COMPLEX NETWORKS BASED ALGORITHM FOR INFECTIOUS DISEASES SPREAD MODELING

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ABSTRACT

In this paper we propose a new algorithm to model the spread of infectious diseases in the population. Through a combination of the Susceptible-Infected-Recovered (SIR) epidemiology model and Small World social network. The model helps to understand the dynamics of infectious diseases in a population, and to identify the main characteristics of epidemic transmission and its evolution over time. Simulations were conducted to evaluate the proposed model. The obtained results were analyzed in order to explore how the evolution of the network influences the spread of the disease also statistical test was applied to validate our model. This model allows health policy makers to better understand the epidemic spread and to implement relevant control strategies.

Keywords: *SIR model, Complex networks, Infectious diseases, Influenza.*

1. INTRODUCTION

How to deeply understand the spread of infectious diseases, and then design effective prevention and control strategies is now an urgent task or threat to organizations or public ministries [1, 2]. Despite advances in treatment and prevention, infectious diseases continue to spread within the population and there are very few such diseases that are eradicated. This leads to a need for monitoring and control of the spread of infectious diseases in order to better understand them.

In 1760, Daniel Bernoulli developed a mathematical model of a smallpox epidemic for an analysis of smallpox mortality and the benefits inoculation to prevent it. His model is known as the first mathematical model in the history of epidemiology. Numerous studies have followed this work in order to understand the mechanism of the spread of infectious diseases [3-5]. In this perspective, using mathematical models seems to be a good tool for understanding the mechanism of the spread of infectious diseases. Several models exist in the literature allowing to represent the various statuses of the disease during the infection [6]. They divide the population into several classes representing different health states during a disease, such as: SI (Susceptible, Infected), SIR (Susceptible, Infected, Removed), etc. In this paper, we are particularly interested in the SIR compartmental model, since this latter can model a large series of infectious diseases. Thus,

each person of the population can be in one of three different compartments. Those who are susceptible to the disease are in the Susceptible compartment, those who are infected and can transmit the disease to others are in the Infected compartment and those who have recovered and are immune and those who are removed from population are in the Recovered compartment. Some infectious diseases are described with models that have different number of compartments like SIS model (Susceptible Infected Susceptible) where individuals cannot have long lasting immunity and there- fore Recovered compartment does not exist.

Traditional epidemiological models assume that the transmission of infection in a homogeneous population increases easily depending on the number of individuals and a risk of random infection [7, 8]. In addition, the transmission of an epidemic occurs through direct contact between a susceptible and infected person. Moreover, the topology of the social structure of individuals has a direct impact on the transmission of the disease. This latter is not just a simple list of nodes and edges, but rather a system where the links interact some with the others and undergo transformations and changes in the time and space. Consequently, the integration of the social network in the SIR model seems interesting [9].Some studies in epidemiology have focused on the structure of the social network and the processes of transmission and diffusion of information within social

networks [10-14].In this paper, we are interested in modelling the phenomenon of the spreading of the epidemic by integrating both the biological and the social aspects of the population. We highlight the phenomenon of transmission and the complexity of disease dynamics on one hand, and the structural complexity of the social network and its influence on the dynamics of the system on the other. The work of [15] in the literature worked on the combination of SEIR model and SW using the probability P_k , however the aim of our paper is to combine the Susceptible-Infected-Removed (SIR) epidemiology model with Small World social networks using the average degree of distribution $\langle k \rangle$, which represents the average number of neighbors that an individual can have in the population, and propose a new propagation algorithm able to approach the number of the infectious in the population in each time t. We use then the results of the algorithm to study the influence the parameter $\langle k \rangle$ in the infection dynamics, and identify the main characteristics of epidemic transmission and its evolution over time. This modelling contributes, effectively, to the simulation of a category of complex systems integrating both the biological aspect and the social aspect of the population. In addition, it provides a simulator (generator) based on the propagation model to generate simulated data.

This paper is organized as follows: Section 1 is the introduction. Section 2 we defined the SIR model and Small World network, in section 3 we generated three algorithms, the first is for the regular ring network, in the second we add the probability P to the regular network and obtained the small world network and the third is for the combination of small world and SIR model. In section 4we compared the model with real data using the kolmogorov-smirnov test in order to validate the proposed model, and present a discussion of the results with numerical simulations.

2. COMPUTATIONAL MODEL

2.1 Sir Model

The SIR model considers three groups of individuals in the population: S refers to healthy individuals in the population concerned (or susceptible to infection), I refer to those infected, and R is those who are recovered and cannot more infected. This system can be represented graphically by a set of three compartments connected by individuals flows which pass from one to the other, figure 1,
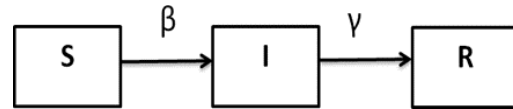

Figure 1: The Different Compartments Of The SIR Model

Each compartment is associated with a state variable: S, I and R. Thus, compartment R agents can no longer be infected again (R can also be called the compartment of reinstated individuals when we assume that these individuals are not dead). When R is not dead but has acquired immunity, the total population $(S + I + R)$ is constant. The model assumes that individuals in compartment S are sometimes infected by a contact with individuals in the compartment I and change at R at a constant rate. The model is thus written:

$$
\begin{cases}
ds(t)/dt = - \beta \, Ns(t)i(t) \\
di(t)/dt = \beta \, Ns(t)i(t) - \gamma i(t) \\
dr(t)/dt = \gamma i(t)
\end{cases}
\quad (1)
$$

Where β is the infection rate from S to I and γ is the healing rate from I to R. γ is inversely proportional to the mean infectious period, R0, is the average number of people infected by a single infected person when the population has no immunity and no control over infection [3] [10, 11]. In the model of differential equations SIR, the basic reproduction number is given by the formula $R_0 = \beta / \gamma$. On the other hand, if $R_0 > 1$, the infection occurs and spreads. On the other hand, if $R_0 \leq 1$, there is convergence of the disease as shown in the figure 2. Therefore $R_0 = 1$ is a propagation threshold [16, 17].
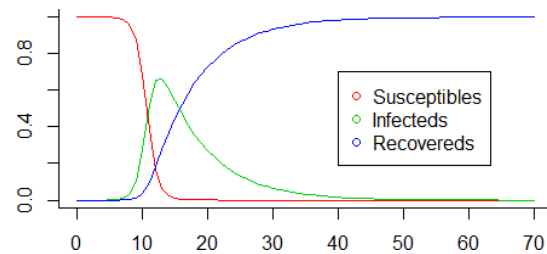

Figure 2: Simulation of the stochastic SIR epidemiology model (β = 1.4247, γ = 0.14286).

2.2 The Social Network Model: Smallworld

Complex networks are present in many domains: biology, sociology, psychology, computer science, etc. We use complex networks to considerate interactions that allow the transmission of infectious diseases between individuals. Such network scan predicts the outcome of an epidemic and thus helps to test and improve public health policies. A social network can be defined as:" a group of persons or groups of people possessing patterns of contacts or interactions between them" [18]. It's on the basis of this type of network that the modelling of the real world was introduced empirically through the Milgram experiment [19, 20].

The small-world effect is now observed on many networks coming from the real world. Several works are interested in the study of structural properties to reveal characteristics of the nodes or the structure. In the Small World graphs, two measures must be identified:

- the average length of the shortest path connecting two nodes of the graph (average path length), making it possible to measure the overall accessibility of the graph;

- cohesion (clustering coefficient), making it possible to measure the probability that two nodes connected to a third are connected to each other, which implies the constitution of the cliques within the network

The network assumes that the presence or absenceof an edge between two nodes is independentof the presence or absence of another edge, sothat each edge can be considered to have an independent probability P. The number of edges connected to any particular node is called the degree k of that node, and has a probability distribution P_k [21] given by:

$$P_k = \binom{N}{k} P^k (1 - P)^{N-k}$$

And the average degree distribution of small world network $\langle k \rangle$:

$$\langle k \rangle = \sum k \, P_k$$

The constitution of the Small World model starts with a regular ring network, and then a number of additional nodes is added to the middle of the network that is connected to a large number of sites (Nodes) randomly selected on the main regular network. Figure 3 represents the construction of a small world network.
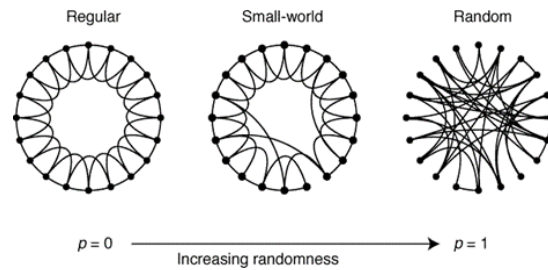

Figure 3: Illustration Of Different Classes Of Networks [22].

In order to model social contacts between human populations, we build a Small World network by representing individuals through nodes and social relationships through links. Formally, a network is described by a graph $G = (V, E)$ consisting of a set of vertices V (also called nodes) where $V = \{i \in N\}$ and aset E of links (also called edges) where $E = \{(i,j)/i, j \in V\}$ hence i: source node and j: target node.

The degree of a node i, represents the neighbors of the node in an unoriented graph denoted $k(i)$. It is the number of links between the node l and the other nodes of the graph, hence $k(i) = \{j/(i,j) \in E\}$. We denote by $T = \{t_0, t_1, ..., t_m\}$, the time sequence as the disease is transmitted, and $G = \{G_{t0}, G_{t1}, ..., G_{tm}\}$ the network sequence, where each element represents the network state at time t. V is a set of individuals, and E_{ij} is the set of edges present in the graph at time t.

3. ALGORITHMS

3.1 Definition Of Assumptions

In order to develop our model, we have to put forward some hypotheses:
- The population size is N, assumed to be fixed;
- The time variable t is of the discrete type, such that $\{t \in T\}$or T is the total time;
- The time period Δt can be days or weeks;

- At each instant t, the population is divided into three classes: $S(t)$: set of susceptible individuals, $I(t)$: set of infected individuals and $R(t)$: set of recovered individuals;
- An infected individual can no longer become susceptible again.

3.2 Sw Network Generation

The proposed process is based on the construction of the Small-World network, that involves two main stages. The construction of a regular ring

network and the reconnection of the edges of the graph (network).

Build a regular ring network
We begin by creating a regular ring network $G(t)$ with N nodes (the total number of nodes representing the total population) and k edges per node (k denotes the degree of a node or the number of its neighbors). We assume that the contact networks to be constructed will be unoriented graphs in such a way that the link between two nodes (source, target) is the same as (target, source). We set up an algorithm to build the regular network:

Algorithm 1: Building the regular ring network
Input: N, k
Output: G(t)
Begin
    for k < (N-1)/2 do
        if N is even then
            if k is even then
                for i = 1 to N do
                    for j = 1 to k/2 do
                        d = i + k/2
                        if d > N then
                        Connect node[i] with node[d-N]
                        else
                        Connect node[i] with node[d]
                        end
                    end
                end
            else
                for i = 1 to N do
                    for j = 0 to (k-1)/2 do
                      d = i + N/2-j
                      if d > N then
                      Connect node[i] with node[d-N]
                      else
                      Connect node[i] with node[d]
                      end
                  end
                end
            end
        else
            for i = 1 to N do
                for j = 0 to (k-1)/2 do
                    d = i + j
                    if d > N then
                    Connect node[i] with node[d-N]
                    else
                    Connect node[i] with node[d]
                    end
                end
            end
        end
    end

Return G(t)
END

Reconnect the edges with random probability(P) the process that follows the algorithm is:
In the graph G (t), some edges are randomly reconnected with a probability of reconnection (P) byreplacing $(Node[i], Node[j])$ by $(Node[i], Node[m])$ to avoid loops and duplicate link. This construction allows us to switch between the regular network $(P = 0)$, the random network $(P = 1)$, and the intermediate network $(0 < P < 1)$ as the Small World network.

Algorithm 2: Connect the edges randomly with a probability (P)
Input: G(t), k, P
Output: G(t +1)
Begin
    for i = 0 to N do
        for j = (i+1) to i+k/2 do
            if j > N then
                j = j - N
            end
            Chance =Uniform random variable between 0 and 1
            if P > Chance then
                Node[m] chosen uniformly randomly
                Disconnect Node[i] and Node[j]
                Connect Node[i] and Node[m]
            end
        end
    end
end
G(t) Envolves G(t +1)
END

3.3 Proposed Model

In this section we combine the Small world network with the SIR model. During the disease, Individuals can have three states:
- s(t): Rate of susceptible people in the population with s(t)= S(t)/N
- i(t): Rate of infected people in the population with i(t)= I(t)/N
- r(t): Rate of recovered people in the population with r(t)= R(t)/N

$$\begin{cases} ds(t)/dt = -\beta Ns(t)i(t) \\ di(t)/dt = \beta Ns(t)i(t) - \gamma i(t) \\ dr(t)/dt = \gamma i(t) \end{cases} \quad (3)$$

The term βNs(t) implies that all the infectious can contact all the susceptibles, in other words the graph modelling the population is completely connected. However, in the Small world complex network [22], of which each node has $\langle k \rangle$links onthe average, the assumption of complete connectionseems unreasonable. Hence the interest of replacingthe term βNs(t) with $\beta \langle k \rangle s(t)$, where $\langle k \rangle$ is the average degree of distribution [21], that represent the average number of neighbors that an individual can have in the population. The differential equations of this model become:

$$\begin{cases} ds(t)/dt = -\beta \langle k \rangle s(t)i(t) \\ di(t)/dt = \beta \langle k \rangle s(t)i(t) - \gamma i(t) \\ dr(t)/dt = \gamma i(t) \end{cases}$$

The numerical method used to solve the differential equations is the Euler method based on the discretization of the variable t. The problem

then amounts to an iterative calculation. To perform this numerical calculation, we need:

1. The duration of the numerical calculation;
2. The initial conditions s(0), i(0) and r(0);
3. The discretization step $\Delta t = 1$ time unit in our case.

$$\begin{cases} s(t+1) = s(t) - \beta\langle k\rangle s(t)i(t) \\ i(t+1) = i(t)(1 + \beta\langle k\rangle s(t) - \gamma) \quad (4) \\ r(t+1) = r(t) + \gamma i(t) \end{cases}$$

Algorithm 3: SIR-Small World model
Input: G(t), k, β, γ
Output: i(t)
BEGIN
Time: t ⟵ t_0;
i(t) = 0;
Initially: infect some nodes;
While i(t)≠ 0 do

    i increases by β⟨k⟩s(t)i(t) with

$$\langle k\rangle = \sum k\,P_k$$

    i decreases by $\gamma i(t)$;
    s decreases byβ⟨k⟩s(t)i(t);
    r increases by $\gamma i(t)$;
    t = t +1;
    G(t) evolves to G(t +1);
end
Return i(t)
END

3.4 The Basic Reproduction Rate: R_0

The dynamics of person-to-person transmis (5) vector transmission can be summarized by two main parameters: reproduction rate and serial or intergenerational interval [7]. A parameter of interest of the proposed model is the reproduction rate R_0, which is interpreted as the average number of new cases generated by an infectious subject in a susceptible population. This parameter is very important in the study of the spread of epidemics, it makes possible the evaluation of an infectious disease, for exa (6) the monitoring of the evolution of virule case of maximization of the value of R_0 and, consequently, to predict the evolution of the epidemic over time. The more the basic (7) reproduction number R_0 increases the more the epidemic spreads. In the case of a seasonal influenza epidemic, this parameter is measured between$R_0 = 1$, and $R_0 = 2$ [23]. To calculate R_0 we use the next generation (8)

matrix FV^{-1} [24]. Mathematically the basic reproduction number is defined as:

$$R0 = \rho(FV^{-1})$$

Where, F is the nonnegative matrix, of the new infection terms, and V is the M-matrix, of the transition terms associated with the model (2). We are interested in working only with positive orthant since the solutions are positive. The system has a unique Disease Free Equilibrium which is obtained by setting i = 0, given by:

$$DFE = (s^*, 0, 1 - s^*)$$

From the system we have:

$$F = \beta\langle k\rangle s^* \text{ and } V^{-1} = -\gamma$$

Hence the basic reproduction number is given by:

$$R_0 = \frac{\beta\langle k\rangle s^*}{\gamma}$$

In the case of R0 > 1, the introduction of an infectious agent into a susceptible population triggers an epidemic. Otherwise if R0 ≤ 1, the epidemic goes out, so the stability of the DFE.

This is the famous threshold theorem, the threshold concept was used by Ross elementally in his" mosquito theorem" [25] and was developed to the famous threshold theorem of Kermackand McKendrickin 1927 [26]. In our study, we calculated the value R0 =0,352. This means that the state of equilibrium of the epidemic wave of influenza is endemic.

4. SIMULATION AND VALIDATION

4.1 Methods And Results

The database used in this study is for the 2004and 2005 seasonal influenza illness in the United States. This is an epidemiological database obtained from the CDC Centers for Disease Control and Prevention. It consists of 157,759 samples that have been tested in people with influenza-like symptoms. The first week, N = 0, corresponds to the last week of September [27], as represented in figure 4.
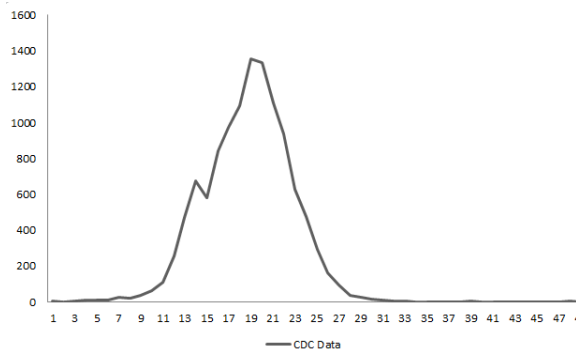

Figure 4: The Evolution Of The Number Of Infected With Influenza.
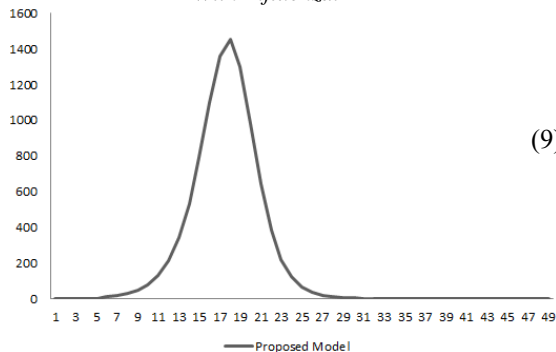

Figure 5: The Evolution Of The Number Of Infected According To The Proposed Model.

To validate our model, we performed simulation experiments with the recommended parameters [13], figure 5. We compare the evolution of the incidence curves obtained by our model and the influenza data for the epidemic wave. A statistical analysis was applied to validate the proposed model. This is an important step in any model construction process. We used linear correlation analysis and the Kolmogorov-Smirnov test to compare observed (real) data with numerical simulations.

4.2 VALIDATION TESTS

4.2.1 Correlation

Correlation is widely used in science as a measure of the degree of linear dependency between two variables. In this work, linear correlation is used to measure the degree of similarity between the simulated data obtained by our model and the observed (real) data, Correlation coefficient= 0,88371. We find that the proposed model fits precisely the dynamic behavior of observed data used in our study.

4.2.2 The kolmogorov-smirnov test

The Kolmogorov-Smirnov test is a non-parametric matching test used to determine whether a sample follows a given law known by its continuous distribution function F(x) or whether two samples follow the same law. Indeed, it is an adjustment test to check whether the observed data are compatible with a given theoretical model. Let $x_1,x_2,... ,x_n$, a sequence of n realizations of the random variable x independent and identically distributed. We assume the following hypotheses:

H_0: x follows the law of F against H_1: x follows another law ie. F = F0 against F ≠ F0.

The Kolmogorov-Smirnov test is defined by the test statistic:

$$\boldsymbol{Dmax} = \boldsymbol{Sup}|\boldsymbol{F_n(x)} - \boldsymbol{F_0(x)}|/\boldsymbol{x} \in \mathbb{R} \tag{9}$$

It consists in rejecting the hypothesis H_0 if $\boldsymbol{Dmax} \geq \boldsymbol{Dn(n,a)}$. In other words, the larger the test statistic D, the more likely it is to reject H_0. Thus, the Kolmogorov-Smirnov test for a sample size (n)at a level of significance (a) has a critical value of $\boldsymbol{Dn(n,a)}$. In our study, we apply the Kolmogorov-Smirnov test for simulated data and actual data, forthe influenza wave. Then we

will test the hypothesisfor the distributions of the two sets of data to be identical, ie, both functions have the same distribution functions, which confirms the null hypothesis (H₀). The statistic test, here designated $\boldsymbol{Dmax}$, was the maximum deviation between the cumulative proportions of the two functions, $\boldsymbol{Dmax} = \boldsymbol{0,03663}$ $\boldsymbol{Dn(n,a)} = \boldsymbol{0,285}$.Indeed, $\boldsymbol{Dmax} < \boldsymbol{0,285}$, the null hypothesis (H₀) is accepted at a level of 5% confidence. Both distributions are compliant.

4.2.3 Analysis of the model

This section showshow the degree of distribution $\langle \boldsymbol{k} \rangle$ could directlyinfluence the evolution of a disease in a social network.The following figure 6 shows the results of thesimulation of the proposed model with different k.
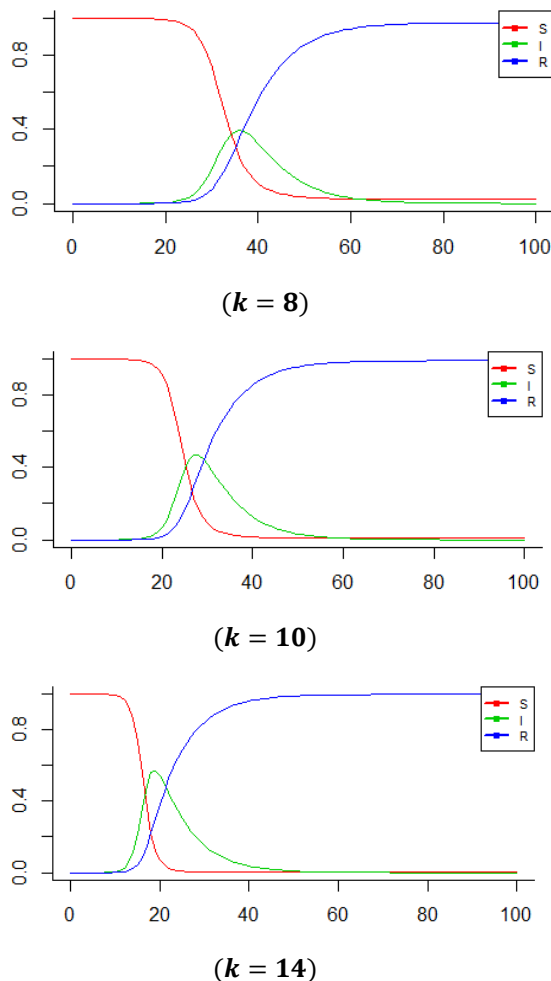

$(\boldsymbol{k} = \boldsymbol{8})$

$(\boldsymbol{k} = \boldsymbol{10})$

$(\boldsymbol{k} = \boldsymbol{14})$

Figure 6: Simulation Of The SIR-Small Worldmodel With Different K.

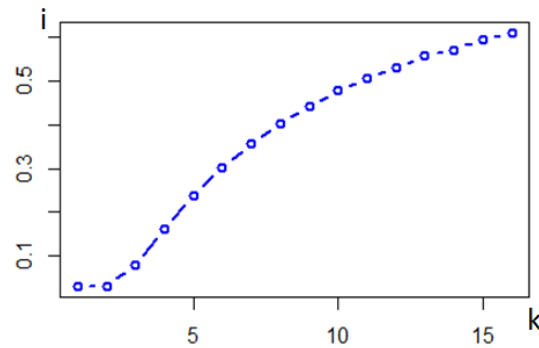*Figure 7: Curve Of I As A Function Of K.*

By comparing the above figures, we notice that the average degree distribution $\langle \boldsymbol{k} \rangle$ has a significant impact on the topology of the network. When the value of this factor increases, the structure of the network becomes more complex. We also observe that the epidemic spreads rapidly in densely connected groups of individuals as illustrated in figure 7. Therefore, within asocial network, the number k must be kept low because the disease spreads faster if this value of degree becomes important. Decision makers and public health professional scan simulate different epidemic scenarios using this model. This allows them to understand the mechanism of spread and to predict the final outcome of the disease by modifying the parameters of the epidemic and the state variables.

5. CONCLUSION

In this work we propose a new algorithm that includes not only the biological aspect but also the social aspect of the population. Our new algorithm allows us to better understand the evolution of the epidemic propagation process and indicates the importance of looking at the dynamics of the disease in real world networks. Our algorithm can, if necessary, support populations structured in age, metapopulations, etc. It also constitutes a platform supporting improvements such as the study of an epidemic of several strains. The results of this algorithm will be very beneficial in analyzing propagation behaviors and in helping health professionals improve understanding of dissemination mechanisms in order to develop effective strategies for confining actual epidemics in the future.

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