Ecological Modelling 222 (2011) 1651–1655

Contents lists available at ScienceDirect

Ecological Modelling

journal homepage: www.elsevier.com/locate/ecolmodel

Short communication

A vaccination game based on public health actions and personal decisions

P.H.T. Schimit[a], L.H.A. Monteiro[b,a,*]

[a] Universidade de São Paulo, Escola Politécnica, Departamento de Engenharia de Telecomunicações e Controle, Av. Prof. Luciano Gualberto, travessa 3, n.380, 05508-900 São Paulo, SP, Brazil

[b] Universidade Presbiteriana Mackenzie, Escola de Engenharia, Pós-graduação em Engenharia Elétrica, Rua da Consolação, n.896, 01302-907 São Paulo, SP, Brazil

ARTICLE INFO

Article history:
Received 2 December 2010
Received in revised form 15 February 2011
Accepted 17 February 2011
Available online 23 March 2011

Keywords:
Complex network
Game theory
Probabilistic cellular automaton
SIR model
Vaccination

ABSTRACT

Susceptible-infective-removed (SIR) models are commonly used for representing the spread of contagious diseases. A SIR model can be described in terms of a probabilistic cellular automaton (PCA), where each individual (corresponding to a cell of the PCA lattice) is connected to others by a random network favoring local contacts. Here, this framework is employed for investigating the consequences of applying vaccine against the propagation of a contagious infection, by considering vaccination as a game, in the sense of game theory. In this game, the players are the government and the susceptible newborns. In order to maximize their own payoffs, the government attempts to reduce the costs for combating the epidemic, and the newborns may be vaccinated only when infective individuals are found in their neighborhoods and/or the government promotes an immunization program. As a consequence of these strategies supported by cost-benefit analysis and perceived risk, numerical simulations show that the disease is not fully eliminated and the government implements quasi-periodic vaccination campaigns.

1. Introduction

Probabilistic cellular automaton (PCA) (e.g. Wolfram, 1994) has been used for modelling the spreading of contagious diseases (e.g. Ahmed et al., 1998; Monteiro et al., 2006a; Moreno et al., 2002; Newman, 2002; Schimit and Monteiro, 2009; Sirakoulis et al., 2000; Yakowitz et al., 1990). In this approach, each cell composing the PCA lattice usually corresponds to an individual, which is in one of three states, *S*, *I* or *R*, as in the classical SIR model proposed by Kermack and McKendrick (1927). The state *S* represents the individual that is susceptible and therefore subjected to the infection; the state *I* is related to the individual that is infective and hence can transmit the disease for those that are susceptible; the state *R* is associated with removal from the process of disease propagation, meaning that the individual is either cured and immunized or dead. In this epidemiological context, disease-causing contacts form a connection network among the individuals. The corresponding coupling topology can be considered as regular (e.g. Ahmed et al., 1998; Monteiro et al., 2006a; Sirakoulis et al., 2000; Yakowitz et al., 1990) or irregular (e.g. Moreno et al., 2002; Newman, 2002; Schimit and Monteiro, 2009). Networks with regular and local couplings are convenient to model a contagious disease with short-range character. When long-range interactions must also be taken into account, random coupling topologies (e.g. Barrat et al., 2008) are often employed.

The numerical values of the probabilities concerning the transitions among the states *S*, *I* and *R* and the choice of the topology used for representing the contacts among the individuals define a SIR model based on PCA. This framework has been utilized for investigating, for instance, the spatio-temporal evolution of the number of individuals in each PCA state (e.g. Ahmed et al., 1998; Doran and Laffan, 2005; Monteiro et al., 2006a; Slimi et al., 2009; Yakowitz et al., 1990); the relations among topological properties and features of the epidemiological dynamics (e.g. Cohen et al., 2003; Holme, 2004; Moreno et al., 2002; Newman, 2002; Schimit and Monteiro, 2009); the effects of attitudes commonly adopted against infection spreadings, like use of face masks against airborne infections (e.g. Schimit and Monteiro, 2010), restrictions on long-range population movement (e.g. Sun et al., 2010), and vaccination (e.g. Ajelli and Merler, 2009; Liu et al., 2003; Sirakoulis et al., 2000).

Vaccines are used for controlling the propagation of contagious diseases. They can be administered to the susceptible population either continually, which is called constant vaccination, or periodically, which is called pulse vaccination (e.g. Shulgin et al., 1998). Both these modes of distributing vaccines have been implemented. For instance, the first mode was worldwide used for eradicating smallpox (e.g. Behbehani, 1983); the second mode was employed in Brazil to control the occurrence of poliomyelitis (e.g. Risi, 1984). Of course, if the susceptible portion was reduced to zero by vaccination, then the disease would disappear (by assuming that there are not natural reservoirs of the corresponding pathogen in other species and by supposing that the vaccine confers perfect and

* Corresponding author at: Universidade Presbiteriana Mackenzie, Escola de Engenharia, Rua da Consolação, n.896, 01302-907 São Paulo, SP, Brazil.
Tel.: +55 11 2114 8711; fax: +55 11 2114 8600.
E-mail addresses: pedro.schimit@poli.usp.br (P.H.T. Schimit), luizm@mackenzie.br, luizm@usp.br (L.H.A. Monteiro).

0304-3800/$ – see front matter © 2011 Elsevier B.V. All rights reserved.
doi:10.1016/j.ecolmodel.2011.02.019

sustained protection). In many theoretical studies supported by differential equations, the proportion of vaccinees is a parameter not influenced by the opinion of the citizens about the immunization program (e.g. Liu et al., 2008; Piccardi and Lazzaris, 1998; Shulgin et al., 1998; Sun and Hsieh, 2010; Yip et al., 2007). Even when individuals are represented as nodes of regular (e.g. White et al., 2007) or random graphs (e.g. Ajelli and Merler, 2009; Ben-Zion et al., 2010; Cohen et al., 2003; Holme, 2004), the public perception of the benefits and the perils of vaccination are usually not take into account.

In practice, vaccination of the entire susceptible population is impossible to achieve. Besides economic and logistical reasons associated with launching a large-scale vaccination campaign, individuals can resolve to remain unvaccinated when the perceived risk of getting sick from vaccination is similar or higher than the perceived risk of acquiring the disease in their regular social contacts (e.g. Seale et al., 2010). This scenario has inspired works on modelling vaccination as a game, in the sense of game theory (e.g. Webb, 2007). For instance, Bauch and Earn (2004) analyzed a vaccination game by using a SIR model written in terms of differential equations. They supposed that parents decide to vaccinate or not to vaccinate their children in function of their personal evaluations. Consequently, the vaccine coverage level results from the decisions of all parents. This can be considered a "game against the field" (e.g. Webb, 2007), because players influence each other, acting according to the situation of the environment (the field). Bauch and Earn (2004) concluded that disease eradication is not possible through voluntary vaccination if the parents only think in their own interests. d'Onofrio et al. (2007) and Reluga et al. (2006) also combined game with epidemiological models and showed that sustained oscillations can appear in the infective population, via Hopf bifurcation (e.g. Guckenheimer and Holmes, 1983) caused by a variation, for instance, in the vaccination coverage. In these games, all individuals share the same information about the epidemic.

Social adhesion to a vaccination program is affected by the information disclosed by the mass media and by the messages coming from the neighborhood. Hence, the knowledge concerning the disease and the vaccine is usually not uniform over the population. Here, we investigate a vaccination game by considering as players the newborns (simulating a childhood epidemic) and the "government" (that is, the public health agency responsible for planning and accomplishing immunization programs). Thus, in this game, the field is also influenced by the government, which intends to maximize its payoff, trying to minimize the expenses for combating preventable-vaccine diseases.

The cost of a vaccination campaign is an important factor neglected in other studies. Here, we find that if the government acts only according to the cost-benefit survey of voluntary vaccination versus treatment, then the corresponding disease is not eradicated. We observe that outbreaks recurrently appear and vaccination campaigns are quasi-periodically performed.

This manuscript is organized as follows. In Section 2, the network topology employed for representing social contacts, the PCA used as epidemiological model, and the proposed vaccination game are described. In Section 3, results of numerical simulations are presented. In Section 4, the relevance of this study is stressed.

2. Vaccination model

2.1. Contact network

In our network model (Monteiro et al., 2006b; Schimit and Monteiro, 2009), each cell of the PCA lattice corresponds to an individual and an edge between two cells represents a social contact. The neighborhood matrix of a cell is defined as the square matrix of size $2r+1$ centered on such a cell. From each cell, m connections start to other cells pertaining to its neighborhood matrix. The maximum radius where a connection can be made is r. The case $r=1$ including all 8 surrounding cells is known as Moore neighborhood of unitary radius (e.g. Wolfram, 1994). The cells with Moore radius equal to i form the layer i. The probability q_i of creating a connection between a cell and any cell pertaining to the layer i of its neighborhood matrix is given by $q_i = 2(r+1-i)/[r(r+1)]$, where $i = 1, 2, ..., r$ (Schimit and Monteiro, 2009). For instance, for $r=4$, then $q_1 = 4/10$, $q_2 = 3/10$, $q_3 = 2/10$, $q_4 = 1/10$. Thus, the probability of linking a cell to any of the 8 cells composing the layer $i=1$ is 40%, to any of the 16 cells composing the layer $i=2$ is 30% and so on. Two or more connections between the same two cells are allowed. Our random network of connections is mainly locally connected (Schimit and Monteiro, 2009) like graphs called "small-worlds" (Watts and Strogatz, 1998), because the clustering coefficient c is "high" (that is, $c \gg m/N$, where N is the number of cells constituting the lattice) and, since "long-range" interactions are allowed, the average shortest path length l is "small" (that is, $l \sim \ln(N)/\ln(m)$).

2.2. SIR model

In our epidemiological model, individuals live in a square matrix formed by $n \times n = N$ cells with periodic boundary conditions. Each cell in the PCA lattice represents an individual that can be in one of three states: susceptible (S), infective (I) or recovered (R). The time evolution of the population is governed by the following set of probabilities of state transitions (Schimit and Monteiro, 2009). At each time step, there is a probability P_i of a S-cell being infected according to $P_i(\nu) = (1 - e^{-k\nu})$, where ν is the number of connections with distinct infective neighbors and k is a parameter related to the disease infectivity. Each I-cell has probability P_c per time step of becoming cured; if not cured, then it has probability P_d per time step of dying because of the disease. And, at each iteration, a R-cell may die due to other causes with probability P_n. When I and R-cells perish, susceptible ones replace them. Therefore, the total number of individuals $N = n^2$ remains constant; an appropriate assumption for modelling diseases spreading quickly and/or populations where the deaths are balanced by the births. The states of all cells are simultaneously updated throughout the simulation.

The basic reproduction number R_0 is a bifurcation parameter able of predicting the course of an epidemic (e.g. Anderson and May, 1982). In our PCA model, it can be numerically determined by (Schimit and Monteiro, 2009):

$$R_0 = \frac{N\Delta I(t)_{S\to I}}{S(t)[\Delta R(t)_{I\to R} + \Delta S(t)_{I\to S}(1 - \Delta R(t)_{I\to R}/(I(t)\Delta t))]} \quad (1)$$

where $S(t)$ and $I(t)$ are the numbers of susceptible and infective cells at the instant t, respectively; $\Delta I(t)_{S\to I}/\Delta t$ is the increase per time step of infective cells due to the contagion process; $\Delta R(t)_{I\to R}/\Delta t$ is the increase per time step of recovered cells due to the healing process; $\Delta S(t)_{I\to S}/\Delta t$ is the increase per time step of susceptible cells due to the death caused by the infection. The disease-free stationary state is asymptotically stable if $R_0 < 1$ and unstable if $R_0 > 1$; the endemic stationary state is unstable if $R_0 < 1$ and asymptotically stable if $R_0 > 1$ (Schimit and Monteiro, 2009). For instance, Fig. 1 exhibits the dynamical behaviors of the normalized concentrations of S, I and R-cells. At $t=0$, these cells are randomly distributed over the lattice according to the proportions $S(0)/N = 99.5\,\%$, $I(0)/N = 0.5\,\%$ and $R(0)/N = 0\,\%$. The parameter values used in this numerical simulation are $n=200$, $m=5$, $r=5$, $P_c = 60\%$, $P_d = 30\,\%$, $P_n = 10\,\%$ and $k=1$. In this case, $R_0 \simeq 3.6$. Notice that, in the permanent regime, the endemic state is reached. In fact, the disease persists in the population because $R_0 > 1$.

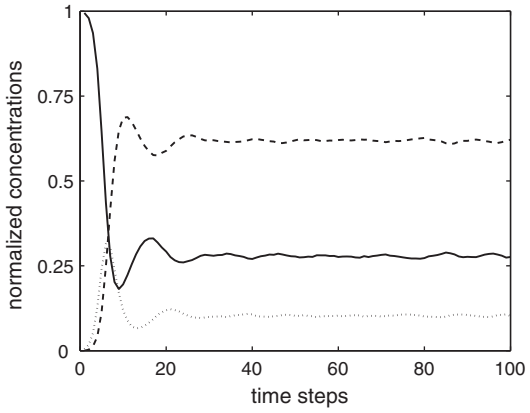

Fig. 1. Time evolutions of $S(t)/N$ (solid line), $I(t)/N$ (dotted line) and $R(t)/N$ (dashed line) obtained from a PCA simulation. The parameter values are $n = 200$, $m = 5$, $r = 5$, $P_c = 0.6$, $P_d = 0.3$, $P_n = 0.1$ and $k = 1$. The initial condition is $S(0)/N = 0.995$, $I(0)/N = 0.005$ and $R(0)/N = 0$. As $R_0 \simeq 3.6 > 1$, the endemic stationary state is reached in the permanent regime; that is, $\lim_{t\to\infty} I(t)/N \neq 0$.

2.3. Vaccination game

Here, vaccine is administered to the newborns, which are vaccinated only when there exist infective neighbors (in their neighborhood matrixes) and/or when the government promotes a vaccination campaign. In fact, a healthy individual does not usually take vaccine against a disease with low incidence or little disclosure (e.g. Luman et al., 2002; Reluga et al., 2006).

At each time step, the vaccination game is played by each newborn (I- and R-cells that perished at $t-1$ and were replaced by S-cells at t) and the government. The newborn strategies are labeled by V and NV, meaning vaccination and non-vaccination; the government strategies are labeled by C and NC, meaning campaign and non-campaign. Table 1 is the payoff matrix. Each element of this matrix is composed by two numbers: the first is the government payoff; the second is the newborn payoff. We assume that there are no side-effects associated with the vaccine, which confers immediate and permanent protection against the infection. Thus, the risk α of becoming sick due to the vaccine is nil. The consequences of $\alpha > 0$ were already explored by other authors (e.g. Bauch and Earn, 2004; d'Onofrio et al., 2007; Reluga et al., 2006). They concluded that oscillation in the infective population is a natural consequence when $\alpha > 0$.

Let γ be the cost per individual of promoting a vaccination campaign and δ the cost per individual of treating a sick person. Typically, $\delta \approx 10\gamma$ (e.g. Bocchini et al., 2008; WHO, 2005). Let the vaccine uptake level during a vaccination campaign be β. Typically, $\beta \approx 90\,\%$ (e.g. Coudeville et al., 2005; LeBaron et al., 1997). Let σ be the average probability of a susceptible individual being infected. The value of σ is determined in a PCA simulation by $\Delta I(t)_{S\to I}/(N\Delta t)$; that is, σ is the number of infections occurring at t divided by the total number of individuals.

In the game presented in Table 1, if the government plays C and the newborn plays V, then the first receives $-\gamma$ and the second receives 0, since there is no risk of becoming sick after vaccination. If the government plays NC and the newborn plays V, then the first gets 0, because there are no campaign expenses; and the second also gets 0. When the government chooses C and the individual chooses NV, then the newborn payoff is $-P_iP_d$, where P_i is the probability of getting sick and P_d is the probability of dying because of the disease. When the government chooses NC and the newborn chooses NV, then the government payoff is $-\delta\rho$, where ρ is taken as the highest value between P_i and σ (if there is no infective neighbor, then $P_i = 0$ and $\rho = \sigma$; if there is infective neighbor, then $\rho = P_i$ if $P_i > \sigma$ or $\rho = \sigma$ if $P_i < \sigma$). When $P_i = 0$ and in the absence of a vaccination campaign, it is assumed that a newborn plays NV due to lack of motivation. During a campaign, there is a probability β of a newborn playing V. Vaccination corresponds to the state transition $S \to R$.

Table 1
Matrix payoff of the vaccination game.

Government	Newborn	
	V	NV
C	$-\gamma$, 0	$-\gamma$, $-P_iP_d$
NC	0, 0	$-\delta\rho$, $-P_iP_d$

Let x_j be a binary variable, where $x_j = 1$ if the government payoff for launching a vaccination campaign for the newborn j is higher than its payoff for not launching such a campaign; and $x_j = 0$ otherwise. Notice that if $P_i \neq 0$, then $x_j = 0$ if $\gamma > \delta\rho$ and $x_j = 1$ if $\gamma < \delta\rho$. The average value of x_j is denoted by x, and this number is considered the probability of accomplishing a campaign in the next time step.

If S-, I- and R-cells are homogeneously distributed over the lattice, then the PCA is equivalent to the following set of ordinary differential equations:

$$\begin{aligned}\frac{dS(t)}{dt} &= -aS(t)I(t) + c\,[1-p(t)]\,I(t) + e\,[1-p(t)]\,R(t)\\ \frac{dI(t)}{dt} &= aS(t)I(t) - bI(t) - cI(t)\\ \frac{dR(t)}{dt} &= [b + p(t)c]\,I(t) - e\,[1-p(t)]\,R(t)\end{aligned} \tag{2}$$

where a is the infection rate constant; b is the recovering rate constant; c is the death rate constant related to the disease; e is the death rate constant related to other causes; and $p(t)$ is the percentage of vaccinated newborns. The case $p = 0$ was already analyzed by Schimit and Monteiro (2009). The total number of individuals $N = S(t) + I(t) + R(t)$ is constant, because $dS/dt + dI/dt + dR/dt = 0$. By taking $p(t) = p = constant$, stability analysis of Eqs. (2) shows that $R_0 = aN/(b + c)$. Therefore, this parameter can be estimated by Eq. (1) (Schimit and Monteiro, 2009) during a vaccination campaign (when $p(t) \simeq \beta$) or between two consecutive campaigns (when $p(t) \simeq 0$). Notice that R_0 does not depend on p.

3. Simulation results

In the figures presented in this section, the values of the epidemiological parameters used in the numerical simulations are $P_c = 60\,\%$, $P_d = 30\,\%$, $P_n = 10\,\%$ and $k = 1$; the initial condition is $S(0)/N = 99.5\,\%$, $I(0)/N = 0.5\,\%$ and $R(0)/N = 0$; the lattice size is $n = 200$ (thus, $N = 40000$); the values of topological parameters are $m = 4$ and $r = 8$, and $m = 14$ and $r = 6$ (other simulations were performed with different values, but the results were not qualitatively distinct from the ones reported here). The values of the game parameters are $\gamma = 1$, $\delta = 10$ and $\beta = 90\%$.

Figs. 2 and 3 show oscillations in the numbers of S-, I- and R-cells and in the accomplishment of vaccination campaigns. In these figures, the occurrence of campaigns is represented by gray areas; the absence, by white areas. In Fig. 2, $m = 4$ and $r = 8$, and $R_0 \simeq 3.0$; in Fig. 3, $m = 14$ and $r = 6$, and $R_0 \simeq 7.2$. An important result is: the higher the value of R_0, the higher the frequency of the observed oscillations.

These dynamical behaviors can be explained by the following reasoning. Suppose that initially there are just a few cases of infective individuals in the population (in fact, the initial condition is $I(0)/N = 0.5\,\%$). In this situation, most newborns present $P_i = 0$ and the government does not perform a vaccination campaign, because treating the rare patients is cheaper than making such a campaign. Due to this weak control, the disease spreads and an epidemic arises. Now, the government gets a higher payoff by launching

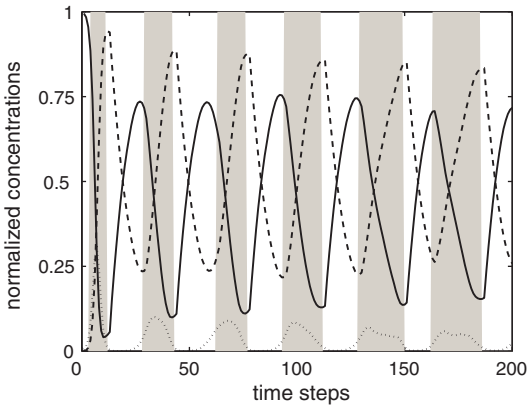

Fig. 2. Time evolutions of $S(t)/N$ (solid line), $I(t)/N$ (dotted line) and $R(t)/N$ (dashed line) in the vaccination game obtained from a PCA simulation. The parameter values are $n = 200$, $m = 4$, $r = 8$, $P_c = 0.6$, $P_d = 0.3$, $P_n = 0.1$, $k = 1$, $\gamma = 1$, $\delta = 10$ and $\beta = 0.9$. The initial condition is $S(0)/N = 0.995$, $I(0)/N = 0.005$ and $R(0)/N = 0$. The gray areas correspond to the time steps where a vaccination campaign is accomplished. Absence of campaign is represented by white areas.

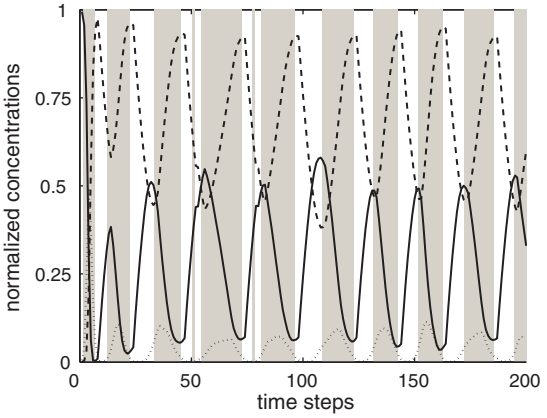

Fig. 3. Time evolutions of $S(t)/N$ (solid line), $I(t)/N$ (dotted line) and $R(t)/N$ (dashed line). The parameter values are $m = 14$ and $r = 6$ (the other values are equal to those used in Fig. 2). Gray areas correspond to vaccination campaign; white areas to no campaign.

a campaign, which causes a decrease in the number of infective individuals, returning to the initial situation.

A similar analysis can be performed from the point of view of game theory. In Table 1, there is a unique Nash equilibrium, given by the strategies (*NC*,*V*). These choices represent a Nash equilibrium (e.g. Webb, 2007) because neither newborns nor government can increase their payoffs by unilaterally adopting another strategy. Such a solution would be really found if people were conscious about the importance of vaccinating, even in the absence of campaigns. However, this ideal scenario is not commonly observed (e.g. Seale et al., 2010). For a contagious disease with low incidence, the strategies usually found are (*NC*, *NV*). These attitudes favor disease propagation, leading to $P_i \approx 1$. Then, $x \approx 1$ and the actions shift to (*C*,*V*). However, when $\gamma > \delta\rho$, the government stops the campaign, playing *NC*. Without this stimulus and because the perceived risk of getting sick is low, the individual prefers to play *NV*. Simulations show that this cycle is quasi-periodically repeated.

4. Conclusions

Mass vaccination campaigns aim to reduce morbidity and mortality caused by contagious infections. Obviously, these public health interventions are economically and logistically limited and some of them are only implemented in response to specific outbreaks (e.g. Goodson et al., 2009; Krause et al., 2002). Here, we concluded that a strategy based on cost-benefit analysis of voluntary vaccination versus treatment can temporarily control an epidemic but it can not eradicate the corresponding disease. In the PCA simulations, successive epidemics are observed, which happen because individuals tend to not take vaccine when the risk of contracting the pathogen in their social contacts is low and/or the disease rarely appears in the mass media. Observe that quasi-periodic vaccination is a consequence of our epidemiological model. Observe also that outbreaks arise even for vaccines conferring complete and sustained immunity. Of course, outbreaks could also be caused by suboptimal efficacy of vaccines (e.g. Franco et al., 2004).

The parameters values used in these simulations do not correspond to a particular disease affecting a specific city or country; however, they allow a qualitative understanding of the model. We found that the higher the value of R_0, the higher the frequencies of campaigns and recurrent epidemics. Therefore, chickenpox ($R_0 \simeq 8.7$) requires more efforts to be controlled than rubella ($R_0 \simeq 6.3$) (according to the estimations of R_0 performed by Anderson and May (1982)). In our study, the targets of the vaccination programs are the newborns and not the whole population to simulate the fight against childhood diseases. In fact, for a limited supply of vaccines, targeted immunization usually yields better results than random immunization (e.g. Patel et al., 2005).

Outbreaks can arise after interrupting vaccination campaigns (e.g. Ajelli and Merler, 2009). Therefore, additional or different strategies seem to be necessary if the goal is eradication. For instance, in order to increase the vaccine coverage, vaccination could be made compulsory instead of voluntary, but there are ethical problems with this approach (e.g. Salmon et al., 2006). We suggest that voluntary vaccination could be spontaneously stimulated by the mass media (that is, without cost to the government), emphasizing the relevance of immunization policies. Notice that the average cost per dose under routine immunization can be about three times less expensive than under vaccination campaign (e.g. Shepard et al., 1989). Therefore, education and motivation seem to be the ways for achieving eradication.

Acknowledgement

L.H.A.M. is partially supported by CNPq.

References

Ahmed, E., Agiza, H.N., Hassan, S.Z., 1998. On modeling hepatitis B transmission using cellular automata. J. Stat. Phys. 92, 707–712.
Ajelli, M., Merler, S., 2009. An individual-based model of hepatitis A transmission. J. Theor. Biol. 259, 478–488.
Anderson, R.M., May, R.M., 1982. Directly transmitted infectious diseases: control by vaccination. Science 215, 1053–1060.
Barrat, A., Barthélemy, M., Vespignani, A., 2008. Dynamical Processes on Complex Networks. Cambridge University Press, Cambridge.
Bauch, C.T., Earn, D.J.D., 2004. Vaccination and the theory of games. Proc. Natl. Acad. Sci. U.S.A. 101, 13391–13394.
Behbehani, A.M., 1983. The smallpox story: life and death of an old disease. Microbiol. Rev. 47, 455–509.
Ben-Zion, Y., Cohen, Y., Shnerb, M.N., 2010. Modeling epidemics dynamics on heterogenous networks. J. Theor. Biol. 264, 197–204.
Bocchini, J.A., Bernstein, H.H., Bradley, J.S., Brady, M.T., Byington, C.L., Dennehy, P.H., Frenck, R.W., Glode, M.P., Keyserling, H.L., Kimberlin, D.W., Long, S.S., Rubin, L.G., 2008. Prevention of influenza: recommendations for influenza immunization of children, 2007–2008. Pediatrics 121, E1016–E1031.
Cohen, R., Havlin, S., ben-Avraham, D., 2003. Efficient immunization strategies for computer networks and populations. Phys. Rev. Lett. 91, 247901.
Coudeville, L., Brunot, A., Szucs, T.D., Dervaux, B., 2005. The economic value of childhood varicella vaccination in France and Germany. Value Health 8, 209–222.
d'Onofrio, A., Manfredi, P., Salinelli, E., 2007. Bifurcation threshold in an SIR model with information-dependent vaccination. Math. Model. Nat. Phenom. 2, 26–43.
Doran, J.R., Laffan, S.W., 2005. Simulating the spatial dynamics of foot and mouth disease outbreaks in feral pigs and livestock in Queensland, Australia, using

a susceptible-infected-recovered cellular automata model. Prev. Vet. Med. 70, 133–152.
Franco, E., Giambi, C., Ialacci, R., Maurici, M., 2004. Pertussis vaccination for adolescents and adults. Expert Opin. Biol. Ther. 4, 1669–1676.
Goodson, J.L., Wiesen, E., Perry, R.T., Mach, O., Kitambi, M., Kibona, M., Luman, E.T., Cairns, K.L., 2009. Impact of measles outbreak response vaccination campaign in Dar es Salaam, Tanzania. Vaccine 27, 5870–5874.
Guckenheimer, J., Holmes, P., 1983. Nonlinear Oscillations, Dynamical Systems and Bifurcations of Vector Fields. Springer, New York.
Holme, P., 2004. Efficient local strategies for vaccination and network attack. Europhys. Lett. 68, 908–914.
Kermack, W.O., McKendrick, A.G., 1927. Contribution to the mathematical theory of epidemics. Proc. Roy. Soc. Lond. A 115, 700–721.
Krause, G., Blackmore, C., Wiersma, S., Lesneski, C., Gauch, L., Hopkins, R.S., 2002. Mass vaccination campaign following community outbreak of meningococcal disease. Emerg. Infect. Dis. 8, 1398–1403.
LeBaron, C.W., Chaney, M., Baughman, A.L., Dini, E.F., Maes, E., Dietz, V., Bernier, R., 1997. Impact of measurement and feedback on vaccination coverage in public clinics, 1988–1994. JAMA 277, 631–635.
Liu, X., Takeuchi, Y., Iwami, S., 2008. SVIR epidemic models with vaccination strategies. J. Theor. Biol. 253, 1–11.
Liu, Z., Lai, Y.C., Ye, N., 2003. Propagation and immunization of infection on general networks with both homogeneous and heterogeneous components. Phys. Rev. E 67, 031911.
Luman, E.T., McCauley, M.M., Stokley, S., Chu, S.Y., Pickering, L.K., 2002. Timeliness of childhood immunizations. Pediatrics 110, 935–939.
Monteiro, L.H.A., Chimara, H.D.B., Berlinck, J.G.C., 2006a. Big cities: shelters for contagious diseases. Ecol. Model. 197, 258–262.
Monteiro, L.H.A., Paiva, D.C., Piqueira, J.R.C., 2006b. Spreading depression in mainly locally connected cellular automaton. J. Biol. Syst. 14, 617–629.
Moreno, Y., Pastor-Satorras, R., Vespignani, A., 2002. Epidemic outbreaks in complex heterogeneous networks. Eur. Phys. J. B 26, 521–529.
Newman, M.E.J., 2002. Spread of epidemic disease on networks. Phys. Rev. E 66, 016128.
Patel, R., Longini, I.M., Halloran, M.E., 2005. Finding optimal vaccination strategies for pandemic influenza using genetic algorithms. J. Theor. Biol. 234, 201–212.
Piccardi, C., Lazzaris, S., 1998. Vaccination policies for chaos reduction in childhood epidemics. IEEE Trans. Biomed. Eng. 45, 591–595.
Reluga, T.C., Bauch, C.T., Galvani, A.P., 2006. Evolving public perceptions and stability in vaccine uptake. Math. Biosci. 204, 185–198.
Risi, J.B., 1984. The control of poliomyelitis in Brazil. Rev. Infect. Dis. 6 (suppl. 2), S400–S403.
Salmon, D.A., Teret, S.P., MacIntyre, C.R., Salisbury, D., Burgess, M.A., Halsey, N.A., 2006. Compulsory vaccination and conscientious or philosophical exemptions: past, present, and future. Lancet 367, 436–442.
Schimit, P.H.T., Monteiro, L.H.A., 2009. On the basic reproduction number and the topological properties of the contact network: an epidemiological study in mainly locally connected cellular automata. Ecol. Model. 220, 1034–1042.
Schimit, P.H.T., Monteiro, L.H.A., 2010. Who should wear mask against airbone infections? Altering the contact network for controlling the spread of contagious diseases. Ecol. Model. 221, 1329–1332.
Seale, H., Heywood, A.E., McLaws, M.L., Ward, K.F., Lowbridge, C.P., Van, D., MacIntyre, C.R., 2010. Why do I need it? I am not at risk! Public perceptions towards the pandemic (H1N1) 2009 vaccine. BMC Infect. Dis. 10, 99.
Shepard, D.S., Robertson, R.L., Cameron, C.S.M., Saturno, P., Pollack, M., Manceau, J., Martinez, P., Meissner, P., Perrone, J., 1989. Cost-effectiveness of routine and campaign vaccination strategies in Ecuador. Bull. World Health Organ. 67, 649–662.
Shulgin, B., Stone, L., Agur, Z., 1998. Pulse vaccination strategy in the SIR epidemic model. Bull. Math. Biol. 60, 1123–1148.
Sirakoulis, G., Karafyllidis, Ch., Thanailakis, I.A., 2000. A cellular automaton model for the effects of population movement and vaccination on epidemic propagation. Ecol. Model. 133, 209–223.
Slimi, R., El Yacoubi, S., Dumonteil, E., Gourbiere, S., 2009. A cellular automata model for Chagas disease. Appl. Math. Model. 33, 1072–1085.
Sun, C., Hsieh, Y.H., 2010. Global analysis of an SEIR model with varying population size and vaccination. Appl. Math. Model. 34, 2685–2697.
Sun, G.Q., Liu, Q.X., Jin, Z., Chakraborty, A., Li, B.L., 2010. Influence of infection rate and migration on extinction of disease in spatial epidemics. J. Theor. Biol. 264, 95–103.
Watts, D.J., Strogatz, S.H., 1998. Collective dynamics of small-world networks. Nature 393, 440–442.
Webb, J.N., 2007. Game Theory: Decisions, Interaction and Evolution. Springer, London.
White, S.H., del Rey, A.M., Sanchez, G.R., 2007. Modeling epidemics using cellular automata. Appl. Math. Comput. 186, 193–202.
WHO, 2005. Immunization against diseases of public health importance. World Health Organization. http://www.who.int/mediacentre/factsheets/fs288/en/index.html.
Wolfram, S., 1994. Cellular Automata and Complexity: Collected Papers. Westview Press, New York.
Yakowitz, S., Gani, J., Hayes, R., 1990. Cellular automaton modeling of epidemics. Appl. Math. Comp. 40, 41–54.
Yip, P.S.F., Watson, R., Chen, Q., 2007. Estimation of vaccine efficacy and the vaccination threshold. Stat. Med. 26, 4475–4488.