Since January 2020 Elsevier has created a COVID-19 resource centre with free information in English and Mandarin on the novel coronavirus COVID-19. The COVID-19 resource centre is hosted on Elsevier Connect, the company's public news and information website.

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Short communication

On the spread of SARS-CoV-2 under quarantine: A study based on probabilistic cellular automaton

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ABSTRACT

Currently, SARS-CoV-2 (severe acute respiratory syndrome coronavirus 2) is a major worldwide public-health problem. Here, its propagation is modeled by using a probabilistic cellular automaton (PCA). In this model, sick individuals can either remain asymptomatic during the infection or become symptomatic. In order to derive an analytical expression for the basic reproduction number R_0, a mean-field approximation written in terms of ordinary differential equations (ODE) is proposed and analyzed. By considering time-constant and time-varying parameters in both approaches (PCA and ODE), numerical simulations are performed in order to evaluate the impact of distinct quarantine regimes on the SARS-CoV-2 pandemic.

1. Introduction

Severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) is the pathogen responsible for the current outbreak of coronavirus disease 2019 (COVID-19). The interpersonal transmission mainly occurs through infectious aerosols and droplets, via skin-to-skin contact, and after touching contaminated objects (Lai et al., 2020; Singhal, 2020; Sohrabi et al., 2020; Velavan and Meyer, 2020). Besides its potential lethality, this virus became a truly global problem due to its impact on businesses, family relationships, jobs, schools, temples, theaters; in short, on virtually all kinds of human activities. A major reason for this impact in our routine comes from the possibility of transmission by pre-symptomatic and asymptomatic carriers; that is, by apparently healthy individuals (Lai et al., 2020; Singhal, 2020; Sohrabi et al., 2020; Velavan and Meyer, 2020). Without COVID-19 testing, the identification of these carriers is a challenge (Day, 2020a, 2020b; Yu and Yang, 2020). Hence, as part of a control strategy, all non-sick individuals are supposed to be potential carriers.

To reduce the encounters among susceptible and unidentified infected individuals, public health authorities have recommended increasing the physical distancing and restricting the movement of people. Hence, in the latest months, cities and countries have implemented policies of quarantine and lockdown (Bruns et al., 2020; Iacobucci, 2020; Sjodin et al., 2020; Yang et al., 2020). The contagion can be also reduced by practicing good hygiene habits (such as frequent hand-washing, regular surface disinfection) and by wearing face masks in public places. Other ways for containing the virus propagation involve the development of a vaccine to prevent the infection and the use of a drug for successfully treating COVID-19 patients (Lai et al., 2020; Singhal, 2020; Sohrabi et al., 2020; Velavan and Meyer, 2020). A vaccine would decrease the number of susceptible individuals; an antiviral drug would shorten the infectious period of sick individuals.

Despite the recent appearance, the spread of SARS-CoV-2 has already been theoretically studied (Gostic et al., 2020; Kochanczyk et al., 2020; Monteiro, 2020; Volpert et al., 2020; Yang and Wang, 2020; Zhou et al., 2020). These studies are based on the analyses and simulations of systems of ordinary differential equations (ODE). In fact, epidemic models are usually described by ODE (Anderson and May, 1992; Keeling and Rohani, 2008).

Probabilistic cellular automaton (PCA) has been considered as an alternative approach to study the propagation of contagious diseases (Ahmed et al., 1998; Boccara et al., 1994; Doran and Laffan, 2005; Ferreri and Venturino, 2013; Fuentes and Kuperman, 1999; Nie and Li, 2020; Slimi et al., 2009; Zhang et al., 2018). Here, an epidemic model formulated in terms of PCA is proposed to investigate the propagation of SARS-CoV-2. In this discrete-time model, probabilistic rules specify the possible changes in the health status of the individuals. The transmission by individuals who do not develop symptoms and by the individuals who experience symptoms are both taken into account. To derive analytical results, a mean-field approximation for the PCA model,

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written as a set of ODE, is proposed. A similar ODE system was already analyzed (Monteiro, 2020). In our previous work (Monteiro, 2020), asymptomatic individuals can cause asymptomatic and symptomatic infections, and symptomatic individuals can also cause asymptomatic and symptomatic infections; thus, there are four rate constants weighting these four contagion pathways. In this work, there are three rate constants (and three contagion pathways) related to the virus spread. In addition, the infections caused by either asymptomatic or/and symptomatic individuals are divided into a fraction of x individuals that will become asymptomatic and a fraction of $1-x$ individuals that will become symptomatic. This way of representing the process of disease spread facilitates the estimation of ODE parameters (and the predictions obtained from the analytical expressions derived from the ODE model) from PCA simulations. Besides, here the main aim is to examine the effects of distinct quarantine regimes on the course of the COVID-19 pandemic. For this reason, numerical simulations were carried out with the PCA and ODE models by considering time-constant and time-varying contagion rates.

Usually, epidemic models on SARS-CoV-2 consider the latent health state (Gostic et al., 2020; Kochanczyk et al., 2020; Yang and Wang, 2020; Zhou et al., 2020). As in our previous work (Monteiro, 2020), here, the latent state, corresponding to individuals who have been infected but are not infectious yet, is not explicitly considered. However, the corresponding latent (incubation) period is taken into account. The originality of the model proposed here is also in the way of representing the virus propagation.

This paper is organized as follows. In Section 2, the PCA model is introduced. In Section 3, the corresponding ODE model is derived and analyzed. In Section 4, results obtained from computer simulations with both models are presented. In Section 5, the possible relevance of this epidemic study is discussed.

2. The PCA model

Let a human population living in a given geographical area be represented by a two-dimensional lattice $n \times n$ with periodic boundary conditions (that is, the top and bottom edges are connected and the left and right edges are also connected in order to avoid edge effects). Each cell composing this lattice represents an individual, which maintains social contact with the eight surrounding neighbors. This coupling topology is known as Moore neighborhood of unit radius (Wolfram, 1994).

At each time step t, each individual is in one of four health states: susceptible (S), asymptomatic infected (A), symptomatic infected (I), or recovered (R). The time evolution of this SAIR epidemic model is driven by seven probabilistic rules of state transitions: two rules for S-individuals, two rules for A-individuals, two rules for I-individuals, and one rule for R-individuals. For S-individuals: at each time step, there is a probability $P_i = 1 - e^{-(k_1 \nu_1 + k_2 \nu_2)}$ of a S-individual being infected due to the presence of A and/or I-neighbors. In this expression for P_i, ν_1 is the number of A-neighbors, ν_2 is the number of I-neighbors, k_1 expresses the infectivity of A-individuals, and k_2 expresses the infectivity of I-individuals. Note that $P_i = 0$ if $\nu_1 = \nu_2 = 0$ and $P_i \to 1$ if $k_1 \to \infty$ and/or $k_2 \to \infty$. If a S-individual is infected, the probability of becoming asymptomatic (that is, an A individual) is P_x; consequently, the probability of becoming symptomatic (that is, an I-individual) is $1 - P_x$. For A-individuals: at each time step, there is a probability P_{b_1} of an A-individual being cured and becoming a R-individual. If this A-individual remains infected, there is a probability P_{c_1} of dying (due to other causes). For I-individuals: at each time step, there is a probability P_{b_2} of an I-individual being cured and becoming a R-individual. If this I-individual remains infected, there is a probability P_{c_2} of dying due to the infection. For R-individuals: at each time step, there is a probability P_d of a R-individual dying (due to other causes). When A, I, or R-individuals die, S-individuals replace them. Therefore, since the deaths are balanced by the births, the total number of individuals $N = n^2$ remains constant.

Note that there is no transition from A to I; thus, A-individuals are those who remain asymptomatic during the whole course of the infection. Note also that the exposed state, usually denoted by the letter E in epidemiological models, is not considered; thus, the incubation period (the time from being infected to becoming infectious) is incorporated in the recovery and death rate constants of A and I-individuals. In addition, R-individuals are assumed to have acquired a protective immunity against this pathogen. However, if this diseases does not confer long-lasting immunity, then P_d must also include the probability per time step of losing this protection. In a computer simulation, the health states of all individuals are simultaneously updated in the end of each time step. Similar epidemic models formulated in terms of PCA can be found in literature (Chaves and Monteiro, 2017; Ferraz and Monteiro, 2019; Monteiro et al., 2006; Ramos and Schimit, 2019; Schimit and Monteiro, 2009; Silva and Monteiro, 2014).

3. The ODE model

Let $S(t)$, $A(t)$, $I(t)$, and $R(t)$ be the numbers of S, A, I, and R-individuals living in a given geographical region at the instant t, respectively. If these four subpopulations are homogeneously mixed in this region (Turnes and Monteiro, 2014), then a mean-field approximation for the PCA model can be written in terms of the following set of ordinary differential equations:

$$\begin{aligned}\frac{dS(t)}{dt} &= -a_1 S(t)A(t) - a_2 S(t)I(t) - a_3 S(t)A(t)I(t) \\ &\quad + c_1 A(t) + c_2 I(t) + dR(t)\end{aligned} \tag{1}$$

$$\frac{dA(t)}{dt} = x[a_1 S(t)A(t) + a_2 S(t)I(t) + a_3 S(t)A(t)I(t)] - b_1 A(t) - c_1 A(t) \tag{2}$$

$$\frac{dI(t)}{dt} = (1-x)[a_1 S(t)A(t) + a_2 S(t)I(t) + a_3 S(t)A(t)I(t)] - b_2 I(t) - c_2 I(t) \tag{3}$$

$$\frac{dR(t)}{dt} = b_1 A(t) + b_2 I(t) - dR(t) \tag{4}$$

The parameters a_1, a_2, a_3, b_1, b_2, c_1, c_2, d, and x are positive numbers. The rate constants a_1, a_2, and a_3 are respectively related to the contagion of S-individuals caused only by A-individuals, only by I-individuals, and simultaneously by A and I-individuals. In other words, the terms $a_1 S(t)A(t)$ and $a_2 S(t)I(t)$ respectively correspond to the disease propagation due to encounter of S-individuals with either A or I-individuals. The inclusion of the term with the parameter a_3 is necessary because, in the cellular automaton (and in the real-world; for instance, in a supermarket), both A and I-individuals can simultaneously be in the neighborhood of a S-individual.

The parameter x is the fraction of S-individuals who were just infected and will remain asymptomatic (that is, will be A-individuals); therefore, $1-x$ is the fraction of S-individuals who were just infected and will develop symptoms (that is, will be I-individuals) during the infection. The parameters b_1 and b_2 express the recovery rate constants of A and I-individuals, respectively. The parameters c_1, c_2, and d express the death rate constants of A, I, and R-individuals, respectively. Note that the terms of deaths in Eqs. (2)–(4) represent births of S-individuals in Eq. (1). Thus, as in the PCA model, the total number of individuals N is kept constant. This can be verified by observing that $dS(t)/dt + dA(t)/dt + dI(t)/dt + dR(t)/dt = 0$; therefore, $S(t) + A(t) + I(t) + R(t) = N$. As $R(t) = N - S(t) - A(t) - I(t)$, the ODE model can be rewritten as a third-order dynamical system:

$$\begin{aligned}\frac{dS}{dt} &= -a_1 SA - a_2 SI - a_3 SAI + c_1 A + c_2 I + d(N - S - A - I) \\ &\equiv f_1(S, A, I)\end{aligned} \tag{5}$$

$$\frac{dA}{dt} = x(a_1SA + a_2SI + a_3SAI) - b_1A - c_1A \equiv f_2(S, A, I) \tag{6}$$

$$\frac{dI}{dt} = (1-x)(a_1SA + a_2SI + a_3SAI) - b_2I - c_2I \equiv f_3(S, A, I) \tag{7}$$

A similar model based on ODE for SARS-CoV-2 was already analyzed (Monteiro, 2020). The difference is in the terms representing the virus transmission.

In the dynamical systems theory (Guckenheimer and Holmes, 2002), a solution (S^*, A^*, I^*) for Eqs. (5)–(7) satisfying $f_1(S^*, A^*, I^*) = 0$, $f_2(S^*, A^*, I^*) = 0$, and $f_3(S^*, A^*, I^*) = 0$, in which S^*, A^*, and I^* are constants, is called stationary solution. The stationary solution given by $(S_1^*, A_1^*, I_1^*) = (N, 0, 0)$ represents a disease-free steady-state, because $A_1^* = 0$ and $I_1^* = 0$. A solution given by (S_2^*, A_2^*, I_2^*), with $A_2^* \neq 0$ and $I_2^* \neq 0$, represents an endemic steady-state. Obviously, $R_1^* = 0$ and $R_2^* = N - S_2^* - A_2^* - I_2^*$. By neglecting the term with a_3 in the equations $f_1 = 0$, $f_2 = 0$, and $f_3 = 0$, then $I_2^* \approx qA_2^*$, with:

$$q \approx \frac{(b_1 + c_1)(1-x)}{(b_2 + c_2)x} \tag{8}$$

This approximate expression can be used to evaluate the proportion between symptomatic and asymptomatic individuals in the endemic steady-state (achieved in a simulation of the PCA model or found in real-world data).

The basic reproduction number R_0 of this model is given by:

$$R_0 = \frac{xa_1N}{b_1 + c_1} + \frac{(1-x)a_2N}{b_2 + c_2} \tag{9}$$

Recall that R_0 expresses the average number of infections directly caused by a single sick individual introduced into a susceptible population (Anderson and May, 1992; Keeling and Rohani, 2008). The expression found for R_0 corresponds to the largest eigenvalue of the next generation matrix $\mathbf{G} = \mathbf{F}\mathbf{V}^{-1}$ (Diekmann et al., 2010; van den Driessche, 2017), in which the matrix $\mathbf{F}$ is related to the appearance of new infections in the infected subpopulations (which are A and I) and the matrix $\mathbf{V}$ is related to other state transitions occurring in these (two) subpopulations. If $R_0 < 1$, the disease is naturally eradicated; if $R_0 > 1$, it persists in the host population. Observe in Eq. (9) that the contribution of A-individuals (related to a_1) is added to the contribution of I-individuals (related to a_2). If $x = 0$, then $R_0 = a_2N/(b_2 + c_2)$, an expression already derived in other epidemiological studies (Ferraz and Monteiro, 2019; Monteiro et al., 2006; Schimit and Monteiro, 2009; Silva and Monteiro, 2014).

The ODE system can approximately reproduce the dynamical behavior observed in numerical simulations of the PCA model if the values of $a_1, a_2, a_3, b_1, b_2, c_1, c_2, d$, and x are consistently obtained from $k_1, k_2, P_{b_1}, P_{b_2}, P_{c_1}, P_{c_2}, P_d$, and P_x. Here, consistent choices are $b_1 = P_{b_1}$, $b_2 = P_{b_2}$, $c_1 = (1 - P_{b_1})P_{c_1}$, $c_2 = (1 - P_{b_2})P_{c_2}$, $d = P_d$, and $x = P_x$. The values for a_1, a_2, and a_3 in the ODE model are influenced by the values of k_1, k_2, and by the coupling network of the PCA lattice. They can be estimated from a simulation of the PCA model as follows:

$$a_1 \simeq \frac{-\Delta S_1}{S_{av}A_{av}} \tag{10}$$

$$a_2 \simeq \frac{-\Delta S_2}{S_{av}I_{av}} \tag{11}$$

$$a_3 \simeq \frac{-\Delta S_3}{S_{av}A_{av}I_{av}} \tag{12}$$

Note the number of infections caused by the virus propagation rule in the PCA model, which depends on P_i (which is function of k_1 and k_2), is split into three terms in the ODE model, which depend on a_1, a_2, and a_3. In these expressions, ΔS_1, ΔS_2, and ΔS_3 are respectively the differences between two consecutive time steps of susceptible individuals due to the contagion when there are only A-individuals, only I-individuals, and both A and I-individuals in the neighborhoods of S-individuals in the PCA lattice (for instance, if between two times steps, 18 S-individuals were infected and there were only A-individuals in their neighborhoods, then $\Delta S_1 = -18$). Also, S_{av}, A_{av}, and I_{av} are the asymptotic values of $S(t)$, $A(t)$, and $I(t)$; that is, the values after the transient phase of a simulation of the PCA model. In practice, S_{av}, A_{av}, and I_{av} are taken as the average values computed in the last 52 time steps of simulations with 520 time steps (which are enough to the system achieving its long-term behavior).

The other relations between the ODE parameters and the PCA parameters can be inferred by comparing the mathematical terms of the ODE model to the corresponding state transition rules of the PCA model. For instance, recall the b_1 is the recovery rate constant of A-individuals. From Eq. (6), note that $b_1 = \Delta A_{A\to R}/A_{av}$, in which $\Delta A_{A\to R}$ is the number of A-individuals who recover between two consecutive time steps. Therefore, the fraction $\Delta A_{A\to R}/A_{av}$ corresponds to the percentage of recovered A-individuals; in other words, the probability of an A-individual being cured. Hence, $b_1 = P_{b_1}$. The parameter c_1 denotes the death rate constant of A-individuals. Note that $c_1 = (1 - P_{b_1})P_{c_1}$, because only A-individuals that were not cured (with probability $1 - P_{b_1}$) can die (with probability P_{c_1}).

Suppose that, in a simulation, the PCA converges to an endemic steady-state. If the values of the ODE parameters are estimated from this simulation as described above, then $S_2^* \simeq S_{av}$, $A_2^* \simeq A_{av}$, $I_2^* \simeq I_{av}$, and $R_2^* \simeq R_{av}$; that is, the ODE system converges to the same steady state achieved in the PCA lattice. Similar studies with PCA and ODE can be found in literature (Ferraz and Monteiro, 2019; Monteiro et al., 2006; Ramos and Schimit, 2019; Schimit and Monteiro, 2009; Silva and Monteiro, 2014).

4. Simulation results

Numerical simulations were performed with the PCA model by taking the initial condition $S(0)/N = 99.99\%$, $A(0)/N = 0.005\%$, $I(0) = 0.005\%$, and $R(0) = 0\%$; therefore, the disease is introduced by $A(0)/N + I(0)/N = 0.01\%$ of infected individuals. Also, at $t = 0$, there is no R-individual. One time step in the PCA model corresponds to one week of real time. The PCA simulation follows the pseudocode presented in Appendix.

If the recovery from this infection confers long-term immunity and the average life expectancy of the host population is 80 years, then $P_{c_1} = P_d = 1/(80 \times 52)$. If the immunity lasts for 1/3 of one year, on average, then $P_d = 1/(80 \times 52) + 3/52$. Unfortunately, the immune response to SARS-CoV-2 is not yet fully understood. By including the incubation period, the recovery periods for A and I-individuals are about 2 and 3 weeks (Hu et al., 2020; Singhal, 2020), respectively; therefore $P_{b_1} = 1/2$ and $P_{b_2} = 1/3$. The death probability (per time step) of I-individuals is supposed to be $P_{c_2} = 1/50$, because, on average, 6% of I-individuals die in 3 weeks (Giangreco, 2020; Wu et al., 2020). If, when a S-individual gets sick, there is a 80% chance of remaining without symptoms, then $P_x = 4/5$. With these choices, $q \approx 0.36$; therefore, the infected subpopulation in steady state is composed of 74% of A-individuals (and 26% of I-individuals), which are values compatible with those observed in two countries (Day, 2020a; 2020b). If $P_x = 1/5$, then $q \approx 5.8$ and the fraction of A-individuals in the infected subpopulation is 15%, which is a number compatible with other works (Mizumoto et al., 2020; Singhal, 2020). The values of the infectivity parameters k_1 and k_2 must be chosen in order to obtain $R_0 \in [1.5, 6.5]$ (Gostic et al., 2020; Singhal, 2020; Tang et al., 2020; Wu et al., 2020). Here, $k_1 = 1$ and $k_2 = 4$. Note that $k_2 > k_1$, because I-individuals are supposed to be more infectious than A-individuals. Recall that k_1 and k_2 affect a_1 and a_2 (and a_3) and, consequently, R_0, which is determined by Eq. (9).

First, consider R-individuals with long-term immunity (that is, $P_d =$

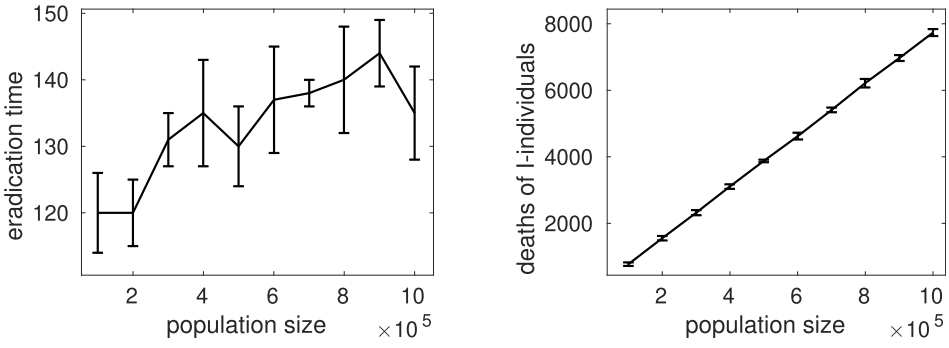

Fig. 1. Eradication time T (at left) and number of deaths D of I-individuals (at right) found in a PCA lattice by taking $P_{b_1} = 1/2$, $P_{c_1} = P_d = 1/(80 \times 52)$, $P_{b_2} = 1/3$, $P_{c_2} = 1/50$, $P_x = 4/5$, $k_1 = 1$, and $k_2 = 4$. In these simulations, $n = 316, 447, 548, 632, 707, 775, 837, 894, 949$, and 1000 (thus, the difference between two consecutive values of N is about 100,000). Five simulations were run for each value of n from the initial condition $S(0)/N = 99.99\%$, $A(0)/N = 0.005\%$, $I(0)/N = 0.005\%$, and $R(0)/N = 0\%$. The disease naturally disappears in all cases.

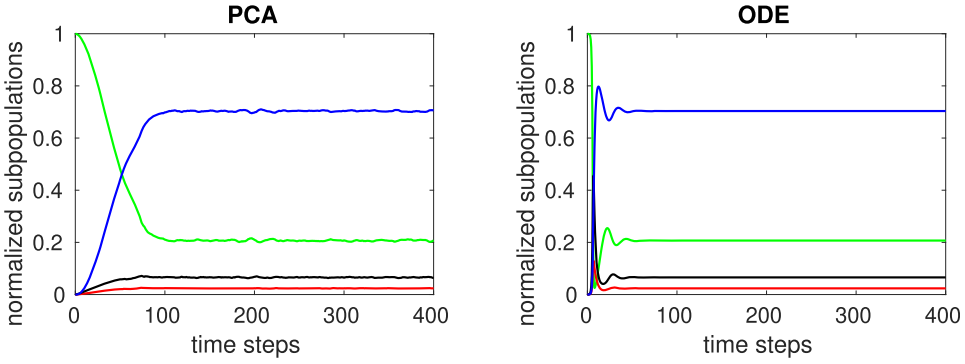

Fig. 2. Time evolutions of $S(t)/N$ (green line), $A(t)/N$ (black line), $I(t)/N$ (red line), and $R(t)/N$ (blue line) from $S(0)/N = 99.99\%$, $A(0)/N = 0.005\%$, $I(0)/N = 0.005\%$, and $R(0)/N = 0\%$ obtained from PCA (left) and ODE (right). In the PCA, $n = 400$, $P_{b_1} = 1/2$, $P_{c_1} = 1/(80 \times 52)$, $P_{b_2} = 1/3$, $P_{c_2} = 1/50$, $P_d = 1/(80 \times 52) + (3/52)$, $P_x = 4/5$, $k_1 = 1$, and $k_2 = 4$. In the ODE, $b_1 = 1/2$, $c_1 = (1 - 1/2) \times (1/(80 \times 52))$, $b_2 = 1/3$, $c_2 = (1 - 1/3) \times 1/50$, $d = 1/(80 \times 52) + (3/52)$, $x = 4/5$, $a_1 N \simeq 1.946$, $a_2 N \simeq 1.596$, and $a_3 N^2 \simeq 21.02$ (these last three numbers were computed by using Eqs. (10)–(12)). In both approaches, as time passes by, $S(t)/N \to 0.207$, $A(t)/N \to 0.066$, $I(t)/N \to 0.024$, and $R(t)/N \to 0.703$. In this case, $R_0 \simeq 4.0 > 1$; hence, the disease persists in the host population. (For interpretation of the references to color in this figure legend, the reader is referred to the web version of this article.)

$1/(80 \times 52)$) and $n = 400$ (thus, the population is composed of 160,000 individuals). Simulations reveal that, in this case, the disease is naturally eradicated at the time step T; that is, $A(t) = 0$ and $I(t) = 0$ for $t \geq T$. The eradication occurs for $T \simeq 130$ for $P_x = 4/5$ and $P_x = 1/5$. In the first case, the number D of deaths of I-individuals (that is, the number of transitions $I \to S$) is $D \simeq 1.2 \times 10^3$; in the second case, $D \simeq 5.1 \times 10^3$. These are average numbers that were obtained in five simulations for each value of P_x. Observe that the higher the value of P_x, the lower the value of D. Fig. 1 illustrates how T (Fig. 1 at left) and D (Fig. 1 at right) vary with $N = n^2$ for $P_x = 4/5$. Note that $D \simeq 0.0078N$; that is, D linearly increases with N. Also, $T \simeq 130$; that is, the disease disappears after about 130 weeks in lattices with $99856 \leq N \leq 1,000,000$.

A quarantine regime can be simulated by reducing the values of k_1 and k_2. Assume that $k_1 = 1$ and $k_2 = 4$ are both multiplied by the factor σ. For $\sigma = 1/4$, the disease disappears after about 170 weeks and the number of deaths is about $D \simeq 0.0076N$. Therefore, the relation between D and N is practically the same for $\sigma = 1$ and $\sigma = 1/4$.

Now, consider R-individuals with short-lasting immunity (that is, $P_d = 1/(80 \times 52) + 3/52$) and $n = 400$. Fig. 2 shows the time evolutions of $S(t)/N$ (green line), $A(t)/N$ (black line), $I(t)/N$ (red line), and $R(t)/N$ (blue line) obtained in a simulation with the PCA model and by numerically integrating the ODE model with the 4th-order Runge-Kutta method with integration time step of 0.01. In the PCA model, for $P_x = 4/5$, $k_1 = 1$, and $k_2 = 4$, then $S(t)/N \to S_{av}/N \simeq 0.207$, $A(t)/N \to A_{av}/N \simeq 0.066$, $I(t)/N \to I_{av}/N \simeq 0.024$, and $R(t)/N \to R_{av}/N \simeq 0.703$ as time goes by. Note that $I_{av}/A_{av} \simeq 0.36$, which coincides with the value estimated for q from the ODE system by using Eq. (8). By numerically integrating Eqs. (1)–(4) with $a_1 N \simeq 1.946$, $a_2 N \simeq 1.596$, $a_3 N^2 \simeq 21.02$ (recall that a_1, a_2, and a_3 are computed by using Eqs. (10)–(12)), $b_1 = 0.5$, $c_1 \simeq 0.00012$, $b_2 \simeq 0.33333$, $c_2 \simeq 0.01333$, $d \simeq 0.05793$, and $x = 0.8$, the system converges to the endemic steady-state $S_2^*/N \simeq 0.207$, $A_2^*/N \simeq 0.066$, $I_2^*/N \simeq 0.024$, $R_2^*/N \simeq 0.703$. Observe that the asymptotic behaviors found in PCA and ODE are identical (recall that the ODE parameters were identified from the long-term behavior found in the PCA simulations; hence, there is the good agreement of both approaches when they reach their stationary solutions. The match in the transitory phase, however, is not good). In this scenario, from Eq. (9), $R_0 \simeq 4.0$. Also, there were $D \simeq 2.5 \times 10^4$ deaths of I-individuals in 520 times steps.

In the PCA, for $P_x = 1/5$, $S(t)/N \to 0.141$, $A(t)/N \to 0.018$, $I(t)/N \to 0.102$, $R(t)/N \to 0.739$ as time passes by. Similar numbers are found from the ODE system. In addition, $q \simeq 5.7$, $R_0 \simeq 6.2$, and $D \simeq 11 \times 10^4$ in 520 times steps. Note that, for $P_x = 1/5$ and for $P_x = 4/5$, the disease endemically persists in the host population; however, the smaller the value of P_x, the greater the values of q, R_0, and D. Recall that $1 - P_x$ is the probability of an infected individual developing symptoms.

Table 1 presents the average percentages S_{av}/N, A_{av}/N, I_{av}/N, and R_{av}/N obtained in the last 52 time steps (one year of real time) of simulations with 520 time steps under distinct quarantine regimes, for $n = 400$. In addition, the ratio I_{av}/A_{av}, the basic reproduction number R_0, and the number D of deaths of I-individuals occurring in the whole simulation are also shown. Table 1 summarizes the results of eight scenarios: in four scenarios, σ is kept constant and equal to 1, 3/4, 1/2, and 1/4; in four scenarios, σ periodically varies between 1 and 3/4, between 1 and 1/2, between 1 and 1/4, and between 1 and 0 (note that $\sigma = 0$ means full lockdown). The period of this variation is taken as 8 time steps (thus, there occurs a periodic alternation between one month with quarantine and one month without quarantine). As expected, Table 1 shows that the lower the value of σ, the higher the value of S_{av} and the lower the values of A_{av}/N, I_{av}/N, R_0, and D. Note

input paramater values n, k_1, k_2, P_{b_1}, P_{c_1}, P_{b_2}, P_{c_2}, P_d
set matrix $M(i, j)$ $(i, j = 1, \ldots, n)$
set the cells (i, j) equal to S, A, I, or R according to the initial proportion
if $(i, j) = S$, then compute $P_i = 1 - e^{-(k_1 v_1 + k_2 v_2)}$
% v_1 is the number of A-neighbors
% and v_2 is the number of I-neighbors at this time step
  if random number $< P_i$
  then $(i, j) = A$ with probability P_x or $(i, j) = I$ with probability $1 - P_x$
  else $(i, j) = S$
  endif
endif
if $(i, j) = A$
  if random number $< P_{b_1}$
  then $(i, j) = R$
  else $(i, j) = A$
  endif
  if $(i, j) = A$ and random number $< P_{c_1}$
  then $(i, j) = S$
  else $(i, j) = A$
  endif
endif
if $(i, j) = I$
  if random number $< P_{b_2}$
  then $(i, j) = R$
  else $(i, j) = I$
  endif
  if $(i, j) = I$ and random number $< P_{c_2}$
  then $(i, j) = S$
  else $(i, j) = I$
  endif
endif
if $(i, j) = R$
  if random number $< P_d$
  then $(i, j) = S$
  else $(i, j) = R$
  endif
endif
go to the next cell without updating the state of this cell
update simultaneously the states of all cells
go to the next time step

Algorithm 1. Pseudocode for computing one time step of the PCA simulation.

that $I_{av}/A_{av} \simeq 0.36$, which agrees with the value of q estimated from Eq. (8). In these eight scenarios, the disease is not eradicated. Other simulations show that it can be eradicated by imposing a permanent quarantine with $\sigma \leq 2/25$ or a periodic quarantine with σ switching, for instance, between 1/4 and 0. In this case, $T \simeq 55$ and $D \simeq 20$.

5. Discussion and conclusion

Here, an epidemic model based on PCA for the spread of SARS-CoV-2 was proposed and simulated. From its mean-field approximation written in terms of ODE, analytical expressions for the basic reproduction number R_0 and the ratio q between symptomatic and symptomatic individuals were derived. These expressions can be useful to characterize the dynamical behavior observed in computer simulations with the PCA model. It is relevant to stress that the values of R_0 and q obtained in these simulations are similar to those found in real-world observations.

The simulations were performed by using realistic parameter values. They revealed that, in the case of long-lasting immunity, the disease is naturally eradicated. As expected, the number of deaths D linearly grows with N; here, $D/N \approx 0.8\%$. However, D/N is not significantly reduced by decreasing σ. Also, the eradication occurs after $T \approx 2 - 3$ years from an initial condition with 0.01% of sick people. Note that the population size does not significantly affect the eradication time T nor the proportion of deaths D/N. Fig. 1 illustrates these results.

For the short-lasting immunity considered in this work, the disease endemically persists with $R_0 \simeq 2 - 4$. As presented in Table 1, by reducing the infectivity parameters k_1 and k_2, then A_{av}/N, I_{av}/N, R_0, and D are reduced and S_{av} is increased. Note that the simulation with σ switching between 1 and 0 is surprising. Even by imposing a periodic quarantine regime since the beginning of the spread (which starts with 8 A-individuals and 8 I-individuals), the pathogen chronically persists after 10 years (520 time steps) with about $A_{av}/N + I_{av}/N \simeq 1\%$ of infected individuals. Also, in this simulation, $R_0 \simeq 0.8 < 1$. This value was computed from the average values of the infectivity rate constants

Table 1
Average values of $S(t)/N$, $A(t)/N$, $I(t)/N$, and $R(t)/N$ obtained in the last 52 time steps (that is, 1 year of real time) in simulations with 520 time steps (which are enough to the system reaching a steady state). These average normalized numbers are respectively denoted by S_{av}/N, A_{av}/N, I_{av}/N, and R_{av}/N. In these simulations, $k_1 = \sigma \times 1$ and $k_2 = \sigma \times 4$. The other parameter values are the same as used in Fig. 2. Permanent quarantine corresponds to $\sigma = 0.75$, 0.5, and 0.25; periodic quarantine is simulated by varying σ between 1 and 0.75, between 1 and 0.5, between 1 and 0.25, and between 1 and 0. This periodic oscillation is represented below by $\{1, 0.75\}$, $\{1, 0.5\}$, $\{1, 0.25\}$, and $\{1, 0\}$, respectively. The period of this oscillation is 8 time steps, because there is an alternation between 4 weeks without quarantine ($\sigma = 1$) and 4 weeks with quarantine ($\sigma < 1$). Also, the ratio I_{av}/A_{av}, the basic reproduction number R_0, and the amount D of deaths of I-individuals during the whole simulation are given. In all these simulations, the disease endemically persists.

σ	S_{av}/N	A_{av}/N	I_{av}/N	R_{av}/N	I_{av}/A_{av}	R_0	D
1	0.207	0.066	0.024	0.703	0.36	4.0	2.50×10^4
0.75	0.232	0.064	0.023	0.681	0.36	3.5	2.42×10^4
0.5	0.279	0.060	0.022	0.639	0.36	2.8	2.23×10^4
0.25	0. 416	0.048	0.017	0.518	0.36	1.8	1.76×10^4
$\{1, 0.75\}$	0.220	0.065	0.023	0.692	0.36	3.7	2.46×10^4
$\{1, 0.5\}$	0.238	0.063	0.023	0.676	0.36	3.4	2.40×10^4
$\{1, 0.25\}$	0.284	0.058	0.021	0.636	0.37	2.8	2.24×10^4
$\{1, 0\}$	0.900	0.008	0.003	0.089	0.36	0.8	0.20×10^4

a_1 and a_2 in the last 52 time steps. Therefore, naive computations of this parameter can lead to elusive conclusions.

With the parameter values used in Fig. 2, eradication requires either a permanent quarantine with $\sigma \leq 0.08$ or a periodic quarantine with "low" values of σ. For instance, the disease disappears by periodically switching σ between 0.25 and 0. In the time intervals without quarantine, reductions in σ can be attained by improving personal hygiene habits (for instance, frequent hand washing) and/or by changing social behaviors (for instance, by keeping social distancing).

The development of an immune response to SARS-CoV-2, as a consequence of recovery from infection or vaccination, will determine the dynamics of the spread and, consequently, the fate of this infection. Its eradication can require quarantine regimes. Mathematical models based on PCA and ODE can be used by public health authorities to find an optimal quarantine policy for mitigating this pandemic.

The study presented here was about the long-term behavior of this contagious infection and, unfortunately, we are still living its transitory phase. The parameter values used in the simulations were obtained in literature in order to provide realistic predictions. However, it is relevant to stress that underreporting in the number of cases and deaths due to SARS-CoV-2, low testing rates of suspected cases, and the implementation of erratic quarantine/lockdown regimes will make it difficult to derive reliable and unequivocal predictions from any epidemiological model.

CRediT authorship contribution statement

L.H.A. Monteiro: Conceptualization, Methodology, Validation, Formal analysis, Investigation, Resources, Writing - original draft, Writing - review & editing, Visualization, Supervision, Project administration, Funding acquisition. **V.C. Fanti:** Software, Validation, Formal analysis, Investigation, Resources, Data curation, Writing - original draft, Visualization, Funding acquisition. **A.S. Tessaro:** Software, Validation, Formal analysis, Investigation, Resources, Data curation, Writing - original draft, Visualization, Funding acquisition.

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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