

{ Alife Mutants Hackingsession on Systems and Organisms, Bielefeld 2004 }

# Spatial Epidemics & AL

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# Overview

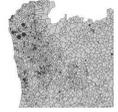
- Epidemic modelling
- Complexity
- AL epidemic models
- My model

# Epidemic Modelling

Spatial Epidemics - "Analysis of the spatial/geographical distribution of the incidence of a disease". (Lawson, A. B., 2001)

Due to its nature, epidemics have interested scientists of the field of diffusion and spatial diffusion study.

Nowadays, with the emergence of AIDS as pandemic disease, the re-emergence of malaria and tuberculosis as global killers, Ebola, Lyme disease and more recently SARS, the interest in epidemic diseases studies was renewed. (Cliff, A.; Hagget, P.; Smallman-Raynor, M., 1998)



# Epidemic Modelling

Spatial Epidemics Models

→

Top Down

→

Bottom Up

→

Emergence

• Deterministic and based on systems of differential equations: Markov Chains, mean field type equations

• Stochastic and based on computer simulations: agent based models, CA, SW, ...

# Epidemic Modelling

Deterministic models:

Type of Epidemic	Epidemic component	Mathematical symbol	Technology transfer equivalent
Simple	Susceptible	S	Potential adopter
General	Infective	I	adopter
	Susceptible	S	Potential adopter
	Infective	I	adopter
	Removed	R	Rejecter

SI model for microparasites.  
<http://hilbert.dartmouth.edu/~m4w02/syl2.htm>

$$\frac{dS}{dt} = -rSI$$

$$\frac{dI}{dt} = rSI - \gamma I$$

$$\frac{dR}{dt} = \gamma I$$

S - susceptible  
 I - infective  
 R - removed  
 r - infection rate  
 U - removal rate  
 $r = r / U$  (threshold value)  
 $S_0 > r$

# Epidemic Modelling

SLIR model (Chen, S., 2001)

$$\Delta S(t) = S(t+1) - S(t)$$

$$\Delta S(t) = -\beta(t) S(t) I(t)$$

$$\Delta I(t) = \beta(t) S(t) I(t) - \gamma(t) S(t+1) I(t) - \nu(t) I(t)$$

$$\Delta I(t) = \beta(t) S(t) I(t) - \gamma(t) S(t+1) I(t) - \nu(t) I(t) - \nu(t) I(t)$$

$$\Delta R(t) = \gamma(t) S(t) I(t) - \nu(t) I(t)$$

$\beta$  Infection force  
 $\gamma$  Latent period  
 $\nu$  Infectious period

rule 110

## { Epidemic Modelling }

**Some objections can be opposed to this kind of models:**

- Diffusive or perfect mixing, and random motion are assumptions not always fulfilled, at least in human populations. (Mansila, R; Gutierrez, J.L.).
- These models tend to incorporate many parameters to explain reality, which increases their complexity and makes them computationally intensive and difficult to analyze!

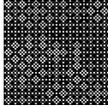
rule 110

## { Complexity }

Contrary of what happens on top down approach, on complex systems approach it is considered that spatial extended systems (dynamical systems in which n spatial dimensions are added) are capable of non-trivial collective behaviour - unexpected behaviour which is observed in macroscopic quantities.

It is assumed that there are several levels of reality: at a microscopic level, interactions may be described by complicated potentials, but at a macroscopic level, the properties of the system are dominated by the aggregated effect of all microscopic interactions.

Human epidemics are strongly related to the dynamics of populations and to the network of social contacts, which is typically a complex system where emergence appears (Nowak, A.; Lewenstein, M.).



rule 110

## { AL Epidemic Models }

Epidemic models using artificial techniques try to capture the complexity of spatial epidemics. Concerning the structure of the model, there have been essentially two approaches:

- The grid approach (cellular automata)
- the graph approach (small worlds)

Some models combine this two approaches and even the compartmental models approach (Chen, S., 2001).

rule 110

## { AL Epidemic Models }

"Cellular Automata (CA) are discrete dynamical systems (in space and time) whose behaviour is completely specified in terms of a local relation" (Toffoli ET AL, 1991).

Some advantages of CA epidemic models:

- treat individuals in biological populations as discrete entities and allow to introduce local stochasticity.
- are very well suited for computer simulations, essentially due to its inherent parallelism and locality.



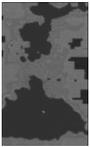
Wolfram's rule 130

rule 110

## { AL Epidemic Models }

There are some examples of CA epidemic models in plant populations (A.C. Newton, G. Gibson & D. Cox). As plants can not move, the pattern of infection is fully determined by local relations

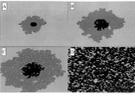
In human populations, CA models also have to deal with the problem of movement of individuals, plus the interaction between individuals. There have been several approaches to this problem in recent studies.



Map of red core severity, with blue areas denoting greater disease incidence (A.C. Newton, G. Gibson & D. Cox)

rule 110

## { AL Epidemic Models }

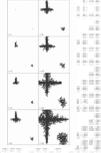


Interaction radius to define the neighborhood and probability transition rules: Fuentes M. A., Kuperman, M. N., 1999.

Probabilistic CA model (Fuentes M. A., Kuperman, M. N., 1999)

Ching Fu, S.; Milne, G.

use neighborhood radius and motion radius to model contagious and movement, with probabilities associated to each one. In this model, a cell represents a equal sized area of the landscape containing individuals.



Epidemic model (Ching Fu, S.; Milne, G.)

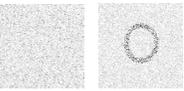
{ AL Epidemic Models }

Lattice Gas Cellular Automata (LCGA):

LCGA - particular class of CA where the dynamics is performed in parallel over all the sites and can be decomposed in two stages: propagation: the particles jump to a nearest-neighbour site according to their direction; collision: the particles entering the same site at the same interaction, interact, producing a new particle distribution. Examples: HPP, FHP (Chopard, B.; Droz, M; 1998)

► Fuks, H.; Lawniczak, T., 2002:

LCGA for reaction-diffusion systems (generic disease); hexagonal grid with a maximum density of individuals in each cell and random movement into adjacent cells.



LCGA model with homogenic and barrier vaccination (Fuks, H.; Lawniczak, T., 2002.)

{ AL Epidemic Models }

Site Exchange Cellular Automata (SE CA):

SE CA - CA networks whose rules consist of two subrules: contagious rule - is a local rule synchronously applied, based on Conway's " Game of life " (describes the behavior of the disease); transport rule: sequentially applied, describes the motion of individuals.



► Mansila, R; Gutierrez, J.L.:

In this model, movement is random, but the degree of mixing between individuals is controlled by a parameter (p).

Deterministic SE model (Mansila, R; Gutierrez, J.L.)

{ AL Epidemic Models }



<http://www.c3.lanl.gov/mega-math/gloss/graph/gr.html>

**Graphs** - A graph G consists of a non empty set of elements (vertices) and a list of unordered pairs of these elements (edges). (Chen, S., 2001)

Graphs can be used to represent all kinds of networks, where vertices represent network elements (like people) and edges represent some kind of a predefined relationship between connected elements (like a transmission route).

The theory of Small worlds, also known as "six degrees of separation", was developed by Duncan Watts and it refers to a particular kind of graphs between order and randomness (Watts, Duncan J., 2003).

{ AL Epidemic Models }

Complex networks, such as social contacts, present some key characteristics:

- short average path length
- high level of clustering
- power law and exponential degree distributions



The spread of a disease occurs by person-to-person contact, and the structure of networks of such contacts has a huge impact on the nature of epidemics. There are some examples of SW epidemic models.

Small World Network (Chen, S., 2001)

{ AL Epidemic Models }

► Moore, C; Newman, M. E.J.:

CA probabilistic model: site and bond percolation. The occupation probabilities of sites correspond to the susceptibility of individuals to the disease and the occupation probabilities of bonds correspond to the transmissibility of the disease. The model is build on a SW network.



Small World network (Moore, C; Newman, M. E.J.)

{ AL Epidemic Models }

► Chen, S (2001):

Foot and Mouth Disease; Two levels: individuals inside the farms, combine compartmental models (mean field assumption) and lattice based methods; agents between farms: SW method. Individuals move randomly inside the farm, between the neighborhoods. Virus transmission routes could be local (neighbor farms) or long-distance (countrywide movement). A variable P (0 to 1) controlled this movement from neighborhood to totally random ( $\alpha$ )



Definition of the local neighbours of a farm (Chen, S., 2001)

**AL Epidemic Models**

► "Modeling Global Epidemics":  
 CA model of the propagation of diseases in the world, where each cell represents the people living in the area. It was assumed that people living in highly populated areas are interacting with more people and that people living in parts of the world with more developed infrastructure tend to travel over large distances (structured diffusion).

$I_{x,y}^{cont}$  - number of people that are contagious in the source-cell X.  
 $S_{y,y}^{cont}$  - number of people that are susceptible in the target-cell Y.  
 $W_{x,y}$  - weight between 0 and 1. It is proportional to the fraction of time a person living in the target-cell X in average spends in the source-cell Y during a time step.  
 $W_{x,y}$  and  $W_{y,x}$  - weight depending on the infrastructure in the source and target-cell respectively.  
 $N_x$  and  $C_x$  - number of interactions and contagiousness.  
 $Area_{cell}$  - area of the cell in  $km^2$ .  
 Weight ( $W_{x,y}$ ) parameter - several traveling possibilities are considered: stay in home cell, neighbour cell, Gaussian travelling (Gaussian distribution centred at the persons home site), SW network.

$\frac{I_{x,y}^{cont} W_{x,y} W_{y,x} N_x C_x}{area_{cell}}$

<http://www.dd.chalmers.se/~f97kahe/epidemicsim/epidemicsim.htm>

**Model**

**Targets:**

- create an individual based model where each cell actually represents an individual, so that locality is not lost by an aggregation effect.
- create a realistic motion model that considers different kinds of movements, that are not totally random, but present a structure, emulating what happens in human populations.
- create a realistic model of the disease, considering latent periods and extra parameters such as age or infrastructure qualities.
- create a coupled GIS-CA model that can put together the visualization and analysis capabilities of GIS with a fast CA model.

SE CA  
 two phases: (i) mixing and (ii) contagious; assuming there is no virus transmission while the individual is moving.

**Model**

**Mixing Phase:**  
 Let Z be the set of integer numbers and be the lattice  $\mathbb{Z}^2$   
 $\mathcal{O} = \{0, 1, \dots, \beta\}$  is the set of the element of the form  $\theta_{i,j} \in \mathcal{O}$   
 where  $\theta_{i,j} \in \mathcal{O}$  represents their subclass (susceptible, infective, removed...) and  $i, j$  the position on the matrix.  
 $\tau: \mathcal{O} \rightarrow \mathcal{O}$  is a function that satisfies the following conditions:  
 Let  $\theta_{i,j} \in \mathcal{O}$  be the image of the element  $\theta_{i',j'} \in \mathcal{O}$  under the application of  $\tau$   
 if  $\theta_{i',j'} = 0 \Rightarrow \theta_{i,j} = 0$   
 for every  $x, x', y, y'$   
 if  $\theta_{i',j'} = 0 \Rightarrow \theta_{i',j'} = 0$

**Model**

**Transport Rules:**  
 The  $\mathcal{O}$  contains a subset of Lattice  $\mathcal{O}$  units  
 The probabilistic rules applied to every  $\theta_{i,j} \in \mathcal{O}$  consider four types of movement:

- neighborhood
- intra
- inter
- SW

with units (blue and black) (grey)

**Model**

Neighborhood Intra Inter SW All

**Model**

**Contagious Phase:**  
 For now, it is used a SIR model, with Moore neighborhood:  
 $\theta_{i,j}^{t+1} = \theta_{i,j}^t \cdot \theta_{i+1,j}^t \cdot \theta_{i-1,j}^t \cdot \theta_{i,j+1}^t \cdot \theta_{i,j-1}^t$   
 The contagious rule is deterministic and totalistic.  
 The states of a cell can be expressed as  $s=i, S, R$   
 Let 1 be an infected site and 0 a non infected site.  
 The transition of a cell to I, is given by  

$$I_{i,j}^{t+1} = S_{i,j}^t \Rightarrow \begin{cases} 1 & \text{if } \sum_{i',j'} \theta_{i',j'}^t \geq 1 \\ 0 & \text{otherwise} \end{cases}$$
  
 The transition of I to R is given by  $I_{i,j}^{t+1} = S_{i,j}^t \cdot R$

