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Mosquito-borne diseases simulated by cellular automata: A review

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Abstract
In this work we review cellular automata models that have been proposed to simulate mosquito-borne disease spread. We summarize some of the strengths or weaknesses of the different cellular automata models. We classify the models according to: those that include human mobility, type of compartmental model, simulations of hypothetical scenarios or those that use real data to fit the model, use of geographic information systems, population densities, seasonality and control strategies to prevent the diseases spread.

Keywords: Mosquito-borne disease spread, cellular automata, control, simulations

Introduction
Causing millions of deaths every year, mosquitos' ability to carry and spread diseases to humans made them to be considered as one of the deadliest animals in the world. In 2015 malaria alone caused 438,000 deaths. Dengue incidence has risen in the past 30 years around the world and at the present time the number of countries reporting their first dengue outbreaks is increasing.
Besides Dengue, Zika, mayaro, chikungunya, and yellow fever are also transmitted to humans by the *Aedes aegypti* mosquito. More than half of the world's population lives in areas where this mosquito species is present. No vaccinations are provided for these mosquito diseases, thus sustained mosquito control efforts are important to prevent outbreaks.
There are several different types of mosquito and some have the ability to carry many different diseases. Beside carrying diseases that affects humans, mosquitoes can also transmit several diseases and parasites to dogs and horses. These include dog heartworm, West Nile virus (WNV) and Eastern equine encephalitis (EEE). Mosquito vectored diseases include protozoan diseases as malaria, filarial diseases such as dog heartworm, and viruses such as dengue, encephalitis and yellow fever. Presently, dengue produces approximately 50-100 million infections annually, but malaria remains the world's most prevalent vector-borne disease. In spite of control efforts, malaria is emerging in areas that in the past were free of the disease and reemerging in areas where control efforts were once effective.
More than a hundred years have passed since Ross's mathematical model [1] to describe malaria transmission was published, and more than 50 years since Macdonald [2] extended Ross's theory and applied to the Global Malaria Eradication Programme (GMEP, 1955-1969). Since then, there has been a growing interest in modeling and simulating the spread of mosquito borne diseases in order to understand the vector dynamics and analyze vector control strategies
Reiner [3] made a systematic review of mathematical models for mosquito-borne spread and they suggest some efforts that must be make to address some issues such as: variation in individual host attributes, heterogeneous biting, poorly mixed mosquito-host encounters and temporal variation. Eder [4] made a review of vector-borne diseases (VBDs) models affecting urban and suburban populations; they identified as key strategies for public health policy and practice, the strengthening of surveillance-control, particularly in asymptomatic cases and mobile population, also the use of early warning tools to predict increasing transmission.
Mathematical models, both deterministic and stochastic, are useful tools to characterize and get a better understanding of the relationship of vector-borne disease to ecological communities.
The starting point in building mathematical models to describe mosquito-borne diseases spread is the compartmental assumption. Here the population is divided in groups: Susceptible, Exposed, Infectious, Recovered, Death, etc. Both the host (human) and the vector (mosquitos)

are classified using the compartmental assumption. Time evolution of the amount of individuals at each compartment is commonly modeled as a system of ordinary differential equations (ode).

The stability analysis of the ode system provides us with one of the most important quantities in disease spread, namely the basic reproduction number R_0 (the number of secondary infections produced by a typical case of an infection in a population that is totally susceptible). This is a threshold; when $R_0 < 1$ the infection will die out in the long run, but if $R_0 > 1$ the infection will be able to spread in a population. This quantity is useful in guiding control strategies.

Brauer *et al*. [5] formulate and analyze two ode epidemic models for vector-transmitted diseases, one appropriate for dengue and chikungunya fever outbreaks and another one that also includes direct transmission appropriate for Zika virus outbreaks. Ordinary differential equations models assume a perfect mixing of the populations and do not account for aspects like: local interactions of vectors and host, host and vector heterogeneous distributions, geolocation, and host mobility.

Some approaches that have arisen to deal with spatial spread include: systems of reaction-diffusion partial differential equations, agent based models, metapopulation methods and cellular automata. Each of these approaches has some advantages and disadvantages.

Agent based models (ABM) for mosquito diseases spread have been implemented in computational codes such that EpiSimS (Los Alamos) [6], EMOD-DTK [7]. Moore [8] employed EMOD-DTK [7] to study local and regional transmission of Chikungunya concluding that increasing spatial granularity improves the fit of the model to temporal incidence patterns. This improvement is derived from the fact that simulations with spatially granular models more appropriately capture spatial heterogeneity in epidemiologically relevant factors, such as mosquito abundance and human demography and mobility. Even though ABM can capture local interactions among the individuals, their computational complexity increases when the number of individuals increases, causing some problems to be practically unmanageable.

Metapopulation models assume that the spatial domain is divided into patches. At each patch a system of ordinary differential equations for the compartmental mosquito-host diseases model is solved, the interactions among the patches are computed using matrices that contain information related to the movement of the hosts, i.e. individual fluxes due to the transportation and mobility structure. In fact Cosner [9] by using Lagrangian and Eulerian approaches have shown that the movement of humans between patches is sufficient to maintain disease persistence in patches with zero transmission. Wang [10] summarizes advances of mathematical models in the studies of epidemic diseases in heterogeneous geography; by considering the invasion of malaria into a population distributed into multi patches, the basic reproduction number of the metapopulation model is established. Bichara and Castillo [11] use a multi-patch and multi-group approach to model host-vector heterogeneous structure; hosts' dispersal is modeled in terms of patch-residence times with the nonlinear dynamics taking into account the effective patch-host size. They computed the residence times basic reproduction number and their numerical simulations underlined the effects of residence times on disease prevalence. Arino [12] defined a SIRS-SI host vector metapopulation model for malaria where they consider partially immune recovered hosts, showing that their calculated basic reproduction number governs the local stability of the disease free equilibrium but not the global behavior of the system because of the potential occurrence of a backward bifurcation. They identified the reservoirs of infection and evaluated the effect of control measures.

The metapopulation models are usually high dimensional and contain many parameters that need to be estimated. They also assume that the population of each patch is sufficiently large so that a deterministic model is appropriate and there is homogeneous mixing within each patch. In this work we focus on reviewing the modelling of mosquito borne diseases transmitted by mosquitos using cellular automata models.

The work is organized as follows: in section 2 we present some of the main assumptions of previously reported cellular automata model for mosquito diseases spread. In section 3 models are classified according to some of their features such as: the use of GIS (geographical information), type of compartmental model, human mobility, simulation of hypothetical cases or fitting the model to real data, mosquito control strategies, population densities and seasonality. In section 4 we include some conclusions of this work.

Cellular automata models to simulate mosquito diseases spread

Due to their simplicity and to the remarkable potential to model complex systems [13-15], cellular automata methods are becoming quite popular among the modeling and simulation community.

A cellular automaton (CA) is a collection of cells (squares, hexagons, triangles) arranged in a grid, such that at each time cell changes state as a function of its state and the state of the neighboring cells according to a defined set of rules.

Due to their ability to reproduce global behavior of a system by using local interactions, their easy implementation and their suitability for visualizing diseases-spreading results, cellular automata approaches have been widely used to model diseases spread propagation as Ortigoza and Lorandi have reported[16].

A cellular automata approach can be suitable to simulate local evaluations of mosquito diseases models where diseases variables (such as mosquito-human and human-mosquito transmission rates, mosquito birth/death rates, extrinsic incubation rates among others) can be assumed to be time and space dependent, allowing in this way an easy implementation of seasonality due to ambient variables as temperature and rainfall. Moreover stochastic effects may be significant at early stages of an outbreak and also when patch populations are small, thus it seems to be reasonable to consider stochastic cellular automata in order to model and simulate mosquito diseases spread. In spite of all these features, only a few cellular automata models have been defined to simulate the spread of specific mosquito-borne diseases. In 2006 Ferreira [17] coupled a continuous differential equations model to a cellular automata in order to simulate the controlling dispersal dynamics of *Aedes aegypti* (the vector responsible for dengue fever). However, the first cellular automata for mosquito borne diseases spread was reported by Gagliardi [18]. They defined a SEIR sei probabilistic cellular automata to run spatio-temporal simulations of dengue diseases spreading, and employing two overlapping rectangular grids in an hypothetical domain, they accounted for the local interactions of infected due to the neighboring cells and global interactions due to mobility population by using probabilities. Merchant [19] proposed a SI si probabilistic

cellular automata model to simulate malaria spread. The implementation of his model can be integrated with EpiSims/TRANSIMS (Epidemic Simulation System/ microsimulator to conduct regional transportation system analyses). Host mobility, vector diffusion and population densities were considered. Santos [20] defined a three-level (three coupled rectangular grids) cellular automata for human, adult and immature vector populations for dengue diseases spread, their probabilistic model includes external seasonality forcing, human and vector mobility and vector control effects. They included seasonality by using time series for weekly rainfall data. Ramchurn [21] introduced a method which combines Google Earth images, SEIR stochastic cellular automata and scale-free network ideas to yield quantitative estimates for the outcome of a localized dengue fever outbreak in Mauritius 2009. They considered an area of 2.9 km x 3.6 km divided into cells each 0.1 km x 0.1 km in size. Using colour image analysis they estimated the number of houses and to estimate the human population in a cell they assumed an average number of five inhabitants per house. Botari [22] defined a probabilistic SIR cellular automata model on a rectangular grid to fit data of a dengue outbreak in Rio de Janeiro. They considered constant density of mosquitos flying by a Lévy flight distribution and a limited number of people at each site (approximately a lot or house with an average of 3 people per site), they assumed the probability of infected mosquitos as being dependent on temperature and rainfall index. Castro [23] developed a stochastic SEIR_sei cellular automata to simulate the spread of dengue fever in a dense community. They defined two rectangular grids for host and mosquitos with house index and human mobility (using a probability of host movement); the movement of mosquitos was implemented using different ranges of Moore neighborhoods. Their numerical experiments show that high house indexes values combined with high/moderate vector/human ratio provide the maintenance of viral transmission. Gerardi and Monteiro [24] defined SIR and SIS cellular automata on rectangular grids; they considered a time step of a week (infectious period) thus transition rules from I to R or I to S are deterministic, they assumed the mosquitos are uniformly distributed over the rectangular space thus they do not consider the mosquito concentration. They used a genetic algorithm to find the infection probabilities by fitting the model to the reported number of cases of dengue fever in Rio de Janeiro for 2007 and 2008. Enduri and Jolad [25] defined a stochastic SEIR sei cellular automata on two overlapping rectangular lattices to reproduce observed peaks and intensity of dengue reported data in India 2006-2012. They included diffusion of vectors and human mobility using a scale free Lévy pattern; they found that although human mobility makes the infection spread faster, there is an apparent early suppression of the epidemic compare to immobile humans. Theodorakos [26] defined a probabilistic SIR si cellular automata on three layer: host, vectors and environment/climate within gridded metapopulations in order to examine influencing factors and high risk regions of dengue in Nicaragua. They employed Moore neighborhoods of varying sizes and commuting host movements in the same neighborhood, moreover a differential evolution was used as a training method to fit time series of geo-referenced host/vector population data. Pereira and Schimit [27] defined a stochastic SIR si celullar automata on two rectangular lattices to model human-mosquito interactions of three coexisting serotypes of dengue fever. They varied the mosquito birth and mosquito bites probabilities to explore dengue control strategies. Dias and Monteiro [28] defined a probabilistic SIR cellular automata on a rectangular grids to simulate the propagation of vector-borne diseases. In their numerical experiments they considered different spatial distributions and time variations of vector abundance to conclude that in clustered distributions the prevalence is lower but the eradication is more difficult to be achieved, as compared to homogeneous distributions. Let us mention that besides the work of Ferreira [17] Syafarina [29], Vogel [30] and Gouvêa [31] also reported the use of a cellular automata to simulate the mosquito population dynamics. Finally, even though it is not a mosquito-borne disease model, the work of Eosina [32] used a Hiden Markov model to find a probabilistic function that represented a cellular automata transmission rule employed to fit spatio-temporal dengue data.

Classifying the models

Table 1 summarizes the attributes of the surveyed cellular automata that have been employed to model the propagation of mosquito borne diseases. All the reviewed cellular automata models were assumed probabilistic, defined on rectangular grids, and based on a compartmental approach for humans and mosquitos. 45% of them are SEIR_sei and only 27% of them did not consider the mosquito diseases dynamics (only compartments for the states of humans), 54% do not consider the *exposed human* compartment (no incubation period for humans); 81% of the models were specifically defined for dengue fever propagation, 9% for malaria, none of them considered horizontal transmission for diseases transmitted by both mosquitos and sexual contact such that zika and mayaro, 63% of the models include mobility, 54% fitting to real cases, 45% include seasonality effects, 36% consider control strategies and only 27% include geolocation (GIS).

Table 1: Models classification

	Type	GIS	Mobility	Hypothetical	Fitting data	Control	Seasonality
				Case	to real case	strategies	
Gagliardi	SEIR sei		X	X			
Santos	SEIR sei		X		X		X
Ramchurn	SEIR sei	X	X		X	X	
Botari	SIR		X		X		X
Gerardi	SIR/SIS				X		
Castro	SEIR sei		X	X		X	
Theodorakos	SIR si	X			X		X
Enduri	SEIR sei		X		X		X
Pereira	SIR si			X		X	
Dias	SIR			X		X	X
Merchant	SI si	X	X	X			

Table 2: Cellular automata and simulation classification

	model approach	Neighborhood	B.C.	Initial Conditions
Gagliardi	Individual	Moore	X	X
Santos	Populated	Moore	closed	random mosquitos
Ramchurn	Populated	X	X	population density
Botari	Populated	X	Periodic	constant average
Gerardi	Populated	Neumann/ Moore	X	constant average
Castro	Populated	Ring	periodic	population density
Theodorakos	Populated	Moore	X	population density
Enduri	Populated	Moore	X	Poisson distribution H/M
Pereira	Individual	Ring	periodic	constant average
Dias	Individual	Moore	periodic	uniform,random,cluster mosquitos
Merchant	Populated	Moore	X	population density

Table 2 summarizes cellular automata and modeling characteristics of the reviewed models. Among these characteristics we indicate if the approach is individual based (each cell is an individual host) or populated (each cell contains several hosts), type of neighborhood, type of boundary condition and type of initial condition. Here the cross mark means that the type was not stated, H stands for humans and M for mosquitos. With respect to the type of neighborhood: 54% used Moore type, 18% ring type (to model vector diffusion movement, ring is the difference between concentric Moore neighborhoods of consecutive radii), 27% did not state the type of neighborhood, 54% of the models did not state the type of boundary conditions, periodic boundary conditions were used at 36% and 9 % used a closed boundary condition. Although the basic reproduction number is of great importance for diseases spread, none of the reviewed models deal with the space variation of the basic reproduction number. Thus we suggest that spatial analysis as the one accomplished by Cissé [33] must be conducted for mosquito-borne disease cellular automata models.

Regarding to the initial conditions, it is clear that in order to run a simulation, an initial seed of infection of humans or mosquitos must be assumed, but it is also important to state the initial spatial distribution of hosts and vectors. This can be achieved in different ways, assuming constant average number of host/vectors per cell, uniform and Poisson distribution or using localized population densities from GIS. Setting appropriate initial conditions is relevant because spatially heterogeneous transmission may arise due to spatial variation in vector habitat and human population density. Relating to boundary conditions, it is important to choose the appropriate ones. For instance, close boundary conditions on an isolated region (no immigration) produce a different behavior in persistence that flow boundary conditions where susceptibles are allow to enter into the computational domain. Cellular automata approaches can model control strategies by including local evaluations of time space dependent probabilities: biting vector to human rate, mosquito birth and mosquito death. Moreover they can provide (at a not substantial computational complexity) valuable extensions of mosquito diseases models such that including heterogeneity in the mosquito biting at each cell by using a negative binomial distribution as the ode model proposed by Kong [34] or even include more compartments. For instance, Pereira [27] used 24 compartments to model dengue spread with 3 strains, Baez [35] defined a 18 compartments ode model for coinfection of Chikungunya and dengue. Isea and Lonngren[36] employed a 30 compartments ode model for a triple (dengue, zika, chikungunya) epidemic outbreak or models that also include eggs, larva, pupa and adult compartments for mosquito cycle life.

Mainly, during winter time, mosquitos are less numerous or even absent, on the other hand during the rest of the year they are more numerous and will present one or two peaks in abundance (summer time). Seasonality (timing and pattern of abundance cycles) affects the impact of mosquitos on infection dynamics. In fact, environmental variables such as temperature and humidity have an effect on the variation of vector numbers and vector activity.

High mosquito abundance is mostly associated with an increased probability of pathogen establishment, this increase in abundance can be used as an early warning of potencial pathogen establishment and transmission. Indeed, activity and mosquito survival are decreased when seasonally unfavorable conditions appeared. Climate seasonal changes are tightly related to development rate and vector activity, this will affect the vector abundance and pathogen transmission [37].

Cellular automata implementations provide flexibility to include seasonality effects into mosquito-host diseases spread models, at each cell and at each time step some important mosquito-borne diseases parameters can be modified according to the changes in weather variables. For instance Zhu [38] and Kakarla [39] reported relations for the extrinsic incubation rate, mortality mosquitos rate, human-vector, vector-human transmission probabilities as functions of temperature. Cellular automata models allow us to consider no homogeneous spatial distributions of humans and vectors; with the exception of Castro [23] and Theodorakos [26], all the other reviewed cellular automata approaches assume that humans are distributed covering the entire domain (non empty human cells) which is an appropriate assumption for high density human populated urban areas but should be relaxed in order to consider semi-urban and rural areas. Dias [28] made simulations with uniform, random and cluster distributions for mosquitos. Population densities are implemented by assuming different number of individuals (host/vectors) per cell. Theodorakos [26] employed population density data from NASA and the European Space Agency. These population densities can also be considered as time varying driven by environmental variables (rainfall, temperature, humidity, etc.).

As the consequences of climate change are becoming evident, climate-based models of disease risk are of growing importance, Tjaden [40] reviewed some of the main climate-based models for mosquito borne diseases spread. Vector habitats and vector densities can be identified and characterized by using satellite-derived environmental variables such as temperature, humidity and land cover type, Kalluri [41] review some remote sensing techniques applied to vector-borne diseases spread. The book of Khormi and Kumar [42] shows how

the use of geographic information systems (GIS) can provide a greater understanding of how vector-borne diseases are spread: monitoring, management and control.

As reported by Schneckenreither [43], cellular automata provide us with an alternative modeling approach to study spatial diseases spread where we can explore local interactions at different scales. Mosquito-borne cellular automata models can be directly derived from ode models. In fact, there is a close relationship between ode and cellular automata approaches, Misici and Santarelli [44] presented simulations where cellular automata shows similar quantitative and qualitative behavior in space and time as the ode SIR model.

As an example, we include a cellular automata model derived from the SEIR sei ode model of Brauer [5] and adapted to Chikungunya disease

$$S' = -\beta S \frac{i_v}{n_v}$$

$$E' = \beta S \frac{i_v}{n_v} - kE$$

$$I' = kE - \gamma I$$

$$R' = \gamma I$$

$$s_v' = \mu n_v - \beta_v s_v \frac{I}{N} - \mu s_v$$

$$e_v' = \beta_v s_v \frac{I}{N} - \eta e_v - \mu e_v$$

$$i_v' = \eta e_v - \mu i_v$$

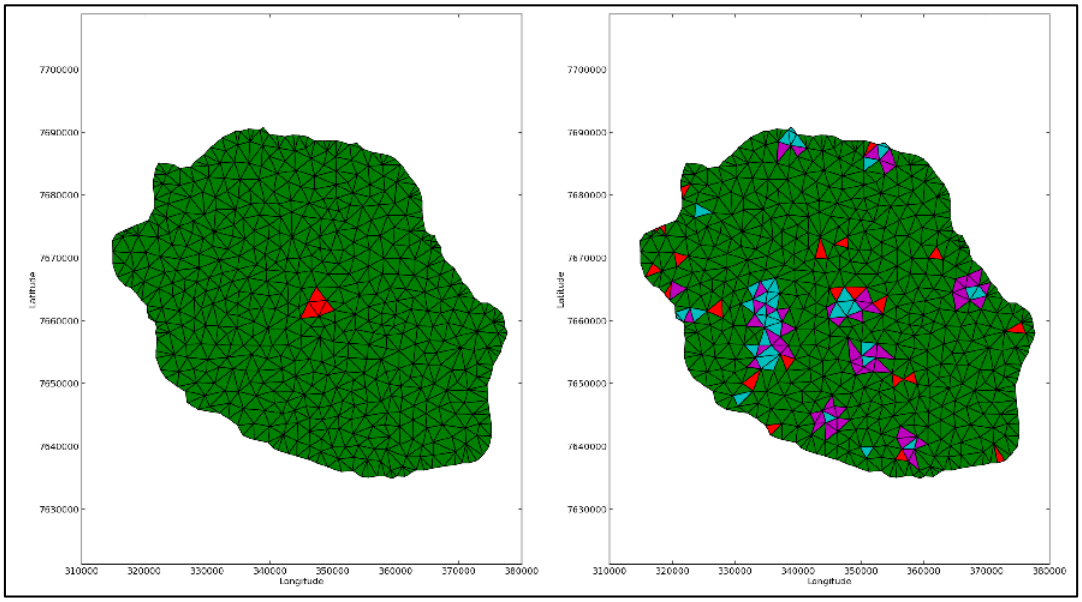

Fig 1: 20 days cellular automata simulation host mobility

Figure 1 shows the spatial diseases spread of a 20 days simulation with human mobility, the spatial domain was discretized by using an unstructured triangular grids, figure 2 shows a comparison of the time evolution of the number of Susceptible, Exposed, Infected, Infected and Recovered humans obtained with an ode SEIR_sei model and a cellular automata defined for the chikungunya outbreak at Reunion Island 2006.

The simulation was run by using the parameters obtained by Yakob and Clements [45], they fitted real data to an ode model. These parameters are summarized at table 3 and a pseudocode is provided.

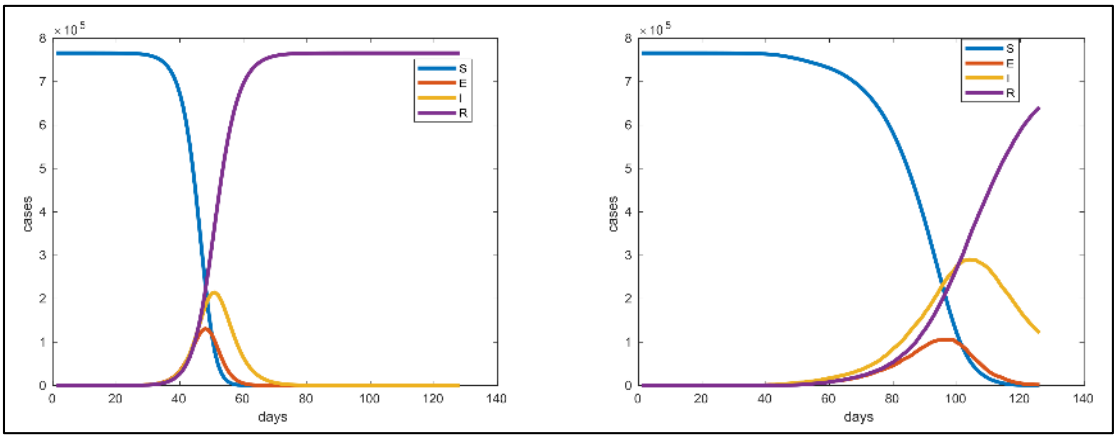

Fig 2: Left ode model, right ca model simulation, mosquitos per human, 766000 total human population, human mobility probability

Update (mesh,dof[], ndof[])
p1=(p2*beta)/(ratemh*betav);
loop over the cells i
if (mobi==1)

do movement of a portion of infected into a random selected cell
end if
Calculate Nvi, Nv infected and total number of mosquitos in the neighborhood if (rand()<(1.0-pow(1.0-p1,Nvi)))
Sh is reduced Sh*Nvi/Nv and Eh augmented end if if (rand()<alpha)
Eh is reduced Eh*alpha and Ih augmented end if
if (rand()<gamma)
Ih is reduced Ih*gamma and Rh augmented
end if
Calculate Nhi, Nh infected and total number of humans in the neighborhood if (rand()<(1.0-pow(1.0-p2,Nhi)))
sv is reduced Sv*Nhi/Nh and ev augmented end if
if (rand()<eta)
ev reduced eta*ev and iv augmented end if
if (rand()<mu)
sv is augmented mu(ev+iv),
ev and iv are reduced mu*ev and mu*iv respectively
end if
end loop over cells

Table 3: Parameters of the Chikungunya spread model

Parameter	Value	Description
β	0.14	mosquito to human transmission
β_v	0.4	human to mosquito transmission
k	0.5	reciprocal of host latent period
η	0.5	reciprocal of mosquito latent period
γ	0.25	host recovery rate per day
μ	0.05	reciprocal of mosquito life span in days

Discussion

All the reviewed models were implemented on rectangular domains using rectangular grids. Merchant [19], Enduri [25], Botari [22] and Castro [23] included the movement of mosquitos in their models, we consider that for low resolution scales this assumption can be relaxed so mosquitos can be assumed to be confined to cells. For instance *Aedes aegypti* usually fly an average of 400 meters during their lifespan also they are unlikely to displace long distances due to wind, the evidence shows that during heavy winds they do not fly, instead they stay closer to the ground. Flight range studies suggest that most female *Aedes aegypti* may spend their lifetime in or around the houses where they emerge as adults. This means that people, rather than mosquitos, rapidly move the virus within and between communities and places. As reported by Stoddard [46] human mobility is an important feature that mosquito-borne diseases cellular automata models must include. On the other hand for high resolution space scales the effect of local mosquito mobility (mosquitos move and tend to aggregate around humans) should be considered as Moulay and Pigné [47] showed in their numerical simulations. With a cellular automata formulation this can be easily accomplished, for instance just by allowing local movement of mosquitos inside the neighborhood, a portion of the total number of mosquitos move to the highest human populated cell of the neighborhood. More realistic simulations can be achieved by implementing a cellular automata on an unstructured triangular grid [48, 49]. Besides the anisotropy reduction (anisotropy is induced by rectangular grids), some of the immediate benefits include the use of computational domains defined by simple polygonal regions (flat bounded domains consisting of straight non-intersecting line segments) with holes and not only the traditional rectangular domains employed in classical cellular automata implementations. In geographical information systems the area of study can be defined by a polygon file. Geographical data such as population density, soil classification, rain fall, temperature, etc. are oftentimes defined by TIN files (triangulated irregular network). Finite element experience with mesh structure of the triangular grids can be employed to provide an easy implementation of the transition function, the neighborhood identification, the setting of initial/boundary conditions and the visualization of the final results.

Conclusions

Cellular automata formulations allow us to consider local interactions of infected due to neighboring cells and global interactions due to human mobility probability. At each cell, control vector strategies are implemented by assuming bitting rates, mosquito birth and death rates that depend on time and space. Moreover space heterogeneities are accounted locally by using population densities and seasonality is easily implemented by considering extrinsic incubation rates, mosquito's birth rate and vector-human transmission probabilities as functions of temperature (time-space dependent). All these features make cellular automata an attractive method to perform mosquito-borne diseases simulations. Nevertheless, spatial analyses of the basic reproduction number must be conducted for mosquito-borne disease spread cellular automata formulations. Individual based cellular automata (a host per cell) seems to be more appropriate to simulate diseases spread in small population regions such that villages, towns or small cities. Populated cells (more than one host per cell) can be employed for simulations over bigger regions. In fact, a domain decomposition method can be adapted to identify big patches such as districts, counties and a local refinement can be used on each sub domain. This naturally lead us to consider parallel implementations such as message passing interface (MPI) and share-memory programming (OpenMP).
The more features that a model includes to mimic the reality, the more complexity it requires for its numerical implementation. Thus a commitment must be made between the computational complexity and the features included in the model, we suggest: host mobility, GIS (geolocation of host and mosquitos using population densities), control strategies, fitting data and seasonality (whenever data are provided) as minimal features to be included in cellular automata mosquito-borne diseases spread models.

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References

1. Ross R. The Prevention of Malaria 2nd ed., (with Addendum) John Murray, London, 1911.
2. MacDonald G. The Epidemiology and Control of Malaria, Oxford University Press, London, 1957.
3. Reiner R, Perkins T, Barker C, Niu T, Chaves L, Ellis A *et al*. A systematic review of mathematical models of

mosquito-borne pathogen transmission: Journal Real Society, Interface. 1970-2010; 10(81).
4. Eder M, Cortés F, De-Siqueira N, Araújo G, Degroote S, Braga C *et al*. Scoping review on vector-borne diseases in urban areas: transmission dynamics, vectorial capacity and co-infection. Infectious Diseases of Poverty. 2018; 71(1):90.
5. Brauer F, Castillo-Chavez C, Mubayi A, Towers S. Some models for epidemics of vector-transmitted diseases. Infectious Disease Modelling. 2016; 1(1):79-87.
6. Bryan C, Mniszewski S, Liu K. Integrating Predictive Visualization with the Epidemic Disease Simulation System (episims). In Proceedings of the IEEE VIS 2014 workshop visualization for predictive analytics, 2014.
7. Soda KJ, Moore SM, España G, Bloedow J, Raybaud B, Althouse B *et al*., DTK-Dengue: A new agent-based model of dengue virus transmission dynamics. Bio Rxiv. 2018, 376-533.
8. Moore S, Bosch Q, Siraj A, Soda L, España G, Campo A *et al*., Local and regional dynamics of chikungunya virus transmission in Colombia: the role of mismatched spatial heterogeneity. BMC Medicine. 2018; 16(1):152.
9. Cosner C, Beier J, Cantrel R, Impoinvil D, Kapitanski L, Potts M *et al*. The effects of human movement on the persistence of vector-borne diseases. Journal of theoretical biology. 2009; 258(4):550-560.
10. Wang W. Epidemic Models with Population Dispersal. En Mathematics for Life Science and Medicine. Berlin Heidelberg: Springer; 2007, 67-95.
11. Bichara D, Castillo-Chavez C. Vector-borne diseases models with residence times-a lagrangian perspective. Mathematical biosciences. 2016; 281:128-138.
12. Arino J, Ducrot A, PZongo. A metapopulation model for malaria with transmission-blocking partial immunity in hosts. Journal of Mathematical Biology. 2012; 64(3):423-448.
13. Sloot P, Hoekstra A. Cellular Automata as a Mesoscopic Approach to Model and Simulate Complex Systems Berlin Heidelberg: Springer, 2001.
14. Batty M. Cities and Complexity: Understanding Cities with Cellular Automata, Agent-Based Models, and Fractals: The MIT Press, 2005.
15. Deutsch A, Dormann S. Cellular automaton modeling of biological pattern formation Boston: Birkhauser, 2005.
16. Ortigoza G, Lorandi A. Modelos de Autómatas Celulares para simular la propagación de enfermedades infecciosas. Revista de Investigación en Ciencias de la Salud, Universidad Veracruzana. 2018; 13(2):6-12.
17. Ferreira C, Pulino P, Yang H, Takahashi L. Controlling Dispersal Dynamics of Aedes aegypti. Mathematical Population Studies. 2006; 13(4):215-236.
18. Gagliardi H, da-Silva F, Alves D. Automata Network Simulator Applied to the Epidemiology of Urban Dengue Fever Berlin: Springer, 2006.
19. Merchant F. Simulating the spread of malaria: a cellular automata based mathematical model and a prototype software implementation: Doctoral dissertation, Virginia Tec, 2007.
20. Santos L, Costa M, Pinho S, Andrade R, Barreto F, Teixeira M *et al*. Periodic forcing in a three-level cellular automata model for a vector-transmitted disease. Physical Review E. 2009; 80(1):016102.
21. Ramchurn S, Moheeput K, Goorah S. An analysis of a short-lived outbreak of dengue fever in Mauritius. Eurosurveillance 2009; 14(34):19314.
22. Botari T, Alves S, Leonel E. Explaining the high number of infected people by dengue in Rio de Janeiro in 2008 using a susceptible-infective-recovered model. Physical Review E. 2011; 83(3):037101.
23. de-Castro-Medeiros L, Castilho C, Braga C, de-Souza W, Regis L, Monteiro A. Modeling the dynamic transmission of dengue fever: investigating disease persistence. PLOS neglected tropical diseases. 2011; 5(1):e942.
24. Gerardi D, Monteiro L. ystem Identification and Prediction of Dengue Fever Incidence in Rio de Janeiro. Mathematical Problems in Engineering. 2011.
25. Enduri M, Jolad S. Dynamics of Dengue with human and vector mobility. arxiv populations and evolution. 2014; 1409:0965.
26. Theodorakos K, Broeckhove J, Willem L. Examination of influencing factors and high-risk regions of dengue in Nicaragua, using spatiotemporal compartmental simulations. Tropical Medicine & International Health. 2017, 156-157.
27. Pereira F, Schimit P. Dengue fever spreading based on probabilistic cellular automata with two lattices. Physica A: Statistical Mechanics and its Applications. 2018; 499:p. 75-87.
28. Dias J, Monteiro L. Clustered Breeding Sites: Shelters for Vector-Borne Diseases. Computational and mathematical methods in medicine. 2018, 2018.
29. Syafarina I, Sadikin R, Nuraini N. Mosquito population dynamics from cellular automata-based simulation. AIP Conference Proceedings. 2016; 1705(1):020051.
30. Vogel U, Lhken R, Kiel E. A Mosquitoes Tool for Simulating the Spread of Invasive, Proceedings of the 28th EnviroInfo 2014 Conference, 2014.
31. Gouvêa-Jr M. Time-spatial model on the dynamics of the proliferation of Aedes aegypti. Communications in Nonlinear Science and Numerical Simulation. 2017; 44:130-143.
32. Eosina P, Djatna T, Khusun H. A Cellular Automata Modeling for Visualizing and Predicting Spreading Patterns of Dengue Fever. TELKOMNIKA Telecommunication, Computing, Electronics and Control. 2016, 228.
33. Cissé B, El-Yacoubi S, Gourbière S. A cellular automaton model for the transmission of Chagas disease in heterogeneous landscape and host community. Applied Mathematical Modelling. 2016; 40(2):782-794.
34. Kong L, Wang J, Li Z, Lai S, Liu Q, Wu H *et al*. Modeling the Heterogeneity of Dengue Transmission in a City. International journal of environmental research and public health. 2018; 15(6):1128.
35. Baez N, Casa M, Danis R, Velasco J. A mathematical model fpr Dengue and Chikungunya in México. [Online]. Disponible en: https://www.biorxiv.org/content/biorxiv/early/2017/03/30/122556.full.pdf.
36. Isea R, Lonngren K. A Preliminary Mathematical Model for the Dynamic Transmission of Dengue, Chikungunya and Zika. arXiv preprint, 2016.
37. Control ECfDPa. The importance of vector abundance and seasonality.
38. Zhu G, Liu T, JX, Zhang B, Song T, Zhang Y *et al*. Effects of human mobility, temperature and mosquito control on

the spatiotemporal transmission of dengue. Science of the Total Environment. 2019; 651:969-978.
39. Kakarla S, Mopuri R, Mutheneni S, Bhimala K, Kumaraswamy S, Kadiri M *et al*., Temperature dependent transmission potential model for chikungunya in India. Science of the Total Environment. 2019; 647:66-74.
40. Tjaden N, Caminade C, Beierkuhnlein C, Thomas S. Mosquito-borne diseases: advances in modelling climate-change impacts. Trends in parasitology. 2018; 34(3):227-245.
41. Kalluri S, GP, RD, SM. Surveillance of arthropod vector-borne infectious diseases using remote sensing techniques: a review. PLoS pathogens. 2007; 3(10):e116.
42. Khormi H, Kumar L. Modeling Interactions between Vector-borne Diseases and Environment Using GIS: CRC press, 2015.
43. Schneckenreither G, Popper N, Zauner G, Breitenecker F. Modelling SIR-type epidemics by ODEs, PDEs, difference equations and cellular automata A comparative study. Simulation Modelling Practice and Theory. 2008; 16(8):1014-1023.
44. Misici L, Santarelli F. Epidemic Propagation: An Automaton Model as the Continuous SIR Model. Applied Mathematics. 2013; 4(10C):84-89.
45. Yakob L, Clements A. A Mathematical Model of Chikungunya Dynamics and Control: The Major Epidemic on Reunion Island, PLoS ONE. 2013; 8(3):e57448.
46. Stoddard S, Morrison A, Vazquez-Prokopec G, Soldan V, Kochel T, Kitron U *et al*. The Role of Human Movement in the Transmission of Vector-Borne Pathogens. PLoS neglected tropical diseases. 2009; 3(7):e481.
47. Moulay D, Pigné Y. A metapopulation model for chikungunya including population mobility on a large-scale network. Journal of theoretical biology. 2013; 318:129-139.
48. Ortigoza G. Unstructured Triangular Cellular Automata for Modeling Geographic spread. Journal of applied mathematics and Computation Elsevier. 2015; 258:520-536.
49. Ortigoza G, Lorandi A, Neri I. ACFUEGOS: An Unstructured Triangular Cellular Automata for Modelling Forest Fire Propagation. En High Performance Computer Applications. Springer. 2016, 132-143.