



On considering the influence of recovered individuals in disease propagations



A.L.S. Moraes^a, L.H.A. Monteiro^{a,b,*}

^a Universidade Presbiteriana Mackenzie, Escola de Engenharia, São Paulo, SP, Brazil

^b Universidade de São Paulo, Escola Politécnica, São Paulo, SP, Brazil

ARTICLE INFO

Article history:

Received 30 May 2015

Revised 12 September 2015

Accepted 2 November 2015

Available online 6 November 2015

Keywords:

Bifurcation

Childhood infection

Epidemiology

SIR model

Varicella

ABSTRACT

Consider diseases transmitted through personal contacts, for which recovery usually confers complete and long-lasting immunity, like some of the common viral infections of childhood. Here, an epidemic model based on differential equations is proposed to evaluate the influence of the recovered (immune) individuals on the spread of such diseases. Indeed, immune individuals can affect the infection rate of susceptible individuals and the recovery rate of sick individuals. The predictive ability of the proposed model is assessed from records concerning the incidence of varicella in three European countries, in a pre-vaccination era.

© 2015 Elsevier B.V. All rights reserved.

1. Introduction

Consider infectious diseases that propagate through social contacts. Specifically, think about typical viral infections of childhood, like varicella (chickenpox). Recovery from this disease commonly confers full and sustained immunity [5,8,27].

Epidemic models on varicella do not usually include the influence of the recovered (immune) individuals on the spread of this disease [4,7,12,16,20,21]. Such an influence, however, should be taken into account. Habitually, immune individuals take care of sick children, reducing the convalescence period. Immune individuals also act as catalysts by facilitating the interaction among susceptible and infective children in clubs, parks, schools. In fact, children generally go out their homes only accompanied by immune adults. Thus, recovered individuals can increase the contagion rate, which is harmful to the host population; but they can also increase the recovery rate, which is beneficial.

Here, these opposite effects are taken into consideration in an epidemic SIR model written in terms of ordinary differential equations. We did not find in the literature any epidemic model about any contagious disease with this feature. The model proposed in this work allows the existence of multiple stable equilibria. The predictions of this model are evaluated by comparing them to real data related to the annual incidence rate of varicella in Belgium [22], Germany [25], and Italy [6], prior to varicella vaccination [15].

This manuscript on the influence of recovered individuals in infection propagation is organized as follows: in Section 2, the proposed model is analyzed; in Section 3, numerical simulations performed with hypothetical and real parameter values are presented; in Section 4, the main results are discussed.

* Corresponding author at: Universidade Presbiteriana Mackenzie, Escola de Engenharia, Rua da Consolação, n. 896, 01302-907, São Paulo, SP, Brazil. Tel.: +55 1121148711; fax: +55 11 2114 8600.

E-mail addresses: 71412621@mackenzista.com.br (A.L.S. Moraes), luizm@mackenzie.br, luizm@usp.br (L.H.A. Monteiro).

2. Model and analytical results

SIR models have been successfully employed in epidemiological studies [1,3,9–11]. This work is based on the following SIR model:

$$\frac{dS(t)}{dt} = f_1 = -aS(t)I(t)[1 + q_1R(t)] + cI(t) + eR(t) \tag{1}$$

$$\frac{dI(t)}{dt} = f_2 = aS(t)I(t)[1 + q_1R(t)] - bI(t)(1 + q_2R(t)) - cI(t) \tag{2}$$

$$\frac{dR(t)}{dt} = f_3 = bI(t)(1 + q_2R(t)) - eR(t). \tag{3}$$

The variables $S(t)$, $I(t)$, and $R(t)$ denote the numbers of susceptible, infective, and recovered individuals at the time t , respectively. It is supposed that these three kinds of individuals are homogeneously distributed over the space [1,23].

The six parameters a , b , c , e , q_1 , and q_2 are positive numbers: a is the infection rate constant, b is the recovery rate constant, c is the rate constant of death of I -individuals, e is the rate constant of death of R -individuals, q_1 is the constant expressing the impact of R -individuals on the contagion of S -individuals, q_2 is the constant denoting the influence of R -individuals on the cure of I -individuals. The analytical form of the terms containing q_1 and q_2 shows a linear variation with $R(t)$, because this is the simplest way of representing the effects of recovered people on a disease spreading. Thus, it becomes the natural choice for such terms in this early investigation.

In this model, it is assumed that deaths of I and R -individuals are balanced by births of S -individuals; that is, when I and R -individuals die, S -individuals replace them. Hence, $dS(t)/dt + dI(t)/dt + dR(t)/dt = 0$; consequently, $S(t) + I(t) + R(t) \equiv N$, in which N is the total number of individuals. Therefore, the size of the population remains constant and equal to N . Note that the terms involving the parameters c and e in Eq. (1) represent the birth rate. Note also that $R(t) = N - S(t) - I(t)$; therefore, this model is a second-order autonomous dynamical system. Hence, the attractors can be either steady states or limit cycles [2,14].

A steady state is a stationary solution. It corresponds to an equilibrium point (S^*, I^*) in the state space $S \times I$, in which S^* and I^* are constants satisfying $f_1(S^*, I^*) = 0$ and $f_2(S^*, I^*) = 0$ for any time t (obviously, $R^* = N - S^* - I^*$). The local stability of an equilibrium point (S^*, I^*) can be inferred from the eigenvalues of the Jacobian matrix obtained from the system of Eqs. (1) and (2) linearized around such a point [2,14]. Hartman–Grobman theorem states that (S^*, I^*) is locally asymptotically stable if both eigenvalues have negative real parts [2,14]. For this second-order system, the eigenvalues $\lambda_{1,2}$ are the roots of the polynomial $\lambda^2 - T\lambda + \Delta = 0$, in which $T = [\partial f_1/\partial S + \partial f_2/\partial I]_{(S,I)=(S^*,I^*)}$ is the trace and $\Delta = [(\partial f_1/\partial S)(\partial f_2/\partial I) - (\partial f_1/\partial I)(\partial f_2/\partial S)]_{(S,I)=(S^*,I^*)}$ is the determinant of the Jacobian matrix computed at (S^*, I^*) . The eigenvalues $\lambda_{1,2}$ have negative real parts if $T < 0$ and $\Delta > 0$.

A limit cycle is a periodic solution. It is represented by a closed and isolated trajectory in the state space $S \times I$. By varying the parameter values, a limit cycle enclosing an equilibrium point can appear via Hopf bifurcation [2,14]. If $T = 0$ and $\Delta > 0$ for an equilibrium point, then a Hopf bifurcation can occur, and a limit cycle with stability opposite to this equilibrium point emerges in the state space.

For the proposed model, the stationary solution given by $(S_{free}^*, I_{free}^*) = (N, 0)$ corresponds to the disease-free steady-state (that is, the steady state without infective individuals). It exists for any values of q_1 and q_2 . For this solution, $T = (R_0 - 1)/(b + c) - e$ and $\Delta = e(1 - R_0)/(b + c)$, with $R_0 \equiv aN/(b + c)$. Therefore, the disease-free steady-state is locally asymptotically stable if $R_0 < 1$ and unstable if $R_0 > 1$. The (bifurcation) parameter R_0 is usually known as basic reproduction number [1,18].

The existence and the stability of endemic steady-states (S_{ende}^*, I_{ende}^*) (that is, steady states with infective individuals) depend on q_1 and q_2 . The scenario with $q_1 = 0$ and $q_2 = 0$ was already analyzed by Schimit and Monteiro [19]. In this case, there is only one endemic steady-state given by:

$$(S_{ende}^*, I_{ende}^*) = \left(\frac{N}{R_0}, \frac{eN}{e + b} \left(1 - \frac{1}{R_0} \right) \right) \tag{4}$$

which is locally asymptotically stable if $R_0 > 1$ and unstable if $R_0 < 1$. Observe that the system experiences a transcritical bifurcation in $R_0 = 1$, because the steady states (S_{free}^*, I_{free}^*) and (S_{ende}^*, I_{ende}^*) of opposite stabilities exchange their stabilities when R_0 is varied around 1 [19].

Here, the case $q_1 > 0$ and $q_2 = 0$ is first investigated; then, it is taken $q_1 = 0$ and $q_2 > 0$; and, finally, it is considered $q_1 > 0$ and $q_2 > 0$.

Case 1: $q_1 > 0$ and $q_2 = 0$ (that is, R -individuals only affect the propagation rate). Endemic steady-states are obtained from:

$$a_0(I_{ende}^*)^2 + a_1 I_{ende}^* + a_2 = 0 \tag{5}$$

with $a_0 = q_1 b(b + e)/e^2$, $a_1 = 1 + b(1 - q_1 N)/e$, $a_2 = N(1 - R_0)/R_0$, and:

$$S_{ende}^* = N - \left(1 + \frac{b}{e} \right) I_{ende}^*. \tag{6}$$

The roots of Eq. (5) are $I_{ende,1}^* = (-a_1 + \rho)/(2a_0)$ and $I_{ende,2}^* = (-a_1 - \rho)/(2a_0)$, with $\rho = \sqrt{a_1^2 - 4a_0a_2}$. Only positive and real roots are biologically meaningful. Observe that $a_0 > 0$. If $a_2 < 0$ (that is, $R_0 > 1$), then the only positive root is $I_{ende,1}^*$; if $a_2 > 0$,

Download English Version:

<https://daneshyari.com/en/article/758007>

Download Persian Version:

<https://daneshyari.com/article/758007>

[Daneshyari.com](https://daneshyari.com)