction of YF, in this work, we formulated an ODE model considering humans that live in an urban area and some of them enter for different purposes to the forest regions where YF is maintained

mainly by monkeys and *Haemagogus spp.* We based our study on the basic reproduction numbers, R_0^w, R_0^m, and R_0^u of monkeys, migrant humans, and urban humans, respectively. These numbers represent estimations of the average number of infections caused by an infective individual when introduced in a whole susceptible population. Besides, we denote by $R_0^s = R_0^w + R_0^m$ the number of secondary cases given by sylvatic monkeys and migrant humans in the forest area. When $R_0^s < 1$ and $R_0^u < 1$, both forest and urban YF epidemics fade out. Furthermore, we have the following cases.

Case I. R0s>1 and $R_0^u < 1$. Forest YF outbreaks lead to urban YF cases carry out by infectious migrant humans i_m.
Case II. $R_0^s < 1$ and $R_0^u > 1$. Forest YF fades out, but urban YF epidemics occurs.
Case III. $R_0^s > 1$ and $R_0^u > 1$. Both forest and urban YF cases reach high levels of indigenous cases independently of migrants due to the occurrence of intrinsic forest and urban epidemics.

Based in our model, we found an expression named r_m to estimate the risk of acquiring YF by migrants during their stay in the forest region. It was calculated numerically for several conditions, and the results showed that r_m increases when the number of migrants increases. Furthermore, the simulations corroborated that, if the migrants proportion is high, the urban reproductive number can increase to levels that lead to epidemics of considerable magnitude and/or to the establishment of the disease. However, if the urban area is relatively safe (ie, R_0^u sufficiently low), the return of infectious migrants can sustain only very low incidence of YF.

At this point, it is worthy to mention that our concept of YF risk is different to the one that is given in the work of Massad et al.[11] In this paper, the authors consider one infected person entering in a city free of YF, while we consider the opposite, namely, healthy persons going into an environment with YF.

ACKNOWLEDGEMENTS

We thank two anonymous referees for their careful reading. Their valuable suggestions greatly improved the quality of this paper. This work was partially supported by Project IN-113716 PAPIIT-UNAM.

ORCID

Lourdes Esteva https://orcid.org/0000-0001-7966-1546
Cristobal Vargas https://orcid.org/0000-0002-5265-3797
Hyun Mo Yang https://orcid.org/0000-0002-1711-363X

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AUTHOR BIOGRAPHIES

María de Lourdes Esteva received the Bachelor's degree in Mathematics from the Universidad Nacional Autónoma de México, Mexico City, Mexico; the Master's degree in Sciences from Rutgers University, New Brunswick, NJ; and the PhD degree in Sciences from the Centro de Investigación y Estudios Avanzados (Cinvestav), Instituto Politécnico Nacional, Mexico City. She is currently a Full-Time Professor with the Faculty of Sciences, Universidad Nacional Autónoma de México. Her research interests are in biomathematics, mathematical epidemiology, and differential equations.

Cristobal Vargas received the Bachelor's degree in Sciences from the Escuela Superior de Física y Matemáticas, Instituto Politécnico Nacional (IPN), Mexico City, Mexico; the Master's degree in Sciences from the University of Wisconsin-Madison, Madison, WI; and the PhD degree in Mathematical Sciences from The University of Texas at Arlington, Arlington, TX. He is currently a Full-Time Professor with the Department of Automatic Control, Centro de Investigación y Estudios Avanzados (Cinvestav), IPN. His research interests are in applied mathematics and numerical analysis.

Hyun Mo Yang received the Bachelor's degree in Physics and the Master's and PhD degrees in Sciences from the Universidade de São Paulo, São Paulo, Brazil. He is currently a Full-Time Professor with the Department of Applied Mathematics, Universidade Estadual de Campinas, Campinas, Brazil. His research interests are in biomathematics, mathematical epidemiology, and differential equations.

How to cite this article: Esteva L, Vargas C, Yang HM. A model for yellow fever with migration. *Comp and Math Methods*. 2019;e1059. https://doi.org/10.1002/cmm4.1059