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Rift Valley fever in West Africa: the role of space in endemicity

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Summary Rift Valley fever is an endemic vector-borne disease in West Africa, which mainly affects domestic ruminants and occasionally humans. The aetiological mechanisms of its endemicity remain under debate. We used a simple spatially explicit model to assess the possibility of endemicity without wild animals providing a permanent virus reservoir. Our model takes into account the vertical transmission in some mosquito species, the rainfall-driven emergence of their eggs and local and distant contacts because of herd migration. Endemicity without such a permanent virus reservoir would be impossible in a single site except when there is a strictly periodic rainfall pattern; but it would be possible when there are herd movements and sufficient inter-site variability in rainfall, which drives mosquito emergence.

keywords Rift Valley fever, Sahel, spatial stochastic model, rainfall, herd movements

Introduction

During recent years, the emergence of infectious diseases has become of great concern, especially in the case of vector-borne viral zoonoses that occasionally give rise to human epidemics (such as West Nile fever, Japanese encephalitis, Chevalier *et al.* 2004). Emergence is characterized by the spread and progressive endemicity of these viral diseases in previously unaffected areas. In most cases, the ecological and epidemiological mechanisms at the origin of this emergence remain poorly known.

One of these emergent vector-borne zoonoses is Rift Valley fever (RVF). RVF virus (RVFV) belongs to the genus *Phlebovirus*; family Bunyaviridae (Murphy *et al.* 1995) and is mainly transmitted by mosquitoes of the *Aedes* (*Aedes vexans*, *Aedes ochraceaus*) and *Culex* (*Culex poicilipes*) genera (McIntosh 1972; Fontenille *et al.* 1998) to a wide range of animals, from rodents to camels (Gerdes 2004). From an economic point of view, the most important hosts are sheep, goat and cattle. Among adult livestock, the infection is often unapparent or mild, but it causes abortions and death of newborns (Wilson 1994). Although mosquitoes may transmit RVFV to humans, most human infections result from contact with infected animals, especially during slaughter. The symptoms range from a flu-like illness to encephalitis or haemorrhagic fever. It is important to note that an epizootic does not systematically give rise to an epidemic.

The RVFV was first isolated in Kenya during 1931 (Daubney *et al.* 1931). Until 1977, RVF was considered as a disease of small ruminants, endemic in East and South Africa, causing abortions and stillborn births in ovine species. Subsequent human infections have sporadically occurred with feverish affections and harmless evolution. Two major epidemics occurred in other regions following dam construction and subsequent major ecological modification of the environment: in Egypt near Aswan in 1977 (Meegan 1979; Meegan *et al.* 1979) and in Mauritania near Diama in 1987 (Digoutte & Peters 1989; Jouan *et al.* 1989; Lancelot *et al.* 1990). In Egypt, the RVFV was not endemic before 1977; it disappeared in 1981 and re-emerged there, as well as in the Arabian Peninsula, after 1997. The epidemic in Mauritania revealed a previous endemic circulation in Western Sahel, which is still going on. Indeed, RVFV had already been isolated in mosquitoes in 1974 in Senegal (Fontenille *et al.* 1998) and RVF infection was monitored among cattle in South Mauritania some months before the 1987 epidemic (Saluzzo *et al.* 1987).

In East Africa, the endemic cycle involves the vertical transmission of RVFV in *Aedes* spp. (Linthicum *et al.* 1985). These mosquitoes lay eggs in the drying mud surrounding temporary ponds (Zeller *et al.* 1997; Fontenille *et al.* 1998). Once embryonated, the eggs can survive dry conditions for long periods until the pool is refilled. If eggs are RVFV contaminated, infected adults emerge after these eggs are flooded. This is why epizootics in East Africa

are correlated with unusually heavy rainfall associated with warm ENSO events (Linthicum *et al.* 1999). In West Africa, conversely, no relationship between epidemics or epizootics and heavy rainfall could be demonstrated (Chevalier *et al.* 2004). The mechanisms sustaining this endemicity remain under debate: how does the virus outlive the dry periods when vectors disappear? Is a permanent virus reservoir (cryptic circulation in wild animals) necessary to explain the maintenance of the virus? Or can domestic animals be the chief actors of this endemicity? What happens during inter-epizootic periods that can last up to 15 years in a given place?

One hypothesis is that, as in East Africa, vertical transmission of RVFV could occur in the West African *Aedes* spp. and that this would be able to trigger local epizootics. Moreover, movements and migration of Sahelian herds could provide contacts between herds and ensure RVFV spread. Under this hypothesis, rodents, from which Gora *et al.* (2000) isolated RVFV antibodies, may participate in the amplification of the virus during the epizootic but would not play any role in the endemicity. Our aim was to test whether these hypotheses can effectively explain virus maintenance over a long period of time, and under which conditions. A simple epidemiological model was set up to describe the evolution of the immunological status of herds in the vicinity of pools where *Aedes* sp. mosquitoes lay their eggs. First, we explored the conditions that allow virus persistence in a given location – on a local scale. Then, we studied how spatial dependence could allow the maintenance of the virus.

Model

Local model

The model does not detail the development of the epizootics but considers a yearly update of the immunological status of the cattle. Therefore, we only consider the triggering of the epidemic by infected *Aedes* sp. eggs and not any subsequent transmission by other vectors or aerosol. This is based on the hypotheses described in the Introduction section, which lead to the model depicted in Figure 1 (parameters are summarized in Table 1 and the precise mathematical formulation in Appendix A). The time step of the model is 1 year. The space unit is a region around a pond (of the order of 1 km^2) with an associated population of domestic animals (sheep and/or cow) and of infected eggs. The population of domestic animals is distributed in N classes of age. The first class is the juvenile one, the others are adults. The population of each class is assumed to evolve as a whole. As the model does not describe the dynamic of the epizootic, two immunological

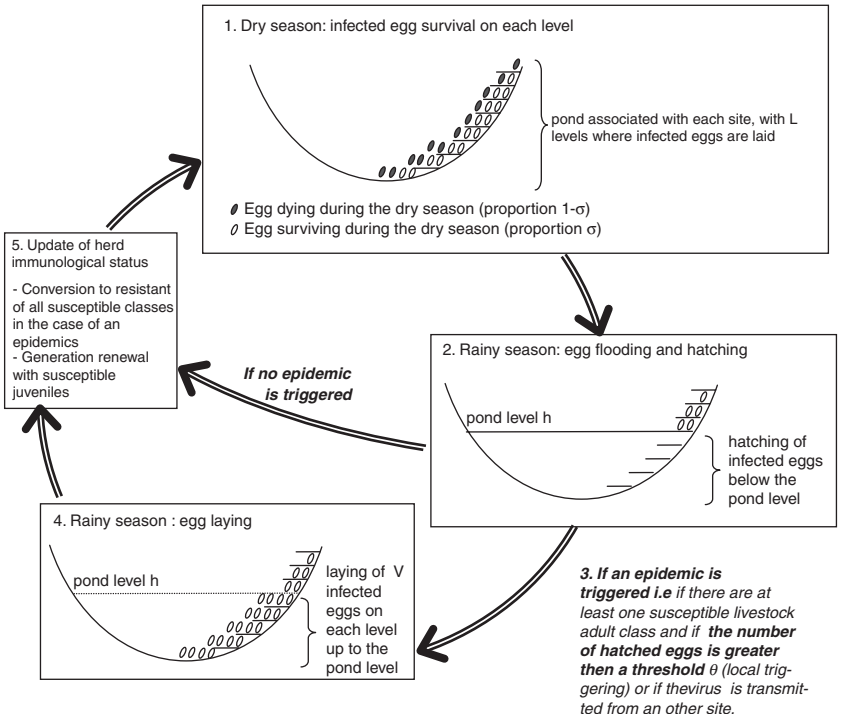

Figure 1 Summary of processes modelled during the 1-year time step.

Table 1 Summary of the model parameters and variables. The nominal values are used except for contrary mentions in the text. The ranges are used for the 1000 randomly drawn parameter sets in the 0-D model with random weather

Variable or parameter	Notation	Nominal value	Range for 0-D model
Water height	$h(t)$		
Minimum pond level	r_0	2	1–9
Pond level	s		
Age class	a		
Number of age classes	N	12	
Number of pool strata	L	10	
Yearly egg survival	σ	2/3	0.2–0.9
Number of infected eggs produced per level	V	10	
Threshold of infected hatched eggs	θ	1	0.5–8.5
Probability of near contacts	α	0.5	
Probability of far contacts	β	0.5	

states are considered: susceptible or resistant. The pond is divided into L layers, on which infected eggs can lie.

- The number of viable eggs decreases exponentially until they are flooded, the yearly survival of dry infected eggs being denoted as σ. The pond is susceptible to filling by rainwater.
- Function $h(t)$ represents the water level reached in the pool at time t, hereafter referred as pond level. It can be a deterministic or a random function of time: the specific forms are detailed in the Methods section. At each time step t, eggs below the pond level $h(t)$ are flooded and hatch.
- If the number of emerging infected vectors is greater than a threshold θ and if there are susceptible adult animals (if there are no susceptible adults, there are no susceptible young livestock either as they are usually protected by maternal antibodies), an epidemic is triggered.
- In the case of an epidemic, a number, V, of contaminated eggs are laid (vertical transmission in *Aedes* spp.) on the edge of temporary ponds on each level up to the pond level $h(t)$.
- The immunological status of the domestic animals is updated (in the case of an epidemic, all susceptible age classes become resistant) and generations are renewed with susceptible juveniles.

The model outputs are (i) the total number of the infected eggs; (ii) the total number of infected livestock age classes (seroprevalence); (iii) the total number of abortions of sheep (clinical prevalence) and (iv) the occurrence of an epizootic or the lack thereof.

2-D model of RFV epizootics

In the 2-D model, the simulation map is a grid of herd accommodation sites i, where for each, the local dynamics of the 0-D model takes place. In addition to the local dynamics, two kinds of contacts between herds are considered to be leading to contagion:

- Local contacts between nearby herds: if an epidemic was locally triggered in one of the four nearest neighbours of a given site whose herd is susceptible, the contagion occurs with a probability α.
- Distant contacts during migration: each herd has a probability β to change places to a randomly chosen site. If an epidemic takes place in this distant site and if the herd is susceptible, the herd acquires the virus and develops an epizootic while getting back to its initial location and associated pond.

Methods

Definition of the endemicity

To determine whether the introduction of a virus can lead to its endemicity, it is first necessary to be able to define mathematically when there is endemicity or not. It is of course a matter of the time of persistence of the virus.

In 0-D and when the pond level function is deterministic, the output of the model is deterministic and reaches a stationary behaviour. Then, endemicity occurs when there is virus transmission during this stationary behaviour.

In 2-D or when the rainfall-driven pond level is stochastic, the time to extinction is a random variable: some virus introductions lead to rapid extinction, others allow persistence over a long period.

Figure 2 compares three typical cumulative distributions of the time to extinction. In one case (epizootic pattern), the curve quickly grows to one: the proportion of long-lasting epizootics is negligible. In this case, for any virus introduction, the antibody prevalence finally goes to zero after a period of fluctuations. In the second case (endemic pattern), the rapid initial growth goes to a value inferior to 1: a proportion of virus introductions lead to the persistence of the virus, where the antibody prevalence reaches a constant value. The probability that a virus introduction in a particular environment finally leads to the persistence is called the endemic potential of the environment and denoted as EP. The last curve (stochastic endemic pattern) shows a plateau but it is less marked than that in the previous case. Nevertheless, it can be considered as characteristic of a possible endemicity. In such curves, the EP is approximated by the probability that a virus

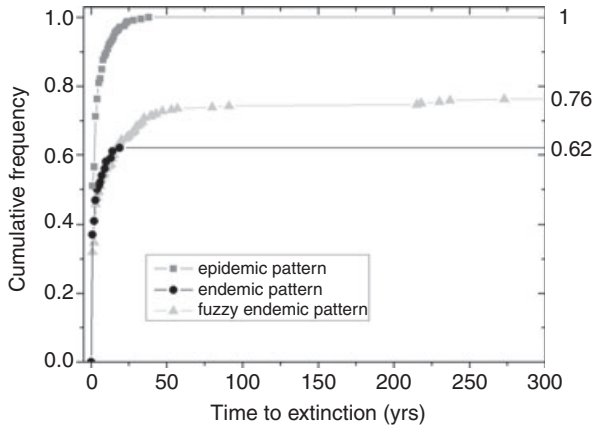

Figure 2 Cumulative frequency distribution of times to extinction, characteristic of an epidemic pattern (EP = 1–1 = 0), a deterministic endemic pattern (EP = 1 – 0.62 = 0.38) and a stochastic endemic pattern (EP = 1–0.76 = 0.24).

introduction leads to persistence over an arbitrary large number of years (here 300). As there are very few points in the plateau, the choice of this limit does not much affect the EP estimation. From a practical perspective, in all cases, the EP is estimated by the proportion of 200 virus introduction, which leads to a persistence of the virus over more than 300 years.

Conditions for local endemicity

We first consider the conditions for endemicity of RVF on a local scale, i.e. with the 0-D model. The possibility of maintenance of the virus is studied with (i) random pond level functions between r_0 (minimal level) and L (maximal level). A total of 1000 random sets of parameters are drawn (see Table 1 for the ranges) and the EP is estimated for each of them. (ii) Periodic pond levels between the levels r_0 and L, with period T, fixed phase ϕ and a random disturbance of the phase of intensity η. Figure 3 displays examples of such time series, whose mathematical formulations are given in Appendix A.

Conditions for spatial endemicity

Next we investigated how the spatial structure of the 2-D model modifies the conditions for virus endemicity. The pond level in one site is the sum of an homogeneous component (the same for every cell, representing the regional climate, being a random variable uniformly distributed between the levels r_0 and $r_0 + r_1$) and of a local component (different for each cell, representing the local variation of climate, being a random variable uniformly

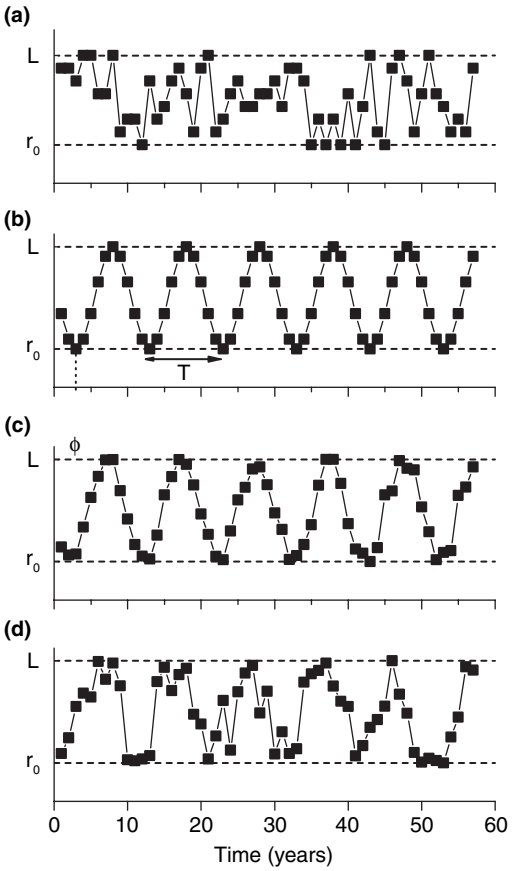

Figure 3 Examples of time series of pond levels. (a) Uniform random distribution between the levels r_0 and L. (b–d) Periodic pond levels with period T and phase ϕ with increasing random phase disturbances.

distributed between 0 and $L - (r_0 + r_1)$. The EP is estimated for different values of r_1, α and β.

Results

Local model

Random weather. Using a random function for the pond level $h(t)$, we find three main patterns of RVF infection (Figure 4): (i) no infection, there is no secondary cases; (ii) sporadic infection, there is a single outbreak and (iii) recurrent epizootics. For each of the 1000 random sets of parameters, the EP is null.

Periodic weather. With a deterministic pond level, some bands of periods centred on the first integer values permit virus persistence (Figure 5a). Most correspond to only one value. The widest is around the period 2 years. This is the

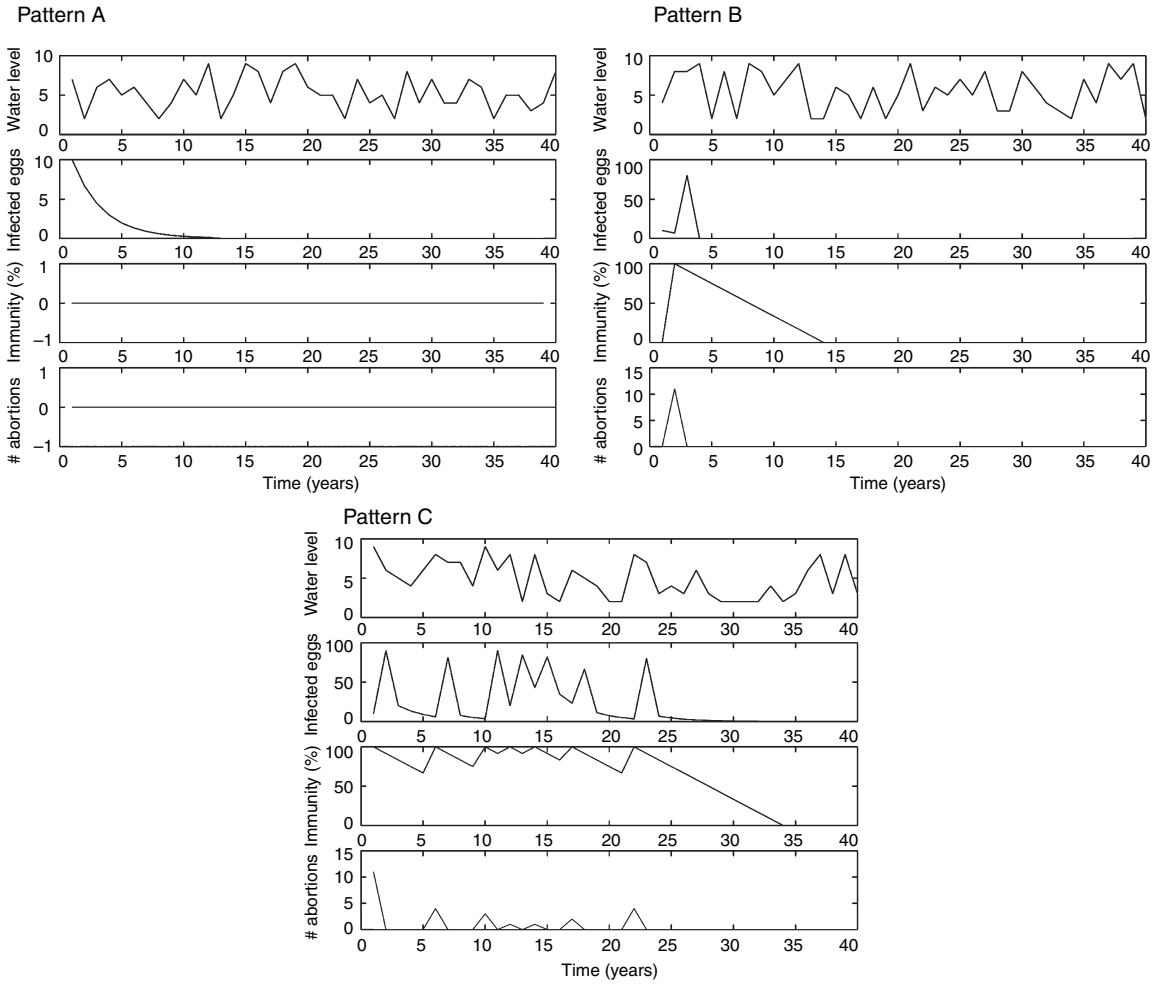

Figure 4 0-D model of RVF with random pond level function. (a–c) Temporal epidemic patterns of RVF in one area: (a) no outbreak, (b) a single outbreak, (c) several successive outbreaks before extinction. For each pattern, the four curves are, from up to down: the temporary ground pond level; the infected eggs density near a pond; the cattle seroprevalence ratio near a pond; the abortion prevalence of the cattle near a pond.

band that best resists the addition of random phase disturbance (Figure 5b–d).

2-D model

Figure 6 displays the evolution of the EP for the different values of α and β, for different values of the weather parameter r_1. In all cases, the site (0,0) corresponds to the 0-D situation and EP = 0 is retrieved. There is a critical curve in the (α, β) space delimiting the epizootic region (EP = 0) and the endemic one (EP > 0). The EP grows continuously from 0 to the highest value from this curve to the point (1,1). The extent of the endemic region is all the more important as r_1 is low, i.e. as the homogeneous component of the weather is small.

Figure 7 displays, for a scenario of endemicity with $\alpha = 0.3$ and $\beta = 0.4$, two examples of the RVF characteristics in one site and averaged over the whole simulation map, one for $r_1 = 0$, the other for $r_1 = 5$. In the first case, the characteristics averaged over the whole space gradually reach quasi-constant equilibrium values. However, in the second case, there are fluctuations over the different characteristics. Some fluctuations appear rather synchronic with the fluctuations of the homogeneous weather component, while others are not. Although at the scale of the whole map the virus circulation is endemic (there are

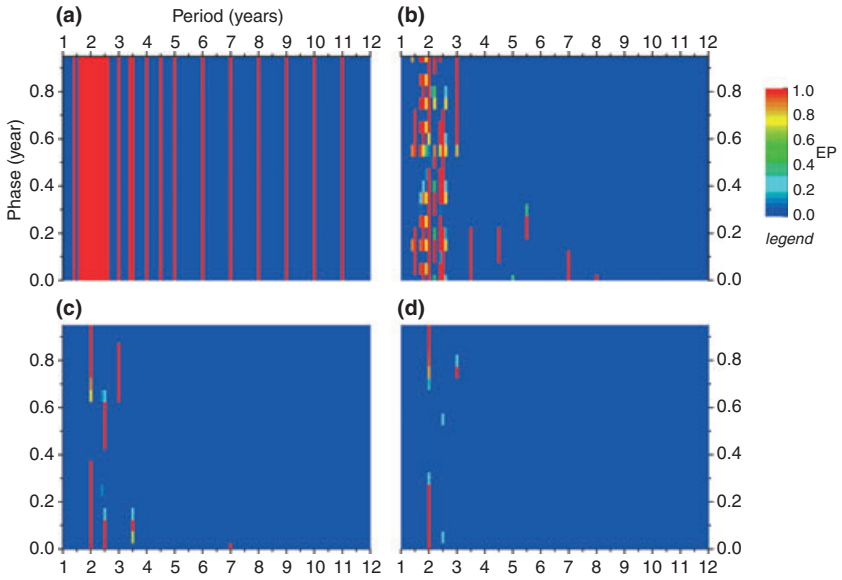

Figure 5 Modal analysis of the 0-D RVF model, representing the EP for periodic pond levels with different periods and phase (Figure 2) and increasing random disturbance r to the periodic pond level function: (a) $r = 0$; (b) $r = 0.1$; (c) $r = 0.3$; (d) $r = 0.5$. The colour scale represents the epidemic potential (see legend).

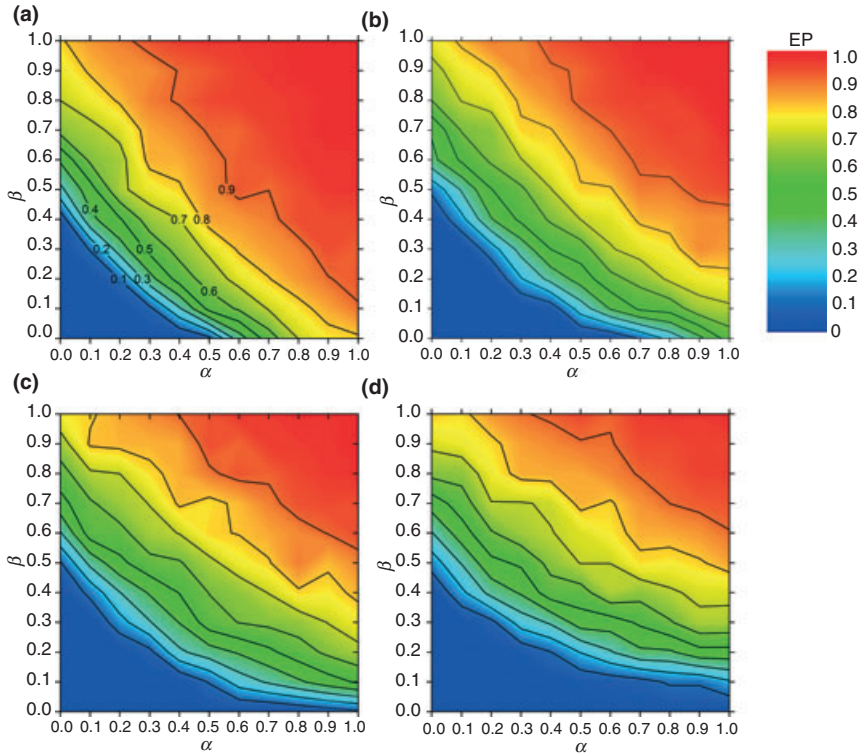

Figure 6 Colour map of the EP in the 2-D model with the parameters of Table 1, different values of the parameters α and β with a random pond level with a homogeneous background (see text and Appendix A) with (a) $r_1 = 0$; (b) $r_1 = 3$; (c) $r_1 = 5$; (d) $r_1 = 7$. Other values are indicated in Table 1.

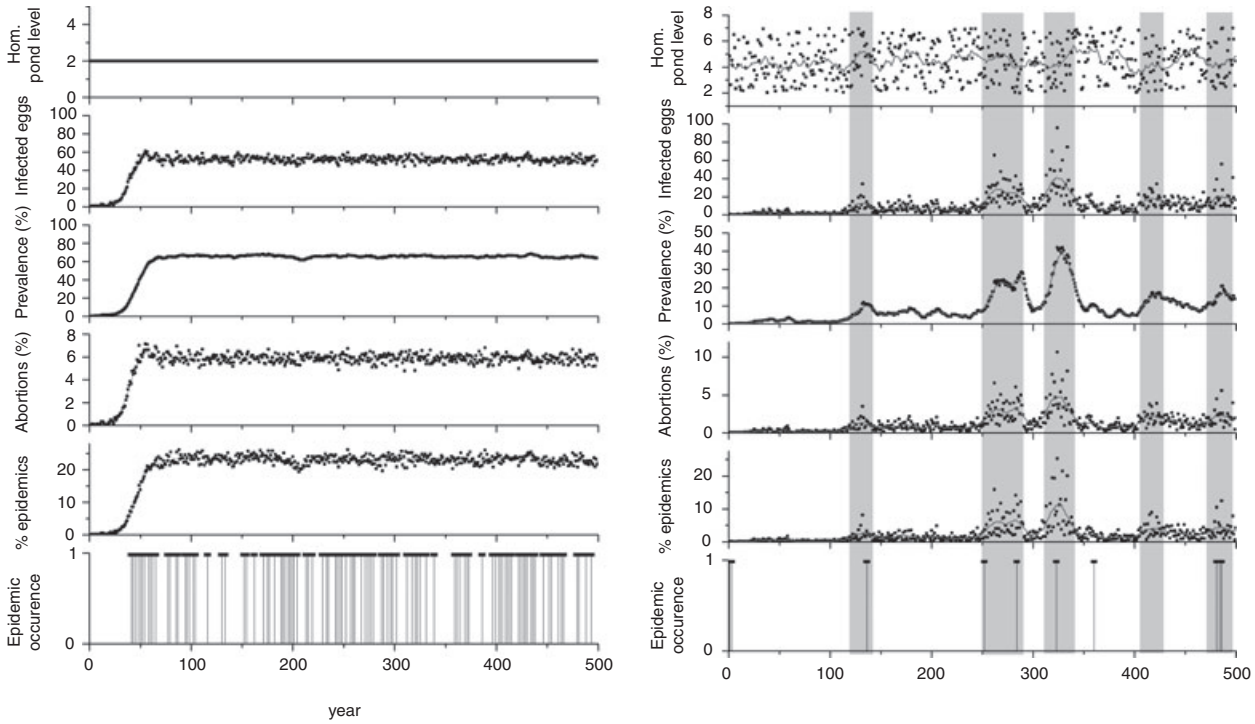

Figure 7 Example of a simulation with the 2-D model (parameters of Table 1, $\alpha = 0.2$ and $\beta = 0.3$). Temporal evolution of the epidemic functions averaged over the whole map and epidemic occurrence in a test site for $r_1 = 0$ (left) and $r_1 = 5$ (right).

always infected eggs and a non-zero immunity), at a given site scale the situation is epizootic with time intervals between successive epizootics long enough for the cattle to renew.

Discussion

A large proportion of epidemiological models to study the influence of space over epidemic or epizootic development is based on qualitative models to represent epidemiological systems where few or no quantitative details are known about the specific mechanisms. Some models consider the development of general epidemics, without reference to a particular disease, from a mathematical (Ball & Neal 2002), physical (Moore & Newman 2000) or ecological (Smith *et al.* 2004) point of view. Others consider particular questions for particular diseases from a theoretical viewpoint (e.g. Koella & Antia 2003; Favier *et al.* 2005). These conceptual mathematical models cannot provide quantitative information about the system nor can they be used for simulation of specific situations. Nevertheless, systematic analyses of the models provide qualitative information about the possible dynamics and epidemic behaviour. We thus propose the present model as a qualitative tool to provide qualitative conclusions about a qualitative question. For example, the assumption that each age class immunological status evolves as a whole is a strong hypothesis, but as it is unfavourable to persistence of endemicity, it does not weaken the results of this study.

Possibility of endemicity without wild animal permanent virus reservoir

The question underlying this work was: can vector vertical transmission of the RVFV lead to its endemicity in the Sahel without wild animals acting as a permanent virus reservoir? The answer our work gives is yes, but under some conditions. At a single site, that is, in 0-D model, endemicity is possible only if rainfall has a strong periodic component of period around 2 years. Obviously, this is not the case in Sahel and persistence of the virus in a single site is therefore impossible. This means that locally the transmission of RVFV can only be epizootic.

The analysis of the 2-D model has shown that, thanks to herd mobility, the spatial structure of the environment can give rise to endemicity with non-periodic pond levels as long as there is enough inter-site variability. Indeed, herd movements ensure virus dissemination while its maintenance is ensured by the fact that epizootics are triggered in some sites while they go to extinction in others. The RVF is

endemic on the global scale but epizootic on the local scale, with inter-epizootic periods that can be of several decades, as shown in real situations (Gerdes 2004). When the pond levels are completely uncorrelated from one site to the other, the endemicity leads to a stationary emergent behaviour with constant characteristics (number of abortions, immunity, number of epizootics). However, when there is a homogeneous rainfall and pond level component, these characteristics fluctuate strongly. In particular, during some periods, the increase in the homogeneous component can induce a simultaneous increase in the mean number of epizootics and, consequently, of the mean seroprevalence among cattle. Such a pattern has been observed in Senegal (Thonnon *et al.* 1999).

In nature, the endemic cycle in West Africa is regularly provided with new RVFV strains, possibly from an endemic circulation inside the rain forest (Sall *et al.* 1999). Taking this into account instead of a single introduction in the onset of the simulation would not change the major conclusions of this study. At a local scale, the rate of introduction of new strains, although not quantified, is certainly not sufficient to sustain the endemicity. At the regional scale, recurrent introduction helps the maintenance of the endemic cycle and makes a wild virus reservoir even less necessary to the maintenance of the endemicity.

Towards a predictive model

This model is an example of what is referred to as 'toy models' in physics: a simple conceptual model which provides interesting hints to qualitatively determine the processes at the origin of virus endemicity, but cannot be used as a predictive tool to guide health policies. A predictive model would require better information about the details of the processes as well as about the dynamics of the pond level.

From the present study, it can, however, be proposed that four main steps are required to upgrade this explicative model into a predictive model. First, it is necessary to characterise the rainfall-induced *Aedes* spp. emergence. In this simple model, it is characterised by a weather-driven pond level. In a predictive model, the local mechanisms that link rainfall and *Aedes* spp. must be quantitatively determined. The temporal and spatial variability of emergence must be studied as well, in relation with rainfall variability and with pond characteristics (Bicout *et al.* 2002; Bicout & Sabatier 2004; Porphyre *et al.* 2005). Secondly, the dynamics of an epizootic must be better described. In particular, it is necessary to determine whether, after the initiation by the epizootic, the main mechanism of transmission is still ensured by insect vectors or simply by aerosol. Thirdly, the possibility of virus transmission between herds must be considered. In our simple model, it is characterized by a single probability. That probability summarizes the probability of changing feeding places, of remaining present in the migration place when an epizootic can be triggered and finally of coming back to its origin before the epizootic is finished. Information about migration habits is necessary to quantify this parameter and improve the description of this process, especially over the period and the locality of the dry season grazing places. This leads to the fourth study to be carried out: the spatial organization of the migration. In the present study, space has been considered homogeneous. Actually, sites are not all equivalent, especially because of the north–south climatic gradient, which implies that migration occurs mainly in this direction.

Connections with other epidemiological models

Whereas this model has been designed to represent a particular system, it shows some similarities with theoretical 'small-world' models (Moore & Newman 2000). In these, as in the present model, the spatial behaviour is characterized by a combination of local and distant contacts. However, small-world models consider the progress of an epidemic, whereas the RVF model plays on a different time scale and addresses the possibility of endemicity.

Other studies have considered the influence of space over long-term dynamics of epidemics, through modelling or analysis of data. Most of them consider or come to the conclusion that local hot spots (regions of high or persistent endemicity) initiate travelling waves of infections in surrounding epidemic areas. These hot spots are often towns where the pathogen remains active and from which it spreads to neighbouring villages, for example, in the 'cities and villages model' for measles (Anderson & May 1991; Grenfell & Bolker 1998) or in urban dengue epidemics (Cazelles *et al.* 2005). In a certain way, this situation is similar to endemic forest cycles occasionally initiating epidemics. This study emphasises a different circulation pattern, where endemicity arises from a collection of coupled sites of similar nature. A similar pattern was proposed by Keeling and Gilligan (2000) for bubonic plague. Here, with local and distant contacts between herds and spatial variability in weather-driven mosquito emergence, it is not necessary to speculate about the existence of a cryptic virus circulation to explain the RVF endemicity.

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Fièvre de la vallée du Rift: Modèle spatialisé pour montrer comment elle peut y être devenue enzootique sans réservoir viral sauvage

La fièvre de la vallée du Rift (RVF) est une arbovirose transmise par des moustiques et originaire d'Afrique orientale, où elle a sans doute un réservoir viral dans la faune sauvage. Au cours du XX siècle, elle s'est répandue en Afrique de l'Ouest où elle affecte les ruminants domestiques et parfois les humains. Nous utilisons un modèle spatialisé pour montrer comment elle peut y être devenue enzootique sans réservoir viral sauvage. Le modèle prend en compte la transmission verticale par certains moustiques, et l'éclosion pendant la saison des pluies d'oeufs infectés. L'enzooticité ne peut exister en un site unique, mais peut être maintenue par la migration des troupeaux à l'échelle régionale si la pluviométrie varie suffisamment entre les sites.

mots cles Fièvre de la vallée du Rift, Sahel, ruminants, modèle spatialisé, pluies, migration ds troupeaux

La fiebre del Rift Valley en África Occidental: el papel del espacio en la endemicidad

La fiebre del Rift Valley es una enfermedad endémica en África Occidental, transmitida por un vector, que afecta principalmente rumiantes domésticos y ocasionalmente a humanos. Los mecanismos etiológicos de su endemicidad aún están siendo debatidos. Hemos utilizado en modelo simple, espacialmente explícito, para evaluar la posibilidad de que haya endemicidad sin la presencia de animales salvajes actuando como un reservorio permanente del virus. Nuestro modelo tiene en cuenta que la transmisión vertical en algunas especies de mosquito, el efecto de las lluvias sobre la aparición de sus huevos, así como los contactos locales y distantes debido a la migración. La endemicidad sería imposible en lugar específico sin un reservorio permanente del virus, excepto cuando hay un patrón periódico de lluvias estricto; pero es posible en presencia de un movimiento de rebaño y una variabilidad suficiente en las precipitaciones de varios lugares, que conllevarían a la aparición del mosquito.

palabras clave Fiebre del Rift Valley, Sahel, modelo espacial estocástico, precipitaciones, movimiento de rebaño

Appendix A: Mathematical formulation of the model

This appendix presents the mathematical description of the model, which has been numerically coded in C language.

Local model

The epidemiological status $p(a,t)$ of an animal of a given age class a, expressed in years (the maximal age class is estimated to 12 years), at time t is defined to be

$$p(a,t) = \begin{cases} 0, & \text{resistant} \\ 1, & \text{susceptible} \end{cases} \quad (1)$$

At time t, a number $o(s, t)$ of RVFV-infected eggs lies in the layer s of the pond (the pond is divided into L layers: $1 \le s \le L$) and the pond level is $h(t)$. With $H(x)$ being the Heaviside step function defined as $H(x) = 0$ for $x < 0$ and $H(x) = 1$ for $x \ge 0$, the function f_1

$$f_1(t) = H\left[\sum_{s=1}^{h(t)} o(s,t) - \theta\right] \quad (2)$$

equals 1 when there are enough infected adults to trigger an epizootic. Similarly, the function

$$f_2(t) = H\left[\sum_{a=2}^{N} p(a,t)\right] \quad (3)$$

equals 1 when there is at least one susceptible adult class. An epizootic is triggered when the product of both functions $f_1(t)$ and $f_2(t)$ equals 1. Then, V infected eggs are laid on each level up to the level $h(t)$. With these parameters, the time evolution of the immunological status of each age class is

$$p(a,t+1) = \begin{cases} 1 & a = 1 \\ [1 - f_1(t)f_2(t)]p(a-1,t) & a > 1 \end{cases} \quad (4)$$

ensuring that p switches from 1 to 0 after an epizootic. The evolution of the number of infected eggs at the level s is

$$o(s,t+1) = \sigma H[s - h(t)]o(s,t) + \{1 - H[s - h(t)]\}f_1(t)f_2(t)V \quad (5)$$

ensuring the decay by mortality of the dry eggs and a renewal of the infected eggs up to the maximum level reached by the pond.

2-D model

The simulation map is a grid of herd accommodation sites i, where, for each of them, the local dynamics of the 0-D model takes place. The pond level h, the variables p and o are now indexed for the site. In the same way as described in the previous section, the functions f_1 (indicating the hatching of enough eggs to trigger an epidemic) and f_2 (indicating the susceptibility of the population to an infection) are defined in each site i

$$f_1(i,t) = H\left[\sum_{s=1}^{h(i,t)} o(i,s,t) - \theta\right] \tag{6}$$

$$f_2(i,t) = H\left[\sum_{a=2}^{N} p(i,a,t)\right] \tag{7}$$

The triggering of an epizootic in the site i is indicated by the function f_3. This can happen, that is, $f_3\ (i,t) = 1$

- if a local epizootic is triggered: $f_1\ (i,t)\ f_2\ (i,t) = 1$;
- with probability α, if an epizootic takes place in one of the four nearest neighbours j, and that the populations of the site i are susceptible, that is, if $f_2(i,t)\sum_j f_1(j,t)f_2(j,t) > 1$
- or if an epizootic is triggered by a distant contact k, that is if $f_1\ (k,t)\ f_2\ (i,t) = 1$. This contact occurs with a probability β and, in this case, a random site k is chosen among the whole simulation map.

The update of the variables is, similarly to equations 4 and 5

$$p(i,a,t+1) = \begin{cases} 1 & a = 1 \\ [1 - f_3(i,t)] \times p(i,a-1,t) & a > 1 \end{cases} \tag{8}$$

$$o(i,s,t+1) = \sigma H[s - h(i,t)]o(i,s,t) + \{1 - H[s - h(i,t)]\}f_3(i,t)V \tag{9}$$

Pond level time series

The equations for the different pond level time series in the local model are

- for the random pond levels

$$h(t) = \lfloor r_0 + (L - r_0 + 1)R(t) \rfloor \tag{10}$$

where $R(t)$ is a sequence of random numbers uniformly distributed over [0,1] and $\lfloor x \rfloor$ denotes the integer part of x

- for the periodic pond levels with phase disturbances

$$h(t) = \left\lfloor r_0 + \frac{(L + 1 - r_0)}{2}\left\{1 + \cos\left[\frac{2\pi}{T}(t + \phi + \eta R(t))\right]\right\}\right\rfloor$$

where $R(t)$ still denotes a sequence of random numbers uniformly distributed over [0,1], η the amplitude of the random disturbance, ϕ the fixed phase and T the period.

In the 2-D model, the pond level function $h(\vec{x},t)$ of a cell $\vec{x}$ is the sum of a global component $h_g(t)$ (the same for each cell) and a local component $h_l(\vec{x},t)$ varying from cell to cell

$$h(\vec{x},t) = \left\lfloor \overbrace{r_0 + r_1 R(t)}^{h_g(t)} + \overbrace{(L + 1 - r_0 - r_1)R_{\vec{x}}(t)}^{h_l(\vec{x},t)} \right\rfloor \tag{11}$$

where $R(t)$ and $R_{\vec{x}}(t)$ are independent sequences of random numbers uniformly distributed between 0 and 1 (r_1 is the amplitude of the stochastic homogeneous component).