On the dynamics of dengue epidemics from large-scale information

Annelise Tran\textsuperscript{a,}\textsuperscript{*}, Marcel Raffy\textsuperscript{b}

\textsuperscript{a}Unité Epidemiologie et Ecologie des Maladies Animales, Centre de Coopération Internationale en Recherche Agronomique pour le Développement, Montpellier, France

\textsuperscript{b}Laboratoire des Sciences de l'Image, de l'Informatique et de Télédétection, Strasbourg, France

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Abstract

A model for the spatial and temporal dynamics of dengue fever is proposed in this article. The vector population dynamics is derived from a diffusion equation that is based on environmental parameters at the scale of a remote-sensing image. Vectors and hosts populations are then classically divided into compartments corresponding to their respective disease status. The transmission processes between hosts and vectors are described by a set of differential equations.

The link between the vector population diffusion model and the compartmental model enables one to describe both the spatial and temporal dynamics of the disease. Simulations in artificial and actual landscapes show the advantage of using remotely sensed and complementary meteorological data for modelling in a realistic way the geographic spread of a vector-borne disease such as dengue fever.

1. Introduction

The geographic spread of epidemics is even less well understood and much less well studied than the temporal development and control of diseases and epidemics (Murray, 2003). Nevertheless, modelling in a realistic way the spatial dynamics of a disease could be of particular relevance for the control of infectious diseases and the understanding of the transmission process. The issue is the manner in which to model the spatial evolution of a disease.

Three different kinds of models for the geotemporal development of epidemics emanate from existing research (Bolker, 1997). The first approach uses “box models” and consists of spatially partitioning the population within compartments (city, neighbourhood) (Anderson and May, 1991). Contacts within a box are assumed to be homogeneously distributed, and the links between boxes are defined according to specific parameters such as geographic mobility (Sattenspiel and Dietz, 1995). In the second approach, the geographic spread of an epidemic is described by “continuous-space models” using a diffusion equation (Källen et al., 1985). More recently, “network models” have been developed in order to reflect all the disease-causing contact processes within a population (Newman, 2002).

Although these models take into account the spatial dimension of an epidemic process, they are not directly linked with data that characterize the reality of the ground, such as, for example, remotely sensed data. Thus, they are not adapted to simulate in a realistic way the geographic spread of an epidemic.

The use of remotely sensed data (satellite images, aerial photographs) in epidemiological studies has become more and more frequent in the last decades: since it provides information on the environment, remote sensing offers an important potential for the study of diseases related to environmental conditions (Cline, 1970; Hay et al., 1997; Curran et al., 2000). Indeed, there is a wide range of remote sensors that provide information about sea and land surfaces at different spatial scales; the use of Geographic Information Systems (GIS) allows researchers to integrate, analyse and display easily spatially referenced data. In most of the studies, a statistical analysis is performed in
order to test the relationship between environmental parameters derived from the images and epidemiological or entomological field data. The inversion of the model then leads to the creation of risk areas’ maps (Beck et al., 1997; Boone et al., 2000; Abdel-Rahman et al., 2001; Mckenzie et al., 2002), but this type of application does not enable one to follow both the spatial and temporal development of a disease.

Finally, remote-sensing data have been proven to be the right scale for the description of insect diffusion (Raffy and Tran, 2005). In this article, we shall approach the issue of a vector-borne disease with a non-static diffusion model based on environmental data that can be derived from remote-sensing images, with a typical pixel size bigger than $20 \times 20 \text{m}^2$. The goal of this paper is more to present and discuss the method than a specific application. The disease studied is dengue fever.

The model is based on the insect population diffusion model described previously (Raffy and Tran, 2005) for the simulation of the vector populations. This model for vector populations is then associated with a compartmental model, which is the natural model used to describe the temporal dynamics of both hosts and vectors populations according to their disease status. We shall see that the link with the vector population diffusion model allows to describe both spatial and temporal dynamics of the disease.

Section 2 presents the epidemiology of dengue fever and previous dengue models; in Section 3, we restate the insect diffusion model. Section 4 establishes the model for the dynamics of a vector-borne disease. Two applications are presented in Section 5 and the method is discussed in Section 6.

2. Dengue fever: epidemiology and modelling

Dengue fever is an arboviral disease transmitted to humans through mosquito bites from the *Stegomyia* genus. It is estimated that 100 million individuals are infected every year (WHO, 1997). There are different forms of dengue infection: an asymptomatic form, dengue fever (DF), and severe forms, dengue haemorrhagic fever (DHF) and dengue shock syndrome (DSS). Four serotypes of the dengue virus have been identified (dengue-1, dengue-2, dengue-3, dengue-4). Infection by one of the serotypes provokes permanent immunity to it, but only a temporary cross-immunity to the others. The *Stegomyia aegypti* mosquito is the most important vector of the dengue virus. For transmission to occur, the female mosquito must bite an infected human during the viraemic phase of illness which lasts for 4–5 days. It becomes itself infective after the extrinsic incubation period (about 10 days). The intrinsic incubation period—by humans—lasts for 5–7 days (McBride and Bielefeldt-Ohmann, 2000). Infected female mosquitoes can also transmit the virus to its descendents (vertical transmission process) (Fig. 1). Today, as there is no vaccine nor specific treatment available, the only solution for dengue surveillance is vector control strategy.

Mathematical models can provide a useful tool to better understand the mechanisms that allow the spread of a dengue epidemic and therefore to increase the efficiency of the vector control strategy.

The natural way to model a vector-borne disease such as dengue fever is to divide both host and vector populations into distinct classes corresponding to each disease status: susceptible individuals (S), exposed (or latent) (E), infective (I), and resistant (R) (Murray, 2003). Then, a set of differential equations describes the flow between compartments. It enables one to follow the temporal evolution of the numbers of individuals (both vectors and hosts) in the different classes. These compartmental models are mostly used for modelling dengue fever transmission, in order to determine the conditions of the endemic equilibrium (Esteva and Vargas, 1998) or to study the influence of different parameters on dengue transmission such as the influence of age structure (Pongsumpun and Tang, 2001), the impact of Ultra-Low Volume (ULV) insecticide applications (Newton and Reiter, 1992), the competition between two virus strains (Feng and Velasco-Hernández, 1997), and the effect of vertical and mechanical transmission in the vector population (Esteva and Vargas, 2000). More recently, a model using a network approach was proposed to study the influence of host movements on the dengue spread (Schmit et al., 2003). The second approach to model dengue is to use an individual-based model for the *S. aegypti* vector (Focks et al., 1995). Finally, dengue transmission risk maps have been derived from different maps of environmental conditions favourable to *S. aegypti* (Carbajal et al., 2001).

In the following approach, we propose to model both the spatial and temporal dynamics of dengue by spatializing different classes of vector and host populations (susceptible, exposed, infective, and, for humans, resistant). We consider only the main vector species, *S. aegypti*. The model relies on remote-sensing data in order to describe in a realistic way the geographic spread of the disease. Indeed, remotely sensed large-scale data have been shown to have the resolution adapted for the description of the diffusion of flying insects, like flies or mosquitoes (Raffy and Tran, 2005). Thus, the elementary unit for the calculations is the image pixel.

First, the vector population dynamics is derived from a diffusion equation that is based on environmental parameters and a remote-sensing image. Then, for each pixel, a
3. Insects diffusion

The dynamics of dengue relies on the *S. aegypti* mosquitoes’ dynamics, which is related to environmental conditions (e.g. temperature, rainfall, relative humidity, vegetation, human density, type of housing). Thus, we shall first examine the *S. aegypti* mosquitoes’ dynamics. In this section, we shall restate the model for insect diffusion. In Section 4, we shall consider the whole epidemic process.

### 3.1. Study area

The area where the study takes place is a region of a few kilometres’ size. We shall call this area $A$ and $\Gamma$ its boundary. Environmental data on area $A$ can be derived from ground measurements, meteorological stations or remotely sensed data. For example, the High-Resolution Visible and InfraRed (HRVIR) multispectral sensor on Satellite Pour l’Observation de la Terre (SPOT) satellites provide information on regions with a width of 60 km and the given spectral information allows to discriminate and map several land cover types. The process is called image classification. The pixel (or picture element) size, corresponding to the smallest area for which the sensor can record data, is $20 \times 20$ m.

### 3.2. The diffusion equation

The global population density of the vectors is estimated using the following diffusion equation (Raffy and Tran, 2005):

$$
\frac{\partial \rho(\Omega, t)}{\partial t} = \text{div} \left[ D_R(\Omega, t) \cdot \nabla \rho(\Omega, t) \right] \\
+ D_W(\Omega, t) \cdot \vec{w} \cdot \nabla \rho \\
+ \text{div} \left[ K_H \cdot \rho(\Omega, t) \cdot \nabla H(\Omega) \right]
$$

$$
\rho(\Omega, 0) = \rho_0(\Omega), \quad \Omega \in A, \quad t > 0,
$$

$$
\rho(\Omega, t) = 0, \quad \forall \Omega \in \Gamma.
$$

The various terms of this equation are as follows:

The average mosquito density of the pixel $\Omega$, at the instant $t$ is $\rho(\Omega, t)$. It is proven in (Raffy and Tran, 2005) that for insects having an average movement of 1 m or less per second, a pixel size of 20–30 m is sufficient to justify a diffusion process. This corresponds to SPOT satellites’ data.

The term $D_R(\Omega, t)$ is a tensor defined by the direct surface effect on insect diffusion. It reflects the landscape roughness on the insect flow, any wind effect or attractive effect excluded. The calculation of $D_R$ includes two operations. First, the image of the area $A$ is separated into significant classes. The knowledge of the visible and near-infrared channels of satellite data leads to separate the following regions: forest, grassland, dry soils, wet soils, cities (Schowengerdt, 1997; Cracknell and Hayes, 1993). Then the $2 \times 2$ $D_R$ tensor is computed as it is detailed in Raffy and Tran (2005).

The large-scale direction and intensity of wind are obtained from meteorological stations and interpolation techniques. This vector field $\vec{W}$ is then transformed by the wind tensor $D_W(\Omega, t)$ which takes into account the landscape roughness. In reality, $D_W(\Omega, t)$ is a $2 \times 2$ tensor correlated to $D_R$.

The term $H(\Omega)$ is the density of human beings in pixel $\Omega$; it causes an attractive force to the insect flux. The intensity of this force, oriented by the term $\nabla H$ in (1) is a function $K_H$ taking a value at each $\Omega$ in $A$. It is estimated by the knowledge of the attractive range of the human on the mosquitoes. Population density index maps can be derived from remote-sensing imagery (Tran et al., 2002).

The term $\alpha(\Omega, t)$ is the birth in $\Omega$ at time $t$; depending on the vector’s biology, it could be estimated from environmental parameters such as temperature, rainfall, and larval habitats availability. For an operational use of the model, on site campaigns associated to high-resolution satellites or air-borne data can enable one to estimate the position of the birth sites. For a simulation use, only the temporal variation of the insect numbers around hatching places is needed.

Finally, $\beta(\Omega, t)$ is the death term; it includes natural mortality and mortality due to insecticide pulverization. This term depends on the vectors density:

$$
\beta(\Omega, t) = m(\Omega, t) \cdot \rho(\Omega, t), \quad m \in [0; 1], \quad \Omega \in A, \quad t > 0.
$$

(2)

The mortality rate $m$ depends on meteorological conditions such as temperature and humidity and on the efficiency of insecticide treatment.

Problem (1) can be numerically solved within an appropriate time step of between 1 h and 1 day, the space discretization step being the pixel size. The details on this point are in Raffy and Tran (2005). Therefore, the set of equations (1) allows to simulate the spatiotemporal dynamics of a mosquito population independently from any presence of a virus.

### 4. Epidemic process

When a virus is introduced into the study site, vector and host populations can be divided into states corresponding to their disease status. A set of differential equations describes the flow between these states, whereas the spatial dynamics of vector populations is assessed by the previous insect diffusion model. Section 4.1 concerns the model for the vector population and Section 4.2 focuses on the host population.
4.1. Vector population

The vector population is divided into three classes, namely safe vectors [with a density of $\rho_s(\Omega, t)$], infected vectors during the extrinsic incubation period [$\rho_I(\Omega, t)$], and infected vectors that can transmit the virus [$\rho_T(\Omega, t)$]. Then we have

$$\rho(\Omega, t) = \rho_s(\Omega, t) + \rho_I(\Omega, t) + \rho_T(\Omega, t),$$

$$\Omega \in A, \ t > 0.$$  \hspace{1cm} (3)

A list of the model parameters is given in Table 1. We note $H(\Omega)$ the human density at the location $\Omega$. $H_S$, $H_I$, $H_V$, $H_R$ are, respectively, the number of susceptibles, infected during the incubation period, viraemic, and recovered persons (Table 1).

The three sub-populations, $\rho_S$, $\rho_I$ and $\rho_V$, follow the same dynamics as the entire vector population with transitions from one state to another. Thus, their densities can be derived from (1). Indeed, Eq. (1) describes the spatial and temporal dynamics of the global vectors populations of which the infected vectors population is a sub-group. The infection process and the transitions between the different disease states led to the addition of modified source and vanishing terms.

We will first detail the calculation of $\rho_I$ and $\rho_V$. Finally, $\rho_S$ is given by Eq. (3).

4.1.1. Infected vectors

The density of infected vectors $\rho_I$ follows (1) with the modified source and vanishing terms $z_I(\Omega, t)$ and $\beta_I(\Omega, t)$:

$$\frac{\partial \rho_I(\Omega, t)}{\partial t} = \text{div}[D_R(\Omega, t) \cdot \nabla \rho_I(\Omega, t)]$$

$$+ \text{div}[K_H \cdot \rho_I(\Omega, t) \cdot \nabla \text{grad} H(\Omega)]$$

$$= z_I(\Omega, t) - \beta_I(\Omega, t), \ \Omega \in A, \ t > 0,$$

$$\rho_I(\Omega, 0) = \rho_{I0}(\Omega), \ \Omega \in A,$$

$$\rho_I(\Omega, t) = 0, \ \forall \Omega \in \Gamma.$$  \hspace{1cm} (4)

$z_I(\Omega, t)$ corresponds to the number of mosquitoes that are infected after biting viraemic persons. It is the product of the density of susceptible vectors by the number of bites per day ($N_B$) and the probabilities of encountering and transmission from host to vector. The probability for a vector to meet a viraemic host in pixel $\Omega$ at time $t$ can be estimated by the rate $H_I(\Omega, t)/H(\Omega)$. The rate of transmission is noted $C_{HV}$ and is estimated by laboratory research.

$$z_I(\Omega, t) = C_{HV} \cdot N_B \cdot \rho_S(\Omega, t)$$

$$\cdot \frac{H_I(\Omega, t)}{H(\Omega)}, \ \Omega \in A, \ t > 0.$$  \hspace{1cm} (5)

$\beta_I(\Omega, t)$ is the disappearance term for infected mosquitoes during the extrinsic incubation period; it includes the mortality (notation: $\beta_{I1}$) and the number of infected mosquitoes that become infective after the extrinsic incubation period (notation: $\beta_{I2}$). Then,

$$\beta_I(\Omega, t) = \beta_{I1}(\Omega, t) + \beta_{I2}(\Omega, t), \Omega \in A, \ t > 0.$$  \hspace{1cm} (6)

Assuming that infected vectors have the same mortality rate as others vectors, their mortality rate can be derived from the global death term $\beta(\Omega, t)$:

$$\beta_{I1}(\Omega, t) = \beta(\Omega, t) \cdot \frac{\rho_I(\Omega, t)}{\rho(\Omega, t)}, \ \Omega \in A, \ t > 0.$$  \hspace{1cm} (7)

We shall call $\tau_{\text{EIP}}$ the extrinsic incubation period. Considering the whole area $A$, the number of infected

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Definition</th>
<th>Unit</th>
<th>Typical value</th>
</tr>
</thead>
<tbody>
<tr>
<td>$\rho$</td>
<td>Vector density</td>
<td>$[L]^2$</td>
<td>—</td>
</tr>
<tr>
<td>$\rho_s$</td>
<td>Density of susceptible vectors</td>
<td>$[L]^2$</td>
<td>—</td>
</tr>
<tr>
<td>$\rho_I$</td>
<td>Density of infected vectors during the extrinsic incubation period</td>
<td>$[L]^2$</td>
<td>—</td>
</tr>
<tr>
<td>$\rho_T$</td>
<td>Density of infected vectors that can transmit the virus</td>
<td>$[L]^2$</td>
<td>—</td>
</tr>
<tr>
<td>$H_S$</td>
<td>Density of susceptible persons</td>
<td>$[L]^2$</td>
<td>—</td>
</tr>
<tr>
<td>$H_I$</td>
<td>Density of infected persons during the incubation period</td>
<td>$[L]^2$</td>
<td>—</td>
</tr>
<tr>
<td>$H_V$</td>
<td>Density of viraemic persons</td>
<td>$[L]^2$</td>
<td>—</td>
</tr>
<tr>
<td>$H_R$</td>
<td>Density of recovered persons</td>
<td>$[L]^2$</td>
<td>—</td>
</tr>
<tr>
<td>$\tau_{\text{EIP}}$</td>
<td>Duration of the extrinsic incubation period</td>
<td>$[T]$</td>
<td>7–12 days</td>
</tr>
<tr>
<td>$\tau_{\text{IIP}}$</td>
<td>Duration of the intrinsic incubation period</td>
<td>$[T]$</td>
<td>4–6 days</td>
</tr>
<tr>
<td>$\tau_V$</td>
<td>Duration of the viraemic period</td>
<td>$[T]$</td>
<td>4–7 days</td>
</tr>
<tr>
<td>$\tau_E$</td>
<td>Mosquito development</td>
<td>$[T]$</td>
<td>12 days</td>
</tr>
<tr>
<td>$C_{VH}$</td>
<td>Probability of transmission from vector to host</td>
<td>$[\cdot]$</td>
<td>0.9</td>
</tr>
<tr>
<td>$C_{HV}$</td>
<td>Probability of transmission from host to vector</td>
<td>$[\cdot]$</td>
<td>0.9</td>
</tr>
<tr>
<td>$C_{V V}$</td>
<td>Probability of vertical transmission</td>
<td>$[\cdot]$</td>
<td>0.003</td>
</tr>
<tr>
<td>$N_B$</td>
<td>Number of bites per vector per day</td>
<td>$[T^{-1}]$</td>
<td>—</td>
</tr>
<tr>
<td>$z_I$</td>
<td>Apparition ($z_I$) and disappearance ($\beta_I$) terms of infected vectors</td>
<td>$[L^{-2}T^{-1}]$</td>
<td>—</td>
</tr>
<tr>
<td>$\beta_{I1}$</td>
<td>Death of infected vectors</td>
<td>$[L^{-2}T^{-1}]$</td>
<td>—</td>
</tr>
<tr>
<td>$\beta_{I2}$</td>
<td>Vectors becoming infective after the extrinsic incubation duration</td>
<td>$[L^{-2}T^{-1}]$</td>
<td>—</td>
</tr>
</tbody>
</table>

The density terms are given for each pixel (a geometrical projection is computed from the actual volumic density).
mosquitoes that become infective after the extrinsic incubation period is the number of vectors that became infected at time \( t + \tau_{\text{EIP}} \), from which the number of vectors dead between time \( t + \tau_{\text{EIP}} \) and \( t \) is subtracted. Then, because these mosquitoes have moved from the place where they were infected between \( t + \tau_{\text{EIP}} \) and the time \( t \), an approximation is made by redistributing the whole population of mosquitoes becoming infective at time \( t \), between the pixels where \( \rho_I \neq 0 \):

\[
\beta_{12}(\Omega, t) = \left[ \sum_{\Omega \in A} \beta_I(\Omega, t - \tau_{\text{EIP}}) \right] \cdot \frac{\rho_I(\Omega, t)}{\rho(\Omega, t)}, \quad \Omega \in A, \quad t > 0.
\]  

This approximation will be discussed later (Section 6).

**4.1.2. Infected vector that can transmit the virus**

In the same way, the density of infected vectors that can transmit the virus \( \rho_I \) follows (1) with the modified source and vanishing terms \( x_I(\Omega, t) \) and \( \beta_I(\Omega, t) \): The term \( x_I(\Omega, t) \) takes into account both horizontal (from host to vector) and vertical transmission (from an infected vector to its descendents). Then, it is the sum of the number of infected mosquitoes that become infective after the extrinsic incubation duration (\( \beta_{12} \) given by Eq. (6)) and the number of infected births, per time unit. The rate of infected births is estimated by the rate \( \rho_I(\Omega, t - \tau_E)/\rho(\Omega, t - \tau_E) \), \( \tau_E \) being the duration of mosquito development. Thus, the number of infected births per time unit is the product of the global birth term \( x(\Omega, t) \) by this rate of infected births and by the probability of vertical transmission \( C_{VV} \):

\[
x_I(\Omega, t) = \beta_{12}(\Omega, t) + C_{VV} \cdot x(\Omega, t) \cdot \frac{\rho_I(\Omega, t - \tau_E)}{\rho(\Omega, t - \tau_E)}, \quad \Omega \in A, \quad t > 0.
\]  

The term \( \beta_I(\Omega, t) \) is the death.

\[
\beta_I(\Omega, t) = \beta(\Omega, t) \cdot \frac{\rho_I(\Omega, t)}{\rho(\Omega, t)}, \quad \Omega \in A, \quad t > 0.
\]  

Finally, the density of the population of susceptible mosquitoes is given by Eq. (3).

**4.2. Hosts**

An estimation of the number of susceptible, infected during the incubation period, viraemic, and recovered persons is given for each pixel by a set of differential equations. The problem is solved by a numeric method (see Raffy and Tran, 2005). The evolution of the number of susceptible persons \( \delta H_S \) during the time step \( \delta t \) is a diminution corresponding to the number of persons who become infected by a mosquito bite. Thus, this number is the product of susceptible human beings by the probability for the person to meet an infected mosquito, approached by the rate \( \rho_V/\rho \), by the number of bites \( (N_B) \) and the probability of transmission from vector to host (noted \( C_{VH} \)):

\[
\delta H_S(\Omega, t) = - \left( C_{VH} \cdot N_B \cdot H_S(\Omega, t) \cdot \frac{\rho_V(\Omega, t)}{\rho(\Omega, t)} \right) \cdot \delta t,
\]

\[\Omega \in A, \quad t > 0.\]  

(11)

The variation of infected hosts in the intrinsic incubation period \( \delta H_I \) during \( \delta t \) is the number of people who become infected (given by Eq. (11)) minus the number of infected persons becoming viraemic after the incubation phase:

\[
\delta H_I(\Omega, t) = \left( C_{VH} \cdot H_S(\Omega, t) \cdot N_B \cdot \frac{\rho_V(\Omega, t)}{\rho(\Omega, t)} - C_{VH} \right)
\]

\[
\cdot \left( H_S(\Omega, t - \tau_{\text{EIP}}) \cdot \frac{\rho_V(\Omega, t - \tau_{\text{EIP}})}{\rho(\Omega, t - \tau_{\text{EIP}})} \right) \cdot \delta t,
\]

\[\Omega \in A, \quad t > 0.\]  

(12)

In the same way, the variation of viraemic hosts \( \delta H_V \) during \( \delta t \) is the number of people who become viraemic minus the number of viraemic persons recovering after the viraemic phase:

\[
\delta H_V(\Omega, t) = \left( C_{VH} \cdot H_S(\Omega, t - \tau_{\text{EIP}}) \cdot N_B \cdot \frac{\rho_V(\Omega, t - \tau_{\text{EIP}})}{\rho(\Omega, t - \tau_{\text{EIP}})} - C_{VH} \right)
\]

\[
\cdot \left( H_S(\Omega, t - \tau_{\text{EIP}} - \tau_{\text{FF}}) \cdot \frac{\rho_V(\Omega, t - \tau_{\text{EIP}} - \tau_{\text{FF}})}{\rho(\Omega, t - \tau_{\text{EIP}} - \tau_{\text{FF}})} \right) \cdot \delta t, \quad \Omega \in A, \quad t > 0.
\]  

(13)

Finally, the variation of recovered host \( \delta H_R \) is given by

\[
\delta H_R(\Omega, t) = \delta H_S(\Omega, t) - \delta H_I(\Omega, t) - \delta H_V(\Omega, t),
\]

\[\Omega \in A, \quad t > 0.\]  

(14)

**Remark.** The spread of a dengue epidemic is due to the movements of both vectors and hosts. Indeed, patients in viraemic phase are likely to move from their home to a place where they can infect susceptible vectors; or susceptible people can move to a place where they can be infected by vectors that can transmit the virus. In the following simulations these movements have not been modelled, but the point is discussed in Section 6.

**5. Simulations**

**5.1. Simulation 1: a dengue epidemic in an artificial landscape**

In order to visualize separately the influence of different phenomena such as wind and host movements on the spatial spread of a dengue virus, the model was tested on a small artificial landscape.

**5.1.1. Description**

Fig. 2 shows the dynamics of the whole vector population (insect density \( \rho \)), the infected vector population \( (\rho_I + \rho_V) \), and the number of susceptible and infected human beings.
and the infected host population \((H_I + H_V)\). The landscape consists of several neighbourhoods, and for simplification purposes of only two birth sites for mosquitoes (Fig. 2: points S1 and S2). One virus serotype was considered, and all the population was supposed to be initially susceptible. The mosquito population evolves according to (1), attracted by the housing areas and carried away by the wind. A viraemic person arrives in a neighbourhood, point \(V_1\). Other neighbourhoods become infected because of the vectors’ dynamics. Finally, certain viraemic hosts move from an infected neighbourhood to \(V_2\). The simulation allows to observe, in a qualitative way, the temporal and spatial evolution of the number of susceptible, infected during the incubation period, viraemic, and recovered persons.

5.1.2. Results

The role of the main factors influencing the dengue virus spatial spread can be analysed by the step-by-step visualization of the distribution of viraemic hosts and infected vectors (Fig. 2).

Provided that the vector density at point \(V_1\) is sufficient when a viraemic host arrives, certain vectors are infected (Fig. 2, \(t = 3\)) and, after a certain time period, such vectors will transmit the virus to susceptible hosts in their neighbourhood (Fig. 2, \(t = 12\)).

Wind: Transported by wind, certain infected vectors are carried away from the first dengue focus and a second neighbourhood is infected (Fig. 2, \(t = 21\)–\(30\)).

Human attraction: Because they are attracted by human beings, the infected vectors will transmit the virus in the closest neighbourhood (Fig. 2, \(t = 35\)–\(40\)).

Host movement: When certain viraemic hosts from an infected district move to the second district, a new dengue focus is created, provided that susceptible vectors are present (Fig. 2, \(t = 50\)).

These very simple results show that our model has a calculable solution and highlight the different mechanisms which play a role in the spatial spread of a dengue epidemic. In a second simulation, we shall test the model on a real landscape.

5.2. Simulation 2: a dengue epidemic in a real urban landscape

A SPOT multispectral image of the municipality of Iracoubo, French Guiana, was used to run a simulation on a real landscape (Fig. 3).

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![Fig. 2. Simulation 1—dynamics of vectors, infected vectors and infected hosts in an artificial landscape.](image-url)
5.2.1. Use of a remote-sensing image to determine certain of the model parameters

An image classification was performed on the SPOT scene in order to map the different land cover types. Five landscape elements were identified: housing areas, bare soils, dense and sparse secondary forest, free water (Fig. 4).

In a second step, the tensors $D_R$ and $D_W$ have been calculated from this classification by afecting to each landscape class a diffusion coefficient $K_R$ and a wind coefficient $K_W$ (Table 2). We adopted an approximate size of near 900 m$^2$/day for the diffusion coefficient (see Raffy & Tran for details). The choice of the coefficients was made assuming that, in our case, Stegomyia mosquitoes easily fly from one house to another or under forest shelter but avoid crossing large roads (bare soil class) or rivers. For simplification, border effects were not taken into account in this simulation.

Finally, a population density index map was also derived from the image, based on the housing area class (Tran et al., 2002).

5.2.1.1. Description. In the simulation, we only considered two birth sites for mosquitoes (points S1 and S2) located in two districts with different urban densities. Wind speed was simulated like in simulation 1.

Again, only one virus serotype was considered, and all the population was supposed to be initially susceptible. At $t = 0$, a viraemic host arrives in the municipality (point $V_1$) (Fig. 5). As in simulation 1, during the simulation some viraemic hosts move from point $V_1$ to $V_2$.

5.2.1.2. Results. Provided that the vector density in $V_1$ is sufficient, the virus spreads to the close neighbourhood, because of the vectors’ mobility, attracted by human being presence (Fig. 5, $t = 3 - 21$). It leads to a creation of an important dengue focus. The movement of viraemic hosts generates a new infection source around $V_2$. Nevertheless, as the landscape structure is less favourable to virus spreading in $V_2$ than in $V_1$ (more bare soils and lower housing density), the extent of the dengue virus spread in $V_2$ is limited and this focus rapidly disappears (Fig. 5, $t = 30$). Because of the transport effect due to wind, certain infected vectors reach new neighbourhoods which in turn become infected (Fig. 5, $t = 40$). As a conclusion, these results appear to reflect in a realistic way the role of the different factors determining a dengue virus spread in a small municipality: host moving leads to the creation of different dengue transmission focuses; and the vectors’ diffusion, due mainly to human presence, landscape structure and wind, determine the extent and the activity of such focuses.

6. Discussion

We proposed a model for the spatial and temporal dynamics of a dengue epidemic based on two remarks. First, models for the geographic spread of vector-borne diseases are needed for study and, above all, control of dengue fever expansion. On the other hand, large-scale data derived from remote-sensing images, meteorological stations, etc., are now available for providing support to
these kinds of models. The proposed model is based on a flying insect diffusion model, taking into account the different phenomena determining vectors dynamics: transport, attractive and repulsive forces, landscape structure. The diffusion model is linked to a compartmental model including hosts movements. Remote-sensing data are used to obtain certain of the model parameters.

As we specified in the introduction, the results are discussed herein in a qualitative way. The simulations show that the different factors that have been taken into account

Fig. 5. Simulation 2—dynamics of vectors, infected vectors and infected hosts in a real landscape.
play their expected role in the spatial spread of the disease. The model has a realistic behaviour in simple theoretic situations, in an artificial or real landscape. Thus, our method appears to give first interesting results and to be relevant for modelling the geographic dynamics of vector-borne diseases.

From a practical point of view, our model could be used to determine in which area the spread of an epidemic will be more important in case of a dengue outbreak and therefore could be a useful tool for vector control agencies, which could optimize their control strategies and test the effects of insecticide pulverizations. Therefore it is adapted for simulations running on few months periods, at a local scale on small geographic areas, conditions of application that are also limited by the duration of the calculation in silico. However, the rapid raise of computer calculation capacities trend to make this factor less and less constraining.

From a methodological point of view our method should be studied in greater detail.

First, we have to adjust the model parameters with field data; in particular, the range and values for $D_R$, $D_W$, $K_H$ coefficients have to be specified. This requires a well-documented study on the disease spread during a dengue outbreak.

Moreover, the model has to be linked to regular surveys in order to obtain the meteorological data as well as the entomological and epidemiological parameters. This implies that the model has to be included in a wider surveillance system to be an efficient control tool. In French Guiana, the S2Dengue (Spatial Surveillance of Dengue) research program associates the different health stakeholders for the real-time collection of all dengue-related information (suspected and confirmed cases, vector densities, etc.). The collected data will allow us to precise the model parameters in the case of dengue surveillance in this area.

Concerning the establishment of the equations governing the infected vectors dynamics (Section 4.4.1), we made an approximation by redistributing the population of vectors becoming infective after the extrinsic incubation period between the population of infected vectors (Eq. (8)). In fact, this assumption can be taken as valid considering that there is no reason for infected vectors in a latent phase to play their expected role in the spatial spread of the disease. The model has a realistic behaviour in simple theoretic situations, in an artificial or real landscape. Thus, our method appears to give first interesting results and to be relevant for modelling the geographic dynamics of vector-borne diseases.

Also, the model was developed considering only one vector species, although more species could be involved in the disease transmission process. It is the case for dengue fever in countries where the two major vector species are present: $S. aegypti$ and $S. albopictus$. In that case, two different sets of diffusion equations have to be resolved, one for each species. Indeed, the dynamics of the two species are different and all parameters (like source and death terms) should be chosen according to the entomological characteristics of each species.

In the simulations only one virus serotype was considered although the co-circulation of different serotypes in one place can occur. In that case, the different sets of equations for vectors and hosts apply separately for each dengue serotype, except that the circulation of one serotype change the immune status of the host population ($H_3$) towards the other virus serotypes. Indeed, after a dengue infection, during a “refractory” period which lasts between 2 and 6 months, it is not possible for a person to be infected by another dengue serotype.

Finally, as we focused in this first step on the spatial point of view, certain others factors which can be relevant have not yet been explicitly included in the model.

**Effect of meteorological parameters such as temperature, rainfall, humidity:** Temperatures have an effect on the duration of the extrinsic incubation period ($\tau_{EIP}$), which could be adjusted by taking this parameter into account. Rainfall is important for larval habitat availability and therefore affects the $\beta$ term. Also, excessive precipitations are against the survival rate. On the other hand, humidity is favourable to the survival rate.

**Immune status of the populations:** The immune status is an important factor because it determines the initial number of susceptible persons ($H_3$). Depending on whether a virus has already circulated in a region or district, the immune status will be different.

**Local practices:** The use of indoor insecticides, mosquito nets or air conditioning are factors that have an effect on the probability of contact between hosts and vectors and thus on the probability of transmission host/vector ($C_{YH}$ and $C_{HV}$).

All of these factors can be determined by spatialized meteorological data and epidemiological surveys and should be introduced in the model.

As a conclusion, further study is needed in order to specify in a realistic way all of the model parameters; this requires an interdisciplinary approach with epidemiologists and entomologists. Nevertheless, the method is promising for introducing a spatial component in epidemiological models.

7. Conclusion

Our results demonstrate that large-scale information data, such as remote-sensing data, could be used for building a model of the spatial and temporal dynamics of a vector-borne disease: dengue fever. A first qualitative
evaluation of the model shows by a visual interpretation that this approach is relevant for describing the vector diffusion and thus the spatial dynamics of an epidemic.

The limitations of the model consist in the movements of the hosts that have not been modelled yet, and the estimation of certain parameters. Further research, which requires an interdisciplinary collaboration, is needed to fix these parameters.

Thus, this kind of model could be improved in the future within wider research programs on the spatial diffusion of the vectors and its role in the dynamics of a dengue epidemic. More generally, the method described in the article constitutes a new approach that could be applied to other human vector-borne diseases.

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