Applied Mathematics Letters 25 (2012) 1056–1060

Contents lists available at SciVerse ScienceDirect

Applied Mathematics Letters

journal homepage: www.elsevier.com/locate/aml

Globally stable endemicity for infectious diseases with information-related changes in contact patterns

B. Buonomo [a], A. d'Onofrio [b,*], D. Lacitignola [c]

[a] Department of Mathematics and Applications, University of Naples Federico II, via Cintia, I-80126 Naples, Italy

[b] Department of Experimental Oncology, European Institute of Oncology, Via Ripamonti 435, I-20141 Milano, Italy

[c] Department of Health and Motory Sciences - Epidemiology Laboratory, University of Cassino, Campus Folcara, I-03043 Cassino, Italy

ARTICLE INFO

Article history:
Received 10 October 2011
Accepted 11 March 2012

Keywords:
Mathematical epidemiology
Information
Behavior
Global stability

ABSTRACT

SIR and SIS epidemic models with information—related changes in contact patterns are introduced. The global stability analysis of the endemic equilibrium is performed by means of the Li–Muldowney geometric approach. Biological implications of the stability conditions are given.

1. Introduction

Feedbacks and global stability are among the most important features of all mathematical models in biology [1,2]. In Mathematical Epidemiology, however, the vast majority of efforts have been devoted to the study of global stability. Indeed, determining under which conditions a disease, independently from the initial burden, either remain endemic or get extinct is probably the most important topic. Recently, however, it is increasingly becoming clear that a realistic epidemic model must include the feedback (FB) that the information about an infectious disease has on its spreading [3–7]. For example, a first type of FB is represented by the case of the so-called *rational exemption*, that is the family's decision to not vaccinate children as a consequence of a 'pseudo-rational' comparison between the risk from infection and the media driven perceived risk from getting damages from the vaccine. This approach has been applied to several epidemic scenarios, see e.g. [8,9,5,10].

A second type of FB, instead, is the one given by the influence of the information on the behavior of healthy subjects. For example, in [4] the authors focus on simple endemic models, by modelling the social contact rate as a decreasing function of the available information on the present and the past disease prevalence. It is shown that social behavior change alone may trigger sustained oscillations. This indicates that human behavior might be a critical explaining factor of oscillations in time-series of endemic diseases.

The modelling idea to represent these distinct FBs is that vaccination decisions, for the first type of FB, and behavioral decisions, for the second type, are formed from an *information set*, summarised by a new state variable mostly based on the publicly available information on both present and also on the recent past spreading of the disease.

In this paper we study the global asymptotic stability of the endemic equilibrium for the second case of FB, and for both SIR and SIS classes of infectious disease models.

* Corresponding author. Tel.: +39 02 94375114; fax: +39 02 57489922.
E-mail addresses: buonomo@unina.it (B. Buonomo), alberto.donofrio@ifom-ieo-campus.it, z20263@ieo.it (A. d'Onofrio), d.lacitignola@unicas.it (D. Lacitignola).

0893-9659/$ – see front matter
doi:10.1016/j.aml.2012.03.016

2. The model

The mathematical description of the information dependence is given by a quantity, M, called *the information index*, [8,9,4,5,10]. This index is described by the distributed delay

$$M = \int_{-\infty}^{t} g(S(\tau), I(\tau))K_a^p(t-\tau)d\tau.$$

The term K_a^p is a delaying kernel, which represents the weight given to past prevalence. Generally, K_a^p is the density function for a gamma distribution:

$$K_a^p(u) = \frac{a^p u^{p-1} e^{-au}}{(p-1)!}, \quad u \geq 0$$

with $a > 0, p = 1, 2, \ldots$. In this case the delay is infinite and centered at p/a (see e.g., [11]), which is the average delay. The function g describes the role played by the state variables in the information dynamics. It may be generally assumed g to be continuous and increasing respect to the I. No hypotheses on the dependence of g on S are made at this stage (actually g might be independent on S). However we set $g(S, 0) = 0$ for all S.

If $p = 1$, we get $K(t) = a\, e^{-at}$, that is a *exponentially fading memory*. In this case we have,

$$\dot{M} = ag(S(t), I(t)) - aM.$$

This possibility to transform an infinite dimensional integro-differential systems into a finite dimensional system of ordinary differential equations is called *linear chain trick*, [11].

In [4] the following model has been introduced

$$\begin{cases} \dot{S} = \mu(1-S) - \beta(M)IS \\ \dot{I} = \beta(M)IS - (\mu+\nu)I \\ \dot{M} = ag(I) - aM, \end{cases} \tag{1}$$

where the state variables are: Susceptible individuals (S), Infectious individuals (I) and the information index (M).

The function β is required to be a positive decreasing function, $\beta'(M) < 0$. The function g is required to be such that $g(0) = 0$, and $g'(I) > 0$. We will prove the global stability result of the endemic equilibrium for g satisfying:

$$g'(I)I \leq g(I). \tag{2}$$

The simplest example is $g(I) = kI$, where $k \in (0, 1)$. Another example is $g(I) = I/(1 + qI)$, where q is a positive constant. The parameters μ, ν, and a, are positive constants.

3. Basic properties

In [4] it has been shown that the set

$$\Omega = \{(S, I, M) : S \geq 0,\ I \geq 0,\ S + I \leq 1,\ 0 \leq M \leq g(1)\}$$

is positively invariant for model (1). Moreover the disease free equilibrium $E_0 = (1, 0, 0)$ is on $\partial\Omega$, as well as its stable manifold, which is the set $\{(S, I, M) \in \Omega : I = 0\}$. As a consequence, the state variables are strongly persistent, i.e. there exists a $\epsilon_0 > 0$ such that if $I(0) > 0, S(0) > 0$, and $M(0) > 0$, then:

$$\liminf_{t\to\infty} I(t) \geq \epsilon_0 > 0; \qquad \liminf_{t\to\infty} S(t) \geq \epsilon_0 > 0; \qquad \liminf_{t\to\infty} M(t) \geq \epsilon_0 > 0.$$

Furthermore, model (1) admits a unique endemic equilibrium,

$$E = (S^*, I^*, M^*)$$

where $S^* = (\mu+\nu)/\beta(g(I^*))$, $M^* = g(I^*)$ and I^* is the unique solution of:

$$\mu\left(\frac{\mu+\nu}{\beta(g(I))}\right) - (\mu+\nu)I = 0.$$

In [4] the following stability result has also been shown.

Theorem 3.1. *Let $R_0 = \beta(0)/(\mu+\nu)$. If $R_0 \leq 1$, then the disease free equilibrium E_0 is globally asymptotically stable. If $R_0 > 1$, then E_0 is unstable and the endemic equilibrium E is locally asymptotically stable.*

4. Global stability of the endemic equilibrium

In this section we analyse the global asymptotic stability of E. The following theorem holds,

Theorem 4.1. *Assume that g satisfies the inequality (2). If $R_0 > 1$ and*

$$\nu > 2\beta(\epsilon_0), \tag{3}$$

where ϵ_0 is the constant of uniform persistence, then the endemic equilibrium E of system (1) exists and is globally asymptotically stable with respect to solutions of (1) initiating in the interior of Ω.

Proof. We first observe that the Jacobian matrix $J(S, I, M)$ corresponding to (1) is:

$$J = \begin{pmatrix} -\mu - \beta(M)I & -\beta(M)S & -\beta'(M)IS \\ \beta(M)I & \beta(M)S - (\mu + \nu) & \beta'(M)IS \\ 0 & ag'(I) & -a \end{pmatrix}.$$

The second additive compound matrix $J^{[2]}(S, I, M)$ is:

$$J^{[2]} = \begin{pmatrix} -2\mu - \nu - \beta(M)(I - S) & \beta'(M)IS & \beta'(M)IS \\ ag'(I) & -\mu - a - \beta(M)I & -\beta(M)S \\ 0 & \beta(M)I & \beta(M)S - \mu - \nu - a \end{pmatrix}.$$

Now we take the function,

$$P = P(S, I, M) = \operatorname{diag}\left\{1, \frac{I}{M}, \frac{I}{M}\right\}.$$

It follows that,

$$P_f P^{-1} = \operatorname{diag}\left\{0, \frac{\dot{I}}{I} - \frac{\dot{M}}{M}, \frac{\dot{I}}{I} - \frac{\dot{M}}{M}\right\},$$

and,

$$PJ^{[2]}P^{-1} = \begin{pmatrix} -2\mu - \nu - \beta(M)(I - S) & \beta'(M)MS & \beta'(M)MS \\ ag'(I)\dfrac{I}{M} & -\mu - a - \beta(M)I & -\beta(M)S \\ 0 & \beta(M)I & \beta(M)S - \mu - \nu - a \end{pmatrix},$$

so that

$$B = P_f P^{-1} + PJ^{[2]}P^{-1} = \begin{bmatrix} B_{11} & B_{12} \\ B_{21} & B_{22} \end{bmatrix},$$

where

$$B_{11} = -2\mu - \nu - \beta(M)(I - S), \qquad B_{12} = [\beta'(M)MS,\ \beta'(M)MS], \qquad B_{21} = \left[ag'(I)\frac{I}{M}, \quad 0\right]^T$$

and

$$B_{22} = \begin{bmatrix} \dfrac{\dot{I}}{I} - \dfrac{\dot{M}}{M} - \mu - a - \beta(M)I & -\beta(M)S \\ \beta(M)I & \dfrac{\dot{I}}{I} - \dfrac{\dot{M}}{M} + \beta(M)S - \mu - \nu - a \end{bmatrix}.$$

Consider now the norm in $\mathbf{R}^3$ as: $|(u, v, w)| = \max\{|u|, |v| + |w|\}$, where (u, v, w) denotes the vector in $\mathbf{R}^3$ and denote by $\mathcal{L}$ the Lozinskiĭ measure with respect to this norm. It follows, [12]:

$$\mathcal{L}(B) \le \sup\{g_1, g_2\} \equiv \sup\{\mathcal{L}_1(B_{11}) + |B_{12}|, \mathcal{L}_1(B_{22}) + |B_{21}|\}, \tag{4}$$

where $|B_{21}|$, $|B_{12}|$ are matrix norms with respect to the L^1 vector norm and $\mathcal{L}_1$ denotes the Lozinskiĭ measure with respect to the L^1 norm[1].

$$\mathcal{L}_1(B_{11}) = -2\mu - \nu - \beta(M)(I - S), \tag{5}$$

[1] i.e., for the generic matrix $A = (a_{ij})$, $|A| = \max_{1\le k\le n} \sum_{j=1}^{n} |a_{jk}|$ and $\mathcal{L}(A) = \max_{1\le k\le n}(a_{kk} + \sum_{j=1(j\ne k)}^{n} |a_{jk}|)$.

$$|B_{12}| = \beta'(M)MS, \qquad |B_{21}| = ag'(I)\frac{I}{M}, \tag{6}$$

$$\mathcal{L}_1(B_{22}) = \frac{\dot{I}}{I} - \frac{\dot{M}}{M} - \mu - a + \max\{0,\ 2\beta(M)S - \nu\}. \tag{7}$$

Taking into account of (4)–(7), the general expressions of g_1 and g_2 for system (1) are thus:

$$g_1 = -2\mu - \nu - \beta(M)(I - S) + \beta'(M)MS, \tag{8}$$

and

$$g_2 = \frac{\dot{I}}{I} - \frac{\dot{M}}{M} - \mu - a + ag'(I)\frac{I}{M} + \max\{0,\ 2\beta(M)S - \nu\}. \tag{9}$$

Observe that system (1) provides the following equalities:

$$\frac{\dot{I}}{I} = \beta(M)S - (\mu + \nu), \qquad \frac{\dot{M}}{M} = a\frac{g(I)}{M} - a.$$

Hence, from (8) one gets,

$$g_1 = \frac{\dot{I}}{I} - \mu - \beta(M)I + \beta'(M)MS,$$

and from (9),

$$g_2 = \frac{\dot{I}}{I} - a\frac{g(I)}{M} - \mu + ag'(I)\frac{I}{M} + \max\{0,\ 2\beta(M)S - \nu\}.$$

Taking into account that $\beta(M) > 0$ and $\beta'(M) \leq 0$ it follows:

$$g_1 \leq \frac{\dot{I}}{I} - \mu.$$

On the other hand, g_2 can be written as

$$g_2 = \frac{\dot{I}}{I} - \mu + \frac{a}{M}[g'(I)I - g(I)] + \max\{0,\ 2\beta(M)S - \nu\}.$$

Taking into account that $S \leq 1$, β is decreasing and that the state variables are strongly persistent, for large t it follows:

$$g_2 \leq \frac{\dot{I}}{I} - \mu + \frac{a}{M}[g'(I)I - g(I)] + \max\{0,\ 2\beta(\epsilon_0) - \nu\}$$

where ϵ_0 is the constant of uniform persistence. From (2) and (3) we obtain, for large t:

$$g_2 \leq \frac{\dot{I}}{I} - \mu.$$

Hence from (4),

$$\mathcal{L}(B) \leq \sup\{g_1, g_2\} = \frac{\dot{I}}{I} - \mu.$$

Therefore

$$\frac{1}{t}\int_0^t \mathcal{L}(B)ds \leq \frac{1}{t}\log\frac{I(t)}{I(0)} - \mu,$$

which implies

$$\limsup_{t\to\infty} \sup_{x_0\in\Gamma} \frac{1}{t}\int_0^t \mathcal{L}(B(x(s, x_0)))ds < 0,$$

so the Bendixson criterion given in [13] is thus verified. #

Remark. Note that (3) and $R_0 > 1$ together imply that $2\beta(\epsilon_0) < \nu < \beta(0)$.

5. A SIS case

In this section we briefly analyse the impact of the information-driven behavior of susceptible subjects on the transmission of a SIS communicable disease.

By including the variable contact rate $\beta(M)$ in the classical SIS model we obtain the following system:

$$\begin{cases} \dot{S} = \mu(1-S) - \beta(M)IS + \gamma I \\ \dot{I} = \beta(M)IS - (\mu+\gamma)I \\ \dot{M} = ag(I) - aM. \end{cases} \tag{10}$$

We can study the model on the plane (limit set) $S + I = 1$. Model (10) reduces to:

$$\begin{cases} \dot{I} = \beta(M)I(1-I) - (\mu+\gamma)\, I = F(I, M) \\ \dot{M} = akI - aM = G(I, M). \end{cases} \tag{11}$$

System (11) has two equilibrium points: a disease-free one, $E_0 = (0, 0)$, and an endemic equilibrium $E = (I_e, kI_e)$, where I_e is the solution of the following equation

$$\beta(kI) = \frac{\mu+\gamma}{1-I},$$

which exists only if $\beta(0) > \mu + \gamma$.

As far as the behaviour of the solutions of system (11), the following proposition holds.

Theorem 5.1. *Let $R_0 = \beta(0)/(\mu+\nu)$. If $R_0 \leq 1$, then the disease free equilibrium E_0 is globally asymptotically stable. If $R_0 > 1$, then E_0 is unstable and the endemic equilibrium E is globally asymptotically stable.*

Proof. From the following differential inequality

$$\dot{I} \leq I[\beta(0)(1-I) - (\mu+\gamma)],$$

it follows that $R_0 \leq 1$ implies the global stability of E_0. On the contrary, simple linearization shows that E_0 is unstable for $R_0 > 1$. The global stability of E may be proven by applying the Bendixson–Dulac criterion. Indeed, by taking the Dulac function $D = 1/I$, it follows that:

$$div\left(\frac{\dot{S}}{I}, \frac{\dot{I}}{I}\right) = -\beta(M)I - \frac{a}{I} < 0,$$

so the locally stable equilibrium E is globally stable. #

6. Conclusions

Our results on the global stability of the endemic equilibrium might have implications of some interest in public health. Indeed, for diseases where $R_0 > 1$, the health authorities can recommend mass vaccination or make it mandatory if the disease is severe or causes economic damage. However, anti-vaccination lobbies could claim that modifying the behavior in the case of an epidemic might be sufficient to eradicate the disease. On the contrary, our results analytically show that this claim is not true for an SIS transmission mechanism, since independently on the initial state the infection will remain endemic in the target population. In the SIR case, we provide the sufficient condition (3), which guarantees the global stability of the endemic state.

References

[1] V. Capasso, Mathematical Structures of Epidemic Systems, in: Lecture Notes in Biomathematics, Springer-Verlag, Berlin, 2008.
[2] J.D. Murray, Mathematical Biology, third ed., in: Interdisciplinary Applied Mathematics, vol. 17, Springer-Verlag, New York, 2002.
[3] C. Bauch, D. Earn, Vaccination and the theory of games, PNAS 101 (2004) 13391–13394.
[4] A. d'Onofrio, P. Manfredi, Information-related changes in contact patterns may trigger oscillations in the endemic prevalence of infectious disease, J. Thor. Biol. 256 (2009) 473–478.
[5] A. d'Onofrio, P. Manfredi, E. Salinelli, Vaccinating behaviour, information, and the dynamics of SIR vaccine preventable diseases, Theor. Popul. Biol. 71 (2007) 301–317.
[6] T. Reluga, C. Bauch, A. Galvani, Evolving public perceptions and stability in vaccine uptake, Math. Biosci. 204 (2006) 185–198.
[7] R. Vardavas, R. Breban, S. Blower, Can influenza epidemics be prevented by voluntary vaccination? PLoS Comp. Biol. 3 (2007) e85.
[8] B. Buonomo, A. d'Onofrio, D. Lacitignola, Global stability of an SIR epidemic model with information dependent vaccination, Math. Biosci. 216 (2008) 9–16.
[9] B. Buonomo, A. d'Onofrio, D. Lacitignola, Rational exemption to vaccination for non-fatal SIS diseases: globally stable and oscillatory endemicity, Math. Biosci. Engin. 7 (2010) 561–578.
[10] A. d'Onofrio, P. Manfredi, E. Salinelli, Fatal SIR diseases and rational exemption to vaccination, Math. Med. Biol. 25 (2008) 337–357.
[11] H. Smith, An Introduction to Delay Differential Equations with Applications to the Life Sciences, in: Text in Applied Mathematics, vol. 57, Springer-Verlag, New York, 2011.
[12] R.H. Martin Jr., Logarithmic norms and projections applied to linear differential systems, J. Math. Anal. Appl. 45 (1974) 432–454.
[13] M.Y. Li, J.S. Muldowney, A geometric approach to global-stability problems, SIAM J. Math. Anal. 27 (1996) 1070–1083.