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Short Communication

The impact of imported cases on the persistence of contagious diseases

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ABSTRACT

Contagious diseases can chronically persist in host populations. Here, this persistence is investigated by using an epidemiological model based on probabilistic cellular automaton (PCA). In this model, imported cases and vaccination are both considered. From a mean-field approximation written in terms of ordinary differential equations (ODE), it is shown that this model has a single stationary solution. This solution corresponds to an endemic equilibrium point, which is locally asymptotically stable. The impact of the imported cases on this attractive endemic solution is analytically estimated from the ODE system and numerically confirmed from computer simulations with the PCA model. The relevance of this study for controlling the spread of infectious diseases is discussed from a public health perspective.

1. Introduction

Why are there pathogens that persistently infect a host species? This question is particularly challenging when recovery from infection confers longlife immunity. For instance, most people who get measles recover fully and no longer contract and, consequently, spread this disease. Also, vaccination against measles, which is available for free as part of routine immunization services in about 85% of countries, is a very effective preventive measure (Dabbagh et al., 2017). Then, why is measles not eradicated, since almost the entire adult population is immune to it? Part of the answer is: several contagious diseases have persisted around the world because big cities have served as reservoirs for them (Bartlett, 1957; Monteiro et al., 2006). It is obvious that measles cannot survive in a small isolated community. In fact, re-introduction can be necessary for its persistence. A real-world example: in 2016, Brazil was declared free of measles; in 2018, a measles epidemic affected the Brazilian states of Amazonas and Roraima (Goldani, 2018; Pacheco et al., 2019). This disease was likely introduced by Venezuelan migrants and its spread was favored by a declining vaccination rate in these Brazilian states.

Usually, in theoretical works, the imported cases of an infection arrive at a constant rate (Bakare, 2016; Brauer and van den Driessche, 2001; Enatsu et al., 2012; Guo and Li, 2012; Li et al., 2006; Li and Cui, 2013; Naresh et al., 2009). Here, it is assumed that the imported cases arrive at a rate proportional to the number of susceptible individuals in a fixed-size population. This assumption can be interpreted in two different ways: (1) the inflow of infected immigrants is compensated by the outflow of susceptible residents and/or (2) residents travel healthy and come back sick. In practice, these migratory movements correspond to spontaneous infections, in the sense that the pathogen is not contracted from social contacts with infected neighbors.

Probabilistic cellular automaton (PCA) has been employed in eco-epidemiological studies (Ahmed et al., 1998; Boccara et al., 1994; Doran and Laffan, 2005; Ferreri and Venturino, 2013; Fuentes and Kuperman, 1999; Slimi et al., 2009; Zhang et al., 2018). Here, a PCA model is proposed to numerically investigate the impact of imported cases on the persistence of contagious diseases. This issue is also analytically examined from a system of ordinary differential equations (ODE) equivalent to the PCA model. The PCA and ODE approaches are presented in the next sections.

2. The model based on PCA

The host population is represented by a two-dimensional lattice $n \times n$ with periodic boundary conditions (that is, the bottom and top edges are connected and the left and right edges are connected too; thus, there are no edge effects). Each cell of this toroidal lattice corresponds to an individual, which contacts the eight surrounding neighbors. This coupling pattern is known as Moore neighborhood of unit radius (Wolfram, 1994). This is a simplistic way of representing the social contacts among the individuals. Other static structures (Newmann, 2002) or even time-varying topologies (Schimit and Monteiro, 2009) could be considered.

At each time step, each individual is in one of three health states:

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susceptible (S), infected (I), or recovered (R). The time evolution of this SIR epidemiological model is driven by six probabilistic rules of state transitions: three rules for S-individuals, two rules for I-individuals, and one rule for R-individuals. For S-individuals: at each time step, there is a probability P_v of a S-individual being vaccinated and becoming a R-individual, which is permanently immune to the disease. If this S-individual is not vaccinated, there is a probability P_s of becoming spontaneously infected, which corresponds to an imported case of the disease. If this individual still remains in the S-state, there is a probability $P_i = 1 - e^{-kv}$ of being infected due to contact with sick neighbor. In this expression for P_i, v is the number of infected neighbors and k is the parameter expressing the pathogen infectivity. Note that $P_i = 0$ if $v = 0$ and $P_i \to 1$ if $k \to \infty$. For I-individuals: at each time step, there is a probability P_c of an I-individual being cured and becoming a R-individual. If this I-individual remains sick, there is a probability P_d of dying due to the infection. For R-individuals: at each time step, there is a probability P_o of a R-individual dying due to other causes. When I or R-individuals die, S-individuals replace them. Thus, the total number of individuals $N = n^2$ is kept constant in this population in which the deaths are balanced by the births. Observe that, since age ranges are not taken into account, it is not necessary to consider the death of S-individuals, because a dead S-individual would be replaced by a newborn S-individual with the same features.

The proposed model can be summarized as:

$$S \xrightarrow{P_v} R \tag{1}$$

$$S \xrightarrow{P_s} I \tag{2}$$

$$S + vI \xrightarrow{P_i} I + vI \tag{3}$$

$$I \xrightarrow{P_c} R \tag{4}$$

$$I \xrightarrow{P_d} S \tag{5}$$

$$R \xrightarrow{P_o} S \tag{6}$$

Throughout the simulation, the health states of all individuals are simultaneously updated in the end of each time step. Similar epidemiological models based on PCA can be found in the literature (Chaves and Monteiro, 2017; Monteiro et al., 2006; Schimit and Monteiro, 2009; 2012; Silva and Monteiro, 2014). Here, it is assumed a time-constant vaccination probability. Certainly, other vaccination schemes could be considered. Such schemes could be based, for instance, in the evolutionary game theory, which has been employed to represent the individual's attitude toward vaccination (Ariful Kabir and Tanimoto, 2019; Bauch and Earn, 2004; Kuga et al., 2019; Schimit and Monteiro, 2011).

3. The model based on ODE

Assume that S, I, and R-individuals are homogeneously distributed over the geographical region represented by the toroidal lattice. By considering the homogeneous mixing assumption (Turnes and Monteiro, 2014), a mean-field approximation of the PCA model can be described by the following set of ODE:

$$\frac{dS(t)}{dt} = -aS(t)I(t) - eS(t) + hR(t) + cI(t) - vS(t) \tag{7}$$

$$\frac{dI(t)}{dt} = aS(t)I(t) + eS(t) - bI(t) - cI(t) \tag{8}$$

$$\frac{dR(t)}{dt} = bI(t) - hR(t) + vS(t) \tag{9}$$

in which $S(t)$, $I(t)$, and $R(t)$ are the numbers of susceptible, infected, and recovered individuals at the instant t, respectively. The six parameters a, b, c, e, h, and v are non-negative numbers: a is the rate constant related to the infection transmission to S-individuals due to contact with I-individuals, b is the recovery rate constant of I-individuals, c is the rate constant of death (supposed to be caused by the disease) of I-individuals, h is the rate constant of death (due to other causes) of R-individuals, v is the vaccination rate constant of S-individuals, and e is the rate constant related to the imported cases.

Observe that the deaths of I and R-individuals are balanced by the births of S-individuals; hence, $dS(t)/dt + dI(t)/dt + dR(t)/dt = 0$. As a consequence, $S(t) + I(t) + R(t) = N$; that is, the total number of individuals N remains fixed. Since $R(t) = N - S(t) - I(t)$, this SIR model based on ODE can be rewritten as the following second-order dynamical system:

$$\frac{dS}{dt} = F(S, I) = -aS(I + \epsilon) + h(N - S - I) + cI - vS \tag{10}$$

$$\frac{dI}{dt} = G(S, I) = aS(I + \epsilon) - bI - cI \tag{11}$$

with $\epsilon = e/a$. In the next section, this model is analyzed from a dynamical systems theory perspective (Guckenheimer and Holmes, 2002).

4. Analytical results

A stationary solution of the system composed by Eqs. (10) and (11) corresponds to an equilibrium point (S^*, I^*) in the state space $S \times I$. The constants S^* and I^* are obtained from $dS/dt = 0$ and $dI/dt = 0$; that is, $F(S^*, I^*) = 0$ and $G(S^*, I^*) = 0$. This model has a single positive equilibrium point given by:

$$S^* = \frac{(b + c)I^*}{a(I^* + \epsilon)} \tag{12}$$

$$I^* = \frac{-\beta + |\beta|\sqrt{1 + (4\alpha\gamma/\beta^2)}}{2\alpha} \tag{13}$$

with $\alpha = a(b + h) > 0$, $\beta = (b + c)(h + v) + a\epsilon(b + h) - ahN$, and $\gamma = a\epsilon hN > 0$. This equilibrium point corresponds to an endemic stationary solution, because $I^* > 0$. Obviously, $R^* = N - S^* - I^*$.

The local stability of this equilibrium point can be determined from the eigenvalues of the Jacobian matrix $\mathbf{J}$ derived from the second-order system of ODE linearized around such a point. According to the Hartman–Grobman theorem (Guckenheimer and Holmes, 2002), this steady state is locally asymptotically stable if both eigenvalues of $\mathbf{J}$ have negative real parts. For this second-order system, the eigenvalues λ, obtained from $\det(\mathbf{J} - \lambda\mathbf{I}) = \mathbf{0}$ ($\mathbf{I}$ is the identity matrix), are the roots of the polynomial $\lambda^2 - T\lambda + \Delta = 0$, in which T and Δ are, respectively, the trace and the determinant of $\mathbf{J}$ computed at (S^*, I^*). Both eigenvalues have negative real parts if $T < 0$ and $\Delta > 0$. In this case:

$$T = \left[\frac{\partial F}{\partial S} + \frac{\partial G}{\partial I}\right]_{(S,I)=(S^*,I^*)} = -a(I^* + \epsilon) - (h + v) - [(b + c) - aS^*] \tag{14}$$

$$\Delta = \left[\frac{\partial F}{\partial S}\frac{\partial G}{\partial I} - \frac{\partial F}{\partial I}\frac{\partial G}{\partial S}\right]_{(S,I)=(S^*,I^*)} = a(b + h)(I^* + \epsilon) + (h + v)[(b + c) - aS^*] \tag{15}$$

Since $aS^* = (b + c)I^*/(I^* + \epsilon) < (b + c)$, then $T < 0$ and $\Delta > 0$; consequently, the endemic steady-state is locally asymptotically stable.

For $\epsilon = 0$ (no imported case), there are two non-negative equilibrium points:

$$S_1^* = \frac{hN}{h + v} \tag{16}$$

$$I_1^* = 0 \tag{17}$$

and:

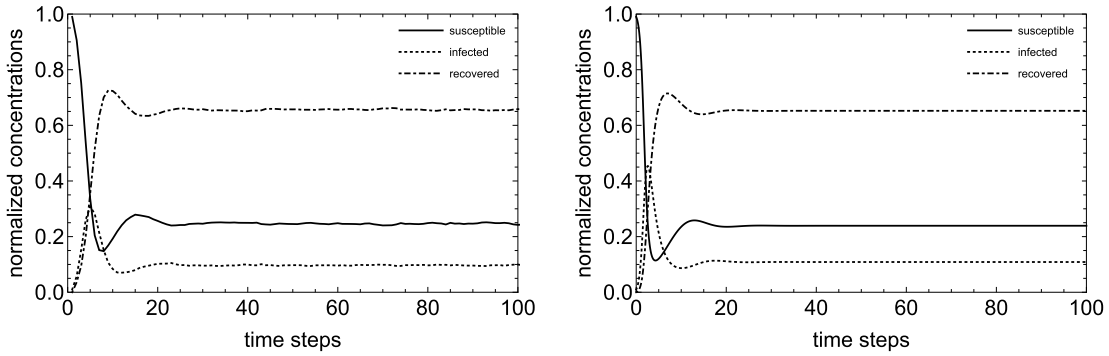

Fig. 1. Time evolutions of $S(t)/N$ (solid line), $I(t)/N$ (dotted line), and $R(t)/N$ (dash-dotted line) from $S(0)/N = 99\%$, $I(0)/N = 1\%$, and $R(0)/N = 0\%$ obtained from PCA (left) and ODE (right). In the PCA, $n = 200$, $P_v = 3\%$, $P_s = 1\%$, $P_c = 60\%$, $P_d = 30\%$, $P_o = 10\%$, and $k = 1$. In the ODE, $v = 0.03$, $a\epsilon = 0.0097$, $b = 0.6$, $c = 0.12$, $h = 0.1$, and $aN \simeq 2.83$. Note that, as time passes by, $S(t)/N \to S^*/N = 0.25$ and $I(t)/N \to I^*/N = 0.10$ (consequently, $R(t)/N \to R^*/N = 0.65$) in both approaches.

$$S_2^* = \frac{N}{R_0} \tag{18}$$

$$I_2^* = \frac{(h+v)N(r_0-1)}{(b+h)R_0} \tag{19}$$

with $R_0 \equiv aN/(b+c)$ and $r_0 \equiv hR_0/(h+v)$.

For the disease-free equilibrium point (S_1^*, I_1^*), $T = -(b+c)[1 - r_0 + (h+v)/(b+c)]$ and $\Delta = (h+v)(1-r_0)$; therefore, it is locally asymptotically stable for $r_0 < 1$ and unstable for $r_0 > 1$. For the endemic equilibrium point (S_2^*, I_2^*), $T = -(aI_2^* + h + v)$ and $\Delta = a(b+h)I_2^*$; hence, it is locally asymptotically stable for $r_0 > 1$ and unstable for $r_0 < 1$. As the equilibrium points (S_1^*, I_1^*) and (S_2^*, I_2^*) exchange their stabilities at $r_0 = 1$, a transcritical bifurcation (Guckenheimer and Holmes, 2002) occurs for this particular combination of parameter values.

The case $\epsilon = 0$, $v = 0$ had been already analyzed (Monteiro et al., 2006; Schimit and Monteiro, 2009; 2012). In this case, $r_0 = R_0$ and the transcritical bifurcation involving the disease-free and endemic steady-states occurs at $R_0 = 1$. Note that R_0 can be considered as the basic reproduction number (for $\epsilon = 0$, $v = 0$) and r_0 as the effective reproduction number (for $\epsilon = 0$, $v > 0$). The basic reproduction number is a bifurcation parameter, which gives an estimate of the amount of secondary infections per sick individual introduced into a completely susceptible population. The effective reproduction number is also a bifurcation parameter, which gives an estimate of the amount of secondary infections per sick individual introduced into a population with immune members (Anderson and May, 1992; Delamater et al., 2019; Keeling and Rohani, 2008; Schimit and Monteiro, 2012). For $\epsilon > 0$, $v > 0$ there is no bifurcation parameter.

Assume that sick people avoid traveling; that is, assume that imported cases are rare. In this scenario, the impact of imported cases, denoted by ΔI^*, can be approximated by:

$$\Delta I^* \equiv I^*|_{\epsilon \to 0} - I_2^*|_{\epsilon=0} \simeq I_2^*|_{\epsilon \to 0} - I_2^*|_{\epsilon=0} + \frac{\epsilon}{r_0 - 1} \tag{20}$$

ΔI^* expresses the difference between the asymptotic values of I-individuals for $\epsilon \to 0$ and for $\epsilon = 0$. In the derivation of this formula, the approximation $\sqrt{1+q} \simeq 1 + (q/2)$ for $q = 4\alpha\gamma/\beta^2 \to 0$ was taken into account in Eq. (13) (observe that $\gamma \to 0$ for $\epsilon \to 0$). Recall that I_2^* is given by Eq. (19). In Eq. (20), $I_2^*|_{\epsilon \to 0}$ is the value of I_2^* computed with the value of a for $\epsilon \to 0$ and $I_2^*|_{\epsilon=0}$ is the value of I_2^* computed with the value of a for $\epsilon = 0$. Eq. (20) is the central result of this work.

Therefore, the impact of imported cases on the endemic steady-state increases for $r_0 \to 1$ in the limit $\epsilon \to 0$. This analytical result is numerically confirmed from computational simulations presented in the next section.

5. Numerical simulations

The analytical results derived in the previous section can be confirmed from PCA simulations if the parameter values of the ODE system are consistently chosen. In fact, the PCA dynamics can be approximately reproduced by the ODE system by considering that $b = P_c$, $c = (1 - P_c)P_d$, $h = P_o$, $v = P_v$, and $a\epsilon = (1 - P_v)P_s$. These expressions show how to obtain five rate constants of the ODE system from the probabilities of state transitions of the PCA model. The rate constant a, which is the only parameter whose value is influenced by the contact network, can be estimated from a PCA simulation from:

$$a \simeq \frac{\Delta I/\Delta t}{S^* I^*} \tag{21}$$

in which $\Delta I/\Delta t$ is the increase per time step of infected individuals due to the contagion of S-individuals by I-neighbors, and S^* and I^* are the asymptotic values of $S(t)$ and $I(t)$. Average values of a are calculated by taking into account the last 50 time steps of PCA simulations with 300 time steps (that is, when the asymptotic solution was already reached). Since the rule of spontaneous infection is applied before the rule of contagion by infected neighbor, a is affected by P_s. The high the value of P_s, the high the value of a, because the number of infected individuals (consequently, the number of infected neighbors) increases with P_s. The value of a for $P_s = 0$ (no imported case) is denoted by a_0.

Fig. 1 shows the time evolutions of $S(t)/N$ (solid line), $I(t)/N$ (dotted line), and $R(t)/N$ (dash-dotted line) in the PCA lattice for $n = 200$ (thus, $N = 40000$), $P_v = 3\%$, $P_s = 1\%$, $P_c = 60\%$, $P_d = 30\%$, $P_o = 10\%$, and $k = 1$. The initial condition is $S(0)/N = 99\%$, $I(0)/N = 1\%$, and $R(0)/N = 0\%$. This figure also shows the numerical solution of the ODE system from the same initial condition by taking $v = 0.03$, $a\epsilon = (1 - 0.03) \times 0.01 = 0.0097$, $b = 0.6$, $c = (1 - 0.6) \times 0.3 = 0.12$, $h = 0.1$, and $aN \simeq 2.83$ (which was obtained from the PCA simulation by using Eq. (21)). The ODE system was numerically solved by employing the 4th-order Runge–Kutta integration method with integration time step of 0.01. Note the good agreement between both approaches. For instance, observe that S^* and I^* calculated from Eqs. (12) and (13) are equal to 0.25 and 0.10, respectively, which are the same values found in the permanent regime of the PCA simulation shown in Fig. 1.

Table 1 presents the results of simulations with the PCA model for $P_s = 0.1\%$ and $k = 1.0,\ 0.7,\ 0.5,\ 0.4,\ 0.3$ (the other parameter values are the same as used in Fig. 1). This value of P_s is adequate to illustrate the validity of Eq. (20); that is, it corresponds to $\epsilon \to 0$. Note that the lower the value of k, the lower the values of a, a_0, and r_0. In this table, the values of $\Delta I^*/N$ found in PCA simulations and the corresponding values estimated from Eq. (20) are shown. Observe that Eq. (20) gives a good approximation of the impact of imported cases on the infection prevalence. Also, as theoretically predicted, $\Delta I^*/N$ increases by decreasing r_0: for $r_0 = 2.89$ ($k = 1$), the impact is practically null; for $r_0 = 1.32$ ($k = 0.3$), the impact is about 2%.

Table 1
Values of $\Delta I^*/N \equiv (I^*|_{\epsilon\to 0} - I_2^*|_{\epsilon=0})/N$ found in PCA simulations and estimated from Eq. (20). In PCA simulations, $n = 200$, $P_v = 3\%$, $P_s = 0.1\%$, $P_c = 60\%$, $P_d = 30\%$, $P_o = 10\%$, and $k = 1.0, 0.7, 0.5, 0.4, 0.3$. The values of a and a_0 were determined from PCA simulations (recall that a_0 is the value of a for $P_s = 0\%$ calculated from Eq. (21)). The parameter values of the ODE system were obtained as explained in the main text.

k	a	a_0	r_0	$\Delta I^*/N$ (PCA)	$\Delta I^*/N$ (ODE)
1.0	2.71	2.70	2.89	0 ± 0.002	0.0004
0.7	2.23	2.20	2.38	0.002 ± 0.002	0.001
0.5	1.83	1.79	1.96	0.003 ± 0.002	0.002
0.4	1.54	1.47	1.65	0.004 ± 0.002	0.005
0.3	1.24	1.08	1.32	0.017 ± 0.002	0.018

6. Discussion and conclusion

As in other works taking into account imported cases of an infectious disease (Brauer and van den Driessche, 2001; Enatsu et al., 2012; Guo and Li, 2012; Li et al., 2006; Naresh et al., 2009), the proposed model admits only one biologically feasible steady-state, which is asymptotically stable and endemic. This qualitative result is not affected by the topological structure of the contact network. As in these cited studies, the consideration of imported cases prevents the existence of a disease-free steady-state.

The impact of rare imported cases on the long-term prevalence can be estimated from Eq. (20), which is the main contribution of this paper. This analytical formula derived from the ODE system was confirmed from numerical simulations with the equivalent PCA model, in which the spatial dimension is considered. Observe that this impact decreases by increasing r_0. Thus, for diseases with high values of r_0, such as measles (Anderson and May, 1992; Delamater et al., 2019), imported cases are more relevant to start an outbreak than to maintain the endemic infection level. In Europe, measles has remained endemic due to an inadequate immunization rate (Lo Vecchio et al., 2019).

In short: to eliminate a contagious disease of a specific geographical region, it is necessary to impose $\epsilon = 0$ (that is, the reintroduction must be forbidden) and $r_0 < 1$ (recall that the value of r_0 decreases by increasing the vaccination rate v). It is hard to control legal and unauthorized migratory movements of infected individuals in a given region (for instance, a country); that is, it is hard to control the value of ϵ. However, by considering the Earth as a single geographical region, then certainly $\epsilon = 0$. Thus, from the public health standpoint, the implementation of optimal immunization coverage by all countries should be enough to reach $r_0 < 1$ worldwide. The idea of "think globally, act locally" is the key for eradicating infectious diseases that can be prevented by vaccination.

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