

Classic And Network Epidemiological Models

Modelos Epidemiológicos Clásicos Y Sobre Redes

C. C. Cortes  ; J. D. Hernández 
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 Artículo de investigación científica y tecnológica

Abstract— In this work is analyzed the environment and the dynamics of the states for a disease within a constant and closed population, represented by a system of ordinary differential equations, in which the individual, besides having the same opportunity to get in contact with any other, can recover or not, acquiring or not immunity through time. With these defined guidelines, the conditions when the disease spreads over time between such models are compared with those represented by a network. As the network can be represented by an adjacency matrix, the dynamics in the epidemiological states depends, besides the conditions in their parameters of the classic models, on largest eigenvalue of such matrix.

Index Terms— Adjacency matrix, epidemic, epidemic threshold, network k -regular.

Resumen—En este trabajo se analiza el entorno y la dinámica de los estados para una enfermedad dentro de una población constante y cerrada, representado por un sistema de ecuaciones diferenciales ordinarias, en que el individuo, además de tener la misma oportunidad de entrar en contacto con cualquier otro, se pueda o no recuperar, adquiriendo o no inmunidad a través del tiempo. Con estos lineamientos definidos, se compara las condiciones cuando la enfermedad se propaga a lo largo del tiempo entre dichos modelos con los representados por una red. Como la red puede ser representado por una matriz de adyacencia, la dinámica en los estados epidemiológicos depende, además de las condiciones en sus parámetros de los modelos clásicos, del valor propio más grande de dicha matriz.

Palabras claves— Epidemia, grafo k -regular, matriz de adyacencia, umbral epidémico.

I. INTRODUCTION

THE epidemiology of viral diseases is a discipline that deals with the study of determinants, predictions and control of factors related to health and disease, as well as

the study of the dynamics and distribution of viral diseases in a population [1].

In order to establish the dynamics of these diseases and to carry out a pursuit or control to these, it is made use of mathematical tools, like the ordinary differential equations, which allows to establish relations between these behaviors by means of variables and parameters or factors that influence the development or extinction of the disease.

There are various mathematical models that explain the dynamics of their epidemiological states by assuming that each individual has the same opportunity to come into contact with any other individual in the population. For example, for diseases such as HIV, there are two types of states: those who have already contracted the disease and remain infected and infectious to others, and those who are susceptible to contracting it because they are in a risk zone. This type of model is known as the IS model and is characterized by the fact that the individual acquires the virus from an infected person without acquiring immunity [2, 3]. Similarly, for diseases such as influenza, can be described by the SIS model [4, 5], which gives way to the individual can move from an infected state to susceptible and be prone to acquire the disease again. For diseases in which the individual acquires immunity are explained by SIR models [6].

One of the major challenges is the spread of diseases for people who are associated with a limited number of individuals in a population, as millions of people now cross the borders of many countries every day, increasing the likelihood of an epidemic or pandemic, as well as the invasion of ecosystems and environmental degradation that can create opportunities for existing and new infectious diseases. Therefore, one of the solutions to this difficulty is to fit an epidemiological model, such as the case of the IS, SIS or SIR, into a network when few infected individuals enter.

Therefore, the aim of this work is to analyze the dynamics of SI, SIS and SIR models, adjusted or not in a network, and to compare the necessary conditions to locate or not the disease in a constant and closed population along the time. In order to understand this process, the following steps are developed: in the second section the classical SI, SIS and SIR models are analyzed. In the third, fourth and fifth section, the construction of the SI, SIR and SIS models in networks, respectively, is shown and an analysis of the short- and long-term dynamics is made to determine the conditions in which

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C.C. Cortes. Mathematician and Master in Mathematics. Universidad Carlos III de Madrid, Spain. E-mail: chcortes@uc3m.math.es

J. D. Hernandez. Mathematician and Master in Applied Mathematics. Currently he is an occasional professor at the Department of Mathematics and Statistics at the Universidad Surcolombiana, Colombia.

E-mail: juan.hernandez@usco.edu.co



the disease spreads or not and to compare them with the conditions of the models described given in the second section. Similarly, construction methods are shown to

II. CLASSIC EPIDEMIOLOGICAL MODELS

By assuming that the nodes and their edges in a network do not present variations over time, a qualitative analysis is then made to the main epidemiological models, represented by a system of ordinary differential equations, without considering birth or natural death rates due to disease.

A. Model SI

Consider a population with N individuals, constant and closed over time, divided into two states, infected when in contact with an infected and susceptible to this. Let $S(t) \geq 0$ and $X(t) \geq 0$ the number of susceptible and infected individuals, respectively, at any one time $t \geq 0$, so that $S(t) + X(t) = N$.

As seen in Fig. 1 **grafo k-regular.**, if $\beta > 0$ represents the rate of infection, and by assuming that the individual has the same opportunity to come into contact with any other individual in the population, the change in X with respect to t is represented by,

$$\frac{dX}{dt} = \frac{\beta SX}{N},$$

where $\frac{\beta SX}{N}$ is the flow of infection between healthy and infectious individuals per unit of time.

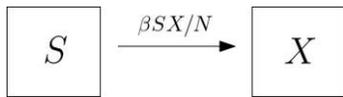


Fig. 1. Construction of (1).

Since $S(t) + X(t) = N$, then change of S per unit of time is given by

$$\frac{dS}{dt} = -\frac{\beta SX}{N}.$$

Thus, the final model is

$$\begin{cases} \frac{dS}{dt} = -\frac{\beta SX}{N} \\ \frac{dX}{dt} = \frac{\beta SX}{N} \end{cases} \quad (1)$$

If $s(t) = \frac{S(t)}{N}$ and $x(t) = \frac{X(t)}{N}$ represent the fractions of susceptible and infected individuals at the time $t \geq 0$ respectively, then $s(t) + x(t) = 1$ and (1) is equivalent to

$$\begin{cases} \frac{ds}{dt} = -\beta sx \\ \frac{dx}{dt} = \beta sx \end{cases} \quad (2)$$

approximate model solutions on the network due to the complexity of finding an analytical solution.

Due to the biological interest in analyzing the dynamics of $x(t)$ since the beginning of the disease $t = 0$. Consider $X(0) := X_0 \leq N$ y $S(0) = N - X_0$ the number of infected and susceptible individuals, respectively, equivalent to $x(0) = x_0 := \frac{X_0}{N}$ and $s(0) = 1 - x_0$ as initial conditions of (2).

Since $s(t) = 1 - x(t)$, from (2) we have

$$\frac{dx}{dt} = \beta x(1 - x), \quad (3)$$

that is,

$$\frac{1}{x(1 - x)} \frac{dx}{dt} = \beta. \quad (4)$$

When integrating with respect to t for both sides of (4), we have to

$$\ln\left(\frac{x}{1 - x}\right) = \beta t + c,$$

where $c \in \mathbb{R}$ is a constant of integration. When considering $x(0) = x_0$ the particular solution of (3) en

$$x(t) = \frac{x_0 \exp(\beta t)}{1 + x_0 (\exp(\beta t) - 1)}.$$

An important question in any epidemic is whether or not the infection spreads over time $t \geq 0$. In the case of propagation, we must determine how it develops over time or when it will start to diminish. Indeed, as

$$\lim_{t \rightarrow \infty} x(t) = \lim_{t \rightarrow \infty} \frac{x_0 \exp(\beta t)}{1 + x_0 (\exp(\beta t) - 1)} = 1,$$

for everything $x_0, \beta > 0$ from (2) an epidemic is shown to always spread and eventually infect all susceptible individuals if $x_0 > 0$. However, if $x_0 = 0$ then $x(t) = 0$ for everything $t \geq 0$, that is, the population will remain in a susceptible state. Similarly, for $x_0 > 0$ fixed, $x(t)$ converge to 1 with a faster propagation speed each time $\beta \gg 0$ as seen in Fig.2.

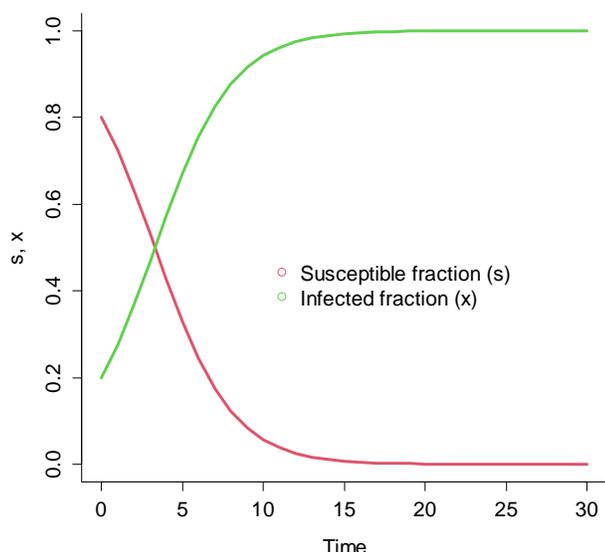


Fig. 2. Dynamics of (2) with $\beta = 0.42$ and $x_0 = 0.2$.

Therefore, the following result has been verified.

Theorem 1 Let $(s(t), x(t)) \in \mathbb{R}_+^2$ a solution of (2) with $\beta > 0$ and initial condition $(s(0), x(0)) = (1 - x_0, x_0)$. Then $x(t) \rightarrow 1$ when $t \rightarrow \infty$ while $s(t) \rightarrow 0$ for $t \rightarrow \infty$, that is, $X(t) \rightarrow N$ when $t \rightarrow \infty$ y $S(t) \rightarrow N$ for $t \rightarrow \infty$.

B. SIR Model

Unlike the SI model, consider that the population at the time $t \geq 0$ is divided into three stationary, susceptible states $S(t) \geq 0$, infected by the disease $X(t) \geq 0$ and recovered without acquiring the disease again $R(t) \geq 0$.

In view of Fig.3 and as stated [7], suppose that the susceptible fraction $s(t) = \frac{S(t)}{N}$ which becomes an infectious fraction $x(t) = \frac{X(t)}{N}$ is proportional to the product of its fractions, that is, the rate of loss of the susceptible fraction is βsx , where $\beta > 0$ is the infection rate. Therefore, the change in the susceptible fraction $s(t)$ regarding time is given by,

$$\frac{ds}{dt} = -\beta sx,$$

where the negative sign represents the loss of the susceptible fraction.

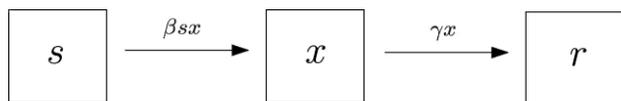


Fig. 3. Construction of (6).

Since βsx also indicates the gain rate of the infectious fraction, and $\gamma > 0$ represents the rate of profit of the recovered fraction, i.e. γx indicates the exit of the infectious fraction, the change of x with respect to time is represented by

$$\frac{dx}{dt} = \beta sx - \gamma x,$$

and the equation that describes the change of the recovered fraction $r(t) = \frac{R(t)}{N}$ es,

$$\frac{dr}{dt} = \gamma x.$$

As the total population N was divided into three states, susceptible, infectious and recovered, you have to $S(t) + X(t) + R(t) = N$ equivalent to $s(t) + x(t) + r(t) = 1$ and therefore the model is represented by

$$\begin{cases} \frac{ds}{dt} = -\beta sx \\ \frac{dx}{dt} = \beta sx - \gamma x \\ \frac{dr}{dt} = \gamma x. \end{cases} \quad (6)$$

The objective is to determine the conditions in which the epidemic spreads or not from $X(0) = X_0 \leq N$, $S(0) = N - X_0$, and $R(0) = 0$, equivalent to $x(0) = x_0 := \frac{X_0}{N}$, $s_0 = 1 - x_0$ and $r_0 = 0$.

From (6) we can be seen that $\frac{ds}{dt} \leq 0$, that is, the susceptible fraction $s(t)$ will decrease as long as there are infectious individuals, and therefore $s(t) \leq s_0$ for everything $t \geq 0$. Similarly, $\frac{dr}{dt} \geq 0$, therefore $r_0 \leq r(t)$, for everything $t \geq 0$, if there are infectious individuals.

On the other hand,

- If $s_0 \leq \frac{\gamma}{\beta}$, this is $s(t) \leq \frac{\gamma}{\beta}$, then $\frac{dx}{dt} = x(\beta s - \gamma) \leq 0$ and $x_0 > x(t) \rightarrow 0$ when $t \rightarrow \infty$, this is, the fraction of infected people decreases.
- If $\frac{dx}{dt} > 0$ when $t_0 > 0$, then $\left. \frac{dx}{dt} \right|_{t=0} = x_0(\beta s_0 - \gamma) > 0$ if $s_0 > \frac{\gamma}{\beta}$. Therefore, the number of infected people will increase and there will be an epidemic. So, for some $t > 0$ there will be an epidemic outbreak if $x(t) > x_0$.

Since the first two equations of (6) do not depend on $r(t)$, then

$$\frac{dx}{ds} = \frac{\beta sx - \gamma x}{-\beta sx} = -1 + \frac{\gamma}{\beta s}, \quad (7)$$

which $\frac{dx}{ds} < 0$ if $s > \frac{\gamma}{\beta}$ and $\frac{dx}{ds} > 0$ if $s < \frac{\gamma}{\beta}$.

By integrating (7) with respect to s , we have to

$$x(s) = -s + \frac{\gamma}{\beta} \ln s + c, \quad (8)$$

where $c \in \mathbb{R}$ is a constant of arbitrary integration.

Considering the initial conditions x_0 y s_0 at (8) we have

$$c = x_0 + s_0 - \frac{\gamma}{\beta} \ln s_0,$$

and therefore the dynamics of (6) on the plane (s, x) in initial condition $x(0) = x_0$ y $s(0) = s_0$ is given by

$$x(s) = x_0 + s_0 - s + \frac{\gamma}{\beta} \ln \left(\frac{s}{s_0} \right). \quad (9)$$

From (9) you must $x(s) = -\infty$ y $x(s_0) = x_0 > 0$ which there is a point s_∞ such that $x(s_\infty) = 0$ with $0 < s_\infty < s_0$. Note that $\frac{ds}{dt}(s_\infty, 0, 1 - s_\infty) = \frac{dx}{dt}(s_\infty, 0, 1 - s_\infty) = \frac{dr}{dt}(s_\infty, 0, 1 - s_\infty) = 0$, that is, the point $(s_\infty, 0, 1 - s_\infty)$ corresponds to a balance of (6).

On the other hand, the maximum number of the infectious fraction, at any moment of time, satisfies $\frac{dx}{dt} = 0$ when $x \neq 0$. From (6) you must $\frac{dx}{dt} = \beta x \left(s - \frac{\gamma}{\beta} \right) = 0$ if $s = \frac{\gamma}{\beta}$ for $x > 0$. When replacing the value of s in (9) you have to

$$x_{max} = x_0 + s_0 - \frac{\gamma}{\beta} + \frac{\gamma}{\beta} \ln \left(\frac{\gamma}{s_0 \beta} \right),$$

as seen in Fig.4.

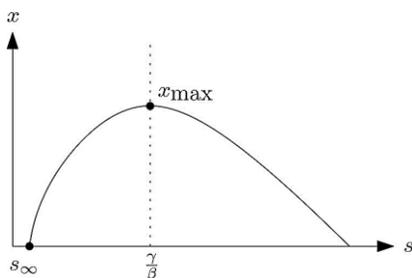


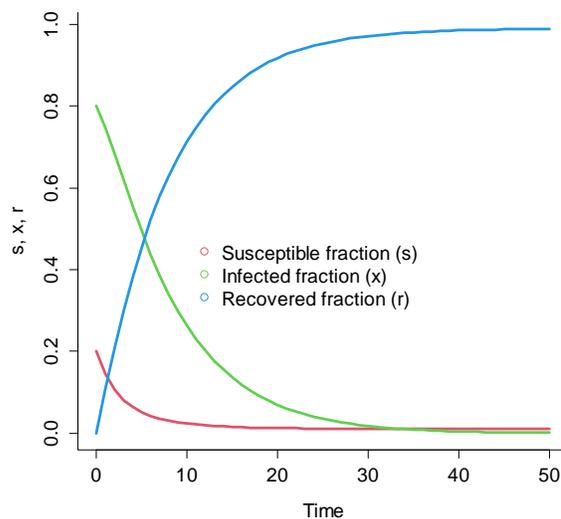
Fig. 4. Function (8).

Note that, from Fig.4, if $\frac{\gamma}{\beta} > 1$ then $\frac{\gamma}{\beta} > s_0$ for everything $0 \leq s_0 \leq 1$ and so $x(t) \rightarrow 0$ when $t \rightarrow \infty$. Therefore, the following result shows the dynamics of the model (6) with respect to the relationship between the parameters $\beta, \gamma > 0$ and its initial conditions.

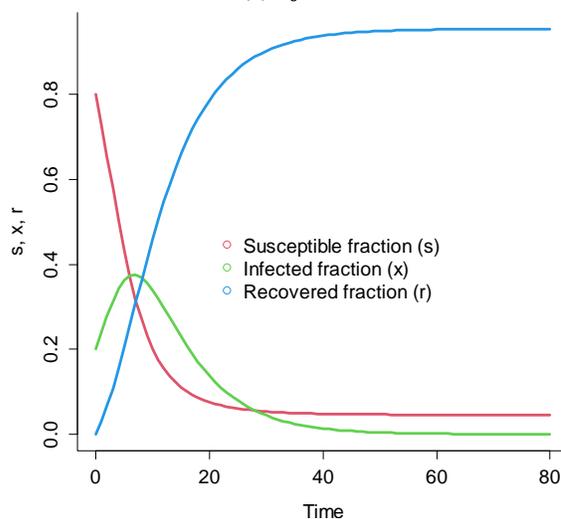
Theorem 2. Let $(s(t), x(t), r(t)) \in \mathbb{R}_+^3$ a solution of (6) with initial condition $(s(0), x(0), r(0)) = (x_0, 1 - x_0, 0)$. If $\frac{\gamma}{\beta} > 1$, $x(t) \rightarrow 0$ when $t \rightarrow \infty$. For $\frac{\gamma}{\beta} < 1$, if $s_0 \leq \frac{\gamma}{\beta}$ then $x(t) \rightarrow 0$ when $t \rightarrow \infty$ and, if $s_0 > \frac{\gamma}{\beta}$ then $x(t)$ first increases until a maximum value is reached $x_{max} = x_0 + s_0 - \frac{\gamma}{\beta} + \frac{\gamma}{\beta} \ln \left(\frac{\gamma}{s_0 \beta} \right)$ and then decreases to zero when $t \rightarrow \infty$. The susceptible fraction $s(t)$ is a decreasing function and the limit s_∞ is the only root in $(0, \frac{\gamma}{\beta})$ of the equation

$$x_0 + s_0 - s_\infty + \frac{\gamma}{\beta} \ln \left(\frac{s_\infty}{s_0} \right) = 0.$$

Fig.5 shows the dynamics of (6) for $\frac{\gamma}{\beta} < 1$ fixed and various initial conditions $(x(0), s(0), r(0)) = (x_0, 1 - x_0, 0)$.



(a) $s_0 = 0.2$



(b) $s_0 = 0.8$

Fig. 5. Solution of (6) with $\beta = 0.42$ and $\gamma = 0.14$.

C. SIS Model

This model extends the IS model by considering that individuals can recover but do not acquire immunity to the disease. In this case, consider $s(t)$ y $x(t)$ as the susceptible and infected fraction, respectively, at the time $t \geq 0$ y $s(t) + x(t) = 1$.

Looking at Fig.6, the following model is proposed

$$\begin{cases} \frac{ds}{dt} = \gamma x - \beta s x \\ \frac{dx}{dt} = \beta s x - \gamma x \end{cases} \quad (10)$$

where $\beta > 0$ is the rate of infection and $\gamma > 0$ recovery rate, with initial conditions $x(0) = x_0$ y $s_0 = 1 - x_0$.

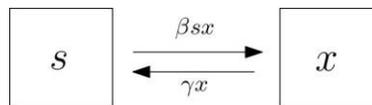


Fig. 6. Construction of (10).

Since $s(t) + x(t) = 1$ for everything $t \geq 0$, the change in the fraction of infected people with respect to time takes the form of

$$\frac{dx}{dt} = \beta x(1 - x) - \gamma x, \tag{11}$$

than by the method of separation of variables, with initial conditions $x(0) = x_0$ y $s(0) = 1 - x_0$, the particular solution of (11) is given by

$$x(t) = \frac{\beta - \gamma}{\beta} \left[\frac{x_0 \exp(t(\beta - \gamma))}{x_0 \exp(t(\beta - \gamma)) + 1 - \frac{\gamma}{\beta}} \right].$$

Therefore, we have the following result,

Theorem 3. Be $(s(t), x(t)) \in \mathbb{R}_+^2$ the solution of (10) with initial condition $x(0) = x_0$ and $s(0) = 1 - x_0$. If $\frac{\beta}{\gamma} < 1$ then $x(t) \rightarrow 0$ when $t \rightarrow \infty$, which tends to disappear to $t \geq 0$. If $\frac{\beta}{\gamma} > 1$ then

$$\lim_{t \rightarrow \infty} x(t) = \lim_{t \rightarrow \infty} \frac{\beta - \gamma}{\beta} \left[\frac{x_0 \exp(t(\beta - \gamma))}{x_0 \exp(t(\beta - \gamma)) + 1 - \frac{\gamma}{\beta}} \right] = \frac{\beta - \gamma}{\beta},$$

and so the disease does not disappear over time.

Fig.7 shows the dynamics of (10) for various conditions on the $\beta, \gamma > 0$.

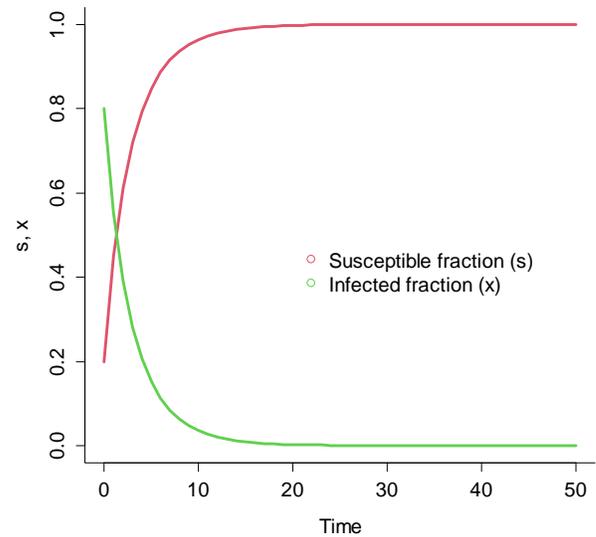
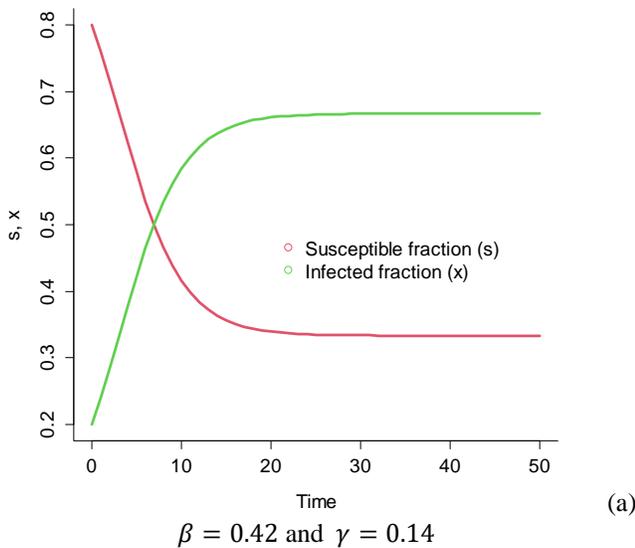


Fig. 7. Computer solution of (10).

III. SI MODEL IN A NETWORK

Consider a network between n individuals, represented by a network G , where the population is represented by nodes and the contact between individuals is represented by edges. This network can be represented by an adjacency matrix $A = [A_{ij}]$ size $n \times n$ which represents the number of edges for each pair of nodes.

Consider that each node i in the moment $t \geq 0$ belongs to an infected state $X(t)$ by a disease or a susceptible state $S(t)$. That is, for a node i arbitrary, $x_i(t) = \text{Prob}(i \in X(t))$ or $s_i(t) = \text{Prob}(i \in S(t))$, where $x_i(t) + s_i(t) = 1$.

If each node i is connected by a neighbor j , the change in probability of an infected node x_i with respect to time is given by

$$\frac{dx_i}{dt} = \beta x_i \sum_{j=1}^n A_{ij} s_j, \tag{12}$$

where $\beta x_i(t) s_j(t)$ is the flow of infection between the infected node $x_i(t)$ and every susceptible neighbor $s_j(t)$ from i in the moment $t \geq 0$ [8].

Since $s_i(t) + x_i(t) = 1$ for everything $t \geq 0$, the change in the probability of a node i susceptible s_i with respect to time is represented by

$$\frac{ds_i}{dt} = -\beta s_i \sum_{j=1}^n A_{ij} x_j.$$

Therefore, the model to be considered is

$$\begin{cases} \frac{ds_i}{dt} = -\beta s_i \sum_{j=1}^n A_{ij} x_j \\ \frac{dx_i}{dt} = \beta s_i \sum_{j=1}^n A_{ij} s_j \end{cases} \tag{13}$$

equivalent to

$$\frac{dx_i}{dt} = \beta x_i \sum_{j=1}^n A_{ij} s_j = \beta x_i \sum_{j=1}^n A_{ij} (1 - x_j) \quad (14)$$

or

$$\frac{ds_i}{dt} = -\beta s_i \sum_{j=1}^n A_{ij} x_j = -\beta s_i \sum_{j=1}^n A_{ij} (1 - s_j). \quad (15)$$

From (13) we can be seen that $\frac{ds_i}{dt} \leq 0$, that is, s_i is decreasing and converge to zero when infectious nodes exist x_j and, x_i is growing and converge to one as there are susceptible nodes s_j . Therefore, the dynamic (13) is equivalent to the dynamic in (2).

Analogous to the initial conditions of the classical SI model, suppose the disease starts with an infected node or a $c \geq 1$ of nodes, chosen at random, such that $x_i(0) = \frac{c}{n}$ and $s_i(0) = 1 - \frac{c}{n}$.

Since $x_i(t)$ in (14) converge to one, the behavior of the system must be analyzed for a short time to determine its propagation speed. Indeed, if t is close to zero, $x_i(t) \approx 0$ and $s_i(t) \approx 1$ for n big enough. Thus, from (14) and ignoring the terms of quadratic order, we have

$$\frac{dx_i}{dt} = \beta \sum_{j=1}^n A_{ij} x_j,$$

equivalent, in matrix form, to

$$\frac{dx}{dt} = \beta Ax, \quad (16)$$

where $x = (x_1, x_2, \dots, x_n)^T$.

If $x(t)$ represented as a linear combination of the own vectors v_r , $1 \leq r \leq n$ associated to the own values $\lambda_1 \leq \lambda_2 \leq \dots \leq \lambda_n$ of the matrix A , this is $Av_r = \lambda_r v_r$,

$$x(t) = \sum_{r=1}^n a_r(t) v_r, \quad (17)$$

where $a_r(t)$ are constants that depend on t , of (16) we have,

$$\begin{aligned} \frac{dx}{dt} &= \sum_{r=1}^n \frac{da_r}{dt} v_r = \beta Ax = \beta A \sum_{r=1}^n a_r(t) v_r = \beta \sum_{r=1}^n a_r(t) Av_r \\ &= \beta \sum_{r=1}^n \lambda_r a_r(t) v_r. \end{aligned}$$

Then,

$$\frac{da_r}{dt} = \beta \lambda_r a_r,$$

with particular solution

$$a_r(t) = a_r(0) \exp(\beta \lambda_r t) \quad (18)$$

By replacing (18) in (17),

$$x(t) = \sum_{r=1}^n a_r(0) \exp(\beta \lambda_r t) v_r \sim \exp(\beta \lambda_n t) v_n$$

and therefore, it is expected that the solution $x(t)$ grows exponentially from short moments of time and, unlike the conclusions in the dynamics of the classical SI model given in Theorem 1, the growth $x(t)$ depends on λ_n and $\beta > 0$. That is, if $\beta \geq 0$, the disease spreads more slowly if the network G is more dispersed, that is, if $\lambda_n \geq 0$ and, for denser networks, this is, $\lambda_n \gg 0$, the disease spreads more quickly.

Fig.8 shows some simulations of (13), made in Matlab, in a network k -regular [9], i.e. with k neighbors for each node i . There is evidence that the contagion spreads throughout the network to t increasingly shorter as the number of neighbors increases.

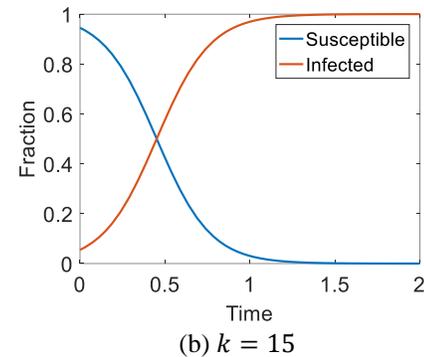
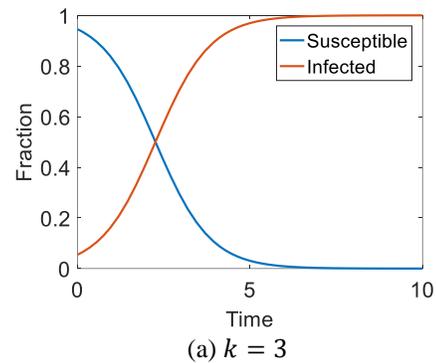


Fig. 8. Fraction of infected and susceptible nodes for a network k -regular with $x_i(0) = 0.055$ in all cases, $\beta = 0.42$ and $n = 10^5$ nodes.

A. Model based on the degree of a node

Since the model (13) cannot be solved analytically, a model must be fitted to approximate the solutions of the SI model in a network whose dynamics in which its states are explained by the degrees of the nodes in the network G [8].

As seen in Fig.9, a network is said to have L components if any L subgraph of G , and the largest component of G is the subgraph L that has the largest number of edges.

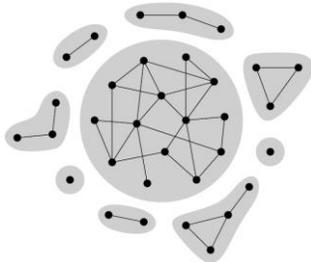


Fig. 9. Components of a G-network.

Since the degree of a node is given by the number of its neighbors, consider $\{p_k\}$ as the distribution in degrees of a network G , where p_k is the fraction of nodes that have degree k , and suppose that the infected nodes make contacts independently of each other, i.e., $\sum_{k=0}^{\infty} p_k = 1$. Consider the probability generating function given by

$$g_0(z) = \sum_{k=0}^{\infty} p_k z^k.$$

The average grade $\langle k \rangle$ is given by

$$\langle k \rangle = \sum_{k=0}^{\infty} k p_k = g'_0(1).$$

More generally, we define the moments

$$\langle k^j \rangle = \sum_{k=0}^{\infty} k^j p_k, \quad j = 1, 2, \dots, \infty.$$

As reference [10], when a node in a network is infected, the infection is transmitted to every connected individual except the edge from which it came. Therefore, the excess grade of a node that is one less than the grade is used. The probability of reaching a node of degree k or excess degree $(k - 1)$, when following a random edge, it is proportional to k , and therefore the probability that a node at the end of a random edge has a degree of excess $(k - 1)$ is a constant multiple of $k p_k$ with the constant chosen to make the sum over k of the probabilities is equal to 1. So the probability of a node having an excess of degree $(k - 1)$ en

$$q_{k-1} = \frac{k p_k}{\langle k \rangle},$$

where

$$\sum_{k=1}^{\infty} q_{k-1} = \sum_{k=0}^{\infty} q_k = 1.$$

This leads to a generating function $g_1(z)$ for excess grade,

$$\begin{aligned} g_1(z) &= \sum_{k=0}^{\infty} q_k z^k = \sum_{k=1}^{\infty} q_{k-1} z^{k-1} = \sum_{k=0}^{\infty} \frac{k p_k}{\langle k \rangle} z^{k-1} \\ &= \frac{1}{\langle k \rangle} g'_0(z). \end{aligned} \tag{19}$$

and the excess of medium grade, denoted by $\langle k_e \rangle$,

$$\begin{aligned} \langle k_e \rangle &= \frac{1}{\langle k \rangle} \sum_{k=1}^{\infty} k(k-1) p_k \\ &= \frac{1}{\langle k \rangle} \sum_{k=1}^{\infty} k^2 p_k - \frac{1}{\langle k \rangle} \sum_{k=1}^{\infty} k p_k \\ &= \frac{\langle k^2 \rangle}{\langle k \rangle} - 1 = g'_1(1). \end{aligned} \tag{20}$$

Note that $g'_1(1) > 1$ in the largest component of G if $\sum_{k=3}^{\infty} (k^2 - 2k) p_k > p_1$, that is, the largest component of G has fewer grade 1 connections.

If a node i is infected and belongs to a small component with few or no connections in G , the probability that the infected node will spread throughout the network is zero. Therefore, an SI model will be adjusted for a large component of G such that $g'_1(1) > 1$. Consider x_k as the probability of a grade neighbor k is infected, and the degree of excess is distributed according to q_k . Then, the average probability that the neighbor is infected is

$$v(t) = \sum_{k=1}^n q_k x_k(t) = \sum_{k=0}^n q_k (1 - s_k(t)), \tag{21}$$

where $x_k + s_k = 1$.

If the node's neighbor i is infected, the probability of the disease being transmitted to the i in the given time interval is βdt . Then the total probability of transmission from a single neighbor during the time interval is $\beta v(t) dt$ and the probability of transmission from any neighbor is $\beta k v(t) dt$ where k is the number of neighbors of i . Furthermore, i is required to be susceptible, which occurs with probability $s_k(t)$, so the final probability that i get infected is $\beta k v(t) s_k dt$ [8]. Therefore, the change of s_k is given by

$$\frac{ds_k}{dt} = -\beta k v s_k, \tag{22}$$

with particular solution in $s_k(0) = s_0$,

$$s_k(t) = s_0 \exp\left(-\beta k \int_0^t v(w) dw\right). \tag{23}$$

If we consider

$$u(t) = \exp\left(-\beta k \int_0^t v(w) dw\right), \tag{24}$$

(23) takes the form

$$s_k(t) = s_0 [u(t)]^k. \tag{25}$$

To calculate s_k an equation must be constructed in terms of u without being dependent on s_k as observed in (24). In effect, from (22) and (25) we have

$$k s_0 u^{k-1} \frac{du}{dt} = \frac{ds_k}{dt} = -\beta k v s_0 u^k,$$

equivalent to

$$\frac{du}{dt} = -\beta u v.$$

When we consider that $\sum_{k=0}^{\infty} q_k = 1$, (21) is equivalent to

$$v(t) = \sum_{k=0}^{\infty} q_k(1 - s_k) = \sum_{k=0}^{\infty} q_k(1 - s_0 u^k) \tag{26}$$

$$= 1 - s_0 g_1(u).$$

Therefore,

$$\frac{du}{dt} = -\beta u[1 - s_0 g_1(u)], \tag{27}$$

is used to determine the values of u .

Finally, to calculate the total fraction $x(t)$ of infected individuals in the network, is averaged over k in this way

$$x(t) = \sum_{k=0}^{\infty} p_k x_k(t) = \sum_{k=0}^{\infty} p_k(1 - s_k(t)) = \sum_{k=1}^{\infty} p_k(1 - s_0 u^k)$$

$$= 1 - s_0 g_1(u).$$

However, the solution of (27) cannot be explicitly calculated, but an analysis can be made for both short- and long-term times. Indeed, when $t = 0$, of (24) must be $u(0) = 1$. Then, $v(t)$ is, by definition, positive and not decreasing, of (24), $u(t) \rightarrow 0$ when $t \rightarrow \infty$. By assuming that the infection starts with only one or a handful of individuals, so $s(0) = s_0 = 1 - \frac{c}{n}$ for some constant $c > 0$, we have $s_0 \rightarrow 1$ when $n \rightarrow \infty$, this implies that, in the long term, (27) it becomes

$$\frac{du}{dt} = -\beta u[1 - g_1(0)] = -\beta u \left(1 - \frac{s_0 p_1}{\langle k \rangle}\right),$$

with general solution,

$$u(t) = K \exp \left[-\beta \left(1 - \frac{p_1}{\langle k \rangle}\right) t \right] \sim \exp \left[-\beta \left(1 - \frac{p_1}{\langle k \rangle}\right) t \right].$$

Then, the long-term behavior of $u(t)$ This is determined by p_1 because grade one nodes are the last to be infected.

On the other hand, as $u(0) = 1$ y $u(t)$ is decreasing, consider $u = 1 - \epsilon$ for short periods of time. Then, from (27),

$$-\frac{du}{dt} = \frac{d\epsilon}{dt} = \beta u[1 - s_0 g_1(u)],$$

and considering that

$$g_1(1 - \epsilon) = 1 - g'_1(1)\epsilon + O(\epsilon^2), \tag{28}$$

we have, ignoring the terms ϵ of higher order, which

$$\frac{d\epsilon}{dt} = \beta [x_0 + (g'_1(1) - 1)\epsilon], \tag{29}$$

where $x_0 = x_k(0) = 1 - s_k(0)$ is the initial value of x_k . Since (29) is a first-order linear equation, using the integral factor $\mu(t) = \exp(\beta(1 - g'_1(1))t)$, that is,

$$\frac{d}{dt} [\exp(\beta(1 - g'_1(1))t) \epsilon] = \beta x_0 \exp(\beta(1 - g'_1(1))t),$$

and by integrating with respect to t , the general solution for (29) is

$$\epsilon(t) = \frac{\beta x_0}{1 - g'_1(1)} + c \exp(\beta(g'_1(1) - 1)t),$$

where c is a constant of arbitrary integration. When considering $\epsilon(0) = 0$, the particular solution of (29) is given by

$$\epsilon(t) = \frac{\beta x_0}{g'_1(1) - 1} [\exp(\beta(g'_1(1) - 1)t) - 1].$$

Therefore,

$$u(t) = 1 - \epsilon = 1 - \frac{\beta x_0}{g'_1(1) - 1} [\exp(\beta(g'_1(1) - 1)t) - 1],$$

with $g'_1(1) > 1$ given in equation (20).

Finally, for short periods of time, this is, $u = 1 - \epsilon$,

$$x(t) = 1 - s_0 g_1(u) = 1 - s_0 g_1(1 - \epsilon)$$

$$\approx 1 - s_0 + s_0 g'_1(1)\epsilon$$

$$= x_0 \left[1 + \frac{\beta g'_1(1)}{g'_1(1) - 1} [\exp(\beta(g'_1(1) - 1)t) - 1] \right],$$

where it is considered $s_0 = 1$ and $g'_1(1) > 1$ as stated in (20). Therefore, as expected, the initial growth of the infection is more or less exponential. Similarly, it is expected that $x(t)$ increase rapidly if $g'_1(1) \gg 1$, that is, $g'_1(1)$ represents how quickly the network branches out as it moves away from the node where the disease first begins.

IV. SIR MODEL IN A NETWORK

Consider $s_i = \text{Prob}(i \in S)$, $x_i = \text{Prob}(i \in X)$ and $r_i = \text{Prob}(i \in R)$. As it is proposed [8] and in an equivalent way to the construction of the classical SIR model and the SI model with network, the changes of s_i , x_i y r_i per unit of time obey the system

$$\begin{cases} \frac{ds_i}{dt} = -\beta s_i \sum_{j=1}^n A_{ij} x_j \\ \frac{dx_i}{dt} = \beta s_i \sum_{j=1}^n A_{ij} x_j - \gamma x_i \\ \frac{dr_i}{dt} = \gamma x_i, \end{cases} \tag{30}$$

where $\gamma > 0$ is the probability per unit of time that an infected individual will recover. In addition, consider in the instant $t = 0$, $s_i(0) = 1 - \frac{c}{n}$, $x_i(0) = \frac{c}{n}$ and $r_i(0) = 0$ [8].

From (30) it can be seen that s_i decreases and r_i grows when infectious nodes exist x_j . Therefore, and equivalent to the SI model in a given network in section 3, the dynamics must be analyzed for short time steps. If $t \approx 0$, $x_i(0) \approx 0$ and

$s_i(0) \approx 1$ when $n \rightarrow \infty$, and so by ignoring the infected of quadratic order, $\frac{dx_i}{dt}$ of (30) can be approximated as

$$\begin{aligned} \frac{dx_i}{dt} &= \beta s_i \sum_{j=1}^n A_{ij} x_j - \gamma x_i \\ &= \beta(1 - x_i) \sum_{j=1}^n A_{ij} x_j - \gamma x_i \\ &= \sum_{j=1}^n (\beta A_{ij} - \gamma \delta_{ij}) x_j, \end{aligned} \quad (31)$$

where

$$\delta_{ij} = \begin{cases} 0, & i \neq j \\ 1, & i = j. \end{cases}$$

In matrix form, (31) takes the form,

$$\frac{dx}{dt} = \beta M x,$$

where

$$M = A - \frac{\beta}{\gamma} I.$$

If v_r is an eigenvector associated with the eigenvalue λ_r of the matrix A , then

$$M v_r = A v_r - \frac{\beta}{\gamma} I v_r = \left(\lambda_r - \frac{\beta}{\gamma} \right) v_r,$$

that is, the value of M associated to the own vector v_r in $\lambda_r - \frac{\beta}{\gamma}$. When considering $x(t)$ as given in (17), we must

$$\frac{da_r}{dt} = (\beta \lambda_r - \gamma) a_r,$$

with particular solution,

$$a_r(t) = a_r(0) \exp((\beta \lambda_r - \gamma)t).$$

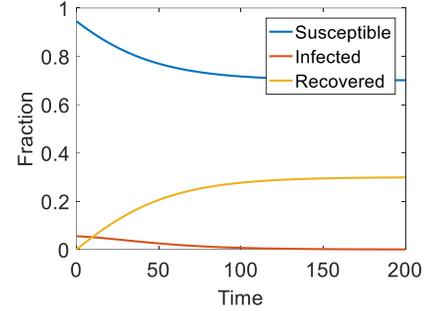
Then

$$\begin{aligned} x(t) &= \sum_{r=1}^n a_r(0) v_r \exp((\beta \lambda_r - \gamma)t) \\ &\sim \exp((\beta \lambda_n - \gamma)t) v_n. \end{aligned} \quad (32)$$

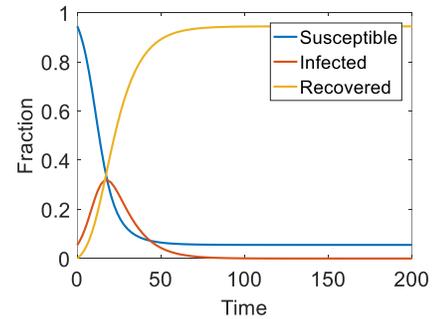
If $\beta \lambda_n - \gamma < 0$, $x(t)$ decreases exponentially to zero. On the other hand, if the main eigenvalue λ_n is small, the probability of infection $\beta > 0$ must be large, or the recovery rate $\gamma > 0$ to make the disease start to spread, equivalent to verifying that $\beta \lambda_n - \gamma > 0$. Therefore, and unlike the conclusions of the dynamics of the classical SIR model given in Theorem 2, the epidemic threshold occurs in $\beta \lambda_n - \gamma = 0$, that is,

$$\frac{\beta}{\gamma} = \frac{1}{\lambda_n}. \quad (33)$$

Fig.10 shows that, when fixing $\beta, \gamma > 0$ for a network k -regular, $x(t)$ decreases to zero as $\frac{\beta}{\gamma} < \frac{1}{k}$, where $\lambda_n = k$ in a network k -regular [11], and $x(t)$ tends to grow to a certain t_0 for k big enough.



(a) $k = 5$



(b) $k = 15$

Fig. 10. Fraction of infected, susceptible and recovered nodes for a network k -regular with $x_i(0) = 0.055$ in all cases, $\beta = 0.02, \gamma = 0.1$ and $n = 10^5$ nodes.

A. Model based on the degree of a node

Consider $s_k(t)$, $x_k(t)$ and $r_k(t)$ the odds that a neighbor with a degree k is susceptible, infected or recovered, respectively, in a time $t \geq 0$. When considering a node i who is a neighbor of a susceptible j , we have to i contracts the disease from one of his neighbors other than j . Then the probability that i infection is given by x_k with k the degree of excess. So that i recovery depends only on the probability that we have been previously infected, which is given by r_k , where k is the degree of excess, and the probability s_k if susceptible can be derived from $s_k + x_k + r_k = 1$ [8].

Analogous to the construction of the SI model based on the degree of a node, the change of s_k , x_k and r_k per unit of time is given by

$$\begin{cases} \frac{ds_k}{dt} &= -\beta k v s_k \\ \frac{dx_k}{dt} &= \beta k v s_k - \gamma x_k \\ \frac{dr_k}{dt} &= \gamma x_k, \end{cases} \quad (34)$$

where the average probability of a neighbor being infected is given by

$$v(t) = \sum_{k=0}^{\infty} q_k x_k(t). \quad (35)$$

If all the degrees of the nodes i on the network G have degree one, the dynamics of (34) can be expressed in an equivalent way as observed in Fig.5.

To find an analytical solution to the model (34), consider

$$w(t) = \sum_{k=0}^{\infty} q_k r_k(t),$$

that is, the average probability $w(t)$ of the neighbors are recovered.

From (34) and (35), we have to

$$\frac{dw}{dt} = \sum_{k=0}^{\infty} \frac{dr_k}{dt} = \gamma \sum_{k=0}^{\infty} q_k x_k = \gamma v, \tag{36}$$

that is,

$$v = \frac{1}{\gamma} \frac{dw}{dt}, \tag{37}$$

that when used in (34), the change of s_k with respect to time is written as

$$\frac{ds_k}{dt} = -\frac{\beta}{\gamma} k \frac{dw}{dt} s_k,$$

equivalent to

$$\frac{1}{s_k} \frac{ds_k}{dw} = -\frac{\beta}{\gamma} k,$$

and whose solution, with respect to the initial condition $s(0) = s_0$ for $w = 0$, is

$$s_k = s_0 \exp\left(-\frac{\beta}{\gamma} kw\right). \tag{38}$$

When considering

$$u(t) = \exp\left(-\frac{\beta}{\gamma} w(t)\right), \tag{39}$$

equivalent to

$$w = -\frac{\gamma}{\beta} \ln u, \tag{40}$$

(38) is rewritten as

$$s_k(t) = s_0 [u(t)]^k. \tag{41}$$

The goal is to find an equation to calculate $u(t)$ without depending on the unknown variables r_k . In fact, when using (40) and (41), we have to

$$\begin{aligned} v(t) &= \sum_{k=0}^{\infty} q_k x_k = \sum_{k=0}^{\infty} q_k (1 - r_k - s_k) \\ &= 1 - w(t) \\ &\quad - s_0 \sum_{k=0}^{\infty} q_k u^k \\ &= 1 + \frac{\gamma}{\beta} \ln u - s_0 g_1(u), \end{aligned} \tag{42}$$

and so on,

$$-\frac{\gamma}{\beta u} \frac{du}{dt} = \frac{dw}{dt} = \gamma v = \gamma \left[1 + \frac{\gamma}{\beta} \ln u - s_0 g_1(u)\right],$$

that is,

$$\frac{du}{dt} = -\beta u \left[1 + \frac{\gamma}{\beta} \ln u - s_0 g_1(u)\right], \tag{43}$$

obtained from (34) and (36). Therefore, the total of the susceptible fraction is of the form,

$$s(t) = \sum_{k=0}^{\infty} p_k s_k = s_0 \sum_{k=0}^{\infty} p_k u^k = s_0 g_0(u). \tag{44}$$

To find the total fraction of x_k , note that of (44),

$$\frac{d}{dt} (\exp(\gamma t) x_k) = \exp(\gamma t) \left(\frac{dx_k}{dt} + \gamma x_k\right) = \exp(\gamma t) \beta k v s_k,$$

than by integrating,

$$\begin{aligned} x_k(t) \exp(\gamma t) - x_0 &= \int_0^t \frac{d}{dt} (\exp(\gamma t) x_k) dt \\ &= \int_0^t \exp(\gamma w) \beta k v(w) s_k(w) dw, \end{aligned} \tag{45}$$

and use (41) and (42), you must

$$\begin{aligned} x_k(t) &= \exp(-\gamma t) \left[x_0 \right. \\ &\quad \left. + \beta k s_0 \int_0^t \exp(\gamma w) [u(w)]^k \left(1 \right. \right. \\ &\quad \left. \left. + \frac{\gamma}{\beta} \ln u(w) - s_0 g_1(u(w)) \right) dw \right]. \end{aligned}$$

To calculate the total recovery fraction, it is enough to see that $r_k = 1 - x_k - s_k$.

Because many times the solution of (43) cannot be explicitly calculated, certain properties are used to analyze the behavior of u . Indeed, by assuming that $s(0) = s_0 = 1 - \frac{c}{n}$, $r(0) = 0$ and $x(0) = x_0 = \frac{c}{n}$, we have to $s_0 \rightarrow 1$ y $x_0 \rightarrow 0$ for $n \rightarrow \infty$. Therefore, from (44) we have, for $t \rightarrow \infty$,

$$r_{\infty} = 1 - s_{\infty} = 1 - g_0(u_{\infty}).$$

If $t = 0$, from (39) we have to $u(0) = 1$ and $\ln u = \ln(1 + (u - 1)) \simeq 1 - u$. Then, for moments of short time and given that u is decreasing, considering $u = 1 - \epsilon$, $s_0 = 1$,

$$g_1(1 - \epsilon) = 1 - g'_1(1)\epsilon + O(\epsilon^2),$$

and ignoring the terms of ϵ of a higher order, it is known that (43)

$$\begin{aligned} \frac{d\epsilon}{dt} &= -\frac{du}{dt} = \beta(1 - \epsilon) \left[1 + \frac{\gamma}{\beta} \ln(1 - \epsilon) - g_1(1 - \epsilon) \right] \\ &\simeq \beta(1 - \epsilon) \left[1 + \frac{\gamma}{\beta} (1 - \epsilon - 1) - g_1(1 - \epsilon) \right] \\ &= \beta(1 - \epsilon) \left[1 - \frac{\gamma}{\beta} \epsilon - 1 + g'_1(1)\epsilon \right] \\ &= \beta(1 - \epsilon) \left[-\frac{\gamma}{\beta} \epsilon + g'_1(1)\epsilon \right] \\ &= [\beta g'_1(1) - \gamma]\epsilon. \end{aligned}$$

Therefore

$$u(t) = 1 - \epsilon(t) = 1 - \exp((\beta g'_1(1) - \gamma)t),$$

and

$$s_k(t) = u^k = [1 - \exp((\beta g'_1(1) - \gamma)t)]^k \simeq 1 - \exp(kt(\beta g'_1(1) - \gamma)). \quad (46)$$

Thus the epidemic threshold is given by

$$\frac{\beta}{\gamma} = \frac{1}{g'_1(1)} = \frac{\langle k \rangle}{\langle k^2 \rangle - \langle k \rangle} \quad (47)$$

with $g'_1(1)$ given in (20), equivalent to the epidemic threshold given in (33) when replacing λ_n by $g'_1(1)$.

V. SIS MODEL IN A NETWORK

In a way equivalent to the construction of the SI model in a network and the classic SIS model, the changes of s_i y x_i per unit of time are given by

$$\begin{cases} \frac{ds_i}{dt} = -\beta s_i \sum_{j=1}^n A_{ij} x_j + \gamma x_i, \\ \frac{dx_i}{dt} = \beta s_i \sum_{j=1}^n A_{ij} x_j - \gamma x_i. \end{cases} \quad (48)$$

Since $s_i + x_i = 1$, the change of x_i per unit of time, given in (48), can be written as

$$\frac{dx_i}{dt} = \beta(1 - x_i) \sum_{j=1}^n A_{ij} x_j - \gamma x_i. \quad (49)$$

Assuming that $x_i(0) = x_0 = 1 - \frac{c}{n}$ for everything i and constant $c \geq 1$ For $n \rightarrow \infty$ and ignoring the terms of x_i of higher order, of (49) must

$$\frac{dx_i}{dt} = \beta \sum_{j=1}^n A_{ij} x_j - \gamma x_i, \quad (50)$$

which is equivalent to (31). Therefore, the epidemiological threshold is given by

$$\frac{\beta}{\gamma} = \frac{1}{\lambda_n},$$

that is, if $\frac{\beta}{\gamma} < \frac{1}{\lambda_n}$ It is expected that $x(t)$ decreases exponentially to zero, while if $\frac{\beta}{\gamma} > \frac{1}{\lambda_n}$, $x(t)$ begins to grow for short moments of time as observed in Fig.11.

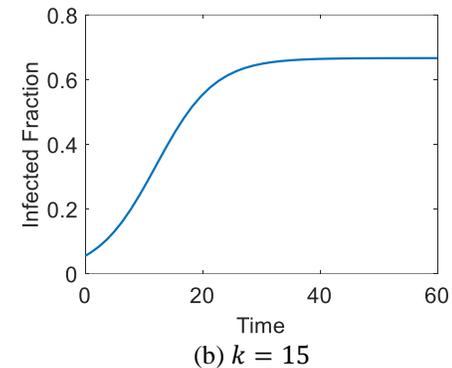
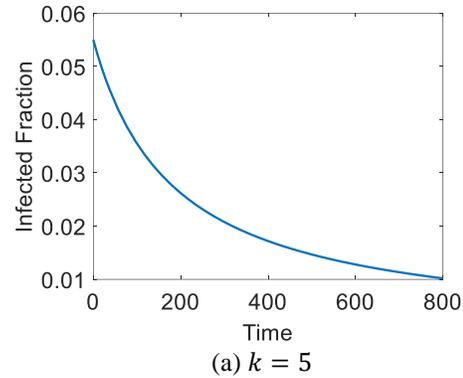


Fig. 11. Fraction of infected nodes for a network k -regulate with $x_i(0) = 0.055$ in all cases, $\beta = 0.02$, $\gamma = 0.1$ y $n = 10^5$ nodes.

A. Model based on the degree of a node

Equivalent to the construction of the SI and SIR models based on the degree of a node, the change of s_k y x_k per unit of time is given by

$$\begin{cases} \frac{ds_k}{dt} = -\beta k v s_k + \gamma x_k \\ \frac{dx_k}{dt} = \beta k v s_k - \gamma x_k, \end{cases}$$

where

$$v(t) = \sum_{k=0}^{\infty} q_k x_k(t). \quad (51)$$

Since $s_k + x_k = 1$, then

$$\frac{dx_k}{dt} = \beta kv(1 - x_k) - \gamma x_k. \tag{52}$$

As in the SIR model based on the degree of a node, if all the degrees of the nodes i on the network G has degree one, the dynamics of (34) can be expressed in an equivalent way as observed in Fig.7.

Because no explicit solution is known for (52), its behavior is analyzed when $t \approx 0$ y $t \rightarrow \infty$. By assuming that $x_k(0) = \frac{c}{n}$ for c constant and, for moments of small time when $n \rightarrow \infty$ and ignoring the terms of x_k of higher order, of (51) and (52) that

$$\frac{dx_k}{dt} = \beta kv - \gamma x_k,$$

which corresponds to a first-order linear equation. When using the integral factor, we have to

$$\begin{aligned} \frac{d}{dt}(x_k \exp(\gamma t)) &= \exp(\gamma t) \frac{dx_k}{dt} + \gamma \exp(\gamma t) x_k \\ &= \beta k \exp(\gamma t) v, \end{aligned}$$

with particular solution in $x_k(0) \rightarrow 0$ given by

$$x_k(t) = \beta k \exp(-\gamma t) \int_0^t \exp(\gamma w) v(w) dw.$$

When considering

$$x_k = ku(t),$$

with

$$u(t) = \beta \exp(-\gamma t) \int_0^t \exp(\gamma w) v(w) dw, \tag{53}$$

it is known from (51) that

$$v(t) = u(t) \sum_{k=0}^{\infty} k q_k = g'_1(1) u(t) \tag{54}$$

The objective is to calculate $u(t)$ without being determined by values x_k . Indeed, since

$$\frac{du}{dt} = -\gamma u + \beta v(t),$$

from (54) we have

$$\frac{du}{dt} = [\beta g'_1(1) - \gamma] u(t),$$

with particular solution in $u(0) = 0$,

$$u(t) = \exp((\beta g'_1(1) - \gamma)t).$$

Therefore, for short term time we have

$$x_k(t) = ku(t) = k \exp((\beta g'_1(1) - \gamma)t),$$

whose epidemic threshold is

$$\frac{\beta}{\gamma} = \frac{1}{g'_1(1)} = \frac{\langle k \rangle}{\langle k^2 \rangle - \langle k \rangle},$$

which corresponds to the same threshold (47) given in the SIR model based on the degree of a node.

When $t \rightarrow \infty$ it is expected that x_k tends to the balance of (52), that is,

$$x_k = \frac{kv}{kv + \frac{\gamma}{\beta}}$$

VI. CONCLUSIONS

The models presented depend on the hypothesis made about the population and the states of the disease. If each individual has the same opportunity to interact with another, a classical epidemiological model could be adjusted, otherwise a network model must be adjusted. Similarly, considering that the disease can have as susceptible, infected and at most recovered state, SI, SIR or SIS models were used. On the other hand, as a comparison between classic and network models was made, a constant and closed population was considered over time, so it was not considered a birth rate or natural or disease mortality.

Since the classical SI model is built by a single parameter representing the infection rate, it is determined that the disease spreads and manages to infect the entire population as long as there is at least one infected, with the difference that the speed with which it spreads decreases when its rate approaches zero. On the other hand, by fitting an SI model into a network, it was determined that the speed of propagation depends both on the infection rate and the largest eigenvalue associated with the adjacency matrix, as a way of represent a dense or dispersed network. Unlike the classical SI model, if the infection rate approaches zero, the disease spreads more rapidly if the network is denser.

For a classical SIR model, regardless of the infection and recovery rate, the infection tends to be eradicated for a long enough period of time. However, if the ratio between the recovery and infection rate is higher than the initial susceptible fraction, the disease spreads until a certain time and then tends to disappear. This dynamic is similar to the SIR model in a network, with the difference that the ratio between the recovery and transmission rate must be greater than the largest own value associated with the adjacency matrix.

On the other hand, for a classic or networked SIS model, the disease does not disappear over time when the ratio between recovery rate and contagion is greater than one or the highest eigenvalue associated with the adjacency matrix, respectively.

Since it is not possible to find an explicit solution for SI, SIS and SIR models in networks, their states are adjusted by the degree of the nodes in the network. Under this

methodology and for short term time, the epidemic threshold for the SIR and SIS models are equivalent to those previously explained by replacing the own value by the excess of the average degree, and for the SI model, the speed of disease propagation is determined by the excess of the average degree above one.

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Christian Camilo Cortes Garcia, Master in Mathematics from the Federal University of Minas Gerais, Brazil and Mathematician from the Universidad Surcolombiana, Colombia. He is currently studying a doctorate in mathematical engineering at the Universidad Carlos III de Madrid, Spain. His research areas are: dynamic

systems, mathematical biology and applied statistics.

ORCID: <http://orcid.org/0000-0002-8955-4530>



Juan David Hernández Ramírez, Master in Applied Mathematics, Universidad Nacional de Colombia, and Mathematician, Universidad Surcolombiana. His areas of interest are numerical analysis and dynamic systems.

ORCID: <http://orcid.org/0000-0003-2410-352>