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Original research article

The co-circulation of two infectious diseases and the impact of vaccination against one of them

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ABSTRACT

An epidemiological model based on probabilistic cellular automaton is proposed to investigate the dynamics of two co-circulating infections. In the model, one of these two diseases compromises the immune response to future infections; however, there is vaccine against this immunosuppressive disease. The goal is to evaluate the impact of the vaccination coverage on the prevalence and on the cumulative deaths associated with both contagious diseases. The performed numerical simulations highlight the importance of vaccination on decreasing morbidity and mortality. The results are discussed from a public health standpoint, by taking into account outbreaks of measles and COVID-19.

1. Introduction

There are about 1400 species of infectious agents that are pathogenic to humans (Woolhouse and Gowtage-Sequeria, 2005) and it is not uncommon to find some of them simultaneously infecting a community. Hence, there are several theoretical models about co-circulating pathogens. These models are usually written in terms of ordinary differential equations. For instance, there are works about the interaction between dengue and leptospirosis (Alemneh, 2020), pneumonia and meningitis (Tilahun, 2019), HIV and hepatitis C (Moualeu et al., 2011), two strains of influenza (Zhang et al., 2013), two strains of dengue (Anggriani et al., 2019).

The co-circulation of infections can be viewed as a complex system, due to the usual large number of variables and parameters composing the corresponding epidemic model. Here, a model with 10 variables and 20 parameters is proposed to study the dynamics of two co-circulating infections. This model is based on probabilistic cellular automaton (PCA), an approach that has been used in several works on ecological and/or epidemiological systems (Ahmed et al., 1998; Boccara et al., 1994; Doran and Laffan, 2005; Ferreri and Venturino, 2013; Nagatani and Tainaka, 2018; Sfa et al., 2020; Slimi et al., 2009).

In this work, disease-1 causes immunosuppression, which impairs the immune response to a subsequent disease-2 infection; however, vaccine against disease-1 is available. The main aim here is to investigate the impact of vaccination against disease-1 on the prevalence and on the cumulative deaths related to both diseases.

This manuscript is organized as follows. In Section 2, the PCA model is introduced. In Section 3, results from computational simulations are presented. In Section 4, these results are discussed by considering measles and COVID-19 as immunity-damaging diseases.

2. The PCA epidemic model

Assume that each cell of a two-dimensional lattice $n \times n$ corresponds to an individual of the host population. The boundary condition of this lattice is taken as periodic, which means that the left and right edges are connected and the top and bottom edges are connected too; therefore, each individual is equivalent from a geographical point of view. Assume also that each individual is socially connected to the eight surrounding neighbors; that is, the interactions among the individuals occur within a Moore neighborhood of unit radius (Wolfram, 1994).

In this population, there occurs the co-circulation of two contagious diseases, called disease-1 and disease-2. Assume that, at each time step $t$, each individual is in one of the following ten health states:

- $S$: susceptible to both diseases;
- $V$: vaccinated against disease-1; susceptible to disease-2;
- $I_1$: infected by disease-1, susceptible to disease-2;
- $I_2$: infected by disease-2, susceptible to disease-1;
- $I_{12}$: infected by disease-1, recovered from disease-2;

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The state transitions are governed by 20 probabilistic rules: 5 rules governing the contagion processes; 1 rule the vaccination process; 5 the recovery processes; 9 the death processes. When infected, vaccinated, or recovered individuals die, susceptible individuals replace them. Therefore, in this population, deaths are balanced by births, so that the total number of individuals \( N = n^2 \) remains constant. Co-infections (simultaneous infection by both diseases) are here not considered. In addition, recovery and vaccination lead to a full long-lasting immunity.

The 5 state transitions related to the spreading of these diseases are:

\[
S + I_1 + I_{12} \xrightarrow{\beta_1} I_1 + I_1 + I_2 \\
S + I_2 + I_{13} + I_{12} - \gamma_1 I_2 + I_1 + I_2 + I_1 \\
R_1 + I_1 + I_{12} \xrightarrow{\gamma_2} I_{12} + I_1 + I_2 \\
R_1 + I_2 + I_{13} + I_{12} - \gamma_2 I_{12} + I_2 + I_2 + I_1 \\
V + I_1 + I_{13} + I_{12} \xrightarrow{\nu_1} I_2 + I_2 + I_1 + I_1
\]

The state transitions related to vaccination against disease-1 are:

\[
S \xrightarrow{\lambda} V
\]

The 5 state transitions related to recovery from these diseases are:

\[
I_1 \xrightarrow{\gamma_1} R_1 \\
I_2 \xrightarrow{\gamma_2} R_2 \\
I_{12} \xrightarrow{\gamma_3} R \\
I_{13} \xrightarrow{\gamma_4} R \\
I_1 \xrightarrow{\gamma_5} R
\]

The 5 state transitions related to death caused by these diseases are:

\[
I_1 \xrightarrow{\delta_1} S \\
I_2 \xrightarrow{\delta_2} S \\
I_{12} \xrightarrow{\delta_3} S \\
I_{13} \xrightarrow{\delta_4} S \\
I_1 \xrightarrow{\delta_5} S
\]

The 4 state transitions related to death caused to other reasons are:

\[
R_1 \xrightarrow{\delta_1} S \\
R_2 \xrightarrow{\delta_2} S \\
R \xrightarrow{\delta} S \\
V \xrightarrow{\delta} S
\]

For S-individuals: at each time step, there is a probability \( \lambda P_1 \) of being infected and becoming an \( I_1 \)-individual and a probability \( (1 - \lambda) P_2 \) of being infected and becoming an \( I_2 \)-individual, with \( x = (i_1 + i_2)/(i_1 + i_1 + i_2 + i_3 + i_4) \), \( P_1 = 1 - \exp[-k_1(i_1 + i_2)] \), and \( P_2 = 1 - \exp[-k_2(i_1 + i_2 + i_3)] \). In these expressions, \( i_1, i_2, i_3, i_4 \), and \( i \) are the numbers of \( I_1, I_2, I_{12}, I_{13}, I \)-individuals in the neighborhood of this \( S \)-individual, respectively. For instance, if \( i_1 = 1, i_2 = 1, i_4 = 1, i_3 = 0, \) and \( i = 0 \), then \( x = 2/3 \), which is the proportion of individuals with disease-1 in the neighborhood of this \( S \)-individual; obviously, \((1 - x) = 1/3 \) is the proportion of individuals with disease-2. Thus, the greater the number of neighbors infected by a specific disease, the greater the probability of the \( S \)-individual getting such a disease. Also, \( k_1 \) and \( k_2 \) are positive constants related to the infectiousness of disease-1 and disease-2, respectively. Note that if \( i_1 + i_2 = 0 \), then \( P_1 = 0 \); therefore, a \( S \)-individual can get disease-1 only if there are neighbors with this disease. Consistently, if \( i_1 + i_2 + i_4 = 0 \), then \( P_2 = 0 \). Note that if \( k_1(i_1 + i_2) \rightarrow \infty \), then \( P_1 \rightarrow 0 \); if \( k_2(i_1 + i_2 + i_4) \rightarrow \infty \), then \( P_2 \rightarrow 0 \). These infection rules correspond to Eqs. (1) and (2), and they are applied only if there is at least one infected individual in the neighborhood of this \( S \)-individual; that is, only if \( i_1 + i_2 + i_4 + i_3 \neq 0 \). If this \( S \)-individual is not infected either by disease-1 or by disease-2, then there is a probability \( \nu \) of being vaccinated against disease-1, as stated in Eq. (6).

For \( I_1 \)-individuals: at each time step, there is a probability \( \beta_1 \) of being cured and becoming a \( R_1 \)-individual. If this \( I_1 \)-individual remains sick, there is a probability \( \gamma_1 \) of dying due to disease-1. These processes of recovery and death are represented by Eqs. (7) and (12), respectively.

For \( I_2 \)-individuals: at each time step, there is a probability \( \beta_2 \) of being cured and becoming a \( R_2 \)-individual. If this \( I_2 \)-individual remains sick, there is a probability \( \gamma_2 \) of dying due to disease-2. These processes of recovery and death are represented by Eqs. (8) and (13), respectively.

For \( R_1 \)-individuals: at each time step, there is a probability \( P_1 = 1 - \exp[-k_1(i_1 + i_2 + i_4)] \) of being infected by disease-1 and becoming an \( I_{12} \)-individual. In this expression, \( k_3 \) is a positive constant. If this \( R_1 \)-individual was not infected, there is a probability \( \delta_1 \) of dying. These processes of infection and death are represented by Eqs. (3) and (18), respectively.

For \( R_2 \)-individuals: at each time step, there is a probability \( P_2 = 1 - \exp[-k_2(i_1 + i_2 + i_4)] \) of being infected by disease-2 and becoming an \( I_{13} \)-individual. In this expression, \( k_3 \) is a positive constant. If this \( R_2 \)-individual was not infected, there is a probability \( \delta_2 \) of dying. These processes of infection and death are represented by Eqs. (4) and (17), respectively.

For \( V \)-individuals: at each time step, there is a probability \( P_2 = 1 - \exp[-k_2(i_1 + i_2 + i_4)] \) of being infected by disease-2 and becoming an \( I_{13} \)-individual. In this expression, \( k_5 \) is a positive constant. If this \( V \)-individual was not infected, there is a probability \( \delta \) of dying. These processes of infection and death are represented by Eqs. (5) and (20), respectively.

For \( I_{12} \)-individuals: at each time step, there is a probability \( \beta_1 \) of being cured and becoming a \( R \)-individual. If this \( I_{12} \)-individual remains sick, there is a probability \( \gamma_1 \) of dying due to disease-2. These processes of recovery and death are represented by Eqs. (9) and (14), respectively.

For \( I_{23} \)-individuals: at each time step, there is a probability \( \beta_2 \) of being cured and becoming a \( R \)-individual. If this \( I_{23} \)-individual remains sick, there is a probability \( \gamma_2 \) of dying due to disease-2. These processes of recovery and death are represented by Eqs. (10) and (15), respectively.

For \( I \)-individuals: at each time step, there is a probability \( \beta \) of being cured and becoming a \( R \)-individual. If this \( I \)-individual remains sick, there is a probability \( \gamma \) of dying due to disease-2. These processes of recovery and death are represented by Eqs. (11) and (16), respectively.

For \( R \)-individuals: at each time step, there is a probability \( \delta \) of dying. This process of death is represented by Eq. (19).

Throughout a computational simulation, the health states of the \( N \) individuals are simultaneously updated in the end of each time step. Similar models based on PCA were already employed in epidemiological studies (Chaves and Monteiro, 2017; Ferraz and Monteiro, 2019; Monteiro et al., 2006; 2020a; Pereira and Schimit, 2018; Schimit and...
state transitions. Red lines are associated with contagion; blue lines with recovery; green lines with death, and cyan line with vaccination.

Fig. 1. Pictorial diagram of the proposed model. The 10 variables are inside the dark circles and the 20 parameters are near the lines representing the possible state transitions. Red lines are associated with contagion; blue lines with recovery; green lines with death, and cyan line with vaccination.

Monteiro, 2009; Silva and Monteiro, 2014). Fig. 1 shows a pictorial representation of the model proposed in this work.

There are 20 positive constants in this model: \(k_1, k_2, k_3, k_4, k_5, b_1, b_2, \beta_1, \beta_2, \beta, c_1, c_2, \gamma_1, \gamma_2, \gamma, d_1, d_2, d, \delta, \) and \(v\). It is infeasible to fully determine how each of these 20 parameters affects the dynamical behavior of 10 variables, which are the numbers of individuals in each health state. Observe that the immunosuppression caused by disease-1 corresponds to \(\beta_2 < \beta\) (the recovery probability of \(I_2\) is lower than the recovery probability of \(I\)) and \(\gamma_2 > \gamma\) (the death probability of \(I_2\) is greater than the death probability of \(I\)). By defining \(B \equiv \beta / \beta_2\) and \(G \equiv \gamma_2 / \gamma\), a compromised immunity after infection by disease-1 corresponds to \(B > 1\) and \(G > 1\). Therefore, the greater the immunosuppression, the higher the values of \(B\) and \(G\).

In this work, the influences of \(B\) and \(G\) are evaluated. Also, the minimum vaccination probability, denoted by \(\nu_m\), for eradicating disease-1 is determined in function of \(B\) and \(G\) for two lattice sizes \(n\). These results are described in the next section.

3. Numerical results

Here, 17 of the 20 parameters are kept fixed. In the simulations, \(k_1 = k_2 = k_3 = k_4 = k_5 = 1\) (thus, the infectiousness is the same for both diseases for \(S, R_1, R_2,\) and \(V\)-individuals), \(b_1 = b_2 = \beta_1 = \beta = 30\%\) (thus, the recovery rate is the same for \(I_1, I_2, I_{12},\) and \(I\)-individuals), \(d_1 = d_2 = d = \delta = 10\%\) (thus, the death rate is the same for \(R_1, R_2, R,\) and \(V\)-individuals). Also, \(n = 100\) (here, the host population is composed of 10,000 individuals) and the initial condition is \(S(0)/N = 98\%\), \(I_1(0)/N = 1\%\), and \(I_2(0)/N = 1\%\). The other parameter values are the same as those of Fig. 2. Note that, disease-1 is equivalent to disease-2; hence, the time evolutions of \(I_1(t) / N\) and \(I_2(t) / N\) are similar, and the time evolutions of \(I_{12}(t) / N\) and \(I_{21}(t) / N\) are also similar. Obviously, in this figure, \(I(t)/N = 0\). In Fig. 3, \(v = 10\% > 0\); therefore, \(I_1(t)/N\) tends to be below \(I_2(t)/N\), and \(I_{12}(t)/N\) tends to be below \(I_{21}(t)/N\), because the vaccine reduces the numbers of individuals vulnerable to disease-1.

Note that, as time passes by, these plots tend to fluctuate around average values, which are denoted by \(\langle I_1/N, I_2/N, I_{12}/N, I_{21}/N, \Gamma \rangle /N\). These constants are the normalized average amounts obtained in the last 200 time steps from a total of 400 time steps. In Fig. 2, \(I_1/N \equiv 0.098, I_2/N \equiv 0.041, I_{12}/N \equiv 0.041,\) and \(\Gamma /N = 0\); in Fig. 3, \(I_1/N \equiv 0.070, I_2/N \equiv 0.011, I_{12}/N \equiv 0.035, I_{21}/N \equiv 0.030,\) and \(\Gamma /N \equiv 0.016\). Let \(Y\) be the sum of these normalized amounts; that is, \(Y \equiv \langle I_1 + I_2 + I_{12} + I_{21} + \Gamma \rangle /N\). For \(v = 0\%\), \(Y \equiv 0.278\); for \(v = 10\%\), \(Y \equiv 0.252\); for \(v = 20\%\), \(Y \equiv 0.216\); for \(v = 30\%\), \(Y \equiv 0.176\). Therefore, vaccination reduces \(Y\). Also, in Figs. 2 and 3, \(n = 100\); however, for \(n = 200\), the same results are obtained. In fact, these normalized amounts do not depend on the lattice size \(n\).

Figs. 4 and 5 show how \(B\) and \(G\) affect the numbers of infected individuals and the numbers of deaths due to both diseases for two values of \(v\). In these figures, \(Y_1 = \langle I_1 + I_{12} \rangle /N\) and \(Y_2 = \langle I_2 + I_{21} + \Gamma \rangle /N\); that is, \(Y_1\) is the normalized number of individuals infected by disease-1 and
Y2 is the normalized number of individuals infected by disease-2, after the system reaching its endemic attractor. The variables \( M_1 \) and \( M_2 \) are the normalized numbers of deaths (the number of deaths divided by the population size \( N \)) caused by disease-1 and disease-2, respectively, during 400 time steps. Thus, \( M_1 \) and \( M_2 \) are related to mortality, and \( Y_1 \) and \( Y_2 \) to infection prevalence. These four variables are affected by the vaccination probability \( \nu \).

Figs. 4 and 5 present average values of \( M_1 \), \( M_2 \), \( Y_1 \), and \( Y_2 \) obtained in three simulations. They reveal that \( Y_1 \), \( M_1 \), and \( M_2 \) increase with \( B \) and \( G \); \( Y_2 \) decreases with \( B \) and increases with \( G \).

The values of \( M_1 \), \( M_2 \), \( Y_1 \), and \( Y_2 \) in Fig. 4, for \( \nu = 0\% \), are usually higher than the corresponding values in Fig. 5, for \( \nu = 10\% \). For instance, for \( B = 3 \) and \( G = 3 \), \( M_1 \approx 9.0 \), \( M_2 \approx 13 \), \( Y_1 \approx 0.17 \), and \( Y_2 \approx 0.15 \) for \( \nu = 0\% \); and \( M_1 \approx 0.8 \), \( M_2 \approx 12 \), \( Y_1 \approx 0.14 \), and \( Y_2 \approx 0.15 \) for \( \nu = 10\% \).

Let respectively \( y(\nu) \equiv Y(0) - Y(\nu) \) and \( m(\nu) \equiv M(0) - M(\nu) \) be reductions due to vaccination \( \nu > 0 \) in the percentage of sick people and in the normalized cumulative deaths, as compared to the case \( \nu = 0 \). Recall that the variables \( Y = Y_1 + Y_2 \) and \( M = M_1 + M_2 \) vary with \( \nu \). From the numbers presented in the paragraph above, a vaccination probability of 10% against disease-1 saved \( mN = (9 + 13) - (6.8 + 12) \times N = 32000 \).
lives of sick people in 400 time steps in a population of $N = 10000$ individuals, which corresponds to 80 lives (0.8% of the whole population) saved per time step. Also, the sick subpopulations were reduced in individuals, which corresponds to 80 lives (0% people and in cumulative deaths increase with Rodrigues and Plotkin, 2020). In the proposed model, this vaccination also the mortality to subsequent non-measles infections (Mina, 2017; without reducing the pre-acquired immunity to other pathogens. an increased incidence of secondary infections (Mina et al., 2019; Pet a significant and prolonged immunosuppression, which can last for infection; however, the post-recovery period is usually characterized by psychological distress (Mahalakshmi et al., 2021), can also suppress host

Table 1 shows the values of $y$ and $m$ for $B = 3$ and $G = 3$ in lattices with $n = 100$ and $n = 200$. Note that $y$ and $m$ increase with $\nu$ and are not significantly affected by $n$. These results are valid for $\nu < \nu_m$. Table 2 presents the minimum vaccination probability $\nu_m$, for which disease-1 disappears in 5 consecutive simulations within the time window of 400 time steps, for $n = 100$ and $n = 200$. These minimum values were determined for $B \cdot G = \{(1, 1); (1, 3); (3, 1); (3, 3)\}$, which are the corners of the surfaces shown in Figs. 4 and 5. Note that, the higher values of $B$ and $G$, the higher the value of $\nu_m$. This qualitative result is also obtained, for instance, for $d_1 = d_2 = d = 5\%$ (instead of 10%). In this case, for $n = 100$, $\nu_m = 11\%$ (instead of 33%) for $B = 1$ and $\nu_m = 14\%$ (instead of 43%) for $B = G = 3$. Reducing $d_1$, $d_2$, $d$, and $\delta$ means increasing the lifetime of immune individuals, which decreases the vaccination effort required to eradication because such reductions contribute to foster a herd immunity. Hence, $\nu_m$ decreases by reducing $d_1$, $d_2$, $d$, and $\delta$.

The simulations reported here were performed with fictitious parameter values. However, realistic values for the proposed model can be obtained from real-world data by employing methods of parameter identification (see, for instance, Monteiro et al., 2020b).

4. Discussion and conclusion

Recovery from measles confers long-life immunity to this viral infection; however, the post-recovery period is usually characterized by a significant and prolonged immunosuppression, which can last for years (Mina et al., 2019; Petrova et al., 2019). As a consequence, there is an increased incidence of secondary infections (Mina et al., 2019; Petrova et al., 2019). Vaccine against measles elicits long-lasting immunity without reducing the pre-acquired immunity to other pathogens. Therefore, this vaccine decreases not only the measles mortality, but also the mortality to subsequent non-measles infections (Mina, 2017; Rodrigues and Plotkin, 2020). In the proposed model, this vaccination effect is reproduced, because reductions in percentages of infected people and in cumulative deaths increase with $\nu$, for $\nu < \nu_m$, as shown in Table 1 and Figs. 2–5.

Table 2 reveals that the higher the values of $B$ and $G$, the higher the critical vaccination coverage $\nu_m$ to eradicate disease-1. In other words: the higher immunosuppression level caused by disease-1, the greater the efforts of public health agencies to encourage vaccination against this disease.

In the model, disease-1 can emulate the role of, for instance, measles or even COVID-19. In fact, COVID-19, in addition to causing respiratory problems, cerebrovascular disorders, neurological injuries, and psychological distress (Mahalakshmi et al., 2021), can also suppress host immunity (Remy et al., 2020), which can increase the vulnerability to future infections as measles does. In the model, disease-2 can be any non-vaccine preventable infection, such as hepatitis C, or a vaccine-preventable infection, but with poor adherence to vaccination, such as hepatitis A (Johnson et al., 2019).

In short, vaccination campaigns against measles/CODVID-19 provide direct protection against measles/COVID-19 and also indirect benefits against other contagious diseases, by preserving the herd immunities of the host population. It is crucial that public health agencies undermine vaccine hesitancy and vaccine refusal. These are threats that can be treated by combating disinformation and fake news.

CRediT authorship contribution statement

G.F. Puga: Software, Validation, Formal analysis, Investigation, Resources, Data curation, Writing - original draft, Visualization, Ecological Complexity 47 (2021) 100941

Table 1

| $\nu$ | $y$ for $n = 100$ | $m$ for $n = 100$ | $y$ for $n = 200$ | $m$ for $n = 200$ |
|-------|-----------------|-----------------|-----------------|-----------------|
| 10%   | 0.03            | 3.2             | 0.03            | 3.3             |
| 20%   | 0.06            | 6.5             | 0.06            | 6.7             |
| 30%   | 0.11            | 10.6            | 0.12            | 10.5            |

Table 2

| $B$ | $G$ | $\nu_m$ for $n = 100$ | $\nu_m$ for $n = 200$ |
|-----|-----|-----------------------|-----------------------|
| 1   | 1   | 33%                   | 35%                   |
| 1   | 3   | 36%                   | 40%                   |
| 3   | 1   | 37%                   | 42%                   |
| 3   | 3   | 43%                   | 43%                   |

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Declaration of Competing Interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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