Applied Mathematical Sciences, Vol. 1, 2007, no. 26, 1277 - 1298

Stability Analysis of a Delay-Difference SIS Epidemiological Model

Alma V. Lara-Sagahón[2,3], Vladislav Kharchenko[3] and Marco V. José [1] [2]

[2]Theoretical Biology Group
Instituto de Investigaciones Biomédicas
Universidad Nacional Autónoma de México
Apdo. Postal 70228, México D.F. 04510, México

[3]FES Cuautitlán, Universidad Nacional
Autónoma de México, Primero de mayo s/n
Cuautitlán Izcalli, 54768

Abstract

We develop a discrete-time SIS model with exponential incidence and where the proportion of infectives takes the form of a delay-difference equation. This model generalizes a model developed by Cooke, Calf, and Level (1977) by allowing the period of infectivity to be of arbitrary length. The model is analyzed to determine the equilibria, its stability and threshold quantities. The endemic equilibrium state is globally asymptotically stable. The solution tends to the endemic equilibrium through infinite damped oscillation if the transmission parameter is greater than a critical value. This critical value is a function of the period of infectiousness.

Mathematics Subject Classification: 92D30, 39A11, 92B05

Keywords: epidemic model; SIS model; Stability analysis; delay difference equations; infectious diseases

1 Introduction

SIS epidemic models have been used to represent the spread of infectious disease that do not confer immunity (e.g. [11] and references therein). We formulate and analyse a simple discrete time SIS model for disease transmission

[1]Corresponding author, email: marcojose@biomedicas.unam.mx

in a closed population. The model is built in the form of a delay-difference equation assuming an exponential incidence and a period of infectivity whose length can be fixed arbitrarily. In epidemic models the rate at which individuals leave the susceptible compartment is given by an incidence function. The most commonly used forms of the incidence function are the standard incidence rate (true mass action) and the simple mass action (pseudo mass action). The standard function states that the incidence is proportional to the fraction of infectives times the number of susceptibles [20, 21]. The proportionality constant of the standard incidence, usually denoted by β, is the average number of contacts sufficient for achieving transmission [8, 20]. On the other hand, the simple mass action is formulated in terms of numbers i.e. the incidence is proportional to the number of infectives times the number of susceptibles. In this case, the proportionality constant of the simple mass action, β, has been considered as a transmission parameter that involves a myriad of epidemiological, environmental and social factors affecting the transmission rates [4], but without a direct epidemiological interpretation [20].

The SIS (susceptible-infected-susceptible) delay-difference model proposed here uses an exponential incidence function. One advantage of the exponential incidence over the mass action is that it always gives biologically sensible well-posed discrete models. In contrast, in discrete models the law of mass action cannot be properly used, and therefore restrictions must be put in the parameters in order to insure not to get absurd solutions (see [2], Lemma 2).

In the ordinary differential equation (ODE) epidemic models the recovery rate is assumed to be a constant, ν, related with the average infectiousness period D by $D = 1/\nu$ [4]. It has been shown that this assumption corresponds to the situation in which the duration of the period of infectiousness is exponentially distributed [8, 20], and it is equivalent to the assumption that the chance to recover is independent of the time in which an individual enters the infective class [4, 25]. It is well known that this principle is mathematically convenient but not necessarily realistic [4, 25], much less if the waiting time is supposed to be a constant. The more general assumption of an arbitrary distribution of the period of infectiousness can be treated in a stochastic setting on chain-binomial models (see [6] chapter 8). Some developments of SIS models deal with stochastic features, where quasi-stationarity and time to extinction are analyzed (see [27] and references therein). If the period of infectivity is held constant, the continuous ODE model becomes a delay-differential equation (e.g. [8, 13, 17, 20] and citations in these works).

The simple SIS model analyzed here is a discrete time model where the period of infectivity is assumed to be a constant. The following discrete or "quantum" principle is of fundamental importance for our approach. We sup-

pose that the individual infection is a consequence of daily contacts, so that, the time step –one day– of our discrete model is a basic cycle of the population life history, a quantum of time. It may be considered neither as an arbitrary convenient small value, as in the discrete models of Allen et al. [1, 2], nor as an infinitesimal one.

In Section 2 we derive the exponential incidence function. Our formulation of the incidence rate is closely related to the derivation of the discrete–time stochastic models of Greenwood and Reed-Frost [1, 12, 24]. We note that the continuous version of the exponential incidence is exactly the law of mass action, but not vice versa, since the "quantification" of the mass action provides an incidence function different from the exponential incidence. The formulation and analysis of the deterministic discrete SIS model with a fixed period of infectiousness are given in Sections 3 and 4, respectively. To find the equilibria, we observed that the initial data have to satisfy an algebraic equation that takes into account the individual infectious history. This equation is invariant under the process defined by the difference system. Therefore, equilibrium states turn out to be the constant solutions of this algebraic equation. In order to analyze the stability properties of our model we use additive perturbation techniques instead of using popular methods, such as the linearized stability analysis [23]. The proof of stability uses some ideas from the paper by Cooke et al. [12]. We extend their results to the delay case.

2 Incidence in homogeneous and uniformly mixing populations

Let us consider the incidence rate function in homogeneous and uniformly mixed populations. A population is uniformly mixed if its members make contact homogeneously with each other. For example, if a person has a total of k contacts per day, then among these there are ky/N contacts with infectives, where N is the population size and Y is the number of infectives. Let c be the probability that a given susceptible becomes infective in one contact with one infective (this is, probably, the main characteristic of the transmission of a disease in a population). Assume that c is a constant for the disease under investigation. Then $1 - c$ is the probability that a given susceptible does not become infective in one contact with one infective.Thus, assuming that each contact is independent of other contacts, the probability that a given susceptible does not become infective during a day (that is in kY/N contacts with infectives) equals $(1 - c)^{kY/N}$. Hence, the probability that a given susceptible becomes infective in one day is $1-(1-c)^{kY/N}$, or equivalently,

$1 - e^{-\beta Y}$, where $\beta = -k\ln(1-c)/N$. Thus, the transmission coefficient β is a parameter that summarizes both population and disease spread characteristics. Now, we may express the incidence as the number of susceptibles, X, times the probability to become infective, i.e.,

$$G(X,Y) = X(1 - e^{-\beta y}). \tag{1}$$

If x, y, and g denote the proportions of susceptibles, infectives and new infectives, respectively, we can write 1 in the form

$$g(x,y) = x(1 - e^{-\gamma y}), \tag{2}$$

where $\gamma = \beta N = -k\ln(1-c)$. It is straightforward to show that the law of mass action is just a first approximation that can be obtained from 1 and 2 by the Taylor series expansion. The foregoing derivation of the incidence function leads to the law of mass action if applied properly to a continuous process. Indeed, if we suppose that during a day the contacts of a person are distributed homogeneously, then for a period of time $[t, t+dt]$ the person will have $kydt$ contacts with infectives. Therefore, the proportion of new infectives for the period of time $[t, t+dt]$ equals $\Delta g(x,y) = x(1 - e^{-\gamma y dt})$. Thus, the continuous version of 2 is

$$g_c = \lim_{dt\to 0} \frac{\Delta g(x,y)}{dt} = \gamma xy, \tag{3}$$

which is exactly the law of mass action expressed in proportions. We cannot say, however, that the incidence 1 or 2 is a discrete version of the law of mass action. Indeed, suppose that we have a continuous process of incidence that is defined by the law of mass action. Then the proportion of new infectives for one day equals a difference $(g_c)_d = y(t+1) - y(t)$, where $y(t)$ is a solution of the incidence equation $\frac{dy}{dt} = \gamma xy = \gamma(1-y)y$. The solution of this differential equation is $y(t) = \frac{Ce^{\gamma t}}{1+Ce^{\gamma t}}$. Now the number of new infectives in one day, the discrete version of mass action, can be calculated by

$$\begin{aligned}
(g_c)_d &= y(t+1) - y(t) \\
&= \frac{Ce^{\gamma t}}{1+Ce^{\gamma t}} \left(1 - \frac{Ce^{\gamma t}}{1+Ce^{\gamma t}}\right) \left(\frac{e^{\gamma} - 1}{1 + (e^{\gamma}-1)\frac{Ce^{\gamma t}}{1+Ce^{\gamma t}}}\right) \\
&= \frac{\gamma_1 xy}{1+\gamma_1 y},
\end{aligned} \tag{4}$$

where $\gamma_1 = e^{\gamma} - 1$. Note that this last result is different from the incidence $g(x,y)$.

The above arguments show that the standard reduction to a continuous process will essentially change the dynamics: $g \neq (g_c)_d$. We remark that in a discrete model developed by Castillo-Chavez and Yakubu [11] the time step equals the period of one generation, whilst in those proposed by Cooke et al. [12] it is equal to the period of infectivity.

Another important note is that in discrete models the law of mass action in its continuous form cannot be properly used. This can be seen in the following example. Suppose that an infection with a period of infectiousness greater than one day, and a parameter γ greater than one, attacks a virgin population. In this way, at day zero the proportion of infectives equals zero and in the first day a proportion p_1 of new infectives ensues. By the mass action principle, in the second day the proportion of infectives would be $p_2 = p_1 + \gamma p_1(1 - p_1)$. If we suppose that $p_1 = (1 + \gamma^{-1})/2$, then by direct calculations it is easy to see that $p_2 = 1 + (\gamma - 1)^2/4\gamma$ which is greater than unity! This contradiction shows that the mass action incidence with $\gamma > 1$ (or $\beta > N^{-1}$ for a population of size N) is not valid for discrete models. This situation is illustrated in Fig. **??** where the exponential incidence and mass action are compared in a discrete SIS model. It is shown that if the mass action is assumed then very small differences in the initial values led to non- sensible biologically results.

3 The discrete SIS model

Herein we present a discrete SIS model based on the discrete principle mentioned in Section 2 with the incidence function 2. We consider a population that has a constant size. For the stability analysis we introduce a perturbation $p(t, x, y, ...)$, which is a function that defines a proportion of new infectives that appears due to some external process (e. g. random effects, preventive actions, etc.) other than the regular contact process in the population. The perturbation may have both positive and negative values. Of course, these values have to be biologically reasonable, that is, the proportion of new infectives by the perturbation cannot be greater than the proportion of susceptibles at the end of the day t and cannot be less than the negation of the proportion of new infectives, i.e., $-g(x, y) \leq p_t \leq x_t - g(x, y)$.

If we assume a constant period of infectiousness, the difference equations of the model are:

$$x_{t+1} = x_t - [x_t(1 - e^{-\gamma y_t}) + p_t] + [x_{t-\sigma}(1 - e^{-\gamma y_{t-\sigma}}) + p_{t-\sigma}] \quad (5)$$

$$y_{t+1} = y_t + [x_t(1 - e^{-\gamma y_t}) + p_t] - [x_{t-\sigma}(1 - e^{-\gamma y_t}) + p_{t-\sigma}] \quad (6)$$

$$x_{t+1} + y_{t+1} = 1. \quad (7)$$

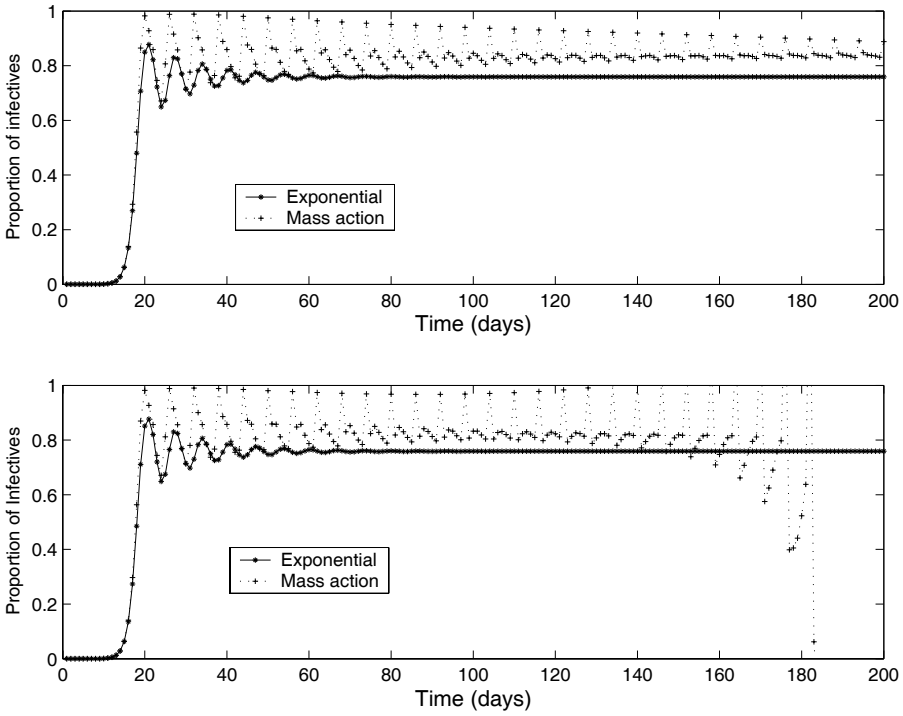

Figure 1: Comparison of the discrete SIS model proposed here and the mass action discrete SIS model with: $\gamma = 1.31$, $\sigma = 5$ days, $y_{-5} = y_{-4} = ... = y_0 = 0$, and $p(t) = 0$ for all time except $p(0)$. A. $p(0) = 5.8e-7$, B. $p(0) = 5.9e-7$. Using the mass action incidence, small differences in the perturbation initial value change the solution towards absurd results.

Because the population size remains constant, the model can be reduced to a single delay-difference equation for the proportion of infectives. This equation defines a dynamic process provided that the following initial values are given:

$$y_{-\sigma}, y_{-\sigma}+1, ..., y_{-1}, y_0, \quad 0 \leq y_i \leq 1, \quad i = -\sigma, ..., 0. \tag{8}$$

The initial values have to satisfy the following equation:

$$y_0 = \sum_{i=0}^{\sigma-1} \left[(1-y_{i-\sigma})(1-e^{-\gamma y_{i-\sigma}}) + p_{i-\sigma}\right] \tag{9}$$

Equation 9 takes into account that the number of infectives at should be equal to the cumulative sum of infectives that acquired the infection in anyone of the previous days. In other words, every infective has its own infectious life history related to the disease under investigation. This condition has to be valid not only at the initial time, but also during all the process. Nevertheless, due to the following lemma, we do not need to postulate this as a general assumption.

Lemma 1 *The dynamic process defined by the equations 5, 6 and 7 with the initial conditions 8 connected by 9 always satisfies the equality*

$$y_t = \sum_{i=0}^{\sigma-1} \left[(1-y_{t+i-\sigma})(1-e^{-\gamma y_{t+i-\sigma}}) + p_{t+i-\sigma}\right], \tag{10}$$

that is, the number of infectives at time t equals the cumulative sum of infectives that had acquired the infection in anyone of the previous days.

Proof. *We only have to show that the equality 10 is preserved when t increases by one. By 6 and 7 and using 10 we have*

$$\begin{aligned} y_{t+1} &= \sum_{i=0}^{\sigma-1} \left[(1-y_{t+i-\sigma})(1-e^{-\gamma y_{t+i-\sigma}}) + p_{t+i-\sigma}\right] + \\ &\quad (1-y_t)(1-e^{-\gamma y_t}) + p_t - \\ &\quad (1-y_{t-\sigma})(1-e^{-\gamma y_{t-\sigma}}) - p_{t-\sigma} \\ &= \sum_{i=0}^{\sigma-1} \left[(1-y_{t+1+i-\sigma})(1-e^{-\gamma y_{t+1+i-\sigma}}) + p_{t+1+i-\sigma}\right] \end{aligned}$$

The lemma is proved. ■

This lemma implies that if one starts with no infections, then $y_{-\sigma} = y_{-\sigma+1} = ... = y_0 = 0$, $p_{-\sigma} = p_{-\sigma+1} = ... = p_{-1} = 0$, and since $t = 0$,

nothing would have to be said about the initial conditions, since (9) is clear and, according to the lemma, 10 is fulfilled automatically.

Note that the proportion of new infectives $g(x_t, y_t)$ will never be greater than the proportion of susceptibles x_t (this condition failed in the example where the law of mass action was applied to a discrete process). Indeed, since $0 \leq 1 - e^{-\gamma y_t} < 1$ for arbitrary positive values of γ and y_t, we have $g(x_t, y_t) = x_t(1 - e^{-\gamma y_t}) \leq x_t$. Therefore, the dynamical process 6, 7 will never give contradictory results like the ones presented in Section 2.

4 Analysis of the model

4.1 Equilibrium states and threshold theorem

The computer simulation experiments show that if after some time the perturbation becomes zero then the solutions of 5, 6, 7 tend to a constant value (Fig. 2). Our rigorous mathematical analysis proves that there is a threshold quantity gauged by the relation $\gamma\sigma = 1$ (or $\beta N\sigma = 1$). If $\gamma\sigma \leq 1$there is only disease free equilibrium state. If $\gamma\sigma > 1$ there is only one endemic equilibrium state. The endemic equilibrium is globally asymptotically stable. The threshold quantity $\gamma\sigma$ is the well-known basic reproductive number usually denoted by R_0 (e.g. [4, 20]).

Theorem 2 *If $0 < \gamma \leq \sigma^{-1}$ then there is just disease free equilibrium state. If $\gamma > \sigma^{-1}$ then there is only one endemic equilibrium which never exceeds $1/(\sigma^{-1} + 1)100\%$ of the total population.*

Proof. *Suppose that $p_t = 0$ for all $t > T$. Then, if $t > T + \sigma$, the relation 10 acquires the form*

$$y_t = \sum_{i=0}^{\sigma-1}(1 - y_{t+i-\sigma})(1 - e^{-\gamma y_{t+i-\sigma}}), \tag{11}$$

therefore we have the following equation for the equilibrium y^*,

$$y^* = \sum_{i=0}^{\sigma-1}(1 - y^*)(1 - e^{-\gamma y^*}), \tag{12}$$

that is

$$\sigma^{-1}y^* = (1 - y^*)(1 - e^{-\gamma y^*}). \tag{13}$$

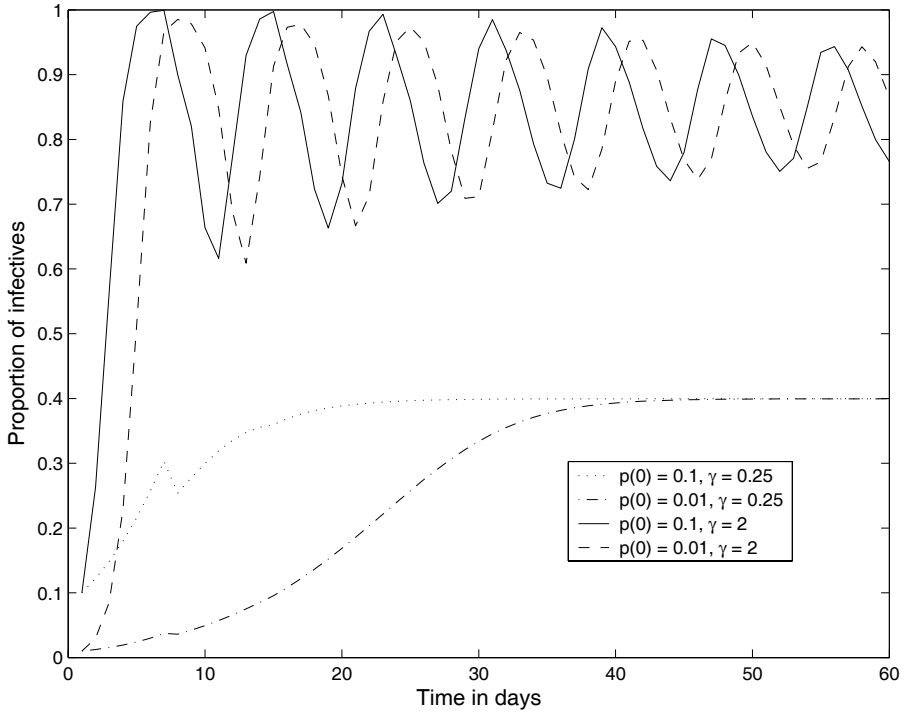

Figure 2: Solutions of the discrete SIS model with various initial values of the perturbation $p(0)$. Parameters values as in Fig. 1 except $p(0)$ and γ as indicated in the figure. From the period of infectiousness we get $\gamma_0 = 0.41$ (see Section 4.3). If $\gamma < \gamma_0$ the solution tends monotonically to the steady state. While If t $\gamma > \gamma_0$ the solution tends to the steady state through infinite damped oscillations.

For given γ and σ we can find a solution geometrically considering two curves, $g_1(y) = \sigma^{-1}y$ and $g_2(y) = (1-y)(1-e^{-\gamma y})$. This is shown in Fig. 3. The intersections of these lines define the solutions of 13. At this point we would like to note that of the two parameters σ and γ, only the parameter σ, can be estimated for a given real disease. At the same time the endemic equilibrium y^* is a parameter that can be measured and estimated. Thus equation 13 allows us to calculate

$$\gamma = -\frac{1}{y^*}\ln\left(1 - \frac{\sigma^{-1}y^*}{1-y^*}\right). \tag{14}$$

In particular, we have $1 \geq \frac{\sigma^{-1}y^*}{1-y^*}$ or, equivalently

$$y^* \leq \frac{1}{\sigma^{-1}+1}. \tag{15}$$

Therefore every value between 0 and $\frac{1}{\sigma^{-1}+1}$ may be an equilibrium state for some parameter γ, while $y^* = 0$ is an equilibrium state for all of them. This proves the second part of the theorem.

Finally, since the second derivative of $g(y) = (1-y)(1-e^{-\gamma y})$ is always negative,

$$g''(y) = -(2 + (1-y)\gamma)\gamma e^{-\gamma y} < 0, \tag{16}$$

the function $g(y)$ is convex, and therefore equation 13 cannot have more than one nonzero solution $y = y^*$. Alternatively, one may multiply 13 by σ/y^* and note that the right hand side is a monotonically decreasing function from $\gamma\sigma$ to 0. The theorem is proved. ■

4.2 Stability analysis

In the following theorem we are going to prove that the endemic equilibrium is globally asymptotically stable.

Theorem 3 *If the perturbation in 5-7 becomes zero since time T then the solution tends to y^*, where y^* is the endemic equilibrium , provided that $\gamma\sigma > 1$, and $y^* = 0$ otherwise.*

Proof. *For all $t > T + \sigma$ equation 14 has the form*

$$y_t = \sum_{i=0}^{\sigma-1} g(y_{t+i-\sigma}), \tag{17}$$

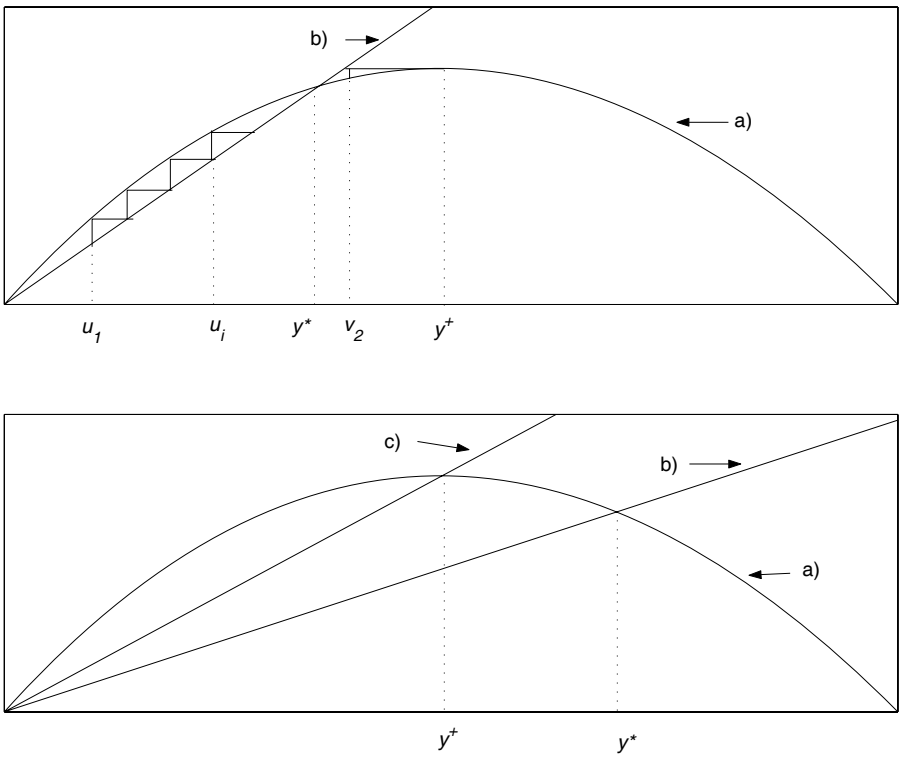

Figure 3: The endemic equilibrium state is given by the intersection of the curves a) $g_1(y) = \sigma^{-1}y$ and and b) $g_2(y) = (1-y)(1-\exp(-\gamma y))$. A. $y^* \leq y^+$. B. $y^* > y^+$, the curve c) is given by $g_3(y) = hy$, where $h = g(y^+)/y^+$. See explanations in Theorem 3.

where $g(y) = (1-y)(1-e^{-\gamma y})$. *Denote by* y^+ *a point where* $g(y)$ *takes its maximal value. Evidently this point is a solution of the equation* $g'(y^+) = 0$. *This equation is*

$$1 - e^{-\gamma y} = \gamma(1+y^+)e^{-\gamma y^+}. \tag{18}$$

Consider the following two essentially different cases:

A*).* $y^* < y^+$. *In this case* $g(y^+) \leq \sigma^{-1}y^+$ *(see Fig. 3A). The function* $g(u)$ *is increasing for* $u \leq y^+$, *so that* $u < w \leq y^+$ *implies* $g(u) < g(w)$. *We define two recurrent sequences,*$v_1, v_2, ...$ *and* $u_1, u_2, ...$ *by the same recurrent relation,*

$$\begin{aligned} v_{k+1} &= \sigma g(v_k); \\ u_{k+1} &= \sigma g(u_k), \end{aligned}$$

but with different initial values,

$$\begin{aligned} v_1 &= y^+; \\ u_1 &= \min\left\{y_{t+\sigma+1}, y_{t+\sigma+2}, ..., y_{T+2\sigma}, y^*\right\}. \end{aligned}$$

Both of these sequences are monotones and they have the same limit y^**(see Fig. 3A). Let us show by induction on* k *that for each* $t > T + k\sigma$ *the following inequalities are valid:*

$$u_k \leq y_t \leq v_k. \tag{19}$$

if $k = 1$, *then* $t > T + \sigma$, *therefore 17 is satisfied. Since by definition* $g(y) \leq g(y^+)$, *we may write*

$$y_t = \sum_{i=0}^{\sigma-1} g(y_{t-\sigma\pm i}) \leq \sigma g(y^+) \leq \sigma\sigma^{-1}y^+ = v_{1,}$$

which proves the right hand side of 19 with $k = 1$. *The left hand side of 19 is satisfied for* $T+\sigma < t \leq T+2\sigma$ *by definition of* u_1. *Let* $k > 1$ *and suppose that 19 is satisfied for a given value* k, *provided that* $T + k\sigma < t \leq T + (k+1)\sigma$. *Then for* $t = T + (k+1)\sigma + 1$ *we have*

$$u_{k+1} = \sigma g(u_{k.}) \leq \sum_{i=0}^{\sigma-1} g(y_{t-\sigma+i}) \leq \sigma g(v_k) = v_{k+1}. \tag{20}$$

In particular for $t = T + (k+1)\sigma + 1$ *both 19 and*

$$u_{k+1} \leq y_t \leq v_{k+1}, \tag{21}$$

are satisfied. Again by 20 with $t = T + (k+1)\sigma + 1$ *we get 19 and 20 for* $t = T + (k+1)\sigma + 2$. *Continuing with this process until* $t = T + (k+1)\sigma + \sigma$ *we will get 21 for all* t, $T + (k+1)\sigma < t \leq T + (k+2)\sigma$. *Thus by induction 19 is proved.*

Denote by k *the integer part of the number* $\frac{t-T}{\sigma}$. *That is* $T + k\sigma \leq t < T + (k+1)\sigma$. *If* t *tends to infinity then* k *does as well. Therefore we may write*

$$y^* = \lim_{k\to\infty} u_k \leq \lim_{t\to\infty} y_t \leq \lim_{k\to\infty} v_k = y^*,$$

which proves the theorem in the case A).

B) $y^* > y^+$. *In this case* $g(y^*) > \sigma^{-1}y^+$, *or, equivalently* $h > \sigma^{-1}$, *(see Fig. 3B) , where by* h *we mean* $\frac{g(y^+)}{y^+}$. *Let us describe firstly some important properties of the dynamics in this case.*

B1) *If for some time* $t_0 > T + \sigma$ *we have* $y_{t_0} > y^+$, *then* $y_t > y^+$ *for all* $t \geq t_0$. *It suffices to show that* $y_t > y^+$ *implies* $y_{t+1} > y^+$. *By means of 6 we have*

$$y_{t+1} = y_t + g(y_t) - g(y_{t-\sigma}) \geq y_t + g(y_t) - g(y^+). \tag{22}$$

By 16 the function $g(y)$ *is convex. Therefore in the interval* $[y^+, 1]$ *we have*

$$0 = g'(y^+) > g'(y) > g'(1) = e^{-\gamma} - 1 > -1. \tag{23}$$

In particular

$$y_t - y^+ \geq g(y^+) - g(y_t). \tag{24}$$

This relation and 22 imply

$$y_{t+1} \geq y_t + g(y_t) - g(y^+) \geq y_t + (y^+ - y_t) = y^+.$$

Thus ***B1*** *is proved.*

B2)*. If the values* $y_{t-\sigma}, y_{t-\sigma+1}, ..., y_{t-1}$ *are greater than or equal to* y^* *then* y_t *is less than* y^*.

Indeed, in this case we have $g(y_{t-\sigma+i}) \leq \sigma^{-1}y^*$, $0 \leq i \leq \sigma - 1$, *and*

$$y_t = \sum_{i=0}^{\sigma-1} g(y_{t-\sigma+i}) \leq \sum_{i=0}^{\sigma-1} \sigma^{-1}y^* = y^*.$$

B3)*. If all values* $y_{t-\sigma}, y_{t-\sigma+1}, ..., y_{t-1}$ *belong to the interval* $[y^+, y^*]$, *then* $y_t \geq y^*$.

Indeed, in this case $g(y_{t-\sigma+i}) \geq \sigma^{-1}y^*$*(see Fig. 3B), and*

$$y_t = \sum_{i=0}^{\sigma-1} g(y_{t-\sigma+i}) \geq \sigma\sigma^{-1}y^*.$$

B4*) The inequality $y_t < y^+$ may not be valid for a long time after the perturbation disappears. Consider a sequence*

$$m_t = \min\{y_{t-\sigma+1}, y_{t-\sigma+2}, ..., y_t\}, \tag{25}$$

Since the function g is convex, we may write

$$\tfrac{g(y)-g(0)}{y-0} > \tfrac{g(y^+)-g(0)}{y^+-0}, \quad y < y^+.$$

In particular, the inequalities $y_{t-\sigma+i} < y^+$, $1 \le i \le \sigma$, imply $g(y_{t-\sigma+i}) \ge hy_{t-\sigma+i}$ (see Fig 3B), where by definition $h = \frac{g(y^+)}{y^+}$. If at the day t there is no perturbation we get

$$y_{t+1} = \sum_{i=1}^{\sigma} g(y_{t-\sigma-i}) \ge h \sum_{i=1}^{\sigma} y_{t-\sigma+i} \ge h\sigma m_t.$$

In this formula $h\sigma > 1$ (see the beginning of the case B). In particular $y_{t+1} > m_t$, and $m_{t+1} \ge m_t$. By means of the same arguments we have

$$y_{t+2} = h\sigma m_t, ..., y_{t+\sigma} \ge h\sigma m_t. \tag{26}$$

This implies, in particular, that $m_{t+\sigma} \ge (h\sigma)m_t$. The iteration of this inequality shows that $m_{t+s\sigma} \ge (h\sigma)^s m_t$. If the perturbation disappears at day $t = T$, we may write

$$y_{T+s\sigma} \ge m_{T+s\sigma} \ge (h\sigma)^s m_T.$$

Since $h\sigma > 1$, the number $(h\sigma)^s m_T$ soon becomes greater than y^+. More precisely, $(h\sigma)^s m_T > y^+$ if $s > \log_{h\sigma} \frac{y^+}{m_T}$. Therefore the inequality $y_t < y^+$may not be valid for a period longer than $\sigma \log_{h\sigma} \frac{y^+}{m_T}$ days.

According to B1 and B4 we may suppose that $y_t > y^+$ for all $t > T$ (increasing T, if necessary). Under this supposition the following property is valid.

B5*) The value y_{t+1} is located between y_t and $y_{t-\sigma}$. Indeed, by means of 6 we get*

$$y_{t+1} = y_t + g(y_t) - g(y_t - \sigma) = y_t + g'(\xi)(y_t - y_{t-\sigma}), \tag{27}$$

where ξ is a point between y_t and $y_{t-\sigma}$. Since $g(y)$ is a convex function (see equation 16) we have

$$0 > g'(\xi) \ge g'(1) = e^{-\gamma} - 1 > -1.$$

Therefore we may rewrite 27 in the form

$$y_{t+1} = y_t + r(y_{t-\sigma} - y_t), \tag{28}$$

where, $r = -g(\xi)$, $0 < r \leq 1 - e^{-\gamma} < 1$.

If $y_{t-\sigma} \geq y_t$, *then* $y_{t-\sigma} - y_t \geq 0$ *and 28 implies,* $y_{t+1} = y_t + r(y_{t-\sigma} - y_t) \leq y_t + (y_{t-\sigma} - y_t) = y_{t-\sigma}$, *while certainly,* $y_t + r(y_{t-\sigma} - y_t) \geq y_t$.

If $y_{t-\sigma} \leq y_t$, *then* $y_t - y_{t-\sigma} \geq 0$ *and* $y_{t+1} = y_t + r(y_t - y_{t-\sigma}) \geq y_{t-\sigma}$, *while* $y_t - r(y_t - y_{t-\sigma}) \leq y_t$. *Thus B5 is satisfied.*

Now we are ready to show that the dynamic is globally stable, that is $\lim_{t\to\infty} y_t = y^*$. *To this end consider the sequence:*

$$m_t = \min\{y_{t-\sigma}, y_{t-\sigma+1}, ..., y_t\}. \tag{29}$$

Since the value y_{t+1} *is located between* y_t *and* $y_{t-\sigma}$, *this sequence is monotone. Therefore it has a limit,* $m_\infty = \lim_{t\to\infty} m_t$. *According to B2 this limit is less than or equal to* y^*. *We claim that* $m_\infty = y^*$.

Suppose on the contrary that $y^* - m_\infty = d > 0$. *Since* m_∞ *is a limit of the monotone sequence, we have* $m_\infty = \lim_{t\to\infty} m_t$ *for all large enough* t, *say* $t > T(\varepsilon)$. *Therefore we have that first*

$$y_t \geq m_\infty - \varepsilon d, \tag{30}$$

for all $t > T(\varepsilon)$, *then each collection of values*

$$y_{t-\sigma}, y_{t-\sigma+1}, ..., y_t, \tag{31}$$

with $t > T(\varepsilon) + \sigma$ *contains at least one element* y_i, *such that* $y_i \leq m\infty$, *and next the property B3 shows that the collection 31 contains at least one element* y_j, *such that* $y_j \geq y^*$. *We will show that these three conditions lead to a contradiction, provided that* ε *is a small enough amount. Let us consider a collection,*

$$y_j, y_{j+1}, ..., y_{j+\sigma}, \tag{32}$$

where $y_j \geq y^*$ *and* $j > T(\varepsilon) + \sigma$. *By means of 28 and 30 we have*

$$\begin{aligned} y_{j+1} &= y_j(1-r) + ry_{j-\sigma} \geq y^*(1-r) + r(m_\infty - \varepsilon d) \\ &= (m_\infty + d)(1-r) + r(m_\infty - \varepsilon d) \\ &= (m_\infty - \varepsilon d) + (1+\varepsilon)(1-r)d \\ &\geq (m_\infty - \varepsilon d) + (1+\varepsilon)e^{-\gamma}d, \end{aligned} \tag{33}$$

since $1 - r \geq e^{-\gamma}$. *In strict analogy, using (33), we have*

$$y_{j+2} = y_{j+1}(1-r) + ry_{j-\sigma+1} \tag{34}$$

$$\geq [(m_\infty - \varepsilon d) + (1+\varepsilon)e^{-\gamma}d](1-r) + r(m_\infty - \varepsilon d) \tag{35}$$

$$\geq (m_\infty - \varepsilon d) + (1+\varepsilon)e^{-2\gamma}d. \tag{36}$$

Continuing with this process we get

$$y_{j+s} \geq (m_\infty - \varepsilon d) + (1+\varepsilon)e^{-s\gamma}d, \tag{37}$$

for all s,$0 \leq s \leq \sigma$. *If* ε *is small enough,* $\varepsilon < e^{-\gamma\sigma}$, *then* $\varepsilon < (1+\varepsilon)e^{-\gamma s}$ *for all* s, $0 \leq s \leq \sigma$. *Therefore*

$$y_{j+s} \geq m_\infty + [(1+\varepsilon)e^{-s\gamma}d - \varepsilon]d > m_\infty.$$

This is a contradiction, since 32 according to 31 has to contain at least one element y_i, $j \leq i \leq j+\sigma$, *such that* $y_i \leq m_\infty$.

Thus, we have shown that $m_\infty = y^*$. *This equality and the definition 29 imply*

$$y_t \geq y^* - \varepsilon,$$

for all large enough t, $t > T(\varepsilon)$. *Since the function* $g(y)$ *is monotone for* $y > y^+$, *we get* $g(y_t) \leq g(y^* - \varepsilon) = g(y^*) + \delta$, *where* $\delta = g(y^* - \varepsilon) - g(y^*)$ *is small enough, provided that* ε *does. Now, for* $t > T(\varepsilon) + \sigma$, *we have*

$$\begin{aligned} y_{t+1} &= \sum_{i=0}^{\sigma-1} g(y_{t-\sigma+i}) \leq \sum_{i=0}^{\sigma-1}(g(y^*) + \delta) \\ &= \sigma g(y^*) + \sigma\delta = y^* + \sigma\delta. \end{aligned}$$

Therefore $y_{t+1} \in [y^* - \varepsilon, y^* + \sigma\delta]$ *for* $t > T(\varepsilon) + \sigma$. *This means exactly that* $\lim_{t\to\infty} y_t = y^*$. *The theorem is proved.* ■

We also observe that the limit when γ tends to infinity is the only bifurcation point. In consequence, increasing the value of γ increases the amplitude and the frequency of the damped oscillations. The return map of the solution obtained with very large values of γ, where the dynamic is near the limiting bifurcation point is illustrated in Fig. 4.

4.3 The critical value γ_0

According to Theorem 3, the condition $y^* = y^+$ is critical for the dynamics. If $y^* \leq y^+$, the dynamics is almost monotonic (it is included between two close monotonic processes, see 19), while if $y^* > y^+$ the dynamic has infinite damped oscillations (see B2 and B3). The critical value γ_0 is a function of the period of infectiousness. In Table 1 we give values of γ_0 for some periods of infectiousness. Note that $\gamma_0 \approx 2/\sigma$ if $\sigma >> 1$. The numerical solution of the

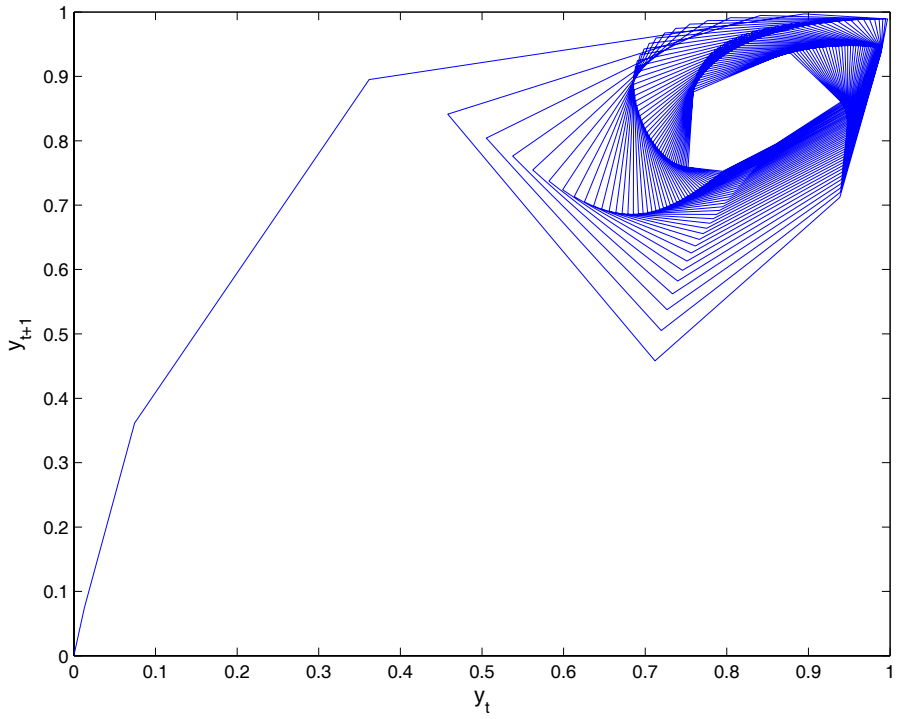

Figure 4: Return map of the discrete SIS model. Initial conditions as Fig. 1 except $\gamma = 5$ and $p(0) = 1e{-}5$. Due to the large value of γ, the solution reaches the steady state through infinite damped oscillations with large amplitude. The map shows only 300 points where the steady state is not yet reached.

σ	γ_0	σ	γ_0	σ	γ_0
1	2.46	5	0.41	15	0.13
2	1.12	7	0.29	20	0.10
3	0.72	9	0.22	30	0.07
4	0.53	10	0.20	40	0.04

Table 1:

equation $y^* = y^+$ can be found as follows. We need to find the intersection of the curves $g_1(y) = (1-y)(1-e^{-\gamma y})$ and $g_2(y) = \sigma^{-1}y$ under the additional condition $g_1'(y) = 0$. We have $g_1'(y) = -(1-e^{-\gamma y}) + (1-y)\gamma e^{-\gamma y} = 0$, that is $1 - e^{-\gamma y} + \gamma y e^{-\gamma y} = \gamma e^{-\gamma y}$. Multiplying by $e^{\gamma y}$ and rearranging, we get $(1-y)\gamma = e^{\gamma y} - 1$. Thus we need to solve the system of equations

$$(1-y)(1-e^{-\gamma_0 y} = \sigma^{-1}y; \tag{38}$$

$$(1-y)\gamma_0 = e^{\gamma_0 y} - 1. \tag{39}$$

Multiplying 38 by γ_0 and substituting 39 in 38 we get $(e^{\gamma_0 y} - 1)(1 - e^{-\gamma_0 y}) = \sigma^{-1}\gamma_0 y$, that is $e^{\gamma_0 y} + e^{-\gamma_0 y} = \sigma^{-1}\gamma_0 y + 2$. Transforming the variable $t = \gamma_0 y$, the equation $e^t + e^{-t} = \sigma^{-1}t + 2$ can be solved by a numerical method of successive substitutions.

5 Discussion

First, we note that in line with our approach the traditional stability proved in Theorem 3 has less importance than the dynamical properties of an active infection, $\gamma > \gamma_0$. Indeed, the properties B2, B3 and B5 mean that the dynamic has infinite damped oscillations, while B1 and B4 state that the proportion of infectives, after a small initial period, will be greater than a critical quantity y^+ (provided that the spread occurs without interventions, $p_t = 0$). The stability may be considered just like a general tendency for the dynamics. More precisely, if we do not accept infinitesimal values for the time step, why should we accept an infinite number of steps? Our proof of stability uses non-constructive "on the contrary" arguments, that is, the stabilisation process may be very slow. Admittedly, the proofs of the global stability in the discrete models considered by Castillo-Chavez et al. [11] and Allen et al. [3], also have non-constructive elements. More concisely, firstly they had proved the non-existence of any m-cycles for $m > 1$, and then applied a result of Cull, that implies stability, (see [11], result 1, p. 157 and [3], Theorem 1. p. 5). Hence the stabilisation, in general, may be a very slow process as well.

Damped oscillations were found in 1929 by H. E. Soper [29] in his classic endemic model [20]. A classic alternative was advanced by Bartlett [7], namely, that undamped oscillations can be understood as a stochastic phenomenon (see detailed discussion in [6], chapter 7). Even though our model is deterministic, it admits stochastic external elements via random perturbation. For example, a relatively small closed organized group (a military unit or a group of workers or tourists) while located in a new place and has random contacts with locals may be considered as a homogeneous and uniformly mixed population with stochastic external perturbation.

The deterministic discrete model described here is different from the formulation of Allen et al. [1]. They used a relative recovery rate to deal with the restrictions imposed by the mass action, and then their time step has to be very small, and it has nothing to do with the population life cycles.

Cooke et al. [12] formulated a discrete model with exponential incidence. However, they analysed exclusively the case in which the time step is equal to the period of infectivity.

The parameters β or γ of the incidences 1 and 2, respectively, have the advantage of an explicit expression in terms of population and disease characteristics. These parameters can be approximated by $\gamma = kc$ or $\beta = kc/N$ if is small enough. The incidence function 2 clearly discriminates the role of infectives and susceptibles, whereas the symmetry of the mass action incidence theoretically allows that susceptibles can transmit susceptibility.

Next, if we compare our model with the continuous mass action SIS model, it can be noted that in both models the threshold quantity, usually called the basic reproductive number and denoted by R_0, is given by the relation $R_0 = \beta N \sigma$. However, the SIS continuous counterpart is always monotonic **??**. In our model, active infections (with $\gamma > \gamma_0$) have more complicated dynamics, which are characterized by the properties B1, B2, B3, B4, B5 and by the important restriction on the endemic equilibrium given in Theorem 2.

The mass action discrete SIS model requires $\gamma < 1$ in order to obtain non-absurd results (see [3], Lemma 2 and also Fig. 4). Nevertheless, if γ is less but close to one, then the fact that the mass action does not lead to absurd results neither means that it may render correct ones. It is likely that in the latter situation the mass action conclusions are far from being reasonable. Only if $\gamma << 1$ we have enough arguments to use this law.

Albeit several infections elicit an immune response, not always this response confers protective immunity against reinfection and, when this happens the infection have a SIS-type spread in human populations. Examples of these disease are: acute respiratory infection caused by influenza virus that do not confer immunity mainly due to the high antigenic variability of the virus; the

streptococcal sore throat that can produce epidemics in small closed communities like military camps or small rural populations, whose etiological agent is the bacteria Streptococcus pneumoniae which has more than 55 serologic types and it is very unlikely that an individual acquires immunity after infection; and gonorrhoea caused by Neisseria gonorrhoea [15]. Our model is also suitable for epidemiological studies of a cohort of individuals infected and reinfected during relatively short periods of time until the individuals acquire protective immunity. An example of this situation are the continued reinfections with rotavirus occurring in young children (from 0.5 up to 3 years of age).

Acknowledgments.

M. V. J. and V. K. were financially supported by PAPIIT IN205307 and PAPIIT IN108306-3, respectively. All authors were also supported by the Multiproyecto: Tecnologías para la Universidad de la Información y la Computación, UNAM, México. We thank Juan R. Bobadilla for valuable technical assistance.

References

[1] L. J. S. Allen, M. A. Jones and C. F. Martin, A discrete time model with vaccination for a measles epidemic, Math. Biosci., 105 (1991), 111-131.

[2] L. J. S. Allen, Some discrete-time SI, SIR and SIS epidemic models, Math. Biosci., 124 (1994), 83-105.

[3] L. S. J. Allen. and A. M. Burgin, Comparison of deterministic and stochastic SIS and SIR models in discrete time, Math. Biosci., 163 (2000), 1-33.

[4] R. M. Anderson and R. M. May, Infectious disease of humans. Dynamics and control. Oxford University Press, 1992.

[5] J. L. Aron and I. B. Schwartz, Seasonality and period-doubling bifurcations in a epidemic model, J. Theoret. Biol., 110 (1984), 665-679.

[6] N. T. J. Bailey, The Mathematical Theory of Infectious Diseases and Its Applications, Griffin: London, 1975.

[7] M. S. Bartlett, Deterministic models for recurrent epidemics, In Proc. 3rd Berkeley,Symp. Math. Stat. Prob., 4 (1956), 81-100. Berkeley: University of California Press, 1959.

[8] F. Brauer, Basic ideas in mathematical epidemiology. In Mathematical Approaches for Emerging and Re-emerging Infectious Disease. An Introduction. Castillo-Chávez, Blower, van den Driessche, Kirschner, Yakubu (Eds). pp. 31-65. Springer-Verlag, 2002a.

[9] F. Brauer, Extensions of the Basic Models. In: Mathematical Approaches for Emerging and Reemerging Infectious Diseases. An Introduction. Castillo-Chavez, Blower, van den Driessche, Kirschner, Yakubu (Eds.), pp. 67-95. Springer-Verlag, 2002b.

[10] S. Bussenberg and K. L. Cooke, Vertically Transmitted Disease. Models and Dynamics. Biomathematics Vol. 23, Berlin, Heidelberg, New York: Springer-Verlag. 1993.

[11] C. Castillo-Chavez and A.–A. Yakubu, Discrete-Time SIS Models with simple and complex Population Dynamics. In: Mathematical Approaches for Emerging and Reemerging Infectious Diseases. An Introduction. Castillo-Chavez, Blower, van den Driessche, Kirschner, Yakubu (Eds.) pp. 153-163. Springer-Verlag, 2002

[12] K. L. Cooke, D. F. Calef and E. V. Level, Stability or chaos in discrete epidemic models. In Nonlinear systems and applications. Lakshmikanthan V. (ed), New York: Academic Press, 1977.

[13] K. L. Cooke and P. van den Driessche, Analysis of an SEIRS epidemic model with two delays, J. Math. Biol., 35 (1996), 240-260.

[14] D. J. Daley and J. Gani, Epidemic Modeling: An Introduction. Cambridge University Press, 1999.

[15] B. D. Davis, R. Dulbecco, H. N. Eisen, H. S. Ginsberg and W. B. jr. Wood, Microbiology. Harper and Row Medical Publishers Inc. Hagertown, Maryland, 1978.

[16] O. Diekmann and J. A. Heesterbeek, Mathematical Epidemiology of Infectious Diseases, John Wiley and Sons, 2000.

[17] P. van den Driessche, Time Delay in Epidemic Models. In: Mathematical Approaches for Emerging and Reemerging Infectious Diseases. An Introduction. Castillo-Chavez, Blower, van den Driessche, Kirschner, Yakubu (Eds.). pp. 119-128. Springer Verlag, 2002.

[18] D. J. D. Earn, P. Rohani, B. M. Bolker and B. T. Grenfell, A simple model for complex dynamical transitions in epidemics. Science, 287 (2000), 667-670.

[19] H. W. Hethcote, Three basic epidemiological models. In: Applied Mathematical Ecology. Gross, T. G. Hallam, T.G., and S. A. Levin (eds.), Berlin: Springer-Verlag, pp. 119-144, 1989.

[20] H. W. Hethcote and P. van den Driessche, An SIS epidemic model with variable population size and a delay, J. Math. Biol., 34 (1995), 177-194.

[21] H. W. Hethcote, The mathematics of infections Diseases, SIAM Review, 42(4) (2000), 599-653.

[22] M. de Jong, O. Diekmann and J. A. Heesterbeek, How Does Transmission of Infection Depend on Population Size? In: Epidemic Models: Their structure and relation to data. Mollison (Ed.). Cambridge University press. 1995.

[23] W. G. Kelley, and A. C. Peterson, Difference Equations. An introduction with applications. Second ed. Academic Press, 1991

[24] C. Lefevre and P. Picard, On the formulation of discrete time epidemic models, Math. Biosci., 95 (1989), 27-35.

[25] A. L. Lloyd, Destabilization of epidemic models with the inclusion of realistic distributions of infectious periods, Proc. R. Soc. Lond. B, 1599 (2001), 985-993.

[26] R. M. May, Simple mathematical models with very complicated dynamic, Nature 261 (1976), 459-467.

[27] L. Nasell, The quasi-stationary distribution of the closed endemic SIS model, Adv. Appl. Prob., 28 (1996), 859-932.

[28] L. F. Olsen and W. M. Schaffer, Chaos versus noisy periodicity: Alternative hypotheses for childhood epidemics, Science 249 (1990) 499-504.

[29] H. E. Soper, Interpretation of periodicity in disease-prevalence, J .R. Statist. Soc., 92 (1929), 34-73.

Received: September 19, 2006